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COMPREHENSIVE OBSTETRICS IN THE TROPICS

Editors

E. Y. Kwawukume

E. Ejiro Emuveyan

January 2002

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COMPREHENSIVE OBSTETRICS IN THE TROPICS

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FOREWORD

In 1986, a group of doctors and dentists in West Africa produced a textbook in Surgery titled "Principles and Practice of Surgery Including Pathology in the Tropics". This book has now gained worldwide recognition on account of its contribution to principles and practice of Surgery in the context of tropical diseases. Fifteen years later, another textbook has emerged in the West Africa region for practice of Obstetrics in the tropics. This noble effort is commendable. Physicians and Surgeons in the sub-region are taking small but very significant steps towards growth of medical education and practice in Africa. They are contributing to growth of knowledge in the field of medicine from their research findings and working experience in the tropics.

Although this book is written primarily for those who study and practice obstetrics in the tropics, it is not limited to such. In a world reduced to a small village by rapid information technology and by rapid movement of people between nations through jet travel, the importance of this book to all medical practitioners and health training institutions worldwide is evident. Tropical medicine is now at the doorstep of every practicing doctor and is within every hospital, as people move from place to place rapidly across the globe. It is therefore necessary that every practicing obstetrician should be well equipped with the information in this book.

The authors of this book have rich experience in the practice of obstetrics in the tropics. Most of them have taught or are teaching the subject in medical schools in West Africa. A number of them are also examiners for the West Africa Postgraduate Medical College. In this book, they have integrated their experience in the field with those of others in other parts of the world to present us with invaluable information covering antenatal and intrapartum care, medical and other complications in pregnancy, surgical procedures in obstetrics and postpartum management. Health problems of particular interest in Africa such as malaria, sickle cell disease and other anaemias and HIV/AIDS have been covered in this book as they relate to obstetrical practice. Nutrition that is crucial to normal development of the fetus and health of the mother and that is of major concern in West Africa where GDP is generally very low and birth rate is high is a topic that should be of interest to all obstetricians. These and other important topics treated here should inform the obstetrician for intelligent and adequate care of the patient.

Being the first edition, it is likely there are areas that may require revision in terms of content and for clarity. The authors and contributors, no doubt, will be very pleased to receive suggestions to improve the quality and usefulness of this invaluable text.

The foreword to this book will be incomplete without noting the immense contribution of Carnegie Foundation in supporting the training of specialists in the field of obstetrics and gynaecology in Ghana. A good number of the authors of this book benefited from the investment of this Foundation.

It is a pleasure for me to support this book. I congratulate the editors as well as the authors drawn from the University of Ghana Medical School, Kwame Nkrumah University of Science and Technology-School of Medical Sciences, College of medicine, University of Lagos, University of Port Harcourt, University of Ibadan, University of Maiduguri Teaching Hospital Nigeria for their achievement. Finally, I recommend this book to you as invaluable source of information about practice of obstetrics in the tropics and of reference for your own study in the areas covered in this book.

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PREFACE

It is becoming clear that with the increasing awareness in women's health, there is a need for a comprehensive textbook to serve as a source of information, reference, direction and resource to health workers. The field of obstetrics is changing rapidly in both the developed and the developing countries. While there are a lot of texts with vital information about obstetric practice in developing countries, there are only few textbooks written by teachers in the sub-region with practical approach to the training of our medical students and postgraduate doctors. Certainly, little of it gets incorporated into the practice of obstetrics to improve the health needs of women.

This book focused on problems and solutions as seen in the tropics and compared with the practice in developed countries. The novelty about the book is the *controversies and discussions* that the postgraduates face on general ward rounds, during examinations and the different opinions expressed by different faculty members and other writers. These views are incorporated with suggestions and solutions.

What needs to be known about pregnant women's health today requires a multidisciplinary approach about the problems encountered in clinical practice and the understanding of high-risk obstetrics. While the practice of obstetrics is changing with new information being gained every day because of advances in electronic technology this textbook has been compiled extensively to present information, as it exists today with probable look into the future.

Comprehensive Obstetrics in the Tropics has been written by teachers in the sub-region to address the gaps in knowledge about what is known and what is still unknown. In this manner, we hope, medical students, general practitioners and those in fellowship programmes in obstetrics, will use the book. It should also serve as a reference to other specialists both in developing and developed countries and thus light a path to future research on women's health.

This is the first edition of *Comprehensive Obstetrics in the Tropics*. Now a neonate, which has completed its months and years of gestation. We would appreciate comments, both positive and negative, from our readers

E. Y. Kwawukume
E. Ejiro Emuveyan

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Finally, all the contributors owe so much to the secretarial support provided by Miss Winifred Dela Asase, Mrs. Mercy Sarpeh, Miss Pearl Amanfu, Mr. Ben Addo and Mr. H.O. Offei of Korle Bu Teaching Hospital, Accra, Ghana.

SECTION 1- ANTENATAL CARE	<i>Page</i>
CHAPTER 1 -the Unborn Patient <i>E Y Kwawukume/Rebecca Acquah-Arhin</i>	1-6
CHAPTER 2 - Preconception And Antenatal Care <i>A. Omigbodun</i>	7-14
CHAPTER 3 - Fetal Surveillance <i>N R K Damale</i>	15-25
CHAPTER 4 - Ultrasonography In Pregnancy <i>E E Emuveyan</i>	26-31
CHAPTER 5 - Normal Physiology In Pregnancy <i>E E Emuveyan</i>	32-37
CHAPTER 6 - HIV In Pregnancy <i>N R K Damale</i>	38-46
CHAPTER 7 - Nutrition In Pregnancy <i>Cecil Klufio</i>	47-58

SECTION 2 - INTRAPARTUM CARE	
CHAPTER 8 - Physiology And Management Of Labour <i>E E Emuveyan</i>	59-67
CHAPTER 9 - The Partograph <i>E Y Kwawukume/ Ali Samba/ J B Wilson</i>	68-76
CHAPTER 10 - Obstructed Labour <i>S A Qbed</i>	77-85
CHAPTER 11 -Ruptured <i>K A Danso</i>	86-92
CHAPTER 12 -Obstetric Anaesthesia & Analgesia <i>Frank Boni</i>	93-104
CHAPTER 13 -Fetal Distress <i>E Y Kwawukume/ O Fakeye</i>	105 -109
CHAPTER 14 -Shoulder Dystocia <i>Sarah Baffoe</i>	110-114

CHAPTER 15 -postpartum Haemorrhage (PPH) <i>E Y Kwawukume</i>	115-121
CHAPTER 16 -vaginal Birth After Caesarean Section <i>A Omigbodun</i>	122-128
CHAPTER 17 - Induction And Augmentation Of Labour <i>E Y Kwawukume</i>	129-134
SECTION 3 - COMPLICATED PREGNANCIES	
✓CHAPTER 18 -Prolonged Pregnancy <i>E E Emuveyan</i>	135-139
CHAPTER 19 -Antepartum Haemorrhage (APH) ✓ <i>E Y Kwawukume</i>	140-150
CHAPTER 20 -Premature Rapture Of Membranes (PROM) ✓ <i>E Y Kwawukume</i>	151-156
CHAPTER 21 -Breech Presentation <i>C T John and A O U Okpani</i>	157-161
CHAPTER 22 -Multiple Pregnancy <i>K Nkyeker</i>	162-172
CHAPTER 23 -Hypertension In Pregnancy <i>E Y Kwawukume</i>	173-184
✓CHAPTER 24 -Intrauterine Growth Restriction <i>K Nkyekyer</i>	185-192
CHAPTER 25 -Intrauterine Fetal Death ✓ <i>S A Obed</i>	193-197
CHAPTER 26 -Preterm Labour ✓ <i>S A Obed Kwawukume</i>	198-204
CHAPTER 27 -Vomitting In Pregnancy <i>Ali Samba/ E Y Kwawukume</i>	205-207
CHAPTER 28 - Cord Prolapse ✓ <i>E Y Kwawukume</i>	208-210
CHAPTER 29 -Ectopic Pregnancy <i>E Y Kwawukume/ A Idrisa</i>	211--218

CHAPTER 30 -Obesity In Pregnancy

CHAPTER 31 - Abortion 226-242
C A Klufio

CHAPTER 32 -Maternal Mortality In The Tropics 234-249
J B Wilson/A T Lassey

SECTION 4 MEDICAL COMPLICATIONS IN PREGNANCY

CHAPTER 33 -Malaria In Pregnancy 250-260
H O Opare/A T Odoi

CHAPTER 34 -Urinary Tract Infection In Pregnancy 261-267
V N Addo

CHAPTER 35 -Cardiac Disease In Pregnancy 268-274
V N Addo/G A Arthur

CHAPTER 36 -Asthma in pregnancy 275-278
V N Addo

CHAPTER 37 -Diabetes In Pregnancy 279-298
C A Klufio

CHAPTER 38 -Anaemia In Pregnancy 299-302
Ivy Ekem/ S A Obed

CHAPTER 39 -Sickle Cell Disease In Pregnancy 303-311
E Y Kwawukume

CHAPTER 40 -Renal Disease In Pregnancy 312-316
J D Seffah

CHAPTER 41 -Thyroid Disease In Pregnancy 317-320
J D Seffah

SECTION 5 - SURGICAL PROCEDURES

CHAPTER 42 -Caesarean Section 321-329
E Y Kwawukume

CHAPTER 43 -Cervical Incompetence 330-339
K Nkyekyer

Chapter 44 - Operative Vaginal Delivery

CHAPTER 44 -Operative Vaginal Delivery <i>HO Opare-Addo/AT Odoi</i>	340-351
CHAPTER 45 - Episiotomy & Third Degree Tear <i>EY Kwawukume/Ali Samba</i>	352-355

SECTION66 POSTPARTUM CARE

CHAPTER 46 -Puerperium ✓ <i>EY Kwawukume/Rebecca Acquah-Arhin</i>	356-362
CHAPTER 47 -Problems Of The Newborn <i>Jenifer Welbeck</i>	363-370
CHAPTER 48 -Family Planning Counselling ✓ <i>Gladys Kankam</i>	371-374
CHAPTER 49 -Family Planning ✓ <i>BDRT Annan/RM Adanu</i>	375-392

SECTION6 7 STATISTICS

CHAPTER 50 -Common Statistics <i>RB BRITWUM</i>	393-403
INDEX	404-412

SECTION 1
ANTENATAL CARE

The Unborn Patient

E.Y. Kwawukume and Rebecca Acquaaah-Arhin

Introduction

In the present century, for a variety of fetal disorders, doctors and parents no longer depend helplessly on what time or luck an intrauterine life presents them with at birth. They are no longer helpless spectators at the death of a baby. The day on which a child is born is not necessarily a simple matter of fate but a matter of whether the baby can be more safely and efficiently cared for in the uterus or out of it.

The World Health Organization's dream of "Health for all" now includes the unborn child, which needs medical treatment as well as scientific observation in a previously "opaque" womb. Studying the unborn child has been made possible because of the transformations in ultrasonography, which lit the path for researchers to scientifically observe the secrets around the fetus in the womb.

Radical changes about the unborn child have been documented in developed countries and some day, in the developing world, brighter prospects will be offered for a wide range of fetal disorders than the present dismal alternatives of neonatal death, abnormalities or therapeutic terminations of affected pregnancies.

Greek and Roman thinkers conceived the idea of a fetus as a Homunculus- a miniature person living and growing within the mother before birth¹. Although this might not explain the biologic transformation of cells in the first trimester to human being, at least it painted the picture of a fetus in the third trimester

The Fetus As A Patient

Technology and advances in research has made it possible for the fetus to become a patient whose illnesses could be diagnosed, investigated and treated in utero. The fetus has come a long way, from the biblical "seed" and mystical "homunculus" to an individual with medical problems that can be diagnosed and treated - **a patient**.

Although the fetus cannot make an appointment

and hardly complains, he/she needs someone to look after him/her. Who is the fetus' physician? The treatment of the unborn child requires the expertise of trained physicians in the care of mothers and babies. There should be a multidisciplinary approach incorporating the obstetrician and the neonatologist in the management of the unborn child.

The Fetus Became A Patient Through These Processes:

- * Development of molecular biology, which provided a conceptual framework.
- * Fetal activity felt by mother or palpated by the physician as a measure of fetal well being
- * Fetal heart beat by auscultation and later by electronic monitors as a reflection of stress and distress
- * Detection of gestational hormones in maternal blood and urine
- * Amniocentesis, which made possible the antenatal diagnosis of many inherited metabolic and chromosomal disorders and assessment of fetal lung maturity and severity of fetal haemolytic reactions
- * Ultrasonography, which is a safe non invasive imaging technique that enables accurate detection of normal and abnormal fetal anatomy with details and provides "live" moving pictures with no harm to mother or fetus. It is used to guide needle puncture and to introduce probes into the uterus for amniocentesis, fetal urine, fetal ascitic and cerebrospinal fluids, fetal blood and tissues.

The diagnosis of fetal diseases was initially treated by abortion until the 1960s when for the first time hydrops fetalis associated with maternal Rh sensitisation was treated successfully by intra-abdominal blood transfusion⁽²⁾.



Ultrasound in diagnosing fetal condition during antenatal care.

The second fetal disease treated in-utero was the devastating respiratory distress syndrome of prematurity by the administration of glucocorticoids to the fetus through the mother to increase the production of fetal surfactant and hasten fetal lung maturity. Other fetal disorders are now being treated including malformations such as hydronephrosis and obstructive hydrocephalus.

Fetal Diagnosis:

1 Congenital Abnormalities And Genetic Counseling:

Evidence from various studies show that majority of early spontaneous abortions especially in the first trimester are chromosomal anomalies and are a way of preventing the inheritance of bad gene mutation.

The causes are Genetic, which are mainly chromosomal aberrations and Environmental, comprising drugs, chemicals, maternal metabolic imbalance, infections and radiation.

Because the genetic ones can recur, counselling is important.

Counselling:

Genetic counselling aims at helping the individual or her whole family to understand the medical facts, including diagnosis, prognosis and possible treatment options, the risk of recurrence and the

reproductive options. This involves presenting medical and genetic information in a manner that they would understand, helping them make an informed choice and assessing and balancing the family's emotional state.

Some of the indications for counselling include:

- * Advanced maternal age
- * Previous baby with chromosomal anomaly
- * One of the couples with balanced chromosomal translocation
- * Mother with X-linked abnormal gene
- * Both parents with same autosomal recessive abnormal gene
- * Previous baby with neural tube defect

There is the need to screen the entire pregnant population by ultrasonography and maternal serum alpha-fetoprotein to detect the above-mentioned abnormalities but these investigations are not available in many parts of the sub-region. The main high-technology screening for our pregnant women is the detection of sickle cell disease in utero but this is limited to few centres though this disease is very common in our sub region.

Complications of counselling might include, anxiety, communicating error in making the diagnosis or confirming it, estimating risk of malformation, risk of miscarriage and stillborn.

2. Chorionic Villus Sampling

It is a method of first trimester antenatal diagnosis of genetic disorders and serves as an alternative to amniocentesis. Amniocentesis is performed in the second trimester and it takes up to 4 weeks for diagnosis to be made. This increases anxiety for the patient and if an abnormality is detected, termination of pregnancy carries increased risks and the methods available might not be acceptable to the patient. Chorionic villus sampling gives reliable results because the trophoblastic tissue has the same genetic constitution as the fetus and samples of chorionic villi reflect the biochemical, chromosomal and DNA status of the fetus. It is done under ultrasound guidance.

Indications are mainly for couples at risk for a fetus with a chromosomal abnormality as outlined above. For cytogenetic analysis, early results are obtained in 24-48 hours but the quality of the

preparations is not optimal for analysis. Reliable results are obtained by culture of the villi and are ready by 7-10 days. All disorders that require DNA probe, such as Duchenne muscular dystrophy, cystic fibrosis and phenylketonuria can be diagnosed with this method because the DNA content of all fetal cells is identical and recombinant techniques can be used for the diagnosis.

Advantages:

- * Early timing
- * Short culture time with results obtained earlier than amniocentesis
- * Early diagnosis and decision making for therapy is quickly made.

Disadvantages:

- * Pregnancy loss
- * False diagnosis
- * Rhesus isoimmunisation
- * Prematurity
- * Birth defects
- * Placental abnormalities.

Amniocentesis is mostly done in the Second trimester and the culture results take longer than chorionic villus sampling. It was the first method used for the diagnosis of chromosomal abnormalities. It is also done under ultrasound guidance and has some complications.

RISKS:

- * Fetal loss,
- * Hip dislocation
- * Talipes equinovarus,
- * Fetomaternal haemorrhage
- * leading to Rh isoimmunisation

Maternal Serum Alpha Fetoprotein

This is an investigation to detect chromosomal abnormalities for high-risk patients. High level is a diagnostic marker for Neural Tube Defects in particular. The test is also positive for other defects such as Open Ventral Wall Defects (gastrochisis, omphalocele), Maternal Serum Alpha-Fetoprotein (MSAFP) levels are low in pregnancies in which the fetuses have Down syndrome and trisomy 18, probably because AFP production in the fetal liver is low or there is rapid removal of AFP from the fetal circulation.⁷

Fetal Blood Sampling

It is done under ultrasound guidance and the cord is entered very close to its insertion site. The main difficulties are maternal obesity, oligohydramnios, polyhydramnios and fetal position with a posterior placenta. Obesity decreases the clarity of the ultrasound image and long needles might have to be used making manipulation difficult.

Oligohydramnios decreases the quality of the ultrasound image and might interfere with the visualisation of the cord insertion. A posterior placenta might compound this. Polyhydramnios with interposition of the fetus and posterior placenta might make access to the cord insertion difficult.

Indications:

- * Prenatal diagnosis of congenital disorders
- * Infections
- * Hemoglobinopathies
- * Rapid Karyotyping
- * Coagulopathies
- * Inborn Errors of Metabolism
- * Assessment of fetal well being
- * Chronic fetal stress
- * Intravenous Fetal Therapy
- * Fetal Pharmacology

Fetal DNA analysis, computed tomography and magnetic resonance imaging are being used in developed countries while in our sub-region ultrasound is the main method available.

The Amniotic Fluid

For the normal development of the unborn child, the amniotic fluid acts as a cushion, allows movements, permits normal lung development, stabilizes the temperature and acts as a barrier against infections. The volume depends on the balance between its production from chorion frondosum, skin, urinary and respiratory tracts and reabsorption from gastrointestinal tract, respiratory tract, amniotic- chorionic interface with maternal uterine wall.

The volume at 12 weeks gestation is about 60mls, at 16 weeks it is 170mls, and at 20 weeks it is 500mls. It reaches its peak at 36-37 weeks with an

average value of 1000mls and subsequently declines to as low as 250mls at 43 weeks^{8,9,10,11,12,13}

Oligohydramnios could be due to diseases such as renal disorders, preterm premature rupture of membranes, intrauterine growth retardation and post maturity. But blood flow velocity waveform may help to differentiate IUGR from impaired placental perfusion from fetal structural anomalies.

Polyhydramnios could be the result of diabetes mellitus, congenital malformations and multiple gestations. The over distension can lead to preterm labour and delivery.

Treatment

Lily's² successful blood transfusions into the peritoneal cavity of the fetus affected with erythroblastosis fetalis might be considered as the beginning of modern fetal surgery. Today, the possibility of treating some fetal disorders in-utero gives an entirely new concept of antenatal care. Treatable disorders are now recognized, and this directly influences clinical management of the pregnancy. The probability of fetal treatment in-utero raises questions about the pathophysiology of fetal organ development and the technical difficulties of management before birth. These new developments raise the questions about complex ethical questions about risks and benefits of the rights of the mother and the fetus as patients. These are the difficult issues to address in the future.

The number of defects and conditions that can be corrected before birth is slowly increasing, though most therapies are still left untreated until after delivery¹⁴.

The common fetal abnormalities, which can be treated in utero, are:

1 Defects treated by selective abortion:

- * Anencephaly, hydraencephaly, holoprocencephaly
- * Severe chromosomal anomalies
- * Bilateral renal agenesis
- * Lethal bone dysplasia.

2 Defects that can influence the mode of delivery due to possible dystocia, requiring

immediate surgery or cannot stand labour:

- * Conjoined twins
- * Giant omphalocele
- * Severe hydrocephalus, large ruptured meningomyelocele
- * Large sacrococcygeal teratoma, cervical cystic hygroma
- * Malformations requiring preterm delivery in the presence of inadequate labour or fetal distress.

3 Defects that may benefit from induced preterm delivery for early correction outside the uterus with recent advances in stimulating fetal surfactant production:

- * Urinary tract obstruction-hydronephrosis
- * Obstructive hydrocephalus
- * Gastrochisis or ruptured omphalocele to prevent thick fibrous inflammatory coat.
- * Hydrops fetalis
- * Intra uterine growth retardation
- * Arrhythmias

4 Defects that may require medical intervention before delivery:

Defects

Anaemia
 Surfactant deficiency
 Metabolic block
 Carboxylase deficiency
 Cardiac arrhythmias
 Endocrine deficiency
 Hypothyroidism
 Adrenal hyperplasia
 Nutritional deficiency
 Haematopoietic defect
 Single gene defect

Intervention

Red blood cells
 Glucocorticoids
 Vitamin B-12
 Biotin
 Digitalis, Propanolol, procainamide
 Thyroxin
 Corticosteroids
 Protein calories
 Stem-cell reconstitution
 Single gene replacement or Stem-Reconstitution

5 Defects that may benefit from surgical intervention before delivery:

Defects	Pathology	Complication
Urinary tract obstruction	hydronephrosis/lung hypoplasia	renal/respiratory failure
Diaphragmatic hernia	lung hypoplasia	respiratory failure
Cerebrospinal fluid obstruction	hydrocephalus	brain damage
Sacrococcygeal teratoma	high-output failure	fetal hydrops/demise
Complete heart block	low out-put failure	fetal hydrops/demise
Ventricular outflow obstruction	pulmonary vascular	pulmonary hypertension
Hypoplasia		

Selective termination of multifetal pregnancies in the first trimester:

With the introduction of antenatal genetic diagnosis and counselling couples now have the option to terminate pregnancies in which the fetus has a birth defect. Some of the techniques used are:

- * Cardiac puncture with Air Embolization
- * Intracardiac/ Intrathoracic
- * Potassium chloride injection
- * Cardiac tamponade
- * Exsanguination
- * Hysterotomy

These advances in technology require special attention to the anaesthetic management of both mother and fetus in order to avoid fetal and maternal morbidity. The objectives include maternal safety, avoidance of teratogenic drugs, and avoidance of fetal asphyxia, fetal anaesthesia and monitoring, uterine relaxation and prevention of preterm labour.

Discussions and Controversies

In most developing countries, the experience in fetal management is limited but the growing knowledge and experiences from the developed world are being followed with keen interest towards the future. Fetal therapy raises a lot of ethical and legal issues. Some of these are whether the refusal of fetal therapy by mothers be respected or overridden by health authorities. In addition, defining the benefits and risks of fetal diagnosis and treatment for the fetus and the mother need precise counselling. Other questions might be whether the risks and benefits of experimental fetal therapy require ethical consideration by prior group review and how should cases for experimental fetal therapy be selected? There are still a lot of unanswered questions about the unborn child. The

date of confinement is still questionable and viability is still uncertain. These are questions that would soon face fetal surgeons in our sub-region. The answers to these problems will be found only with further research. Answers to these will enable obstetricians give comprehensive and intensive care to the fetus and with such findings the fetus may become a "born-again" patient.

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Preconception and Antenatal Care

Akinyinka O. Omigbodun

Introduction

Preconception and antenatal care are forms of health care whose purpose is the improvement of prospects for safe motherhood. To achieve this purpose, both forms of care have to be delivered in the context of comprehensive maternity care that, in addition to preconception and antenatal care, must also include wholesome intrapartum and decent postnatal care (Table 1).

Maternity care has existed in some form for several thousand years. A clear reference is made to the activities of midwives in the book of Exodus (Chapter 2, verses 15-22). Presumably, such activities were restricted to the period of parturition because the passage mentions that the midwives had to explain their inability to enforce the infanticide decree of the ruling Egyptian monarch on the grounds that the Hebrew women had often delivered before the midwives arrived on the scene to assist them. In the past century, maternity care moved from being a totally intrapartum affair into the realms of antenatal supervision and care. This trend commenced in major metropolitan centres like Paris, New York, Adelaide and Edinburgh and later extended to other parts of the world. Eventually, the interest of reproductive health care providers also extended to the period immediately after childbirth and formal postnatal care evolved. Most recently, certainly within the last twenty-five years, preconception care arrived on the scene⁽¹⁾.

In this chapter, the main objectives of preconception and antenatal care will be outlined and the approaches to the organization of these forms of care will be discussed. The potential pitfalls in the provision of such care and the defects in the current programmes of antenatal care will also be mentioned.

Preconception Care

Preconception care can be described as a specialised form of care for women of reproductive age, before the onset of pregnancy, to detect, treat

or counsel them about pre-existing medical and social conditions that may militate against safe motherhood and the delivery of healthy offspring. It involves the taking of a comprehensive health history, a thorough physical examination, appropriate ancillary screening tests and health promotion interventions in a woman planning to embark on the voyage of pregnancy and childbirth.

Table 1

Components of Comprehensive Maternity Care

1. **Preconception Care**
2. **Antenatal Care**
3. **Intrapartum Care**
4. **Postnatal Care**

Rationale for Conducting Preconception Care:

It is becoming more apparent that interventions made during the preconception period are just as crucial as those made in the subsequent nine months of prenatal care in order to achieve the best maternal and fetal outcome⁽²⁾. The most critical period for human fetal organogenesis is between the 17th and 56th days of intrauterine life, a point at which most women are barely coming to terms with the fact that they may be pregnant⁽³⁾. Many of them will not present for antenatal care until several weeks afterwards, by which time a lot of damage may have been done in those at greatest risk⁽⁴⁾. The optimal time to assess these women and manage the situations that are likely to compromise the successful outcome of their pregnancies is before the pregnancy occurs. This is particularly so for many women who abuse drugs such as alcohol and narcotics as these substances tend to inflict maximum damage during the first few

weeks of pregnancy. Bringing the women under supervision before pregnancy allows for their education, counselling and the reduction of the blood and tissue levels of the drugs and their metabolites before the fetus comes under their influence⁽⁶⁾. Some of the agents that are used to get them off their addiction may not be safe for the fetus either. Hence, although preconception care offers a wide choice of possible intervention measures, many of these may be unavailable once pregnancy has commenced.

The women who are likely to benefit the most from a programme of pre-pregnancy intervention include those with pre-existing chronic medical conditions such as diabetes mellitus, epilepsy and coagulation disorders. Others whose social circumstances are difficult, particularly drug abusers and those with nutritional deficiency also stand to derive a lot of benefit from such programmes. Several studies have demonstrated the effectiveness of administering folic acid supplements to women during the periconceptional period in preventing neural tube defects in their babies^(6,7). Thus, a preconception care programme that identifies women with evidence of folate deficiency for special supplements of folic acid or that administers the vitamin to all women planning to get pregnant should help to reduce the incidence of this group of congenital anomalies in the community.

Some could argue that a lot of the women with pre-existing medical problems are already receiving some form of care, either from their family health care provider or even from specialist physicians. Available evidence has however shown that such an informal form of care is seldom effective in improving pregnancy outcome^(8,9). Diabetic women who received a non-formalized form of preconception care had a relatively high incidence of unplanned pregnancies and hyperglycaemia during the first trimester whereas, structured pre-pregnancy care was demonstrated to improve metabolic control in diabetic women in the first and subsequent trimesters, and to reduce the incidence of spontaneous abortions and major congenital abnormalities in their progeny^(9,10,11). Women who attended preconception clinics were also shown to be more likely to book for antenatal care earlier and with lower levels of glycosylated haemoglobin, to have a lower incidence of preterm births, small for gestational age (SGA) babies, lower incidence of macrosomia, fewer admissions into neonatal intensive care and fewer neonatal deaths¹¹. Thus, a pre-conception clinic has a positive impact on both neonatal morbidity and mortality in diabetic

mothers

Apart from having a structured programme of care, the duration of participation of the women also seems to have an impact on outcome. In a study of women with a history of seizures, it was found that the pro-active measures that were taken such as counselling and changing of drug regimen, were demonstrably effective only if the woman was prepared to wait for periods of up to one year to allow for adequate counselling and necessary drug changes⁽¹²⁾. The waiting period afforded the care provider adequate time to withdraw anti-epileptic drugs that carried significant teratogenic risk and substitute them with drugs thought to have less risk. It also provided an opportunity to re-investigate the original illness and ascertain the correct diagnosis. Some of the women originally thought to have epilepsy in the study referred to above⁽¹²⁾ turned out to have other lesions that were best managed before the onset of pregnancy.

One additional major advantage of a structured programme of preconception care is its cost-effectiveness. A group of diabetic women who had preconception care which extended into prenatal care was compared with another group that had only prenatal care⁽¹³⁾. The combined number of outpatient visits during pregnancy for the two groups were similar, but women who had preconception care were less likely to be hospitalised during pregnancy and tended to have shorter inpatient stays. The mean length of stay after delivery was significantly shorter for women who had preconception care; the intensity of care they required tended to be lower and the length of stay of their neonates was shorter than for women who had prenatal care only. The net effect of these differences was an average cost saving of approximately \$34,000 per patient. Hence, preconception care achieved its major intended health benefits and was associated with reduced resource utilization and substantially reduced costs⁽¹³⁾.

Taken together, these advantages of improved maternal health, significant reduction in neonatal morbidity and mortality, an unparalleled opportunity for counselling and other pro-active health activities and substantial cost-savings offer a compelling reason for the wide adoption of preconception care. It is a matter of serious concern that this form of care is yet to be introduced into the maternity care programme of any country in the West African sub-region

Objectives of Preconception Care:

Preconception care offers a unique opportunity for reproductive health care providers, particularly Obstetricians and Gynaecologists, to expand their role in the prevention of maternal and fetal morbidity and in fostering activities directed at health promotion in the female population. The main objectives of a sound preconception care service will include^(1,4):

- * Avoidance of maternal exposure to teratogens during the period of fetal organogenesis
- * Checking basic measurements such as weight and blood pressure before pregnancy begins
- * Introduction of intending mothers to social and medical interventions at a time when they can have maximal effect on the outcome of pregnancy

Good preconception care should be able to identify factors that are subject to modification in a woman that, if left alone, will adversely affect the outcome of pregnancy in such a person. Such factors will include pre-existing medical conditions that are in themselves teratogenic (for example diabetes mellitus) or those for which some of the types of therapy available could be teratogenic (for example phenytoin in seizure disorders and angiotensin converting enzyme inhibitors in hypertension). Other factors such as poor nutrition

and recreational drug use also belong to this category. Appropriate modifications can then be applied.

In many patients, the risk factors that are identifiable are not subject to modification. In such patients, proper counselling is required to ensure that the patient understands the extent of the potential risk and that she is made aware of the possible courses of action she could decide to follow. A summary of these modifiable and fixed factors is provided in Table 2.

Organisation of a Preconception Care Service:

Ideally, a preconception clinic should operate in tandem with the antenatal clinic in a seamless fashion and some have even come up with the concept of a 'periconception' clinic that caters for women planning pregnancies and those who are in the first trimester^(14,15). The only drawback of this approach is that its activities may become so subsumed in antenatal clinic activities that the patients fail to receive the kind of structured care that has been shown to be the most effective approach to preconception care services⁽⁹⁾. Thus, a special time should be allotted solely for preconception clinic activities. A preconception clinic should provide a framework for health promotion and disease prevention that is designed to reach all women with childbearing potential in the community. Specialists in the field of obstetrics and gynaecology should direct such clinics. A multispecialty effort is however required to achieve

Table 2.

Factors Predating Pregnancy with Potential Effects on Pregnancy Outcome

Modifiable Factors

- Drug Abuse
(e.g. Alcohol, Tobacco, Narcotics)
- Medication
- Pre-existing Maternal Diseases
(eg. Diabetes, Epilepsy, HIV/AIDS)
- Nutrition
- Physical Exercise
- Psychosocial Problems
- Socio-Economic Class*

Unmodifiable Factors

- Maternal Age
- Maternal Parity
- Previous Obstetric History
- Maternal Genetic Make-up
- Paternal Genetic Make-up

*This requires a prolonged period of intervention, usually outside the scope of healthcare providers.

the best level of care ⁽⁶⁾. Thus, specialists from various branches of internal medicine (endocrinologists, neurologists, haematologists, psychiatrists), midwives, nurses, clinical psychologists, clinical geneticists and genetic counsellors all have a role to play.

Patient Recruitment:

One of the major stumbling blocks to getting a preconception care service going is the identification of the means by which those that actually need the services can be reached ⁽⁷⁾. Various ways have been suggested for reaching the women in need. Women who have negative pregnancy tests are one such group. In a study of such a group of women presenting to a family practice, more than half of them had risk factors that ought to be addressed in a preconception care setting before they made an attempt at another

pregnancy ⁽¹⁶⁾. Other groups that can form a pool of potential recruits are women attending family planning clinics and those attending the well-women clinics that are often run by health maintenance organizations ⁽²⁾. Certainly many health care providers believe that an opportunistic approach can ensure adequate coverage of the women at risk ⁽¹⁷⁾. In addition to these, certain categories of women (see Table 3) should mandatorily be counselled to accept preconception care.

Activities:

The activities carried out in a preconception clinic should include:

- * screening for genetic carrier states in both potential parents

Table 3:

Indications for Mandatory Referral for Preconception Care	
1. Pre-Existing Medical Conditions	
Cardiac Disease	Congenital Heart Lesions, Rheumatic Carditis, Cardiomyopathy
Systemic Hypertension	
Haematological Disorders	Haemoglobinopathy, Rhesus Isoimmunisation, Coagulopathies, Leukaemia
Autoimmune Diseases	Rheumatoid Arthritis, Systemic Lupus Erythematosus
Genitourinary Disease	Sexually Transmitted Infections
Neurological Disorders	Seizure Disorders, Peripheral Neuritis
Endocrinopathies	Diabetes Mellitus, Hyperthyroidism, Hypothyroidism, Adrenal Disease
Chronic Pulmonary Disease	Tuberculosis, Obstructive Airway Disease
2. Advanced Maternal Age	Screening for Chromosomal Abnormalities, Genetic Counselling
3. Adverse Social History	Use of Psychoactive Drugs, Victim of Domestic Violence
4. Adverse Family History	Genetic Screening and Counselling
5. Adverse Obstetric History	Recurrent Spontaneous Abortions
6. Adverse Medication History	Vaccination with Live Viruses, Use of Anti-Oestrogens (Clomiphene)

- * checking for pre-existing medical conditions such as anaemia, human immunodeficiency virus infection and the acquired immune deficiency syndrome (HIV/AIDS), epilepsy, diabetes mellitus and other endocrinopathies.
- * screening for nutritional deficiency and providing periconceptual vitamin supplementation
- * checking hepatitis B surface antigen and vaccinating those at highest risk (especially drug abusers and health care workers)
- * checking rubella antibody titres and giving immunization. Nearly 25% of women of childbearing age screened in Lagos, Nigeria had no antibodies to rubella ⁽¹⁸⁾, making this an important public health issue.
- * counselling on psychosocial risk factors such as drinking alcohol, smoking, exercise, occupational exposure to teratogens, and avoidance of environmental toxins.
- * contraceptive advice and provision of effective contraception
- * precise dating of pregnancies from encouraging patients to keep menstrual calendars.

Constraints:

There are several obstacles in the way of setting up a successful preconception care service. The most significant is probably the well-known fact that a substantial proportion of all pregnancies are unplanned, leaving no preconception period where the women can present themselves for care. Even among women who are known to be suffering from diabetes mellitus and who may have had pregnancy complications related to the disease in the past, majority of their pregnancies are not planned ⁽¹⁹⁾.

Another difficulty is the attitude of the female population for whom these programmes are designed. A study among women of childbearing age in an area of London, England revealed considerable skepticism about the value of preconception care ⁽¹⁷⁾. Dropout rates from attendance at some preconception clinics approach 50 percent ⁽²⁰⁾, largely because the women were not convinced that attendance was beneficial to them. There is a need for community education in this regard so that the level of acceptance of preconception care will come close

to that of antenatal care, which is now firmly established in the consciousness of the womenfolk in most parts of the world. Reproductive health care providers may also be impeding the introduction of preconception care because of an inherent aversion to something that is unfamiliar. Many of the current practitioners were trained in an era when this form of care was virtually unknown and they may be reluctant to participate in such a venture. Retraining and continuing education activities can help in resolving this.

Antenatal Care

Antenatal care is a specialised pattern of care organised for pregnant women to enable them attain and maintain a state of good health throughout pregnancy, and to improve their chances of having safe delivery of healthy infants at term. It is an indispensable part of an effective maternity care service. It involves a thorough initial assessment to determine the level of risk faced by the mother and the fetus, and continuing follow-up care right through the duration of the pregnancy.

Considering the level of acceptance that antenatal care now enjoys worldwide, it is often easy to forget that the first antenatal clinics were established less than one hundred years ago ⁽²¹⁾. Antenatal care has however undergone remarkable changes in the last thirty years, largely because of two important innovations: the use of real time ultrasonography and the mind-boggling advances that have been made in the field of prenatal diagnosis and fetal therapy. Some of the procedures that are routinely done in pregnant women and their fetuses today were the stuff that would have graced the pages of a science fiction novel as recently as fifty years ago! Yet, for all the benefits that have been attributed to it, the effectiveness of antenatal care in actually reducing maternal and fetal morbidity/mortality has never been scientifically proven ⁽²¹⁾ and, because of ethical considerations, may never be.

Purpose of Antenatal Care:

The main objectives of antenatal care can be summarised thus:

Screening for potential complications in the mother and the fetus

- * prevention of maternal and fetal complications through appropriate proactive measures
- * treatment of any emergent problems in the pregnant woman and her fetus
- * preparation of the parents for the process of

childbirth and child rearing

The various activities that are usually undertaken to achieve these objectives differ from place to place, depending on the hazards that plague a pregnant woman in a particular community. The approach discussed here is particularly useful in the typical low-resource, tropical setting, although some reference will be made to some of the modern advances that ought to be a part of sound antenatal care practices in any institution offering standard reproductive health care.

Conduct of Antenatal Care:*Obstetric Risk Assessment:*

The main purpose of risk assessment in a pregnant woman is to attempt to quantify the potential level of risk she faces in going through pregnancy and childbirth. The steps taken include a definitive diagnosis of pregnancy, an accurate estimation of the gestational age and the anticipated date of delivery, a recording of previous obstetric problems and the problems in the current pregnancy, an evaluation of any pre-existing medical disorders, enquiries into any familial medical problems, an attempt to assess the patient's social circumstances and its potential effect on the pregnancy, a thorough physical examination and the use of appropriate ancillary tests ^(22,23). Pregnancy provides a unique chance to assess a woman's general health and this opportunity should be used maximally for her benefit.

A definitive diagnosis of pregnancy should be made before the patient is booked for antenatal care so as to avoid unwittingly enrolling a woman with pseudocyesis into a prenatal care programme. At the initial visit, a comprehensive health history must be obtained, including a systematic inquiry into past pregnancies, labour and puerpera, and information about lactation and contraception during birth intervals. Information on the babies' birth weights and gender should also be included. The patient should be questioned about any symptoms she may have had during the current pregnancy, especially those suggestive of genital tract infections or pregnancy-induced hypertension. Any history of genital bleeding at any stage of gestation must be noted. The other items of information to obtain at the initial interview include information about pre-existing diseases like heart disease, lung disease, kidney diseases, diabetes mellitus or hypertension. Patient should also provide information about a family history of conditions like diabetes, hypertension, haemoglobinopathies and multiple gestations.

Finally, a detailed social history should be taken, including the demographic details of the baby's father and the use of any medication or psychoactive substances by the mother prior to and during the pregnancy.

After interviewing the patient, she should have a complete general and systemic examination including a recording of maternal weight, height and blood pressure, checks for pallor, oedema, varicose veins and jaundice. There should be a proper breast examination to rule out the presence of pathology and to assess preparedness for breastfeeding. The abdomen should be examined and, in primigravidae, a pelvic examination should be done to exclude soft tissue abnormalities in the lower genital tract.

The ancillary tests to be done should include:

- * Urinalysis for glucose, protein and acetone
- * Urine microscopy (and culture if necessary)
- * Haematocrit estimation, blood grouping,
- * Rhesus typing and tests for immune antibodies.
- * Serological tests for syphilis, rubella and the human immunodeficiency virus (HIV)
- * Maternal serum alpha-fetoprotein test.
- * Papanicolaou smears to detect cervical intraepithelial neoplasia and some microbial infections.

At the end of all these steps, the information obtained helps in categorising the pregnant woman into two groups: High Risk or Low Risk. It is important to emphasise that the risk status is not a static grouping. Events occurring as pregnancy progresses may compel a reassessment of the risk category.

Continuing Care:

Patient is placed on chemoprophylaxis against malaria in endemic areas. An appropriate schedule is worked out for tetanus toxoid administration, usually starting from the day of the first clinic visit with a second dose being administered 4 to 6 weeks later. Iron and folic acid are also prescribed as prophylaxis against anaemia. Having assigned the woman to the appropriate risk category, a

suitable schedule of visits suited to her needs is worked out. There is considerable controversy as to whether the number of antenatal visits has any bearing on pregnancy outcome and there is scant evidence in support of rigid schedules of visits⁽²¹⁾. Nonetheless, there is general agreement that the visits should be more frequent as term approaches because majority of pregnancy complications tend to occur in the third trimester.

At each antenatal clinic visit, the patient should be encouraged to express herself about her state of health and all complaints should be discussed, using this as an opportunity for health education. She should be encouraged to come with her spouse, so that they could be jointly educated about healthy lifestyle choices, good nutrition and the joint formulation of a birth plan. In making the birth plan, the couple should be informed about the expected date of delivery (including the limitations of such predictions!), the signs of danger in pregnancy, the signs to expect once labour has commenced and danger signs to watch out for in their newborn baby. Thereafter, they should decide on where the baby is to be born, who will conduct the delivery, how funds will be set aside for transportation and other expenses at the time of birth, and how they will ensure that there is a safe birth interval before the next pregnancy and delivery.

Each visit is also an opportunity to reassess the risk status of the woman. Parameters that ought to be checked include weight gain, blood pressure, urinalysis for glycosuria and proteinuria, haematocrit (once every trimester except in anaemic patients), and the rate of growth of the symphysio-fundal height, the lie of the fetus, its presentation and fetal heart tones. Any abnormalities are met with commensurate intervention measures.

To ensure that women derive optimal benefits from antenatal care, it is important that they be encouraged to register early. Many women in this environment, particularly the multiparous ones, often delay booking until well into the second and sometimes the third trimester of pregnancy^(24,25). This is a practice that has to be changed through community education and enlightenment. In addition, there is a need for a re-appraisal of our antenatal care practices in the sub-region to identify those measures that are really effective and should be continued, and those that are an unnecessary drain on resources and should be stopped.

Summary

Many pregnancies are predisposed to complications by events that occur in the earliest weeks of gestation when fetal organogenesis is taking place. Often, these complications occur even before the woman knows that she has become pregnant. Preconception care offers patients the opportunity to address these conditions before conception, thereby increasing the potential for a satisfactory pregnancy outcome. The main purpose of preconception care is to allow the introduction of medical and social intervention measures that improve pregnancy outcome even before the onset of the pregnancy, the ultimate form of pro-active care. Since it is a relatively new form of care, concerted efforts are required to overcome skepticism about its value on the part of the female population and resistance to a new form of care among healthcare providers.

When it is confirmed that conception has occurred, it is important that the patient should have a thorough initial evaluation and an accurate assessment of the risk to which she is exposed so that the appropriate level of monitoring is instituted. A patient's exposure to risk may change as the pregnancy progresses, thus periodic revision of risk status is essential. The male partner's cooperation must be enlisted and the couple must be encouraged, as early as possible during the pregnancy, to fashion out a viable birth plan. A meticulous implementation of a standard programme of prenatal care is a crucial step on the road to safe motherhood.

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Fetal Surveillance

NRK Damale

Introduction

The year 2000 deadline the world set to reduce maternal mortality by 50% has come and gone. The target has however not been achieved. Over 98% of these deaths occur in the developing world¹ and continue to occupy health workers and policy makers alike.

In contrast, in the developed world, the significant inroads made into reducing maternal mortality has brought to the fore perinatal morbidity and mortality. In most developed countries the perinatal mortality rates (PMR) have been reduced from one-third to one fourth of those prevalent four decades ago.^(2,3) This has been achieved with the development of fetal medicine units, expensive equipment and new drugs over the last few decades. Modern antenatal care now involves checking both maternal health and fetal well-being and the more successful the outcome of each pregnancy the fewer times women in the developing world would travel on 'the road to death' seeking to complete their desired family size.

Despite its limitations antenatal care in parts of the developing world remains the most effective means of saving maternal and perinatal lives. In Zaria, Nigeria, antenatal care has reduced maternal mortality seven fold and perinatal mortality six fold⁽⁴⁾

However, a great proportion of the population in developing countries dwell in rural areas with only about 35% of all births being assisted by trained attendants in Africa, and 64% in Latin America, as opposed to 93% in East Asia and virtually 100% in North America.⁽⁵⁾ This would in part account for the high perinatal mortality in most developing countries, varying from 25 to 60 per thousand births while most of the developed world record below 10 per thousand births.

In order to tackle the problem in the developing world, it is essential, as already indicated, to adopt a primary care approach to fetal surveillance. This will provide opportunity to the majority of pregnant women to have access to trained health workers

ensuring that high-risk cases identified benefit from locally available health facilities.

Definitions:

Fetal surveillance encompasses all measures taken in pregnancy to determine the well being of the fetus up to the delivery of a healthy baby. Pregnancy and childbirth may still be regarded as a physiological event in the majority of women both in the developed and developing countries of the world. But in some pregnancies, conditions in the mother and the environment appear to predestine problems for the fetus that may lead to its failure to achieve its growth potential or end in a stillbirth.

Low birth weight (LBW) babies defined as those weighing less than 2500gram at birth are a major contributor to perinatal mortality and identifying such pregnancies for surveillance would start addressing the problem.

The LBW babies consist of two distinct groups.

The Preterm infants are those infants born before 37 completed weeks of gestation, from the first day of the last menstrual period. This is mentioned here because it is an important contributor to LBW.

This group of babies while continuing to be a major contributor to perinatal morbidity and mortality and attracting significant research is beyond the scope of this chapter.

Small For Gestational Age (SGA) is defined as birth weight (or anthropometric measurement) less than the 10th percentile for its specific gestational age. Some SGA infants may be constitutionally small and may represent only the tail end of a normal distribution.

In clinical practice the limitation of such a definition is the inclusion of preterm infants as mentioned for LBW and the exclusion of intra-uterine growth retarded infants weighing 2500grams or more.

Intra Uterine Growth Retardation (IUGR) A fetus is growth retarded when it fails to achieve its genetic growth potential. Such a fetus may

stillweigh 2500grams or more and not fit in the category of SGA. The definition is problematic since we are not able to determine the inherent growth potential of the fetus. The incidence is only about 5% rising to 10% in women considered at high risk.

The identification of this group is important because:

These fetuses are at high risk of perinatal morbidity and mortality (>20 fold) compared with the normal weight peer population^{6,7}.

Long term neurological and intellectual impairment has also been reported and it is being suggested that early recognition and delivery (34-35week gestation) could prevent this. The growth retardation intervention trial (GRIT) in the United Kingdom could throw more light on this and is eagerly awaited.

Fetal Surveillance measures are in two parts

Ante-partum fetal surveillance: which are antenatal measures before the onset of labour.

Intra-partum fetal surveillance: this is the more intensive measure during labour at which time uterine contractions introduce a further risk of fetal hypoxia.

In practice however this is a continuum as intrapartum surveillance should soon follow the antepartum measures.

The Aim of Fetal Surveillance:

The aims of fetal surveillance are two fold.

- i) To prevent fetal death or damage from chronic hypoxaemia
- ii) To deter unnecessary intervention and so limit iatrogenic prematurity and unwanted operative delivery.

Aetiology of fetal compromise:

The cause of a stillbirth could be elusive. In a thorough investigation of 3619 cases of stillbirths in the United Kingdom 70.8% were unexplained antepartum deaths and this highlights the magnitude of the problem.⁽⁸⁾

To mount a high level of surveillance in all pregnancies to reduce this may not only be unsuccessful, it would not be possible in developing countries with limited facilities and

personnel. A more practical step would be to identify the conditions, which are associated with fetal growth restriction, fetal loss or damage and low birth weight. The low birth weight group, of relevance to this chapter is those babies born at term hence it is of utmost importance the gestational age is known. This information is unavailable or inaccurate in developing countries because of absence of good data recording and processing systems and lack of skilled staff to investigate perinatal mortality at community, district, regional and national levels. It should be noted that identifying the mother at risk gives no guarantee of success. A condition known to put the fetus at high risk may sometimes have no effect whatsoever while on the other hand some pregnancies with no perceived risk may be complicated by fetal growth restriction and even intra uterine death. In view of this even the low risk mothers should have some level of surveillance during their antenatal clinic visits.

The factors that could identify the high-risk group can be categorised into

- * Maternal factors
- * Fetal factors
- * Placental factors
- * Environmental factors

Maternal Factors

Demographic factors.

Extremes of maternal age; the under-16s and the over-40s; increase the risk of fetal growth restriction. As does nulliparity and grand multiparity. If the woman has delivered a previous growth restricted baby this increases the chance of a subsequent one. Not unexpectedly smaller women deliver smaller babies that are constitutionally small, symmetrical in growth and otherwise healthy and these babies should be regarded as normal. Maternal height and weight are often used to predict this. Maternal and Paternal race are also known to affect the incidence, with birth weights being lowest in the Asian population. With these multiple variables practical difficulties become apparent when using fetal size alone in assessment and management of patients.

Medical Conditions

Commonly sited medical conditions that may affect fetal growth include those that affect utero-placental perfusion or those associated with maternal and therefore fetal hypoxia.

Chronic hypertension

This increases the incidence of fetal growth restriction 2-3 fold. The level of blood pressure (BP), occurrence of proteinuria, level of nutrition and cigarette smoking influence it. Adequate control of uncomplicated chronic hypertension appears to reduce the risk of fetal growth restriction.

Early onset of pre-eclampsia also leads to significant reduction in mean birthweight and a threefold increase in perinatal mortality.

Collagen vascular diseases such as systemic lupus erythematosus (SLE) are associated with an 8-fold risk of fetal growth restriction compared with the general population. SLE is a relatively common disease that has a predilection for the childbearing age group. It is five times more common in non-Caucasians and most common in blacks and should be sought in our region of the world. It is well known that this disease runs a fluctuating course and pregnancy prognosis is worse when the disease is active.

Renal disease of moderate or severe impairment of function before pregnancy has an increased risk of fetal growth restriction. A high rate of hypertension and pre-eclampsia is observed in these patients, which further worsens the incidence of fetal growth restriction. This has been reported in one series as approximately 23%.⁽⁹⁾

Maternal Hypoxemia: The fetus is well adapted to mild hypoxic conditions but manifests growth restriction under more severe hypoxic conditions. These include chronic severe anaemia, which may be nutritional in origin, or associated with haemoglobinopathies such as sickle cell anaemia the latter being very common in developing countries. Chronic pulmonary disease such as severe bronchial asthma may also be associated with fetal growth restriction if control is poor. Finally cyanotic cardiac diseases, though rare should be investigated.

Nutrition

The fetus is well protected against mild under nutrition in the mother but severe nutritional deficiency, as can occur due to natural disasters, social changes and wars, can result in fetal growth restriction. The experience in Holland during the second world war suggests extreme under nutrition in the third trimester has the most effect on fetal growth, hence dietary supplementation of less severe starvation may not

be effective in preventing fetal growth restriction.⁽¹⁰⁾ Underweight women and poor weight gain in pregnancy increases the risk of delivering a fetus less than 2500 grams.⁽¹¹⁾

Maternal Infections

Malaria infestation is endemic in most tropical regions in Africa and Asia. This in pregnancy leads to placental parasitaemia leading to decreased fetal nutrition and low birth-weight apart from the increased maternal morbidity and fetal wastage. There is enough evidence for regular malaria prophylaxis in pregnancy in these areas.⁽¹¹⁾ Other protozoal infections associated with fetal growth restriction includes toxoplasmosis and trypanosomiasis.

A heavy helminthic load may also be significant if the woman is already malnourished.

The most commonly cited viruses reported to be associated with fetal growth restriction are cytomegalovirus, rubella, and varicella zoster.⁽¹²⁾ The incidence of HIV infection is increasing in the developing world and most vertical transmission is thought to occur intrapartum. Recent literature has not confirmed earlier suggestion that it is associated with fetal growth restriction. Any fetal growth restriction found is thought to relate more to the stage of HIV infection and any associated drug or substance abuse.

Some studies have also reported fetal growth restriction with congenital syphilis.

Drugs

The use of alcohol and illicit drugs such as cocaine and opiates as well as other abused substances are on the increase worldwide and are thought to contribute to the incidence of fetal growth retardation.

With the recent actions against tobacco and cigarette smoking in the industrialised world, tobacco advertisement is shifting to the developing world. Cigarette smoking significantly reduces fetal size by the combining effects of carbon monoxide exposure and nicotine inducing the release of catecholamines. The vasoconstrictive effect of this reduces placental perfusion.

Maternal genes have a greater influence on fetal size than paternal genes. Karyotype abnormalities such as trisomy 21 and 18 result in smaller usually symmetrically grown babies. These should be kept in mind as the developing world has no screening

for these but it becomes important if intervention surgery is being planned.

Also in the presence of congenital anomalies the fetus is often growth restricted. Multiple gestations are very common in sub-Saharan Africa and especially monochorionic twins are often growth restricted.

Placental Factors

It is clear that abnormal placental implantation is often associated with fetal growth restriction. The assessment of this using uterine artery Doppler remains largely experimental.

Raised maternal serum alpha foeto protein in the presence of a structurally normal fetus increases the risk of fetal growth restriction. Even though the majority of the developing world does not have access to ultrasonographic diagnosis there is a growing use of this in the region. Some of the placental factors that are associated with fetal growth restriction include single umbilical artery, velamentous cord insertion, bilobed placenta and placental haemangioma. Placental praevia and placental abruption. (managed conservatively) also often manifest fetal growth restriction.

Environmental Factors

Often quoted in the literature is the relationship between increasing altitude and lower birth weight with larger babies born at sea level. This further emphasises the need for locally researched growth charts representative of the local population.

Detecting the fetus at risk

Pregnancy is a natural event with physiological maternal adaptations to bring about a healthy offspring. The majority of pregnancies are thus destined to have an uneventful outcome. One of the challenges obstetricians face is to detect the fetus at risk and mount appropriate surveillance to achieve the delivery of a healthy baby. The approach to this should be two fold.

i) To detect the high risk pregnancy from maternal risk factors, past obstetric history as well as those risk factors developing during the pregnancy.

ii) To implement appropriate antenatal care for the high risk as well as the low risk pregnancy in order to detect fetal compromise and prevent fetal death.

Booking visit:

In the developing world most births are attended by a Traditional Birth Attendant (TBA) who invariably is illiterate and without formal training. The typical woman in the rural setting has also had no contact with the health system prior to pregnancy, while the first antenatal visit may be delayed until the second half of pregnancy and may be the only visit.

Even where facilities would be regarded as adequate and most patients attend regularly, current antenatal care is poorly carried out and only approximately ¼ of small for gestational age babies, defined as birth-weight less than the 10th centile, are detected.⁽¹³⁾ There is the need to improve on policies geared towards improving the antenatal detection of these babies while the research into tools to improve detection goes on.

Home Based Mothers Records (HBMR)

The multiple factors mentioned above as contributing to the Low birth weight baby are simplified in most units into a protocol for risk assessment that is used at the booking visit to assign pregnant women into risk categories. At the district and regional levels in the developing world this screening already forms part of the antenatal booking card with adequately trained staff supervising its implementation. At the community level the majority of pregnant women do not have access to trained health workers. Reducing maternal mortality is the major target but ensuring that these babies are born alive and healthy means the mothers would complete their desired family size with fewer journeys on 'the road to death'. This would also mean less pressure on rather limited obstetric facilities.

Dissevelt et al in Kenya, and Lennox in Papua New Guinea, have shown that using the HBMR already tested and in use in several developing countries improved the rate of detection of at risk pregnant women by rural midwives and their referral pattern became standardised.^(14,15) This could be adapted to highlight risks not only for the mother but also for the fetus. Pregnant mothers with high risk can then be referred to the District for the trained health worker or be seen in the community by the outreach midwife.

Pregnancy dating

Decisions on intervention require accurate knowledge of the gestational age of the pregnancy. This requires an accurate knowledge of the first day

of the last menstrual period (LMP). This may not be known or inaccurate. Time should be spent to ascertain the LMP, relating it to local festivals and events on the calendar and quickening if relevant in order to arrive at the most likely gestation.

With early booking bimanual examination may confirm the dates. Estimating the date of confinement (EDC) may be more difficult and inaccurate for the mothers who book late. EDC from LMP agrees within one week of the best obstetric estimates using all modalities including ultrasound, in only 60% of patients.¹⁶ It is the authors view that as far as possible all pregnancies should have ultrasound dating. Early ultrasound scans before 22 weeks in communities where it is available and affordable would confirm the dates if the LMP is uncertain or the period's irregular.

This also has the advantage of detecting multiple pregnancies, fetal anomalies and low-lying placentas. For follow up antenatal care knowing the accurate gestation would improve the positive predictive value of the symphysis fundal height screening for the small for dates fetus. It also appears to decrease the incidence of post term pregnancies thus reducing the rate of caesarean section and perinatal mortality.⁽¹⁷⁾ Accurate dates are also crucial when a woman is in premature labour with regard to tocolysis and steroid therapy or allowing spontaneous labour.

Continuing antenatal care:

The purpose of routine antepartum fetal surveillance is to detect fetal hypoxic compromise so that timely intervention occurs prior to the development of fetal acidaemia and subsequent intra uterine death. Current antenatal care is poorly carried out and only approximately ¼ of SGA babies are detected.⁽¹³⁾ There is the need to improve on policies geared towards improving the antenatal detection of these babies while the research into tools to improve detection goes on.

There is an excellent discussion on using maternal height and weight including weight gain in pregnancy as a screening tool for the SGA fetus by Lumey LH.^(18,19) The sensitivity and positive predictive value is very low (4% and 14% respectively). It is critical for the health worker at the primary care level who is minimally equipped, to take full advantage of any opportunity to determine the woman's health and nutritional status, any existing anomaly and the risk of adverse outcome for the mother and fetus.

This made the WHO to look at anthropometric measurements again and the reader is referred to an excellent analysis of a multi-centre data.⁽²⁰⁾ This work identified attained weights at 5 or 7 lunar months as the most useful service indicators of LBW with odds ratio of 2.3 to 2.5. This study also found out that low pre-pregnancy weight followed by consistently poor relative weight gain results in a five fold risk of delivering an IUGR baby. A chart to guide the detection of these is shown from the study but locally derived charts may be even more useful.

Continuing antenatal care should therefore aim to identify growth restriction both in the low and high-risk mother by:

- * Checking maternal well-being and weight gain/attained weight in pregnancy
- * Excluding infections such as helminthic infections, malaria, and urinary tract infection
- * Excluding severe anaemia especially in patients with haemoglobinopathies, malaria infestation, urinary tract infection, and its complications, and poor nutrition. In developing countries the evidence is strong for routine iron and folic acid prophylaxis.
- * Checking for fetal well being indicated by fetal movements over a period of time.
- * Clinical examination for symphysis fundal height, presence of adequate liquor and clinical fetal weight estimation.
- * Mothers picked up in the community with these criteria can be seen in the district health centre for ultrasound assessment and further surveillance. The relevance of some of the assessment mentioned is discussed.

Symphysio Fundal Height

This simple and quite objective measurement has been shown to be an excellent guide to fetal growth⁽²¹⁾

Single and sometimes serial measurements may be required to detect the growth-restricted fetus. Several workers have devised different charts but one of the commonest in use is the chart from Calvert et al data.⁽²¹⁾ The criteria often used to

define discrepancy in measurement is 3 cm below or above the mean. About 13 % of the population of mothers would be referred to the district for assessment using this. With local modifications and adjustment of percentile charts to alter the percentage of referrals, low-income countries could apply this at the community level in order not to overwhelm their ultrasound facilities.

Erroneous dating of the pregnancy is a very common cause of discrepancy that could give a false reassurance of satisfactory growth or misclassify a normal growth as growth retarded.

Even in the developing world the use of ultrasound is becoming common and health policy makers should endeavour to make this service available to all pregnant women in all communities.

Serial Measurement of Fetal Growth

This is an important tool to measure specific fetal sections and compare it to the centile distribution for that gestation. Further with comparisons of the centiles attained for the head circumference or biparietal diameter to that of the abdominal circumference disproportionate growth as a result of brain sparing can be diagnosed. Fetal anomaly as a contributor to growth restriction can become evident. With the increasing availability of ultrasound in the developing world, there is the need to mount surveillance on women at high risk of fetal growth restriction such as previous fetal growth restriction, chronic hypertension, early onset pre-eclampsia, multiple pregnancy and other high-risk pregnancies as previously mentioned. This is carried out in addition to the routine SFH measurements during antenatal care. This is performed four weekly from the gestation of viability till term but more often if clinically indicated. With the rate of fetal growth and the inherent error in measurement more frequent than two weekly ultrasound scans are not useful. Needless to say the extent to which this is used depends on availability and affordability where patients pay for medical services.

Low risk patients identified with a SFH below the 10th Centile should also be referred for fetal growth assessment but using routine serial ultrasound as a screening tool for fetal growth restriction in low risk women is not cost effective.

Amniotic Fluid Volume (AFV)

In the absence of fetal anomaly and ruptured fetal membranes, amniotic fluid volume (AFV) may give an indication of the extent of the fetal

cardiovascular response to chronic hypoxic stress. The exact mechanism of this change is not clear although it is known that fetal urine is a major contributor to the amniotic fluid after 16-20 weeks of pregnancy, about 500mls being passed at term. Fetal urine output falls in the presence of chronic fetal hypoxia due to the redistribution of blood from the non-vital body parts and organs such as the limbs and the kidneys to the vital organs such as the brain and heart.

Although the physiology is difficult to explain, it would seem that reduced Δ FV is an important sign of impending fetal demise. Data from Chamberlain and his group illustrates this. ⁽²²⁾ They found increased perinatal loss of up to 110/1000 in the presence of oligohydramnios compared to about 2/1000 perinatal mortality when AFV was normal. The sensitivity of this measurement to predict perinatal death was low at 44% but specificity was 97%. Positive predictive value was also low (6%) but for reassurance the negative predictive value was 99%. The application of this in the developing world in places where this facility is available but expertise not always on site needs further study but this is not specific for detection of fetal growth retardation (FGR).

Fetal Movement Counting

The movements of the fetus start early in life and become recognisable by ultrasound as early as the 7th postmenstrual week but not apparent to the woman until 16 to 20 weeks gestation. ⁽²³⁾ The relationship between fetal movements and fetal health was known since the 16th century. ⁽²⁴⁾ It is however important to note that the number of movements perceived by the mother is subject to a wide variation between mothers and even within an individual from day to day.

To monitor fetal movements, several methods may be used but quite popular is the Cardiff 'count to ten'. The woman is trained to record methodically on a chart the time interval required to feel ten fetal movements ⁽²⁵⁾ The minimum number of fetal movements considered acceptable ranges from three in one hour to ten in 24 hours ^(25,26) The perception of less than ten movements in 10 hours or a significant fall in the total number perceived is an indication for NST and any other fetal assessment available and thought necessary. Although multiple observational studies utilising fetal movement counts suggest a decrease in perinatal mortality ^(26,27) the only randomised controlled trial of fetal movement counting failed to identify any decrease in perinatal mortality in the test group. ⁽²⁵⁾ The absence of a beneficial effect of

the policy of formal fetal movement counting could be explained in part by poor compliance with both recording and reporting alarmingly reduced fetal movements. It is also important to note that Grant et al used 'alarm criteria', that are less stringent than is applied in most units from the authors experience, such as reporting if the women felt fewer than 10 movements in ten hours on two consecutive days. Thus formal fetal movement counting in low risk pregnancy has not reduced perinatal death. However in high-risk pregnancy in the developing countries with lack of equipment for more sophisticated tests, this remains a useful tool for fetal surveillance if appropriately applied. Mothers with reduced fetal movements should be seen for cardiotocographic (CTG) monitoring or a fetal biophysical profile as further assessment of the fetus

Fetal Cardiotocography

1. The Non Stress Test

The cardiotocograph is by no means commonplace equipment in the hospitals and health centres of the developing world. This is because of the cost of the equipment and maintenance difficulties. In the developed world this is the most commonly used equipment to assess fetal well being. The equipment is used to record continuously the fetal heart rate pattern over a period of time 20 to 30 minutes, with the woman in the semi-recumbent position.

The rationale behind this test is that it gives an indication, via cerebrocardiac responses, of fetal cerebral activity, which will become modified in the presence of fetal hypoxia. The conclusion that can be drawn from a normal test is that at best the baby is satisfactorily oxygenated and at worst, the level of hypoxia is not severe enough to produce brain dysfunction.

While normal reactive test in high risk pregnancies may be reassuring of fetal well being, the effectiveness of antepartum non stress test to date has not been subjected to a randomised controlled trial in low risk pregnancy.

With this in mind, CTG's should not be interpreted in isolation. A reactive CTG implies that the neural mechanisms controlling FHR are functioning.

The criteria used to define a normal CTG trace are:

- A normal baseline heart rate of 110-150 beats/minute
- A baseline variability of 5 - 25 beats/minute
- At least two accelerations of an amplitude of 10 - 15 beats/minute over a

15 - 20 minute interval. There should be no decelerations. Shallow spiked occasional decelerations are not an ominous sign. The intra and inter observer variation has led to workers devising scoring systems and computer based assessments of the CTG trace.

When CTG trace is abnormal other fetal assessments are carried out if time permits. If intervention were not immediate, frequent repeat of the CTG trace would reveal when the fetus is becoming compromised. In an unselected population the risk of antepartum fetal death is 1/1000 within one week of a negative cardiotocograph. A normal tracing is therefore not reassuring in all cases.

2. The contraction stress test (CST)

This is not widely practised outside the United States of America but mentioned for the sake of completeness. This procedure is carried out in the same way as the NST. If spontaneous uterine action is not recorded in 20 minutes, syntocinon infusion is commenced to produce contractions at the rate of 3 in 10 minutes. A positive test is where FHR decelerations occur with uterine activity. This is an invasive and time-consuming test requiring expertise that is better used to provide other services in the developing world. Further the incidence of a false positive result is 5-0 % and correlation of a positive test with intrapartum events is poor.⁽²⁸⁾

Theoretically the CST should provide useful additional information both in distinguishing the false positive NST and in the assessment of placental reserve. Some evidence for this comes from a multicentre study in which antepartum death was significantly more common in the NST group (7.8/1000) compared with the CST group (1.1/1000) but as with all multicentre studies the results should be interpreted with caution.

Fetal Biophysical Profile Scoring (fbps)

The purpose of any form of antenatal fetal surveillance must be to recognise accurately fetal and environmental conditions, which adversely influence perinatal mortality and morbidity at an early enough stage to use whatever corrective measures may be available for fetal salvage.

The basis for considering the parameters used in FBPS is that, during and for sometime after acute or active fetal hypoxemia, biophysical variables regulated by the central nervous system outflow are lost. These include fetal breathing movement,

gross body movements, fetal flexor tone, heart rate accelerations with fetal movements (NST) and liquor volume. Response to chronic hypoxemia (oligohydramnios, extrinsic growth failure) is a result of hypoxia induced aortic body chemoreceptor stimulation with subsequent redistribution of fetal cardiac output, often to a profound degree. (29) The effects of this protective reflex change is readily identified in asphyxiated newborns as peripheral cyanosis, apnoea, oliguria,

respiratory distress syndrome and necrotising enterocolitis.

The duration of the observations for this test is quoted to be 30 minutes. Fortunately in clinical practice the average testing time is only 8 minutes, and in more than 40,000 tests abnormal results were encountered in less than 2% of patients. (30, 31) Biophysical profile scoring system. Adapted from Manning et al (1981) (32)

Parameter	Score 2	Score 0
Amniotic fluid volume	>2cm pocket in two perpendicular planes	<2cm pocket in 2 perpendicular planes
Fetal movements	Three or more gross body movements in 30 minutes	Two or less gross body movements in 30 minutes
Fetal breathing movements (FBM)	At least 30 secs. Of sustained FBM in 30 min.	Less than 30 secs. of FBM in 30 min.
Fetal tone	One episode of limb movement from flexion to extension to flexion in a rapid motion	No evidence of flexion movements or fetal movement
Fetal reactivity ²	or more FHR accelerations with fetal movements in 40 min	Fewer than 2 accelerations in 40 min.

The advantage of the FBPS system is the very high proportion of normal tests 97.52 and the very low false negative rate (0.634 per 1000). The risk of fetal death occurring within one week of a normal test was 0.7 per 1000. (31)

This scoring appears to reduce perinatal mortality to a greater extent compared to the other methods discussed but requires equipment not readily available in the developing world. Where facilities and manpower are adequate for example in tertiary units this is worth considering.

Controversy

The controversy with the HBMR is that it is already picking up a high percentage of mothers as being at high risk. In Sri Lanka this figure was 83%, in Papua New Guinea 85 % and in Zimbabwe 64 %. (33)

However including the salient parameters to avoid overwhelming the facilities on the ground while maximising the best use of the available service should be the best approach.

Fetal Movement Counting

This is a simple test in that it is inexpensive. However, given the low prevalence of fetal compromise and an estimated specificity of 90-95% the positive predictive values for fetal compromise are low ranging from 2% to 7%.^(25,27) This would create undue anxiety in several women with infrequent fetal movement but otherwise non-compromised babies. It also has resource implications with increased rate of CTG and hospital admissions. This policy would have to be used on 1250 low risk women to prevent one unexplained antepartum death.⁽²⁵⁾

Symphysio-Fundal Height (SFH)

A large number of mothers to be in the developing world do not remember their LMP, making this measurement less useful than it would have been. This measurement is also affected by such factors as maternal position, whether the measurement is made in the midline or to the superior fetal pole, a full bladder and large uterine fibroids.

There are no standardised criteria to define a size for date's discrepancy and opinion differs as to the use of separate norms for developing countries rather than those used in developed countries.^(34,21)

An estimated 13% of antenatal patients are referred to ultrasound when the SFH measurement is used to screen. Altering the criteria for a positive SFH screen in order to cope with the large numbers referred could alter the sensitivity, specificity, positive and negative predictive value of the test; hence the need for locally derived charts which address local needs.

Routine Antenatal Ultrasound As A Screening Tool For SGA

The incidence of IUGR in the general low risk population is only 5%, reaching a level of 10% in patients who can be considered at risk.⁽³⁵⁾ Because the low risk population is substantially larger group than the high-risk population, approximately one half of all fetuses with IUGR will come from the low risk population. Since ultrasonography measures the fetal size there may be the reasoning that the routine use of this in the low risk population may increase the detection of IUGR. This requires a high level of training to scan, and the cost of equipment and the numbers to screen do not make this a cost effective proposition.⁽³⁶⁾

It was demonstrated that, in spite of using a highly effective method for identifying the SGA fetus with a

sensitivity of 94% and specificity of 90.5%, no clinical benefit accrued at least among low risk mothers.

Non-Stress Test (NST)

The proponents of biophysical profile regard the performance of only NST in the assessment of fetal well being, as inadequate. They argue there is no series of orderly steps by which fetal deterioration proceeds. Hence a change in CTG may be a late change, which could have been detected earlier using ultrasound routinely to assess fetal movements, tone breathing and the liquor volume. Finally an abnormal CTG without the use of ultrasound, especially where routine anomaly ultrasound scan is not practiced, could lead to operative delivery of a fetus with lethal anomalies.

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Ultrasonography In Pregnancy

C.T. John and J.I. Ikimalo

Introduction

An unprecedented impact on obstetric practice was made in the 1960s with the introduction of diagnostic ultrasound scan. Ultrasonography now constitutes an important non-invasive 'third eye' for the obstetrician to evaluate and manage pregnancy from conception to delivery.

The ultrasound machine itself has undergone rapid changes from the A-mode of limited practice to the B-mode scanner of the 1960s with practical two-dimensional imaging and the grey scaling of the early 1970s, which showed movements and raised the potential of fetal biometry. The advent of real-time imaging of the mid 1970s markedly improved imaging and when combined with Doppler waveform analysis of the 1980s and the 3 dimensional (3D) colour imaging of the 1990s, the ultrasound scan has really taken the Obstetrician into the amniotic environment to assess the growing fetus. Poverty and the effects of the World Bank Structural Adjustment Programme (SAP) in the developing countries, particularly the West-African sub-region have made it impossible for us to follow these changes in as rapid a form as in the developed countries.

Physics of ultrasound scan

Sound wave is created with alternate compression and rarefaction of particle in a medium. Wavelength is determined by measurement of one cycle of compression and rarefaction. The frequency of this sound is the number of cycles of compressions and rarefaction, which pass a given point in 1 second. One cycle per second is called one hertz (HZ).

At a frequency greater than 20,000 HZ (20KHZ) sound waves - ultrasound - is inaudible to the human ear and behaves like light waves and travels in a straight line. Those two characteristics made ultrasonography possible. The sound waves are transmitted in a straight line and reflected back in the same way. Diagnostic application range between 2 to 10 Mega (MHZ) frequencies, and most commonly applied are 3.5MHZ and 5MHZ.

Transducers produce high frequency sound by

short burst of electrical stimulation of piezoelectrical crystals located in the transducer. Electrical stimulation causes these crystals to vibrate and produce sound waves. The same piezoelectrical crystals receive the reflected sound waves and convert them to electrical energy, which can be amplified and recorded. Most diagnostic transducers consist of many single piezoelectrical elements spending 1000 times in the receiving mode rather than transmitting waves. Sequential stimulations of these elements produce the real time image by converting the sound wave to ultrasonic image.

The density of various tissues in the body varies and therefore the transmission speed of the ultrasound beam also varies.

When an Ultrasound beam travels through medium of variable density and strikes a very dense object, such as bone, most of the incident wave energy is reflected back and the transmitted waves are attenuated (an echo). Only incident wave reflected back towards the transducer produce images. The speed with which an echo returns and the intensity of returning echoes are proportional to the depth and reflectivity of objects struck by the incident wave. This is basis of grey-scale ultrasound imaging.

Ultrasound scanning in West Africa Sub-Region

The American National Institute of Health sponsored a Consensus Development Conference in 1984 to evaluate the use of Ultrasonography (USS) in pregnancy and concluded that routine screening was not justified. This position is supported both by the American College of Obstetricians and Gynaecologists and a study by Ewiginman.²

The Royal College of Obstetricians and Gynaecologists and the European Committee for Ultrasound Radiation Safety endorse routine prenatal USS. Several other European countries including Germany and Sweden do routine USS.

In the West African region, there are very few

Obstetric units with ultrasound scanners and a few studies have been done to evaluate scanning. On the average, there are few ultrasound-scanning machines in each of the 36 states in Nigeria and most of these machines are sited in private institutions in the main centres. For example colour Doppler imaging may only be available in major centres such as Accra, Lagos and Abuja, where they are available they often turn to be old models acquired from the developed countries.

Ultrasonography was initially done through the abdominal route but the advent of transvaginal probe had improved the images considerably. In using a vaginal probe care must be taken to prevent sexually transmitted disease from one patient to another. The use of the condom to cover the probe is essential.

The modern ultrasound scanning machines like the 3-dimensional and colour imaging are a rarity in the sub-region because of their prohibitive cost, the lack of government investment in health, general poverty and lack of adequate manpower and skill for the use of these equipment.

Inadequate manpower and skill have limited the use of Ultrasound Scan (USS) to level 1. Level II ultrasound scanning aimed at specific organ imaging is available only in specialized tertiary centres. Many doctors and midwives are still unaware of the full range of indications and advantages of ultrasonography. Some even condemn its use and scare patients from having an ultrasound scan. This is because they have neither received any training nor seen ultrasound scan in use. The controversy as to whether or not routine USS should be done for all patients may not be applicable to us now since this technology is not as wide spread as in other regions of the world. Where it is in place, it may not be readily available at all times of the day.⁽⁹⁾ Ultrasound examination is time-consuming and expensive; this may not be affordable to the patients¹⁰ and also to the state.¹¹

Indications For Obstetric Ultrasound

- * Diagnosis of Pregnancy
- * Gestational age estimation
- * Evaluation of fetal growth
- * Vaginal bleeding of unknown aetiology during pregnancy
- * Suspect multiple gestation
- * Discrepancy in uterine size compared with gestational age.

- * Adjunct to amniocentesis
- * Pelvic mass
- * Suspected hydatidiform mole
- * Adjunct to cervical cerclage placement
- * Suspected fetal death
- * Suspected ectopic pregnancy
- * Adjunct to special procedure
- * In vitro fertilization (IVF)
- * Embryo Transfer (ET)
- * Chorionic villus sampling
- * Suspected uterine abnormality
- * Localization of intra uterine contraceptive device
- * Ovarian follicular development and surveillance in patients with infertility
- * Determination of sex of the fetus
- * Biophysical profile
- * Suspected polyhydramnios or oligohydramnios
- * Suspected abruptio placentae
- * Adjunct to external version
- * Estimate of fetal weight, fetal presentation on premature rupture of membranes or preterm labour
- * Abnormal serum fetoprotein values
- * Observation of intrapartum events
- * Management of second twin
- * Manual removal of placenta
- * Follow up observation of identified anomaly
- * Follow up evaluation of placenta location for identified placenta praevia
- * History of previous congenital anomaly
- * Serial evaluation of fetal growth in multiple gestation
- * Evaluation of fetal condition in the late registration for ante-natal care
- * Evaluation of presenting fetal parts

Ultrasound Scan In The First Trimester

Ultrasound scan can be used to visualize an early pregnancy. The gestational sac can be seen as early as 5 weeks using the transvaginal ultrasound and 6 weeks using the transabdominal ultrasound. In early pregnancy, gestational sac and the crown-rump length are used to evaluate the pregnancy. Other early developmental landmarks are shown in the table below.

Table 1**Developmental landmarks by abdominal Ultrasound**

Visualization of	Gestational age from LMP
Gestational Sac	5-6 weeks
Embryonic pole	6-7 weeks
Fetal heart activity	7-8 weeks
Fetal movement	8-9 weeks
Bi-parietal diameter (BPD)	12-13 weeks)

The gestational sac comprises an inner ring made up of decidua capsularis and the chorion laevae and an outer ring made up of decidua vera. The presence of this double echogenic ring in the thickness of the uterus confirms an intrauterine pregnancy. The diameter of the sac is taken once if it is round but if it is ovoid, three dimensions are measured and the average taken.

The gestational sac is not the best to estimate gestational age if the crown rump length (CRL) can be estimated. However, the number of gestation sacs should be noted. The crown rump length is one of the most accurate means of estimating the gestational age of a pregnancy. It is the measurement of the longest length of the fetus excluding the upper and lower limbs.

Earlier than 9 weeks the maximum longitudinal measurement of the fetus may not be easy and after 13 weeks the fetus may flex or extend the spine.

Early Pregnancy Complications**1. Blighted Ovum And Missed Abortion**

Nyberg et al⁽⁹⁾ described ultrasonographic criteria for an abnormal intrauterine gestational sac. A gestational sac >20mm in diameter without a yolk sac or >25mm diameter without an embryo is described as a blighted ovum or an anaembryonic gestation.

Missed abortion on the other hand has an embryo but no detectable fetal heart activity. Some studies have shown that chromosomal abnormalities are common in early pregnancy failures like blighted ovum.⁽⁵⁾ Other reasons are poor implantation and poor vascularisation of the developing sac.⁽⁶⁾

The introduction of transvaginal ultrasound has now shown that a good proportion of the blighted ova are due to an early resolution process of a non-viable embryo.

2. Trophoblastic Diseases

Diseases due to abnormal trophoblastic development include hydatidiform mole, invasive mole, choriocarcinoma and placental site trophoblastic tumour. Pelvic angiography was used in the diagnosis of trophoblastic diseases but the advent of ultrasound scan has provided a simpler non-invasive means of diagnosis.

The typical snowstorm appearance of vesicles in the uterine cavity may not be seen all the time there is a mole. Areas of mixed echogenicity can be seen in incomplete mole, choriocarcinoma or uterine polyp.

3. Abortion

Ultrasound scan is invaluable in the management of patients with abortion. Bleeding in early pregnancy is often alarming to the mother. In threatened abortion the demonstration of a living embryo on sonographic examination shows a greater chance (85-90% chance) of that pregnancy continuing. Ultrasound should be repeated later to localize the placenta.

In incomplete abortion, when some of the products have been passed out, ultrasound will show enlarged uterus with ill-defined gestation sac or internal echoes that are not clearly defined.

In complete abortion a line of central echoes will run through the middle of the uterus representing the decidual reaction.

4. Ectopic Pregnancy

Ultrasound scan is presently playing an increasing role in the diagnosis and treatment of ectopic pregnancy with the use of the vaginal probe. Some of the diagnostic signs that may be observed include absence of an intra uterine sac bordered by two layers of decidua, extra uterine mass, fetal heartbeat and movements outside the uterus. Other signs that are suggestive of ectopic pregnancy are enlarged uterus with thick echogenic endometrium, fluid in the Pouch of Douglas or paracolic gutters in the case of ruptured ectopic pregnancy. A combination of these signs and the use of vaginal ultrasound will increase the chances of a correct diagnosis. Occasionally, separation of the decidua from the myometrium

prior to expulsion of decidual cast and in an incomplete abortion, a double decidual layer looking like an intrauterine pregnancy⁽⁷⁾, the resolution of the scanner and the experience of the person doing the scan also affect detection of double decidual ring. A Pseudogestational sac is another source of false diagnosis. Heterotrophic pregnancy can occur in this sub-region. The ability to make a correct diagnosis especially before a rupture is one of the major benefits of ultrasonography in cases of ectopic pregnancy. Unfortunately this service is not available to many and so ruptured ectopic pregnancy with shock is what we commonly see in the sub-region.

Second And Third Trimester Ultrasound Scan

Most of the pregnant women in the tropics have their first antenatal visit in the second and third trimester. The opportunity for a first trimester ultrasound scan is often missed except there is a problem like threatened abortion or severe abdominal pain. The biparietal diameter, abdominal circumference and the femur length are the most frequently measured parameters as the basis of most obstetric ultrasound. These measurements are used singly or in combination for estimating gestational age, fetal growth and fetal weight.

1. Biparietal diameter (BPD)

This is the measurement of the outer table of the proximal fetal skull and the inner table of the opposite side of the skull. The measurement is taken at the level of the falx cerebrej, the thalamus, the carvum septum pellucidum and the medial cerebral artery. The fetal head is oval in shape at this level and it is best exposed when the fetus is lying in an occipito transverse position. The BPD is useful in the estimation of gestational age.

2. Abdominal Circumference (AC)

Abdominal circumference is measured at the level of the junction of the umbilical vein and the left portal vein, there is an echolucent area looking like a hockey stick. Two diameters D1 and D2 should be measured at right angle to one another. The formula for measuring abdominal circumference AC is $AC = 1.57 (D1 + D2)$. The abdominal circumference can be used to estimate gestational age in the mid-pregnancy or late pregnancy.

Femur Length (FL)

The measurement of the femur length is more commonly used in the second and third trimester

for the estimation of gestational age. Ultrasound determination of gestational age is more correct the earlier in pregnancy it is done than later in pregnancy. Table II shows ultrasonographic methods of dating pregnancy.

3. Table II

Outline of ultrasonographic dating of pregnancy		
WOG	RDM	Accuracy
3-5	None	None
5-6	Gestational Sac (GS)	+ 3.5 days ± 1 wk
6-12	Crown-Rump	± 1 wk
	BPD	± 1 wk
12-20	BPD	± 2wks
FL		± 2 wks
AC		± 3 wks
30-40	BPD	± 3 wks
FL		± 3 wks
AC		±3.5 wks

Weeks of gestation: WOG

Recommended dating measurement: RDM

The Placenta

The image and location of the placenta is better defined as pregnancy progresses. Low lying placenta seen in the early second trimester should be looked at again at 34-36 weeks to see its relationship with the lower uterine segment and the internal os. As pregnancy advances and the uterus enlarges, the placenta matures and 'migrates' with the enlarging uterus and may come off the lower uterine segment.

This maturity is graded on ultrasound scanning from grade 0 to grade III. Grade 0 placenta is most common in the first trimester. The chorionic plate is very smooth and the placental body is homogenous without calcifications.

Grade I placenta appears after 14 weeks until 34 weeks. There is indentation in the chorionic plate, echogenic areas of calcification parallel to the long axis of the placenta.

Grade II placenta first appears from 26 weeks but is more common around 36 weeks. There is more indentation of the chorionic plate but the indentation does not reach the basal plate. Straight-line echoes characterize the placental body with calcifications along the axis of the basal plate.

Grade III placenta appears beyond 35 weeks gestation. The chorionic plate indentations reach the basal plate. There is complete compartmentalisation of the placenta. Extensive echogenic areas represent calcifications. When a grade III placenta is seen before 35 weeks, it is abnormal. Hypertension, intrauterine growth retardation (IUGR) and oligohydramnios are associated with early placental maturation. Diabetes mellitus and Rhesus sensitisation are associated with delayed maturation.

Amniotic Fluid

The most likely cause of polyhydramnios is idiopathic, followed by diabetes mellitus, congenital anomaly, erythroblastosis fetalis and multiple gestation. Ultrasound examination to rule out any fetal anomaly must be done in polyhydramnios. Oligohydramnios is associated with IUGR in 55% of cases and occasionally fetal urinary tract anomalies. Premature rupture of membranes should be ruled out.

Fetal Size

There are many formulae combining Bi-parietal Diameter (BPD), Abdominal Circumference (AC) Head Circumference and Femur Length (FL) to produce fetal weight. The predictive accuracy is only from 14.8% to 20.2% (+25D); its use is limited in this situation.

Intra Uterine Growth Retardation (IUGR)

Clinically, when the symphysio-fundal height is less than the correct gestational age, in the second half of pregnancy intrauterine growth retardation is suspected. Ultrasound evaluation of IUGR is more

correct than clinical examination and combination of both examinations significantly improves the ability to screen for IUGR. Ultrasound parameters for evaluation of IUGR include Oligohydramnios, Biparietal diameter (BPD), Head circumference (HC), Femur length (FL) and Abdominal circumference (AC) fall below the 15th percentile FL/AC > 23.5% HC/AC > 95%.

Post Term Pregnancy

Pregnancy beyond 42 weeks (294 days) from the first day of the last menstrual period is defined as a post term pregnancy.

An early ultrasound dating of pregnancy reduces the incidence of post term pregnancies. Ultrasound assessment of a pregnancy that has gone beyond 42 weeks will include physiological hydramnios, macrosomia, and anatomic survey for congenital normalities.

Complications Of Ultrasound Scanning

There has been no proven harm to the mother or fetus although theoretical risk exists.

Use Of Ultrasound In Invasive Obstetric Practice

Recent advances in tissue culture and genetic engineering coupled with advances in ultrasound technology has made it possible for samples of fetal tissue and fluid to be obtained for diagnostic and therapeutic purposes such as Chorionic Villous Sampling (CVS). Obtaining chorionic villous sample transvaginally is done under ultrasound guidance at a gestational age of 9-12 weeks.

Amniocentesis

Under direct ultrasound vision amniocentesis can be done usually at 16-18 weeks although it can also be done earlier to obtain amniotic fluid and fetoproteins for prenatal diagnosis.

Controversies In The Use Of Ultrasonography In Pregnancy

The ultrasound is a very useful tool in obstetric practice. This is particularly so since there are no proven side effects and the benefits are many. However this tool must be used when necessary and by one with the required expertise. Too many of our women now feel their antenatal care is not complete unless they have one or more abdominopelvic scan during the pregnancy. Doctors who have recently acquired the machine and wish to

encourage their incomes further enhance this. In a number of cases the machines are old, the expertise poor and the results baffling. Taking clinical decisions on such results can be dangerous. On the other hand patients may fail to avail themselves of this useful tool due to ignorance on the part of those advising them.

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Normal Physiology Of Pregnancy

EE Emuveyan

Introduction

Pregnancy is the state of having an implanted embryo in the uterus until such a time that it is terminated by spontaneous or elective abortion or delivery. The fertilized ovum releases the hormone HCG that prevents the corpus luteum from involution and stimulates the secretion of oestrogen and progesterone and initiation of myriads of physiological changes that involve every organ and system in the body.

The anatomical, endocrinological and physiological changes are a positive adaptation of the mothers to accommodate and support the fetus as it grows and develops throughout gestation. At the same time, the mother is prepared for delivery of the fetus and for lactation so that the infant is nourished and supported when it is born.

The physiological changes in pregnancy are a temporary adaptation and produce no permanent deleterious effects on the mother. Virtually all the functional changes in the mother revert to the pre-pregnancy state when the puerperium is complete hence pregnancy is a normal physiological process.

Factors Affecting Maternal Adaptation To Pregnancy

1) Gravidity

This is the most important determinant of the mothers' responses. Inadequate physiological changes are most likely in the first pregnancies but the maternal reaction may also be affected by her age, obstetric history, overt or covert illness (acute or chronic) and the present or earlier environment. One or more previous pregnancies are likely to ensure that adaptation to the current pregnancy will be normal or complete. First pregnancies are less physiological than subsequent ones. This is suggested by the reduced mean birth weight (Thomson al 1968), increase rate of complications and raised perinatal mortality in primigravidae.

2) Maternal Age

The maternal age influences adaptation of pregnancy. In every society, pregnancy induced hypertension is commoner in young mothers. The age range of 18-25yrs is regarded as the probable period for optimal physiological adaptation to pregnancy. In older women, the physiological changes of ageing or concurrent disease may prevent the development of a totally healthy pregnancy. After the age of 35yrs, blood vessels become less flexible and with increasing age both systolic and diastolic blood pressures rise.

3) Social Class

The lower the social class, the worse is the pregnancy outcome.

4) Multiple Pregnancy

The number of fetuses in the uterus determines the extent of many adaptive changes. Mothers carrying twins have more potential stress on almost all of their systems than mothers of singletons.

Physiological Changes Associated With Pregnancy.

General Effects

Pregnancy is associated with marked fluid retention accounting for some 6-8kg of the average maternal weight gain of 11kg. The expansion of all fluid compartments is not equal with a disproportionate increase occurring in plasma volume. The principal determinant of extracellular fluid volume is sodium retention and coupled to this is reduced plasma oncotic pressure found in pregnancy.

Pregnancy is equally associated with hyperlipidaemia and glycosuria. These physiological changes in lipid and carbohydrate metabolism are accom

Together they increase the availability of glucose to the fetus (its preferred source of energy) while conception increases and become most marked in the second half of pregnancy coinciding with increasing fetal requirements for growth.

Systemic Effects Gastro Intestinal Changes

Nausea, vomiting or morning sickness: this typically begins between 4-8 weeks of gestational age and abates by the middle of the second trimester. It is said to be related to the elevated levels of progesterone, human chorionic gonadotrophins and also to relaxation of the smooth muscle of the stomach. Despite this, many patients report dietary cravings during pregnancy. Some patients develop unusual craving for such things as ice, laundry starch or clay.

In general, there is decreased gastrointestinal motility during pregnancy due to increased levels of progesterone. As a result, gastric emptying time is prolonged and there is decreased oesophageal tone and incompetence of the oesophageal cardiac sphincter. This results in gastric reflux and heartburn, a common complaint in pregnancy.

Gall bladder function also decreases in pregnancy with the subsequent cholestasis resulting in an increased tendency to form gallstones.

Elevated liver enzymes and serum protein levels are seen with resultant appearance of spider angioma and palmar erythema, signs that are commonly found in association with liver disease. Serum cholesterol levels are also increased during pregnancy.

Key Gastrointestinal Changes In Pregnancy

Appetite	Usually increased, some times with unusual cravings.
Gastric reflux	caused by oesophageal cardiac splinter relaxation and anatomic displacement.
Gastric motility	Decreases
Intestinal transit time	Slower
Liver	functional unchanged
Gall bladder	Dilated
Bile composition	Unchanged

Pulmonary Changes

Gas transfer is enhanced by the marked increases in pulmonary blood flow associated with pregnancy as well as by the increase in ventilation, predominantly the result of augmented tidal volume. The tidal volume increases by 30-40% and inspiratory capacity increases by about 5%

Vital capacity and inspiratory reserve remain the same as in non-pregnant state. Functional residual capacity, expiratory volume and residual volume are all decreased by about 20%. Total lung capacity is also decreased by about 5% with a resulting increase in minute ventilation of 30-40%. Arterial blood gases reflect this change in pulmonary functions in the following ways; oxygen is increased, carbon dioxide is decreased, and serum bicarbonate is reduced. There is a risk of respiratory alkalosis with the patient aware of dyspnoea, hyperventilation, and a relative decrease in exercise tolerance.

Cardiovascular Changes

The dramatic changes in the maternal cardiovascular system during pregnancy improve oxygenation and flow of nutrition to the fetus.

Cardiac output is increased up to 50% as a result of an increase in both heart rate and stroke volume.

Due to the muscle relaxing effects of increased levels of progesterone during pregnancy, peripheral vascular resistance is decreased. There is a decrease in arterial blood pressure during the first 24 weeks of pregnancy with a gradual return to non-pregnant level by term.

Heamatologic Changes

Plasma volume begins to increase as early as the 6th week of pregnancy and reaches maximum at about 30-34 weeks after which it is stable. The mean increase in plasma volume is about 50% with a greater increase in patients with multiple gestations. Similarly larger babies are associated with a greater increase of maternal plasma volume, whereas a pregnancy complicated by intrauterine growth retardation is often associated with a less than normal increase in blood volume.

There is an increase in red cell mass later in pregnancy but to a lesser degree than does plasma volume. This results in physiologic anaemia.

The white blood cell count also rises as pregnancy advances. This increase is due primarily to increase in the number of granulocytes.

Platelets count during pregnancy may decline slightly but remain within normal range of those of non-pregnant patients.

Pregnancy is considered a hypercoagulate state with an increase risk of thrombo embolism both during pregnancy and the puerperium. This is brought about by increase in fibrinogen level, increase in fibrin splits products and factors VII, VIII, IX and X. Prothrombin (factor II), and factors V and XII remain unchanged during pregnancy. Bleeding and clotting times do not change during normal pregnancy.

Renal Changes

The kidneys are enlarged by about 1cm during pregnancy. Both the renal pelves as well as the ureters are dilated during pregnancy due to the relaxation effects of progesterone and compression effect of the gravid uterus. There is increase in urinary residual volume. Renal plasma flow begins to increase early in pregnancy and increases to as much as 75% over non-pregnant level at term. Glomerular filtration rate equally increases. Creatinine clearance is markedly increased in pregnancy. Plasma osmolality is decreased primarily due to reduction in serum sodium concentration. The plasma Rennin is equally higher than in non-pregnant state. Glucose excretion increases in virtually all pregnant patients.

Key Renal Changes In Pregnancy

Renal plasma flow	increased
Glomerular filtration rate	increased
Urinary output	unchanged
Renin	increased
Angiotensin	increased
24hr protein excretion	unchanged

Skin Changes

The most obvious change in the skin is pigmentation in some areas. The development of mea nigra and darkening of the nipple and areola are almost universal although the depth of pigmentation varies in different people and different races. All are due to increase secretion of melanocyte stimulating hormone. Spider naevi and palmer erythema are also found in most mothers. Both are estrogen effects.

Striae, which develop on the abdomen, breasts and elsewhere, are a response to increased circulating corticosteroids. There is generalized vasodilatation to assist in losing the extra heat produced by maternal, placenta and fetal metabolism.

Fingernails grow more quickly during pregnancy. Hair does not but the rate at which hair is shed is reduced.

Intensely itchy papules sometimes appear during normal pregnancy (pregnancy prurigo) and disappear spontaneously either before or after delivery

Breast Changes

There is increase in blood flow to the breasts resulting in tenderness and tingling sensation. Estrogen stimulates increased ductal growth while progesterone stimulation results in alveolar hypertrophy, thus preparing the breast for lactation.

Changes In The Reproductive Tract

Vaginal discharge is due to vaginal transudation as well as stimulation of vaginal mucosa. There is increase in the weight of the uterus from .70 to 1000g at term.

Endocrinology Changes

The maternal endocrine system is modified during pregnancy by the addition of the feto-placental unit. This produces hCG, hPL and other unique hormones, which affect the mother's, endocrine organs directly or indirectly.

The key endocrine changes in pregnancy are:

1. Thyroid

Thyroxine (T4)	Total increased	Free changed
Triiodothyronine (T3)	Total increased	Free changed
Thyroid binding globulin (TBG)	Increased	

2. Adrenal

Corticosteroid binding Globulin	Increased
Cortisol	Increased
Aldosterone	Increased
Androstenedione	Increased

3. Pituitary

Prolactin	Increased
FSH	Decreased
LH	Decreased
ACTH	Increased
TSH	Unchanged
Oxytocin	Unchanged

4. Ovaries & Placenta

Progesterone	Increased
Estradiol	Increased
Estriol	Increased
HPL	Increased
hCG	Increased (peak btwn 8 & 10 wks)

Diagnosis Of Pregnancy

Symptoms

Amenorrhoea: In a patient who has regular menstrual cycles and is sexually active, a complaint of a period delayed by more than a few days to a week is indicative of pregnancy. Though the vast majority of women experience amenorrhoea during pregnancy until after birth, a few women do experience vaginal bleeding in pregnancy and therefore fail to recognise their condition. One of the early symptoms of pregnancy is nausea and vomiting or morning sickness. This is said to be related to elevated level of progesterone, hCG and also relaxation of smooth muscle of the stomach. Breast enlargement and feeling of painful/tingling sensation are associated with pregnancy. There is enlargement and hyperpigmentation of the areola. The enlarging uterus pressing on the urinary bladder causes urinary frequency.

While none of these symptoms is specific to pregnancy they may in fact alert the woman to the fact that she is pregnant, and can be of value in the assessment of early pregnancy problems such as ectopic pregnancy and miscarriage.

Signs

Breast enlargement, tension and distensions are particularly obvious in the primigravida. There is presence of linea nigra. In early pregnancy bimanual examination shows soft cystic globular uterus with enlargement consistent with the duration of the pregnancy. Congestion and bluish discolouration of the vaginal mucosa (Chadwick's sign) and softening of the cervix (Hager's sign). Palpation of fetal parts and the appreciation of fetal movement and fetal heart tones are diagnostic of pregnancy but at a more advanced gestational age.

Laboratory investigations

The diagnosis of pregnancy should not be made based solely on symptoms and physical findings, which may be misleading in early pregnancy. A pregnancy test is used to make an accurate diagnosis. Even when the pregnancy test is positive, complications such as spontaneous abortion, ectopic pregnancy and trophoblastic disease may make the diagnosis of a normal intra-uterine pregnancy difficult. Several types of urine pregnancy tests are available all of which measure hCG produced by the syncytiotrophoblast of the growing placenta. Serum pregnancy test are however more specific because they test for the unique beta subunit of hCG.

Ultrasound examination: Early ultrasound scanning can diagnose pregnancy once a gestational sac is present (5-6wks of gestation).

Radiologic examination: Advanced pregnancy may very occasionally be accidentally diagnosed on X-ray or Magnetic Resonance imaging but these tests would not be suitable as a method of choice for diagnosis.

Management of Pregnancy (Antenatal care)

✓The aims of antenatal care are:

1. Screening for and diagnosis and management of pre-existing maternal disorders e.g. diabetes, heart diseases etc.
2. Diagnosis and management of obstetric and other maternal complications during pregnancy, including the so-called minor disorders of pregnancy.
3. Detection and management of fetal complications (where feasible).
4. Planning for labour and delivery, care of the newborn and future general and reproductive health, including contraceptives and subsequent pregnancies.
5. Provision of information, advice and education for both mother and father in pregnancy and labour, subsequent health and infant care. Complete obstetric care includes the correct diagnosis of pregnancy followed by an initial thorough assessment early in pregnancy and periodic examinations and screening tests as appropriate throughout the course of pregnancy.

Most pregnant women will deliver healthy infants without any antenatal care. Therefore, obstetric care is designed to promote good health throughout the course of normal pregnancy while screening for and managing any complications that may develop.

Pre-conception counselling: Ideally, obstetric care should commence prior to pregnancy as a preconception visit where a thorough family and medical history for both parents and physical examination of the prospective mother is done. Pre

existing conditions that may affect conception and or pregnancy are identified and appropriate management plan formulated. Unfortunately, the idea of pre pregnancy evaluation is uncommon; instead most women seek care after pregnancy has begun in this environment.

Booking Clinic

At booking, a comprehensive history is taken focusing on previous pregnancy outcome and any medical or surgical condition that may affect pregnancy; information pertinent to genetic screening and information about the course of the current pregnancy is obtained. A comprehensive general physical examination is performed. If medical or obstetric problems are identified during the initial assessment or at any subsequent visit, the pregnancy is designated as "at risk" and appropriate specific management initiated.

Common laboratory investigations usually ordered for are:

- Urinalysis - for glycosuria and proteinuria
- Packed cell volume
- Blood grouping, Rhesus typing and haemoglobin genotype
- Ultrasound at booking is controversial
- Chest X-ray: now abandoned in many developing countries as tuberculosis is almost eradicated.
- Serological testing for syphilis, TORCH syndrome, Hepatitis B virus, Human Immunodeficiency virus.
- Measurement of μ -feto-protein is done especially in developed countries to rule out neural tube defect and Down's syndrome.

Subsequent Antenatal Evaluation:

For a patient with a normal pregnancy, periodic antenatal visits at 4 week interval are usually scheduled until the 28th week at 2 week interval between 28-36 weeks and weekly thereafter. Patients with high-risk pregnancies or those with ongoing complications are usually seen more frequently. At each visit a comprehensive physical examination is carried out to assess maternal and fetal well-being.

Conclusion

Women prepare for pregnancy in the luteal phase of the menstrual cycle. If conception occurs, these changes persist. Adaptation starts before embedding. Although all systems are affected there is not a uniform pattern of change.

The cardiovascular and renal modifications are established in the first trimester and plateau or decrease in the third trimester. Alteration in other systems may appear later in pregnancy and increase to term, while metabolic modification is continuous.

Age, race and other factors may affect the extent or pattern of adaptation. Multiple pregnancy adds another dimension. Most of these changes benefit the fetus, sometimes to the discomfort of the mother but some (such as the general relaxation of smooth muscle) may be a side effect rather than a purposeful adaptation.

The totality of adaptation explains many of the common signs and symptoms of normal pregnancy. Therefore recognition of physiology defines pathology and determines clinical care of pregnant women from conception until at least 6 weeks after delivery.

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Human Immune Deficiency Virus In Pregnancy

Nelson K.R. Damale

The largest epidemic facing mankind today is the Human Immune Deficiency Virus (HIV) infection. Since the first recognition in male homosexuals in 1981, the more significant mode of transmission in the developing countries of Africa and Asia has been established as heterosexual. ⁽¹⁾ In some parts of the developing world ineffective methods of blood screening make blood transfusion also a significant means of transmission. ⁽²⁾

The importance of HIV infection lies in the fact that there is as yet no vaccine, no curative treatment, and the infection is followed by high and long-term morbidity and certain mortality. It has also remained a social stigma with profound social and psychological implications for the woman, her partner and the whole extended family.

The other effect of HIV/AIDS is its potential for producing a large number of uninfected children left as orphans as their infected mothers and fathers die of the disease. On the other hand there are special implications for the HIV positive pregnant woman as quite a significant number of their babies are at risk of transmission of the virus to them during the pregnancy and childbirth with development of childhood Acquired Immune Deficiency Syndrome (AIDS). A major step towards the control of this pandemic requires efforts to establish and control the risk factors that are specific to women of childbearing age as well as reducing the risk of vertical transmission.

A few inroads have been made in developed countries but the advance of this epidemic in the developing countries continues unchecked leaving in its wake not only the orphans and childhood AIDS sufferers but dwindling of the workforce that creates the meagre wealth in Sub-Saharan Africa. HIV is becoming one of the commonest causes of death in the region. ^(3,33)

This chapter considers the epidemiology of the disease, HIV infection in pregnancy in developing countries, the effects of HIV infection on pregnancy, vertical transmission, and the risk of the HIV seropositive pregnant women to health workers.

Epidemiology

For the developing world to reduce the transmission of HIV and limit the magnitude of the epidemic, individual nations need to assess and appreciate the magnitude of the problem, identify the modes of spread and institute measures within their resources to reduce all known modes of spread.

To date size of this pandemic is still uncertain because despite recent improvements, considerable under-reporting still occurs in the developing world. This in part is due to inadequacies in the healthcare system and the deficiencies in the surveillance systems for monitoring the spread of HIV. It is also a rapidly changing field and figures quoted today may be out of date in a few months as new surveys are published.

Of the over five million incident infections in adults in 1999, women accounted for 47% and most of these women are of childbearing age. ⁽⁴⁾ Worldwide it is estimated about 2.4 million infected women give birth annually, and 1600 infants acquire HIV infection every day. Thus in 1999 alone about 570,000 children became infected with HIV, primarily through mother to child transmission. Even though the majority of these children reside in Africa the epidemic is fast spreading in other developing countries notably in the Far East. ⁽⁵⁾

Two serotypes of HIV exist. HIV-1 found worldwide and HIV-2 which was identified in and is prevalent mainly in West Africa. HIV-2 may be less virulent than HIV-1 with a much longer incubation period to AIDS. In other respects, however, the two serotypes behave similarly hence the use of HIV in this chapter refers to both serotypes.

HIV virus has been isolated from blood, semen, saliva, female genital tract secretions and breastmilk and transmission can theoretically occur through exchange of any of these body fluids. There is also good evidence that some of the common tropical infections are associated with increased risk of HIV transmission. ⁽⁶⁾

Globally, 70-80% of HIV infection is estimated to be acquired sexually, 8-15 % parenterally through transfusion of blood and blood products and injection of illicit drugs. The rest 5-10% is acquired through perinatal transmission. ⁽⁷⁾ The highest rates of infection are seen in sexually active young women with a peak at age 35 years. 

Most of the available data on prevalence in the developing world is on reported AIDS cases but it is clear that with the long incubation period to AIDS there are more HIV positive cases than there are AIDS patients. Since the prevalence of this infection varies depending on behavioral patterns it is difficult to estimate the prevalence in the community hence the need to specify groups in the community and their specific prevalence. 

Studies carried out on pregnant women attending urban antenatal clinics have provided the most accurate source of information on prevalence of HIV infection in low risk populations of fertile, sexually active women. Prevalence has ranged from low levels in the western world to high levels like 40% in parts of East Africa. ⁽⁸⁾

The initial infection with HIV may be asymptomatic or may produce an acute glandular fever like seroconversion illness. Following this the individual remains apparently healthy for the incubation period of 8-10 years or more from the time of seroconversion.

The different epidemiology of HIV/AIDS in the developed world compared with less developed countries has been attributed to a wide variety of factors, including transfusion practices and repeated use of needles that may be contaminated, scarification, high prevalence of concomitant sexually transmitted diseases (STD), immunological activation due to endemic infectious diseases, and high mobility of the workforce dictated by economic necessity. This list is by no means exhaustive.

Heterosexual transmission being the main mode of spread in the developing world, the high STD prevalence in many sub-Saharan African countries and the demographic structure of the population where teenagers and young adults constitute a high proportion of the total population needs particular mention. Ulcerative STD's such as Chancroid, syphilis and genital Herpes make HIV transmission more likely. ^(9,10) Chancroid was the most common cause of genital ulcerative disease in studies in Nairobi Kenya and Rwanda with prostitutes serving

as the main reservoir. ⁽¹⁰⁾

The non-ulcerative STD's namely gonorrhoea, chlamydia infection and trichomoniasis were also found in a Kinshasa prostitute cohort to predispose to HIV seroconversion with odds ratios ranging from 2.7 to 3.5. ⁽⁶⁾ Traditional healing practices such as scarifications and the 59 practice of inserting various substances (herbs, Aluminium Hydroxide powder, and stones) in the vagina for self treatment to tighten the vagina leads to ulceration. These practices were highly associated with HIV infection in a study in Zambia. However the cross-sectional study in Malawi reported only a weak positive association between HIV infection and the use of such topical agents. ⁽¹¹⁾ The contribution of this to viral transmission from infected men to women and vice versa needs further research. It is also noteworthy that in a Nairobi study, uncircumcised men have an eightfold risk of seroconversion compared to those circumcised and is thought to be of higher infectivity to their female partners. ^(12,13) The presence of the preputial cavity with the retention of genital secretions is considered a factor that facilitates the survival of both HIV and the causative agents of genital ulcerative disease as well as recurrent balanitis. ⁽⁶⁾

Pregnancy in the HIV positive woman

Epidemiological studies suggest that on a population basis, pregnancy rates among HIV positive women who have not yet developed AIDS are probably comparable with those among uninfected women with similar demographic characteristics. However women who develop AIDS with opportunistic illness are considerably less likely to become pregnant. ⁽¹⁶⁾

Initial concern that pregnancy may increase the rate of progression of HIV disease has not been completely resolved but the consensus is that pregnancy has no major effect on the course of the disease. The CD4 lymphocyte is the main target cell of HIV and destruction of these cells lead to suppression of the immune system and increased susceptibility to common infections. The CD4 lymphocyte count, which is used as a surrogate marker for progression of the disease, does not fall in normal pregnancy, and pregnancy does not seem to have any long-term effect on the rate of decline in HIV positive pregnancy. ⁽¹⁷⁾ Furthermore the survival time of pregnant patients with AIDS was not affected by the pregnancy. ⁽¹⁸⁾

Similarly several effects on the newborn to HIV

positive mothers were reported in the earlier literature notably prematurity and intrauterine growth retardation. These were not confirmed in subsequent research; hence the current issue on pregnancy in HIV positive women is the risk of transmission to the baby and is addressed below.⁽¹⁹⁾

Vertical Transmission

One of the main implications of HIV in pregnancy is possible transmission to the baby and this is the major contributor to paediatric AIDS. The basic mechanisms, which have been proposed, for vertical transmission are,

- Intrauterine (transplacental)
- Intrapartum (at the time of delivery)
- Postpartum (mainly through breast-feeding).

The literature is now replete with factors known to be associated with increased transmission risk in pregnancy among which, are the following:⁽²⁰⁾

1. Mothers with evidence of advanced infection both measured clinically (AIDS) and immunologically (low CD4 count) are 2 to 3 times more likely to transmit the virus than those with intact immune system.
2. Viral load and presence of virulent mutant strains of the HIV virus.
3. The presence of lesions associated with other STD's and chorioamnionitis.
Very premature infants are at high risk (the rate after about 35 weeks gestation is stable)
4. The duration of ruptured membranes, labour, second stage, and the first born (vaginally) in a multiple pregnancy.
5. Elective Caesarean section was found to reduce risk of transmission by 50 % (The European Collaborative Trial)⁽²¹⁾
6. The use of episiotomies, forceps or vacuum extractors has not been directly associated with increased risk.
7. Breast-feeding (probably contributes to the higher vertical transmission rates in Africa compared to Europe by 10-20%.)

UNAIDS estimates that 1.2 million children were living with HIV-1 infection at the end of 1999 and

three times that number had died from the disease⁽⁴⁾.

According to the United Nations Population Division, AIDS will cause 64% of all deaths of under-5-year olds in Botswana between 2000 and 2005. This illustrates one of the worst scenarios but the rate of vertical transmission varies worldwide.

For example estimates in 1993 before the onset of anti-retroviral treatment were as shown below (Table 1)

Table 1.
Estimated vertical transmission rate in 1993.

Continent	Vertical Transmission Rate (%)
Europe	15-29
USA	15-30
Africa	25-35

The belief that the risk of vertical transmission depended on the viral load led in 1994 to the PACTG 076 Trial.⁽²²⁾ This led to a significant reduction of 68% in vertical transmission rate at 18 months. Reduction in vertical transmission will be discussed under antiretroviral therapy, non anti-retroviral measures, and breast-feeding

Antiretroviral therapy

The developed world now offers HIV positive pregnant women anti-retroviral therapy to reduce mother to child transmission. Most of the developing world with limited resources can least afford this treatment in pregnancy, intrapartum and post delivery. The PACTG 076 trial mentioned earlier was one of the first studies.

This was a double blind, placebo controlled, randomized clinical trial in non-breastfeeding mothers, with enrollment criteria as shown below in Table 2.

Table 2, PACTG 076 Trial: Enrollment Criteria

Pregnant 14-34 weeks gestation
HIV Positive
CD4 lymphocyte count >200/mm ³
No previous Zidovudine use
No medical indication for Zidovudine use

Patients were randomised to receive antepartum 100mg Zidovudine orally five times daily starting at 14-34 weeks gestation (median time 26 weeks). This was continued intravenously during labour at 2.0mg /kg over 1 hour and then 1mg /kg /hr thereafter until delivery. The neonate was given oral Zidovudine 2mg/kg, 6 hourly starting 8-12 hr. after birth for 6 weeks.

Vertical transmission rate at 18months was 25.5% with placebo compared to 8.3% with Zidovudine, a reduction of 68%. This is the most effective regimen for reducing transmission but it is expensive and complex, requiring intravenous administration and the long regimen could result in compliance problems.

Less complex, cheaper and shorter Zidovudine regimen in the Abidjan, Ivory Coast trial achieved 37% reduction in transmission at three months.⁽²³⁾ The developing countries will best be served by a cheap, simple, easy to administer regimen. In this light, the HIVNET 012 trial⁽²⁴⁾ in Kampala Uganda using Nevirapine in breastfeeding mothers deserves mention. This trial compared a single 200mg oral dose at the onset of labour and a single 2mg/kg oral dose to the newborn at age 48-72hr with oral Zidovudine given intrapartum and for one week to the infant. Transmission at 14-16 weeks was 13.1% with Nevirapine compared to 25.1 % with Zidovudine; a reduction of 47% over Zidovudine.⁽²⁴⁾ The plea to the developed world and the pharmaceutical companies to reduce the cost of antiretrovirals and treatment for opportunistic infections associated with AIDS has resulted in the recent reduction in the cost of some selected treatments but this is still not within the means of most of these countries.

More research into such short course effective therapy that can meet the budget of developing countries is needed.

The potential adverse effects of these drugs such as teratogenicity, carcinogenesis, and mitochondrial dysfunction in the short term are rare and clearly outweighed by the proven benefit of significant reduction in a lethal infection.⁽²⁵⁾ Subsequent drug resistance in monotherapy and long term effects of these drugs however need further research.

Further research on HIV immunoglobulins and HIV vaccines are also eagerly awaited. HIV vaccines would not only prevent further transmission in adults of child bearing age and therefore vertical transmission, but also reduce postnatal transmission by passive and active immunization of neonates at risk.

Non antiretroviral intervention

Antiretroviral prophylaxis is not yet affordable in the developing world. There is therefore the need to look at simple inexpensive and effective alternatives. Chlorhexidine has been shown to neutralize HIV in vitro but intrapartum washing of the cervicovaginal mucosa and newborn washing with 0.25% Chlorhexidine did not reduce perinatal transmission. In this study, it however significantly reduced overall maternal and neonatal mortality and morbidity.⁽²⁶⁾ Further research in higher concentrations of Chlorhexidine and Benzalkonium Chloride vaginal suppositories is in progress.

Breastfeeding And Vertical Transmission

The risk of transmission of infection from mother to baby over and above intrauterine and intrapartum infection attributed to breastfeeding is between 10-20%. To reduce this WHO recommends that HIV positive mothers refrain from breastfeeding if safe alternatives are affordable and available (WHO1992).

In the developing world artificial milk is expensive and bottle-feeding could lead to infective diarrhoea, gastroenteritis and often-infant mortality. The promotion of breastfeeding would therefore benefit the majority of women who are HIV negative. Breastfeeding promotion has been so successful and so widely practiced that mothers who do not breastfeed may be suspected to be HIV positive. With all the extended family joining in celebration of a new addition to the family, this perceived suspicion might tempt HIV positive mothers to breastfeed in public and bottle-feed at home. This mixed feeding could introduce contaminated fluids

that may cause gastroenteritis and inflammation that could compromise gastrointestinal mucosal integrity, facilitating HIV-1 transmission.

In a study in Nairobi Kenya, formula feeding with cup and spoon reduced postnatal transmission by 44% compared with breastfeeding. However mortality was quite high in both groups (24% and 20% in breastfeeding and formula fed infants respectively) despite facilities with good running water.⁽²⁷⁾ Thus socio-cultural realities have to be considered when counselling against or for breastfeeding.

Stopping breastfeeding also has other consequences in the developing world that need research, such as effects on fertility and birth spacing and the effects of this on maternal health.

Detecting the HIV Positive Pregnant Patient

It is paradoxical that the degree of screening worldwide seems to be inversely proportional to the prevalence of HIV infection.

Since anti retroviral therapy and appropriate intrapartum and postpartum management has been shown to reduce vertical transmission more needs to be done to identify HIV positive pregnant women before measures can be offered to reduce paediatric HIV infection.

The following screening methods could be used.

- Universal testing of pregnant women
- Voluntary named testing
- Selective (targeted) testing

With the high prevalence of HIV in developing countries, universal testing of all pregnant women would have been ideal but for the cost.

Counselling and voluntary named testing, which would reduce cost, is also not yet affordable in most health services and would miss some cases due to participation bias. Some women do not return to hospital for their results hence there is the need to test and confirm positive results at the same visit. The cases the Obstetrician in these countries is likely to manage would be those diagnosed before pregnancy and those investigated in pregnancy on clinical grounds.

To increase this number, prenatal and antenatal education of women in the health units and using the mass media could create the awareness, for

self-funded voluntary testing by mothers in the interest of their babies. There are facilities in urban centers for testing but these services are unlikely to reach 60-80% of the population in most developing countries who dwell in rural areas, where health facilities are not available or are inadequate.

Selected (targeted) testing involves case finding by noting risk factors from the history and offering selected patients counselling and testing. At the present stage of this epidemic in the developing world, women affected heterosexually may be unaware that they are at risk and may also conceal their risk activities from health staff. It has been shown in two studies that four out of ten seropositive pregnant women (40%) denied risk factors at the screening interview.⁽²⁸⁾

The concern with screening is the emotional trauma and the social stigma the patient and her family have to go through. This could be quite strong in the developing countries where the cooperation of these women towards reduction of transmission may not be optimum. In many developing country antenatal settings, there is lack of privacy for counselling, testing and discussion of the report and this will make testing less acceptable to patients.

Some studies have reported encouraging acceptance of screening that leaves some hope for the future. Of more than 5700 pregnant women in Abidjan, and 4000 in Bobo-Dioulasso both in Cote d'Ivoire, 78% and 92% respectively accepted an HIV test and 58% and 82% respectively of those tested returned for the results.⁽²⁹⁾ Other studies have not reported such high acceptance but further research in this area is awaited.

Management Of The Hiv Positive Pregnant Woman.

In Africa the reduced availability of diagnostic techniques for HIV, opportunistic infections, STDs, viral load estimation, and surrogate markers for the progression of HIV/AIDS severely limits the capabilities of the health system to manage HIV positive pregnant women. The cost of antiretroviral therapy, and the fact that a large percentage of the population lack access to basic health care facilities also contribute to this situation.

HIV positive women who are pregnant should be jointly followed by an HIV specialist, an obstetrician, a midwife with expertise and interest in these patients, and a paediatrician where such

health personnel are available. The immediate family should be encouraged to provide good social support.

Early booking should be encouraged and implications of pregnancy with HIV discussed. With the high risk of transmission to the baby and lack of resources to provide antiretroviral therapy, problems of breastfeeding, the risk of opportunistic disease, as well as the option of termination of pregnancy should at least be discussed with the couple. Confidentiality is of utmost importance and such couples should be managed by as small a health team as is necessary for optimum care. From a personal experience this has worked reasonably well in the western world thereby encouraging others who want to give the best chance to the unborn baby to volunteer for antenatal testing.

For couples that can afford it, the management options include prophylaxis against opportunistic infections, antenatal antiretroviral therapy, and peripartum Nevirapine therapy. These being the cheapest for now. Combination therapy to avoid resistance is being more commonly used in the developed world.

In addition to the routine antenatal care, screening for Hepatitis B, Syphilis and common STDs in the community, and cervical cytology is recommended. The rest of antenatal care remains the same for all.

Intrapartum Care



Since most transmission occurs intrapartum a substantial benefit might be gained by the single dose Nevirapine therapy for the mother and baby and couples could be advised to save money towards this. The European Collaborative study has demonstrated a trend towards lowered vertical transmission rates when delivery is by caesarean section but operative procedures in the developing world risk more complications. A retrospective study has also indicated these HIV positive women may be at a higher risk of postoperative complications. The practice of elective caesarean section to reduce vertical transmission cannot be implemented widely in the developing world without local research to confirm the reported benefits. It is noteworthy that double gloving reduces the risk of puncture by a factor of six and the use of blunt needles and tissue holding forceps further reduces the risk of needle stick injury.

During labour gloves, aprons, and face protection

should be employed. The fetal membranes should be left unruptured, thereby minimising fetal contact with maternal secretions. Clinicians should as much as possible avoid invasive procedures such as amniocentesis, fetal scalp electrodes, amniotomy, and peripartum cord blood sampling during labour. All procedures including assisted delivery that could cause a break in skin continuity for the mother and baby theoretically increase the risk of transmission to the baby. All fluid spillage and the placenta should be disposed of with great care to avoid contamination and reduce risk of infecting others.

Early cord clamping and early bathing of the baby may also reduce the risk of transmission. The baby should be seen by the paediatric team and therapy commenced if available and followed up for serological tests. Breastfeeding, its advantages and drawbacks have been discussed in this chapter. The author holds the view that in the known HIV client there should be a place for individualised advice on alternative methods of infant feeding with full counselling depending on all perceived risks including the dangers of mixed feeding. This is the way forward if transmission through breastfeeding is to be reduced without losing the gains of breastfeeding in non-infected mothers.

Postpartum Care

After delivery there is no evidence suggesting these women should be isolated, but careful disposal of contaminated sanitary towels, lochia, dressings and drains is important. Soiling of toilet seats and ward floors require decontamination with freshly prepared sodium hypochlorite solution (10,000 ppm available chlorine) for at least ten minutes before cleaning or mopping. Needless to say all healthcare units should have their own protocol for sterilisation of all equipment, linen, boots etc used for known HIV positive or high-risk cases.

In the developed world, combination viral chemotherapy is continued in all HIV positive women to reduce and maintain the viral load at undetectable levels. For the fetus this would reduce the risk of peripartum viral transmission and for the mother this would protect her immunological system. This is desirable as maternal survival improves and ensures higher infant survival.⁽³³⁾ The cost of this therapy and the large number of HIV positive women in the developing world put such management beyond the budget of most developing countries. Thus for individual countries

various guidelines and treatment frameworks have to be adopted and more research in this area may help ensure better use of available resources.

Postnatal care and counselling of the HIV positive woman and her partner should include advice on safer sexual practice and appropriate contraceptive plans. Use of the sheath may reduce the risk of viral transmission to the spouse but the failure rate of this method of contraception is high (3.1-4.8/100 woman years).

In the absence of antiretroviral treatment, survival long enough to look after more children to adulthood is reduced.

The Health Worker And HIV

Maternity care by its nature is probably the discipline in which the health worker is most exposed to body fluids including blood, urine, liquor and genital secretions. The risk of exposure to the virus is even greater in the developing world with the high prevalence rate of HIV and the crowded labour wards. The risk of transmission from patient to health worker is low provided sensible precautions are instituted. A survey of staff at the Mama Yemo Teaching Hospital in Kinshasa, Democratic Republic of Congo, over a two-year period showed that increase in HIV infection was not related to occupation. Administrative staff and general workers showed the same rate of increase as doctors, surgeons, nurses and midwives.⁽³¹⁾ This study should encourage all health workers to discharge their professional duties the best they can for all patients whether or not they are HIV positive. In the situations of known HIV positive or high risk patients, all health workers should continue to maintain a very high level of practice to prevent the spread of infection to other patients and themselves.

In all deliveries, plastic aprons and gloves should be worn, as should be facemasks, spectacles and plastic boots. From the author's personal observation this has not always been adhered to on the busy labour ward in the developing world. Wearing of gloves is also advised in less risky situations if the health worker already has a skin abrasion, open wounds or severe eczema. All spilt body fluids should be decontaminated and cleaned.

For all deliveries the use of a long sleeved gown is the minimum safe protection and elbow-length gloves should be available for manual removal of placenta and exploration of the uterus.

'Sharps' receptacles should be strategically placed in all delivery rooms and other items, which have

had contact with body fluids, should be carefully disposed of.

All operative procedures including suture of perineum carry the additional risk of needle injury and double gloves should be worn.

If a needle-stick injury occurs, gloves should be removed and the hand and puncture site washed in running tap water (warm enough to encourage bleeding). This area should be covered with waterproof adhesive. Testing the source patient and post exposure drug prophylaxis should be considered but this is not usually available in the developing world because of cost. A course of AZT 1000mg per day for three weeks costs 265 pounds sterling approximately.

Discussion And Controversies.

The prevention of further spread of HIV currently depends upon education of the developing world not only to achieve a high level of awareness that HIV is a problem but also awareness of the various modes of transmission, the risk factors mentioned and the special problem of mother to baby transmission. The population should understand safer sex as an important means of prevention of transmission. Health workers and policy makers should let the public know the overall impact of HIV on the population and the workforce and the importance of adhering to safe sexual behaviour in order to limit spread of the disease.

There is evidence that some simple non-expensive procedures can reduce the spread of the disease and these should be implemented. Cheaper reliable tests for HIV that the developing world can afford would help offer the test to those who would want to know their HIV status. There is recent evidence suggesting the knowledge of seroconversion can result in sexual behavioural change^(14,15)

The future of HIV in the developing world is difficult to predict. The available information to change the course of the disease should be applied widely and further research into the modes of transmission and into vaccines should receive priority and ultimately an affordable cure would be most welcome in the developing world.

Herbalists all over Africa lay claim to cures for AIDS and their patients have seen at least some improvement in their symptoms. There is the need to investigate to ascertain possible existence of antiretroviral agent in these herbal preparations that might be more affordable than western medicine.

The literature is silent on the health of HIV positive women during and after delivery, and that of the infants when treatment is not available. This scenario is the reality in most developing countries. The literature however cites the plight of the rapidly growing population of AIDS orphans and the problems they encounter.

Obviously their survival depends on the extent of immunosuppression and for the infant whether or not there was vertical transmission. During the period of 1990-1994 a study in Kampala and Harare from the second trimester of pregnancy to two years after delivery found that mortality and morbidity requiring hospitalisation during pregnancy were not significantly different. However, HIV positive women in Kampala were 31 times more at risk of dying between forty-two days and two years after delivery than were HIV negative women. In Harare the relative risk was 18.⁽³⁰⁾ In a similar study in the Gambia the relative risk of mortality in infants from 18 months to two years were 2.5.⁽³²⁾ The survival times are much shorter than for their counterparts in the developed world who have a full range of treatments available. More research into this area is required. If the developing world could afford present day antiviral agents and the treatment for opportunistic infections this could reduce this high morbidity and mortality in the women and their children.

Conclusion

The HIV pandemic continues to be a growing problem especially in developing countries. Continuing education of the population about the various modes of transmission, safer sex, control of STD's, safe practices in western and traditional medicine has to be stepped up. Vertical transmission is a significant contributor to new cases but effective interventions have remained unimplemented. The issues of Screening for HIV in pregnancy, the implementation of anti-viral prophylaxis to reduce vertical transmission and affordable and safe alternatives to breastfeeding are crucial to fighting the spread of this lethal virus in the developing world.

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Nutrition In Pregnancy

Cecil A. Klufio

Introduction

DEFINITION: "Nutrition is the science that deals with nutrients and other food substances, and with how the body assimilates them".⁽¹⁾

Nutrients are used to build tissues (i.e. to build, maintain, and repair protoplasm), and to produce energy. The body uses energy to perform vital cellular functions and to generate heat to maintain body temperature. Adequate nutrition in pregnancy is reflected in satisfactory weight gain during the pregnancy. The recommended total weight gain in pregnancy is 10.2kg.⁽²⁾ or 0.3kg/wk. Weight gain cannot, however, be used as a measure of nutrition if occult or overt oedema is present.

In determining nutritional requirements in pregnancy (Appendix 1), we must consider the various maternal adaptations in pregnancy, especially changes in absorption, metabolism, and excretion of the various nutrients, the rise in the basal metabolic rate, and the changes in the level of physical activity that occur as pregnancy advances. Prenatal stores, chronic bacterial and parasitic infestations, as well as social, cultural and dietary taboos should also be taken into account. We must therefore do more than just add requirements in the non-pregnant state to the nutritional requirements of the fetus in arriving at recommended allowances. An appropriate diet is also an essential part of the management of diabetes in pregnancy.

Classes of Foodstuffs

The five main classes of nutrients are carbohydrates, fats, proteins, vitamins, and minerals.

Carbohydrates And Energy (calories)

Energy is measured in calories and kilocalories (kcal.), or in joules and kilo-joules (kJ.). One kcal is the heat energy required to raise the temperature of 1kg of water one degree Celsius (from 14.5°C to 15.5°C). One kcal is equal to 4.2kJ. The pregnant woman requires about 2,500kcal per day.

Three of the five main nutrients, (proteins,

carbohydrates, and fats), yield energy. Though carbohydrates yield less energy than fats, they are the most cost-effective, and the chief, source of energy. This is because they are the most abundant and the cheapest of the three major foodstuffs and their metabolism to produce energy is the most efficient. One gram of pure carbohydrate or pure protein yields 4cals, whilst one gram of pure fat yields 9cals.

Carbohydrates occur as complex starches and sugars. Starch is found mainly in cereals, such as rice, wheat, maize, sorghum, and millet, and in tubers and starch roots, such as yam, cassava, cocoyam (taro), and potato, and in unripe fruits, such as plantain (cooking banana). Sugar is found in ripe fruits, such as mango, banana, orange and tangerine, pawpaw, and guava, and in sugar cane. Unrefined cereals contain some protein, and because they are consumed in large amounts, they make a significant contribution to the total protein intake. Unrefined cereals also contain large amounts of fibre, i.e. indigestible cellulose (roughage) that provides the bulk needed by the intestines to make them propel their contents forwards. They thus aid digestion and prevent constipation. After consumption they produce a slow, rather than a rapid, rise in blood sugar. They are therefore useful in the dietary management of diabetes. Kenkey (boiled fermented maize dough) is especially effective in this respect.

Protein And Body Building

Protein is the chief bodybuilding nutrient. Animal foods are the richest sources: meat, poultry, fish, milk, cheese, eggs, and roe. Good plant sources are peas, beans, nuts (e.g. peanut), whole cereals (e.g. maize, sorghum, and millet), and leafy-green vegetables, e.g. cabbage and cassava leaves. Animal foods are also the best sources of protein because they contain all the eight essential amino acids.

Fats And Oils

When animals are well fed, they store any excess food energy as fat for future use should food not be available in adequate quantities. Plants also store fats and oils in their fruits, seeds, and nuts.

Fats and oils (liquid fats) are a concentrated source of energy. One kilogram of fat contains 9,000cals. Although, weight for weight, fats produce more than twice the energy produced by carbohydrates (9cals/gram versus 4cals/gram) fats are scarcer and more expensive.

Fats are the source of the fat-soluble vitamins (vitamins A, D, E and K). Fats are also the source of the essential fatty acids. These are polyunsaturated fatty acids that must be present in the diet for optimal health. They are linolenic, oleic, and arachidonic acids. Animal sources of fat are stored body or meat fat, e.g. suet, lard and mutton fat and fish oil e.g. cod liver oil and milk fat. Plant sources are seeds and nuts, e.g. olive, sunflower, peanut, and soybean oils.

The carbon atoms in fats of animal origin carry all the hydrogen atoms they can carry; they are "saturated" with hydrogen atoms. They are therefore solid. On the other hand, fats of plant origin are "unsaturated". Unsaturated fats decrease the concentration of cholesterol and low-density lipoproteins, and increase the concentration of high-density lipoproteins in the blood. Unsaturated fats therefore protect against atherosclerosis and its sequelae. Saturated fats promote atherosclerosis.

Vitamins

Vitamins are organic substances, which are required by the body in small amounts to maintain life and health. Vitamins act as catalysts in the formation of hormones, enzymes, blood cells, neuro-transmitters, and genetic material. They are essential to complete the metabolism of the major foodstuffs carbohydrates, proteins and fats. Apart from vitamin D, which can be manufactured by skin in the presence of sunlight, and vitamin K, which can be manufactured in small amounts by intestinal organisms, the body cannot manufacture vitamins. They must therefore be ingested in the diet.

Fat-soluble vitamins: These are vitamins A, D, E, and K. They are digested and absorbed with the help of dietary fat. The fat-soluble vitamins can be stored in fatty tissue and in the liver for long periods. Therefore, they do not need to be eaten daily.

Vitamin A (retinol) is needed for strong bones and teeth, healthy skin and healthy mucous membranes, good vision, and reproduction. Its derivative, retinol, is a component of rhodopsin, the visual pigment in the red cells of the retina. Vitamin A is an anti-oxidant, which at low oxygen tensions

protects cells from free oxygen radicals. Early deficiency of vitamin A causes night blindness. Further deficiency causes keratinization of epithelial tissues and a reduction in mucous secretions, leading to: dry skin, dry eyes, thickening of the corneal epithelium and blindness (xerophthalmia), growth retardation, and susceptibility to infections, especially respiratory infections and gastro-enteritis.

A study in Malawi showed greater risk of mother-to-child transmission of HIV in women with vitamin A deficiency. It however, remains to be demonstrated whether vitamin A supplementation actually prevents transmission.⁽³⁾

Sources: Vitamin A is present as the precursor - carotene in green and yellow fruits and vegetables, such as spinach, yellow maize, peas, beans, broccoli, carrots, sweet potatoes, and palm oil. The carotene is converted to vitamin A by the liver, which stores it. Animal sources of the vitamin are milk and milk products, egg yolk, liver, and fish liver oil.

Vitamin D promotes intestinal absorption of calcium; it is important in calcium and phosphorus metabolism and in the deposition of these in bones and teeth. It is formed when ultra-violet light falls on sterols in the skin.

Sources: Vitamin D is present in milk and its products, egg yolk, liver, tuna, and fish oils. Deficiency of vitamin D causes rickets in children and osteomalacia in adults.

Vitamin E (-tocopherol) is important for reproduction. It is also an anti-oxidant. At high oxygen tensions, it acts synergistically with glutathione to prevent oxidation of unsaturated fats and to protect cells, including erythrocytes, from free oxygen radicals. It enhances storage of vitamin A by the liver. Inadequate vitamin E intake by the pregnant or lactating mother can cause anaemia in the newborn.

Sources: Vitamin E is found in leafy vegetables, green peas, vegetable oils, germ of cereals, and liver.

Vitamin K is essential for production by the liver of the blood clotting factors prothrombin and Factors VII, IX, and X.

Sources: It is synthesised by intestinal bacteria and it is present in green leafy vegetables, the germ of cereals, tomatoes, carrots, potatoes, and in egg

yolk. It is absorbed in the presence of bile. Obstructive jaundice can therefore cause a deficiency and a clotting defect. Long-term treatment with broad-spectrum antibiotics can kill the bacteria that synthesise it, and produce a deficiency that may lead to a clotting defect.

Water-soluble vitamins: These are the eight B vitamins and vitamin C.

Sources: Many plant and animal foods contain the water-soluble vitamins. These include brewer's yeast, leafy green vegetables, whole (not polished) cereals, berries, nuts, peas and beans; organ meats (liver, heart, kidney), lean meats, and eggs. The B complex of vitamins form parts of co-enzymes in tissue enzyme systems that are concerned with releasing energy from foodstuffs carbohydrates, fats and proteins. The water-soluble vitamins, unlike the fat-soluble vitamins, are not stored in the body and must therefore be ingested daily to prevent deficiencies. They are fragile and are destroyed by overcooking.

B₁ (thiamine) is needed to absorb pyruvic acid and to metabolise carbohydrate to release energy. In addition, it is required in the synthesis of nerve-regulating hormones. Deficiency causes beriberi.

B₂ (riboflavin) is needed in the metabolism of carbohydrates, fats, and respiratory proteins (flavoproteins). It also plays a role in maintaining the health of mucous membranes.

B₃ (niacin, nicotinic acid) deficiency causes pellagra. In addition to sources common to the other B vitamins, niacin is obtained from poultry, tuna, and nuts. The body also converts tryptophan to nicotinic acid.

B₆ (pyridoxine) is needed to absorb and metabolise certain amino acids. It is also used in the metabolism of fats and in the formation of red blood cells. In addition to sources common to the other B vitamins, pyridoxine is obtained from avocados and bananas. B₆ deficiency has been suggested as a cause of hyperemesis gravidarum.

Folic acid (folacin) is necessary for DNA synthesis. It is therefore needed in the formation of tissue proteins and in the division of cells. Its deficiency causes macrocytic anaemia. It has been conclusively shown in randomised controlled double-blind trials that folic acid supplementation before conception and in the early weeks of pregnancy significantly reduces the incidence of

neural tube defects in the babies of subjects.⁽⁴⁾ Major dietary sources of folic acid are leafy vegetables, brewer's yeast and liver. Folic acid is absorbed in the upper jejunum and stored in the liver. If intake ceases, the stores will last only 6 weeks.

Causes of folic acid deficiency in pregnancy include:

1. Inadequate intake
 - i. Food lacking in folic acid
 - ii. Overcooking, i.e. boiling food for more than 15 minutes, destroys it
2. Poor absorption
 - i. Diseases of the jejunum: coeliac disease, tropical sprue
3. Extensive small bowel resection
Increased demand
 - i. Pregnancy, particularly multiple pregnancy
 - ii. Increased rates of haemopoiesis; e.g. in sickle cell disease, malaria, and other haemolytic anaemias.
4. Competitive inhibition of dihydrofolate reductase enzyme system by some drugs, e.g. pyrimethamine
5. Antiepileptic drugs
 - i. Phenytoin
 - ii. Primidone

B₁₂ (cyanocobalamin) is needed for DNA synthesis. Sources: Unlike the other B vitamins, B₁₂ is obtained only from animal sources: liver, kidneys, meat, fish, eggs, and milk. It is absorbed in the ileum and this requires intrinsic factor, which is secreted by the parietal cells of the stomach. Deficiency of intrinsic factor causes pernicious anaemia. This is a condition that affects the elderly and is rare in reproductive-age women.

Vitamin C (ascorbic acid) is important in the synthesis and maintenance of collagen, the backbone of connective tissue. It is therefore needed for healthy bones, teeth, gums, and blood vessels. It also helps in reducing ferric iron in plant foods to the ferrous form so that it can be absorbed. Its deficiency results in scurvy, which causes haemorrhages and affects the gums and teeth, mucous membranes, blood vessels, skin, and the long bones of growing children.

Sources: The major dietary sources of vitamin C are fresh citrus fruits, pineapple, guava, watermelon, broccoli, tomatoes, cabbage and green peppers.

Minerals

Iron: The body's total Iron content of 2g is distributed between: (a) functional components - haemoglobin, myoglobin, some enzymes and co-enzymes, and transferrin, and (b) storage components ferritin and haemosiderin. The body achieves Iron balance by controlling the amount of dietary Iron absorbed. The body has no control over Iron losses. Without intervention, haemoglobin levels in pregnancy are lower than the levels in the non-pregnant.

There are 3 reasons for the lower plasma levels in pregnancy. First, physiological haemodilution occurs because plasma volume increases by 50% whilst red cell mass increases by 30%. Second, iron demands of pregnancy may not be met by dietary intake. Third, folic acid demands of pregnancy may not be met by dietary intake. The demand for Iron is only partly offset by the nine months' amenorrhoea of pregnancy and by increased Iron absorption during pregnancy.

The total iron demand in pregnancy is about 1,000mg

Distribution of Iron in pregnancy:

Increased red cell volume - 500mg

Extra blood in the uterus and products of conception - 500mg

Extra blood in lactating breast and in milk - 180mg

Loss at delivery - 200mg

Menstrual blood loss during 9 months amenorrhoea saved - 220mg

The daily requirement of Iron in pregnancy is 20mg per day. The demand is greatest in the last trimester of pregnancy because fetal demands are highest at this time.

Sources: The following are good sources of iron: liver, meat, eggs, and peas. A well-balanced diet contains about 15mg iron per day and up to 10% (1.5mg) of this is absorbed daily. The average Ghanaian diet is not rich in iron. Many of our women start pregnancy with low iron stores, or are already anaemic (haemoglobin levels 10.0g/dL). Supplementation with medicinal iron is therefore necessary. Tablets of 200mg ferrous sulphate will give 65mg of elemental iron. This is given three times a day to correct anaemia and to replenish iron stores.

Causes of Iron deficiency include:

1. Inadequate intake, e.g. foodstuffs that are poor in iron.
2. Poor absorption, e.g. phytates in cereals bind iron; reduction in acid content of gastric juice.
3. Increased demand by pregnancy, particularly multiple pregnancies. Increased blood loss, e.g. from heavy hookworm infestation.

Calcium: Calcium is needed to build and maintain the rigidity of bones and teeth. It contributes to the formation of intercellular cement and cell membranes. It takes part in blood coagulation. It is also an intracellular mediator (a second messenger) and takes part in the regulation of nervous excitability, muscular contraction, and hormone release. Nearly all (99%) of the body calcium is in bone. The rest is in plasma in a concentration of 2.5mmol/L. About 1.2mmol/L of plasma calcium is bound primarily to albumin, and secondly to globulin. The remaining plasma calcium is free; i.e. it is ionised and diffusible. The metabolism of calcium is under the control of three primary hormones: parathyroid hormone (PTH), 1-25-dihydroxycholecalciferol, which is the active metabolite of vitamin D, and calcitonin. PTH raises plasma calcium by osteoclastic mobilisation of calcium from bone and by increasing renal tubular re-absorption of calcium; it also increases tubular secretion of phosphate, and thereby, urinary excretion of phosphate. Vitamin D is first hydroxylated in the liver to 25-hydroxycholecalciferol, and the latter is hydroxylated in the proximal tubule to 1-25-dihydroxycholecalciferol. The latter raises plasma calcium levels by increasing absorption of calcium by the intestine and renal tubules and by mobilising calcium from bone. Calcitonin is secreted by the parafollicular cells (clear cells or C cells) of the thyroid gland. It lowers plasma calcium levels by inhibiting bone resorption and by increasing urinary excretion of calcium.

Calcium deficiency reduces the amount of calcium deposited in bone; i.e. it reduces the amount of calcium accretion per unit bone matrix. This causes rickets in children and osteomalacia in adults.

In osteoporosis, osteoblastic activity does not match osteoclastic activity. As a result, both bone matrix and bone mineral are lost, with loss in bone mass and strength, and increased risk of fractures.

Intestinal absorption of calcium increases early in pregnancy and has doubled by 24 weeks, whilst urinary losses are reduced below normal levels.

The total calcium that goes into formation of the fetal skeleton is only 2.5% of the calcium content of the maternal skeleton. This can be provided by maternal skeletal stores⁽⁵⁾.

Sources: Major sources are milk and milk products, e.g. cheese, butter, and in chewable bone.

Iodine is a component of the thyroid hormones. Iodine deficiency causes goitre and hypothyroidism in the mother, and cretinism in her baby. Significant reversible changes in thyroid morphology and histology occur in normal pregnancy.

The changes include the following:

- * Oestrogen-induced increase in thyroxine binding globulin (TBG) results in elevation of serum total thyroxine (T4) and triiodothyronine (T3) concentrations
- * High hCG levels in first trimester results in raised free T3 and suppressed TSH
- * Increased plasma volume results in increased T4 and T3 pool size
- * Increase in placental Type III 5-deiodinase results in increased degradation of T4 to reverse T3
- * Increased renal clearance of iodide.

As a result, the thyroid enlarges to trap more iodine. According to ultrasound scan measurements, the magnitude of the increase is about 13%. This is usually not detectable by physical examination⁽⁶⁾. If dietary iodine is not enough, thyroid enlargement is greater and becomes physically detectable.

Iodine deficiency exists in some parts of West Africa. Inadequate intake during pregnancy causes cretinism. The introduction and promotion of iodised salt is the most effective preventive measure.

Sources: Its chief sources are seafood and iodised table salt.

Selenium is a trace element. Selenium has important functions:

- * It is an integral prosthetic group in the anti-oxidant enzyme, glutathione peroxidase,

which is present in erythrocytes and other tissues. The reduced form of this enzyme, catalyses the destruction of hydrogen peroxide (H_2O_2) and lipid hydroxyperoxides, thereby, protecting membrane lipids and red blood cells against oxidative damage by peroxides. In this antioxidant function, glutathione and vitamin E act synergistically.

- * It is required for the production of pancreatic juice, which is necessary for the digestion and absorption of fats and the fat-soluble vitamins, including vitamin E.
- * The Type I 5'-deiodinase contains selenocysteine in which the sulphur in cysteine is replaced by selenium. Type I deiodinase converts T4 to T3, and is responsible for most of the T3 in plasma.

Selenium deficiency causes goitre and reduced thyroid-hormone levels. If it coexists with iodine deficiency, both deficiencies will have to be corrected to achieve normal thyroid function.

Sources: The chief source of selenium is plants, and their selenium content depends on selenium concentrations in soil.

Dietary Advice in Pregnancy

In counselling a pregnant patient on what to eat, a dietary history should first be taken to determine:

- Her knowledge of nutrition and the effects of cooking on different food types
- Her socio-economic status and the types of food she can afford
- The foods available on the market
- Any foods that custom, or, her religion, forbids her to eat
- What she and her family can grow at home
- What and how much she personally eats, and what the family or household eat
- Whether she has pica

In advising a lay patient how she can eat a balanced diet, it is not very useful to talk in terms of proteins, carbohydrates and fats/oils. It is more meaningful to group foodstuffs and advise her to include a serving from each group in her daily meals.

- Cereals and cereal products: maize, rice, wheat, sorghum, and millet. These are high in carbohydrate (calories), and the whole grains contain the B vitamins, iron, some protein and fibre.
- Tubers and starch roots: These are very rich in carbohydrates, contain minerals and some vitamins, but contain very little protein and no fat. They also contain some fibre.
- Beans, peas and nuts: These are rich in carbohydrate and contain more protein and fat than cereals do.
- Fruits and vegetables: These are a good source of most of the vitamins and minerals, and contain some protein and fat. They also contain fibre. Overcooking, however, destroys some vitamins, e.g. vitamin C and folic acid.
- Meat and dairy products: meat, poultry, fish, milk, yoghurt, ice cream, cheese, and eggs. These are rich sources of protein, calcium, iron, phosphorus, zinc, and the B vitamins.

Discussion And Controversies

From conception to birth, the nutrients the fetus needs to grow from the weightless fertilised ovum to become the heavier than 3kg term baby, are supplied totally by the mother. The mother eats intermittently (3 meals in a day) but the fetus feeds continuously. It has been estimated that the normal term baby contains 400g of protein, 220g of fat, 80g of carbohydrate, 25g of calcium, 16g of phosphorus, and 0.4g of iron. Additionally, the placenta contains 55g protein, and the mother accumulates some 500g of protein in her breasts and uterus. Basal metabolic rate increases by 25% or by some 350 kcal/day. The demands of pregnancy on the mother are therefore great. Many births and short birth intervals can only increase the demands and make replenishment/reparation before the next pregnancy impossible.

It is also important to remember that in exclusive breastfeeding, the amounts of nutrients the mother loses daily in the milk are more than her requirements during pregnancy.

In non-primate animals, adequate nutrition during gestation is critical. Because fetal growth rate is rapid in these animals, caloric restriction in

pregnancy has a pronounced and immediate effect on the fetus. In humans, the effect is not so dramatic. That the pregnant or lactating woman needs sufficient calories at regular intervals during the day, as well as a well-balanced diet, is obvious. Even when the mother is malnourished, the fetus will extract what it needs, for it is the "complete parasite".⁽⁷⁾

During the reproductive years, a woman accumulates fat when she is not pregnant as an insurance against any lean period that may occur should she become pregnant. Unless the proportion of fat in her body composition is 20% or higher, a woman does not ovulate, and cannot therefore conceive. Because of these safeguards, a state of near starvation during the index pregnancy, especially in the latter half, is required to produce any deleterious effect on the fetus. In normal pregnancy and on a normal diet, the pregnant woman stores nutrients and gains an average of 300-400g in weight per week. By 30 weeks, she has stored over 3.5kg of fat, chiefly in the subcutaneous tissues of the thighs and abdomen. In later pregnancy, under the lipolytic influence of increasing amounts of human placental lactogen, this fat can be mobilised, resulting in a rise in the maternal plasma levels of free fatty acids. Further, the placenta accumulates nutrients, except glucose, and holds them in reserve to be supplied to the fetus when the need arises in late pregnancy.⁽⁸⁾

Acute starvation for many months during pregnancy can cause a significant decrease in birth weight. This occurred in the Dutch famine quasi-natural experiment towards the end of World War II.⁽⁹⁾ However, no other serious adverse effects, such as increased incidence of perinatal mortality, congenital anomalies, and long-term neurological/intellectual deficit, were demonstrated in the children of the mothers who suffered the famine.^(9,10) The duration of pregnancy was not significantly shorter during the Dutch famine. However, others have found an association between low maternal pre-pregnancy weights, (i.e. weight 50kg), and poor weight gain, (i.e. 0.24kg per week) during pregnancy, and an increased risk of preterm birth.⁽¹¹⁾

The civil wars in many parts of Africa and the severe conditions of refugees can be expected to produce similar results.

In many areas of the developing world, average birth weights are 400g or more below that in the developed world. This contributes to the higher

perinatal and infant morbidity and mortality levels in the developing world. ⁽¹²⁾ The reasons for the difference in birth weight are many and include maternal under-nutrition and malnutrition, infections, poor housing conditions, and generally poor health. Maternal malnutrition may be the commonest cause of fetal growth restriction, particularly in parts of the developing world. ⁽¹³⁾ On the other hand, evidence of good nutrition predicted a well-grown baby. ⁽¹⁴⁾ Among term infants, maternal weight gain accounts for the largest proportion of variation in infant birth weight. ⁽¹⁵⁾ In a study in Nigeria, mothers who gained less than 5kg had low birth weight babies; no mother who gained 15kg had a low birth weight infant, and no mother who gained 10kg had a macrosomic infant. ⁽¹⁵⁾

Protein calorie supplementation of the diet of pregnant women to prevent inadequate nutrition would seem appropriate. However, this is controversial. Some studies have concluded that dietary intervention can be beneficial ^(17,18,19), others have concluded that it has only a moderate, an insignificant, or no effect ⁽²⁰⁾, whilst still others have concluded that it can even be harmful. ⁽²¹⁾

Nutritional advice in pregnancy:

Should pregnant women be advised what to eat and how much to eat? Is such advice feasible? Does it make any difference? Kramer reviewed the relevant literature and concluded that nutritional advice appeared effective, but no health benefits for the mother, or her baby could be demonstrated. ⁽²¹⁾ Obviously, nutritional advice would be most beneficial in deprived populations than in well-nourished women. Unfortunately, low-resource women may not have the means to follow the advice. As pointed out by Lindmark, such advice "can be expected to be useful only if the women have the real possibility of increasing their food intake, or altering the composition of their diet. Another prerequisite is that staff at primary antenatal clinics have adequate knowledge of nutrition and have enough time to give practical advice. These conditions are rarely found in under-resourced settings". ⁽²²⁾

The calorie needs of the pregnant woman take precedence over her protein needs. Therefore, in low-resource and poor communities, the emphasis should be on adequate intake: she should eat enough of the foodstuffs she can get, and can afford, and already eats. In other words, she should have "more of the same". That is not to say practical knowledge of nutrition is not useful. The woman is the manager in the home, and as the

message is reinforced at each visit and in each pregnancy, she is bound to take something home. This will benefit the whole family, especially the children.

Food supplementation during pregnancy:

Supplementation can be given with the following objectives: (a) To improve the quality of the habitual food by increasing its protein content without increasing the total calories per day. (b) To increase the total calories per day (i.e. the quantity) without changing the constitution of the food. (c) To increase both the protein content and the total calories. Some terms that will be used in the discussion will now be defined.

Isocaloric protein supplementation: Where the protein content of the supplement replaces an equal quantity of non-protein energy. To illustrate, let us say that the total energy of the habitual food is 1,500kcal/day, and that protein forms 10% (i.e. 150kcal/day) of the total calories, with non-protein (carbohydrates and fats) forming the remaining 90%. We have isocaloric supplementation if, without increasing the total energy, we increase the protein contribution to 20% (i.e. 300kcal/day) so that non-protein energy now forms 80% (1,200kcal/day) of the total energy of 1,500kcal/day.

Balanced protein supplementation: This is when the protein content of the supplement is less than 25% of the energy content of the supplement.

Isocaloric balanced protein supplementation: Where the protein content of the supplement replaces an equal quantity of non-protein energy, as long as the protein supplies less than 25% of the calories in the supplement.

Balanced protein-energy supplementation: Where the total daily energy intake is significantly increased (by 300 to 850kcal/day) with protein contributing less than 25% of the calories in the supplement. To illustrate: Let us say that the total energy of the habitual food is 1,500kcal/day with protein contributing 10% of the energy, i.e. 150kcal/day. We shall have balanced protein-energy supplementation if we add 500kcal to the daily energy intake to make a total of 2,000kcal/day and if the protein content of the supplement is less than 25% protein, say it is 20% (100kcal/day). Should pregnant women be given additional calories to supplement their habitual food intake?

Balanced Protein-energy Supplementation In Pregnancy:

Kramer reviewed the literature on balanced protein-energy supplementation in pregnancy, i.e. where the total daily energy intake is increased with a supplement in which the protein content is less than 25% of the energy content of the supplement. He concluded that supplementation modestly improved maternal weight gain, fetal growth and birth weight, but was unlikely to be of long-term benefit to pregnant women or their babies (18). On the other hand, Prentice et al. observed greater beneficial effects of energy-dense food supplementation in a group of pregnant women in rural Gambia who would have been in negative energy balance, as judged by poor weight gain and loss of subcutaneous fat during the wet season. The loss of subcutaneous fat was demonstrated by decreases in mid-upper arm circumference and triceps fold measurements.

Calorie supplementation significantly increased mean birth weight by 22556g. The proportion of low birth weight decreased from 23.7% to 7.5%.⁽¹⁷⁾ In a study in Guatemala, maternal food supplementation reduced the prevalence of low birth weight from 29% to 13% among women with the lowest socio-economic score⁽¹⁹⁾. Prentice et al. make the point that the beneficial effect of calorie supplementation will be seen when only truly at-risk groups are selected for intervention. These are women who are in negative energy balance in the second and third trimesters of the index pregnancy, as shown by failure to gain weight and loss of subcutaneous fat.⁽¹⁷⁾ The technology for this intervention need not be beyond the means and capacity of developing countries. The feasibility and sustainability of such an intervention in low-resource settings has been demonstrated.⁽²³⁾

However, it is possible that improved nutrition may hasten the return of fertility, and result in shortening of the birth interval. This will defeat the aim of the intervention as short birth intervals have adverse effects on the mother and her children.

Iso-caloric protein supplementation during pregnancy:

During pregnancy, a high-protein diet without an increase in daily energy intake might have adverse effects on the mother and her baby. Studies were done in which extra protein was given to *replace* some of the energy provided by non-protein in the habitual food, but with the total protein in the supplement being less than 25% of the energy content of the supplement.

This resulted in a decrease in maternal weight gain, a decrease in mean birth weight, and an increased risk of small for gestational age births⁽²²⁾.

Food supplementation and cephalo-pelvic disproportion: It is possible that food supplementation may produce babies who are too big for mothers who had suffered malnutrition from birth into adulthood and who are, as a result, physically under-developed. Women born and raised in developing countries who emigrated to the United States as adults were compared with women born in the United States with respect to caesarean birth for dystocia from cephalopelvic disproportion. The former group of women were found to be at higher risk of caesarean delivery for disproportion⁽²⁴⁾.

Maternal iron supplementation: Should women receive supplementary iron in pregnancy? In normal pregnancy, plasma volume starts to rise from the 6th week and by 32 weeks has increased by 1250ml above the non-pregnant level of 2600ml, an increase of 48%. This level is maintained until term. In normal pregnancy and in the absence of iron supplementation, red cell volume starts to rise from the 10th week and continues to rise until term, by which time it has increased by 450ml above the non-pregnant value of 1400ml, an increase of 32%. The increases in plasma and red cell volumes cause the blood volume to increase by about 40%. The magnitude of the blood volume increase is influenced by the weight of the baby, and by the number of fetuses. Multiple pregnancy and heavier babies are associated with greater increases. On the other hand, the increase is much smaller, or may not occur at all, in pre-eclampsia (proteinuric pregnancy induced hypertension) and in women with a history of repeated spontaneous abortions, or stillbirths. Routine administration of therapeutic doses of iron (100mg elemental iron daily) causes larger increases in red cell volume and in blood volume but not in plasma volume.⁽²⁵⁾ In normal pregnancy, cardiac output also starts to rise in the first 10 weeks and by term has increased by 1.5L (45%) above the non-pregnant value of 4.5L.

Because, normally, the increase in plasma volume is much larger than the increase in red cell mass, the haemoglobin concentration, the haematocrit (PCV), and blood viscosity fall. These changes may have beneficial effects. The decrease in blood viscosity and peripheral resistance will lead to increased perfusion, (including increased utero-placental and intervillous blood flow), lower blood pressure, and a reduction in the work of the heart.

Because of the increase in red cell volume and total haemoglobin, the oxygen-carrying capacity of the blood does not fall; indeed, the arterio-venous difference narrows. The "physiological" changes in the haematological indices suggest iron deficiency anaemia: fall in haemoglobin, haematocrit, and serum iron, and sometimes a fall in mean corpuscular haemoglobin concentration, and a rise in total iron binding capacity.

Routine iron supplementation raises, or maintains the serum ferritin level above 100g/L. It results in a substantial reduction in the proportion of women with a haemoglobin level below 10 or 10.5g/dL in late pregnancy and at 6 weeks postpartum. It also reduces the need for postpartum blood transfusion. However, routine supplementation had no detectable effect on any substantive measures of either maternal or fetal outcome.⁽²⁶⁾ It has therefore been suggested that the fall in haemoglobin is "physiological" and should not be prevented or reversed by administration of therapeutic doses of iron during pregnancy.

Some studies have even found adverse perinatal outcome, as measured by the incidence of stillbirth and low birth weight, in women whose haematocrit remained high.^(26,27) The relationship between stillbirth and haemoglobin concentration as recorded in the first trimester was studied in Sweden. It was found that the risk of a stillbirth when the booking haemoglobin was over 14.5g/dL was twice the risk associated with a lower haemoglobin value; the higher the haemoglobin value the stronger the association. The association persisted after controlling for pre-eclampsia⁽²⁸⁾. However, the outcome for women with very low haemoglobin levels is commonly worse. For example, in a study done in Port Moresby, Papua New Guinea, the highest stillbirth rate (94/1000 births) occurred in women whose lowest recorded haemoglobin value was 6g/dL; the rate was 22.5/1000 when the lowest haemoglobin was 12g/dL.⁽²⁹⁾

In addition to the 30% increase in maternal erythrocyte mass, the growing fetus and the placenta demand iron, especially in the last trimester. If the mother's diet is not rich in iron, and if her iron stores are low, these demands will produce anaemia. In the developing world, many women start pregnancy already anaemic, or with low iron stores. Anaemia in pregnancy is common and an important cause of maternal morbidity and mortality and of perinatal mortality in these countries. In a study of postpartum women who did

not have a haemoglobinopathy at the Korle-Bu Teaching Hospital, Ghana, the lowest haemoglobin value recorded during the index pregnancy was less than 10g/dL in 31%, and it was 8g/dL in 7%.⁽³⁰⁾

Non-haemoglobinopathy anaemia in pregnancy in the developing world is caused by a number of factors, which may combine. The factors include: a diet poor in iron and folic acid, malaria and the tropical splenomegaly syndrome, hookworm infestation, chronic infections, and births that are so many and so frequent that the mother is not able to replenish her iron stores before the next birth. Iron supplementation is therefore an essential intervention in antenatal care, not only for the current pregnancy but also to replenish the iron stores to carry the mother through lactation and into the next pregnancy. A dosage of 60mg to 100mg elemental iron daily has been recommended, but we prefer to give therapeutic doses unless, or until, the haemoglobin concentration is above 11g/dL.

Folic acid supplementation: It has been conclusively shown that routine folate supplementation raises, or maintains serum and red cell folate levels and substantially reduces the incidence of anaemia in late pregnancy. It possibly reduces the incidence of low birth weight, as well.⁽³¹⁾ Malaria or sickle cell disease shortens the life span of red cells, demanding increased rates of erythropoiesis to prevent severe anaemia. This in turn demands folic acid. Therefore, in West Africa, where both malaria and sickle cell diseases are common, adequate intake of folic acid is particularly important.

Ghanaians, for example, eat some amount of green leafy vegetables, but in preparing the food, the leaves are boiled for hours and this destroys the folic acid they contain. Folic acid supplementation is therefore important. Supplementation before conception and in early pregnancy is effective in the preventing neural tube defects.^(4, 32) The Centers for Disease Control (CDC), USA, has recommended 400g folic acid daily for supplementation.⁽³³⁾ In Ghana, folic acid preparations containing this amount are expensive. The readily available 5mg tablets are far cheaper and can be given twice a week.

Vitamin A: A study in Malawi showed greater risk of mother-to-child transmission of HIV in women with vitamin A deficiency. It however remains to be demonstrated whether vitamin A supplementation actually prevents transmission.⁽³⁾ Maternal deficiency of vitamin A may also have an

association with infant morbidity and mortality. In the Kassena-Nankana District of northern Ghana, vitamin A deficiency (serum retinol 0.70mol/L) is common. Supplementation with a high dose of vitamin A every 4 months reduced mortality in children aged 6 months to 7 years by 19%.⁽³⁴⁾ The concentration of vitamin A is greater in fetal serum than it is in maternal serum. Fetal liver stores of vitamin A may protect the baby in the first few months of life. Maternal deficiency, therefore, may affect the health of the baby.

Maternal iodine supplementation in areas of iodine deficiency: Iodine deficiency is the leading preventable cause of intellectual impairment in the world. In some areas of the world the prevalence of endemic cretinism may reach 3-15%.⁽³⁵⁾ Iodine deficiency is common in some parts of Ghana. It can be corrected or prevented by adding iodine to the salt of the population at risk; salt can be iodated with potassium iodate, or iodised with potassium iodide. Alternatively, individuals can be given oral, or injectable iodised oil.

Iodine supplementation before or, during pregnancy was studied in areas of iodine deficiency. Supplementation was associated with a significant reduction in deaths during infancy and early childhood, in reduction of both the prevalence of endemic cretinism at age 4 years, and in an increase in psychomotor development scores at 4-25 months of age.⁽³⁶⁾ Iodated salt was introduced in Ghana in 1997. The difficulty is to convince those who are at greatest risk of iodine deficiency to use the iodated salt, instead of the much cheaper traditionally-produced salt.

Twinning and dietary status: It was suggested that dietary differences might explain the wide differences in twinning rates among neighbouring rural villages in some parts of Nigeria.⁽³⁷⁾

Nutrition and the male/female sex ratio:

A low male/female ratio has been associated malnutrition in animals. It has therefore been postulated that malnutrition may affect the human sex ratio. The male/female sex ratio at birth is lower in African populations than in Caucasians. The difference has been attributed to genetic, behavioural, and environmental factors. A study of babies born to women in a rural African society found short maternal stature and obesity to be independently associated with a low sex ratio.⁽³⁸⁾

This may indicate that nutrition has some effect on the sex ratio, although the association may also be genetic.

Conclusion: The nutritional demands of pregnancy are great. When these demands are not met, it is the mother who first suffers. Severe deprivation, however, does affect the fetus. Energy is the foremost requirement, but deficiency of certain nutrients, for example, iodine, can have severe irreparable consequences.

In the reproductive years, a woman only ovulates, and can therefore only become pregnant, when her fat stores have reached a certain threshold (20kg). If nutrition is adequate during pregnancy, she continues to store fat and to gain weight at a rate of about 0.3kg per week.

The pregnant or lactating mother must first consume enough calories. She must be advised to eat enough of the food she already eats. Secondly, she must try to eat a balanced diet. Some nutrients cannot be stored for long periods and must be available in the diet on a daily basis. Many Ghanaian women start pregnancy with depleted iron stores. They need iron, as well as folic acid supplementation.

Appendix 1. Nutritional Requirements in Pregnancy

Total weights gain	102kg
Energy	2550kcal
Protein	38g
Iron	28mg
Calcium	1.2g
Vitamin A	750g
Vitamin D	10g
Folic acid	400g
Ascorbic acid	30mg

Source: WHO 1974 Handbook on Human Nutritional Requirements. Monograph Series No. 61, WHO, Geneva

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SECTION 2

INTRAPARTUM CARE

Physiology And Management Of Labour

E. Ejiro Emuveyan

Introduction

The birth of a mature and healthy infant depends on a mechanism which ensures that the uterus stays quiescent during pregnancy while the fetus is developing and then at the appropriate time initiates the powerful and coordinated uterine activity and softening of the cervix which cause cervical dilatation and ultimately delivery of the infant. The vital importance of this control mechanism and its reliability is illustrated by the fact that 85% of human infants are born at term.

It is remarkable how, in each species, the uterus remains quiescent throughout a pregnancy for whatever duration is necessary for the full development of the fetus. Then, and usually only when the fetus is fully matured, does the quiescent uterus become contractile, the cervix softens and dilates and the transition from an intrauterine to an extra uterine existence is completed by the expulsion of a fetus or fetuses capable of maintaining independent existence.

Of all the experiences of the human condition, birth surely represents the most important. The spectrum of maternal experiences of childbirth extends from exhilarated, fulfilled and enriched mothers, to those women who are permanently crippled physically or emotionally and even, still all too commonly, pay for the experience with their lives.

The simple objective of every pregnancy is the delivery of a healthy baby to a healthy mother. The fullest possible understanding of the birth process, its perturbations and appropriate management policies is central to that objective.

Definition

Labour is defined as the occurrence of uterine contractions of sufficient frequency of one contraction every two to three minutes, intensity, and duration of 40 to 60 seconds resulting in effacement and dilatation of the cervix.

Labour is traditionally divided into three stages:

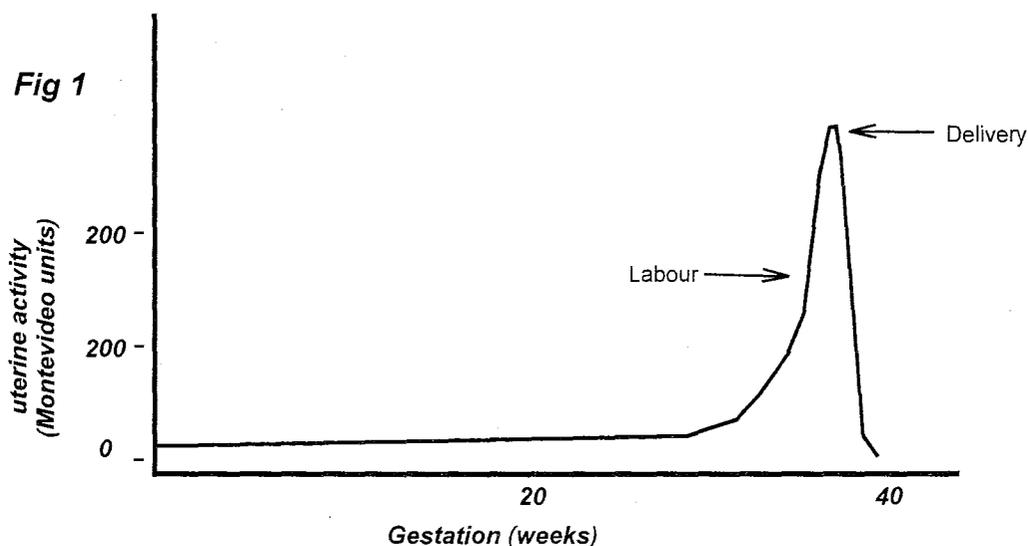
First Stage	- represents the period from onset of labour to full cervical dilatation
Second Stage	- is the period from full cervical dilatation to delivery of the Infant
Third Stage	- represents the period from delivery of the infant to the delivery of The placenta.

Control Of Parturition And Integration Of Control Pathways

Labour represents the transition of the uterus from a quiescent capacitance compartment into an efficient contractile unit capable of effecting birth.

Traditionally, the process is divided into 3 stages as mentioned earlier. However, the transition from capacitance to contractile unit is not as sudden as these traditional definitions might suggest. Rather, it evolves gradually over the last few weeks of pregnancy during a phase termed prelabour, and from a physiological perspective this component of the process perhaps should be added to our traditional categorization of the process.

Prelabour and labour consist of two distinct but nonetheless interlinked processes - cervical ripening and increasing myometrial excitement. The increased myometrial capacity for excitement of the prelabour phase is perhaps best documented by the classical studies of Caldeyro - Barcia (1959). Using intramyometrial balloon catheters to record contractions, he showed that the uterus is never entirely quiescent. Its spontaneous contractility during pregnancy, first described by Braxton Hicks in 1872, increases both in frequency and intensity over the last 5-6 weeks of pregnancy, climaxing in labour and delivery. This is illustrated schematically in Fig.1.



However, this might also be used to show the contraction of myometrial gap junctions, cervical ripening, and increase in uterine oxytocin sensitivity, which develop over the same time. Cervical ripening and myometrial excitement develop in synchrony and progress to the regular uterine contractility and cervical dilatation of labour.

Cervical Ripening

Cervical ripening is the process by which the cervix becomes softer and more easily distensible.

Unlike the uterine corpus, the cervix contains only a small amount of smooth muscle (10% to 15% of the cervical tissue, with the content of smooth muscle decreasing from the upper to the lower segment of the cervix. The underlying stroma is mainly connective tissue made of collagen fibrils (mainly of collagen types I and III) bound together into dense bundles which confer on the cervix the rigidity which is characteristic of its non-pregnant state and early pregnant state. The ground substance consists of large molecular weight proteoglycan complexes. Proteoglycans consist of a protein core to which are attached glycosaminoglycans (GAG) branches. There are a variety of GAGs, such as heparin, heparin sulphate, dermatan, chondroitin and hyaluronic acid. The predominant GAGs found in the cervix are chondroitin sulphate and dermatan sulphate.

In the last few weeks of pregnancy, important changes occur in the cervix, making it softer and more pliable. A change in the relative content of GAGs occurs with an increase in the concentration of hyaluronic acid and a decrease in the concentration of chondroitin and dermatan sulphate. Hyaluronic acid is hydrophilic, and by

attracting water molecules, makes the cervix softer. The decrease in dermatan sulphate concentration causes a decrease in its bridges between collagen fibrils. The end effect is rearrangement of the collagen fibres into looser and more disorganised network. Concomitantly, an increase in cervical collagenase (also known as metalloproteinase - 1) activity occurs. Hence cervical collagen degradation also contributes to the ripening process.

The trigger responsible for the initiation of cervical ripening is not entirely understood. However, recent studies of a number of inflammatory mediators, notably interleukin - 8 (IL - 8), Monocyte Chemoattractant Peptide (MCP - 1) have focussed attention on neutrophils and monocytes recruited from the circulation as the likely factors in the process. Neutrophils are a rich source of collagenases and elastase, matrix metalloproteinases that play a critical role in the breakdown of cervical collagen.

One attractive hypothesis (Kelly 1994), implicates PGE_2 as mainly responsible for vasodilatation of cervical capillaries and increasing their permeability to circulating neutrophils, which are captured by surface adhesion molecules in the capillaries and drawn into the cervical stroma under the chemoattractant influence of IL - 8. This chemokine is also responsible for stimulating their degranulation within the tissues to release these collagenolytic enzymes. Monocytes are also recruited into the cervix by MCP - 1 and might potentially play a unifying role as a source of both PGE_2 and IL - 8).

Activation Of The Myometrium

The myometrium consists predominantly of smooth muscle cells arranged in bundles embedded in connective tissue matrix made principally of collagen, which helps to transmit the generated tension throughout the tissue. The individual myometrial fibre contracts when its two filaments of actin and myosin are combined by phosphorylation by the enzyme **myosin light chain kinase** to form action myosin.

This reaction requires the increased availability of intracellular calcium, released from stores within the smooth muscle cell (mainly the sarcoplasmic reticulum) which may be provoked by oxytocin or PGE_{2a} or both through the second messenger inositol triphosphate. Additionally, extracellular calcium may be transported into myometrial cells via calcium channels. The factors, which start the process of labour spontaneously, have not yet been fully elucidated. Several biological agents have been proposed as being involved in the initiation of the onset of labour. However, the following agents require special mention.

Prostaglandins

Prostaglandins are produced by the amnion, decidua and uterine myometrium. However, the prostaglandins involved in stimulating uterine activity appear to be produced mainly in the amnion and decidua and not the myometrium. The myometrium produces prostaglandins but mostly PGI₂.

The central role of prostaglandins in the onset of labour relates to their known action as myometrial stimulants, their ability to change cervical compliance and especially to reduce its resistance, and their influence on gap junction formation in the myometrium. Three major lines of evidence support the contention that they play a central role in the onset of labour. First, there is an increase in the concentration of these substances in the amniotic fluid, and of their metabolites in plasma and urine in late pregnancy. Antiprostaglandin agents such as aspirin or indomethacin can prolong pregnancy. Second, the myometrium is extremely sensitive to exogenous prostaglandins. They also increase the sensitivity of the myometrium to oxytocin.

Placental Steroids

There is some evidence that prior to the onset of labour, progesterone secretion diminishes and, at the same time oestrogen output increases

significantly. Oestrogen has a stimulatory effect on the myometrium by reducing the resting membrane potential of the cells and increasing the formation of gap junctions. Progesterone has an inhibitory effect on uterine contractility.

Oxytocin

Prior to onset of labour there is no more than a low constant concentration of oxytocin in the blood. It is believed that the initiation of labour does not result directly from the stimulus of maternal oxytocin, though it may be involved in some facilitatory mechanism.

Integration Of Control Pathways

While we are aware of several components of the physiologic systems controlling labour, the mechanism behind human parturition remains enigmatic. As emphasized above, the transition from pregnancy to labour develops gradually during a month or more of 'pre-labour'. From early naive concepts, which credited the mother as responsible for initiating labour by producing oxytocin from her posterior pituitary, the hypothesis has gradually been developed whereby control is initiated and largely vested with the fetoplacental unit.

The key components appear to be the fetal brain whose influence is exerted on the fetoplacental endocrinology via the hypothalamopituitary-adrenal axis. Activation of corticotrophin (ACTH) stimulates adrenal production of:

- (i) Cortisol, which brings about maturation of the fetal lungs with the generation of pulmonary surfactant.
- (ii) Dehydroepiandrosterone sulphate (DHEAS).

DHEAS, a key precursor of placental oestradiol production, ordains a shift in oestrogen to progesterone ratio in favour of oestrogen and provokes an endocrine cross talk between fetus, placenta, membranes and uterus. Cortisol promotes maturation of the fetal lungs and this, together with similar events in the fetal kidneys, may modify the content of the amniotic fluid and thereby activate the fetal membranes (amnion and chorion), particularly in respect of prostaglandin production. By means of such biological changes in the fetal components, (fetus, placenta, amniotic fluid and the membranes) a new dialogue is created with the uterine (maternal) tissues, which envelop them (the decidua, myometrium, and cervix) producing a positive cascade of interactions between prostaglandins, cytokines and oxytocin.

The Course And Mechanisms Of Labour

In describing the phenomenon of labour the uterus may be divided into two functional segments. The upper segment, which comprises muscle fibres that have the ability to contract, relax and retract. The lower segment, consisting of the lower part of the body of the uterus and the cervix, can contract but is relatively passive as compared to the upper segment. The upper and lower segments are not fully formed until the end of the first stage of labour when they can be clearly seen and the transition between them is quite abrupt.

For a clear understanding of the course of labour, knowledge of the components of labour is necessary. These components are the powers, passages and the passengers.

Powers

This refers to the forces that expel the baby. They can be divided into primary and secondary forces. The primary forces refer to the uterine myometrial contractions. The power generated by the upper uterine segment contractions must be associated with a degree of uterine polarity whereby the retractile effects of the upper uterine segments can overcome the stretching and relaxation properties of the lower uterine segment and cervix.

Braxton Hicks contractions, which can be observed during the latter part of pregnancy, and which precede labour, are ineffective with regard to cervical dilatation or descent of the presenting part. Moreover, they do not increase their frequency or duration. Neither do they cause retraction of the upper segment. In these respects they differ from the primary forces of parturition.

The primary forces of labour (contractions) gradually increase their frequency, duration and power. Eventually, they occur approximately one every 3 minutes and there is 1-2 minutes effective relaxation between them. The fibres of the lower segment become elongated and thinned during the process, and gradually incorporate the supravaginal portion of the cervical canal, subsequently, the infravaginal cervix, which is at first effaced, dilates progressively. Correct pressure relationships between the fetal head and the lower uterine segment are important to this mechanism. Uterine contractions begin at the cornual region of the uterus from where, in a circular distribution, they spread outwards and downwards as a peristaltic wave.

Intrauterine pressure recordings taken during labour indicate that the resting tone is usually about 5mmHg and does not increase by more than about 5-10mmHg between contractions. Cervical dilatation is associated with intrauterine pressures above 20 mmHg and it may be observed that patients often experience abdominal discomfort when pressures exceed 25mmHg.

Forceful uterine contractions may increase the intrauterine pressure to about 77mmHg above the resting tone. After the primary forces have achieved full dilatation of the cervix and descent of the fetal presenting part to the pelvic floor, the patient in natural childbirth experiences a spontaneous and overwhelming desire to contract the voluntary muscles of her diaphragm and abdominal wall, in an effort to expel the baby from her body. These are the secondary forces.

Passages

The passages through which a mother expels the baby from her body during natural labour include the soft passages of the birth canal, supported by ligaments, fascia and fat, and the hard bony passage of the pelvis.

Passengers

The passengers include the fetus, the placenta and the membranes. It is the size of the fetal skull that usually determines the ease with which parturition occurs.

The Course Of Labour

Parturition is usually a continuous process but traditionally it is divided into three stages. The first, second and third stages of labour

First Stage

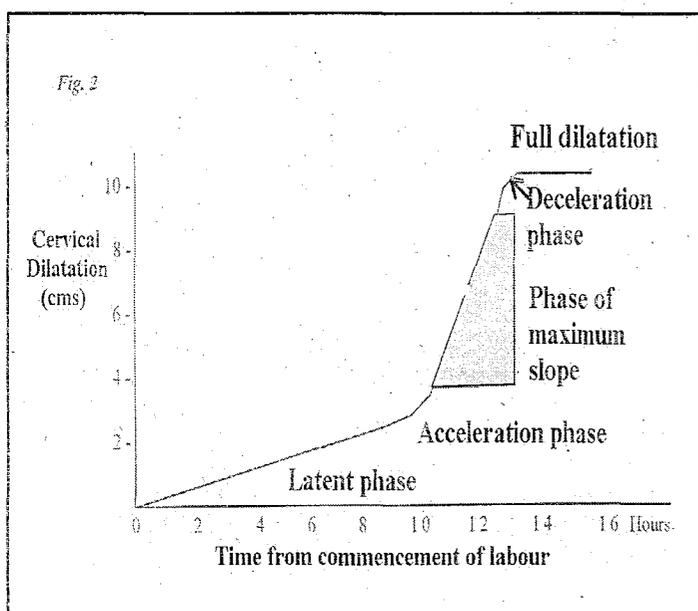
Commonly, early in the first stage, the mucus plug within the cervix is extruded and appears at the vulva as a jelly-like substance streaked with blood (the 'show'). Rupture of the membranes can occur at any time during the first or second stage. Usually, membranes rupture spontaneously in the later part of the first stage. Progress during the first stage of labour is determined by dilatation of the cervix, and/or descent of the presenting fetal part through the maternal pelvis.

Friedman (1955) analysed the progress of spontaneous labour by plotting graphically cervical dilatation against time. He described:

- (a) A latent phase of variable duration; followed by
- (b) A phase of acceleration at about 3 - 4cm dilatation of the cervix:

- (c) The phase of maximum slope; followed by
 (d) A short deceleration phase, which led to full cervical dilatation (10cm). It should be noted that all investigators do not accept the deceleration phase

The average cervical dilatation rate in primigravidae is 1cm/hour whereas for multigravidae it is 1.2cm/hour. The average duration of first stage of labour in primigravidae is 10-12 hours and for multigravidae 6 - 8 hours. The mechanism by which the cervix dilates deserves consideration.



Myometrial contraction and retraction even in the presence of polarity and fundal dominance are unlikely to promote significant cervical dilatation unless the upper pole is fixed relative to the lower one. The fetus probably acts as a central column for the uterus. Thus, while the head cannot escape from the lower uterine area, myometrial retraction occurring in the upper segment pulls upwards the more elastic tissues of the lower uterine segment. Eventually, when the cervix is fully dilated, myometrial retraction continues but the stretched lower pole of the uterus now becomes fixed relative to its upper pole, so the fundus is gradually pulled downwards.

Second Stage

This stage starts from full cervical dilatation to delivery of the infant. Second stage of labour has been traditionally restricted to 2 hours for nulliparous women and 1 hour for multiparous women. In the second stage the resistance offered by the lower uterine segment has been overcome

and the presenting part can be pushed down onto the pelvic floor. The resistance of the pelvic floor is overcome by the uterine contractions, aided by the action of the voluntary muscles of the abdominal wall and diaphragm

Third Stage Of Labour

The third stage of labour is the period from the birth of the infant to delivery of the placenta. Normally the third stage lasts less than 30 minutes. As the cavity of the uterus becomes smaller following the delivery of the infant, the area of the placenta site is diminished so that the placenta is sheared off the spongy layer of the decidua basalis. When the placenta has separated it presses on the pelvic floor, causing the women to have an involuntary desire to bear down. The placenta is expelled from the vagina, followed by the membranes. There is generally an escape of less than 200ml of blood as the placenta is delivered.

There are two ways in which the placenta is delivered. In the Schultz method the placenta presents by its centre, which comes out first dragging the membranes behind it. In the Matthews Duncan method the placenta presents by an edge and slips out of the vulva sideways.

Mechanism Of Labour

The term mechanism refers to the series of changes in position and attitude, which the fetus undergoes during its passage through the birth canal. The cardinal movements with vertex presentation are engagement, flexion, descent, internal rotation, extension, restitution and external rotation.

When the fetal presenting part enters the pelvis, it is said to have undergone engagement if the presentation is cephalic. The head will then undergo flexion, which allows the smallest diameter (vertex) to present to the pelvis. Descent of the vertex will occur with passage of the head down into the pelvis. With descent into the mid pelvis, the fetal vertex will internally rotate so that the sagittal suture is parallel to the antero-posterior diameter of the pelvis. As the vertex passes beneath and beyond the pubic symphysis, it will extend to deliver. When internal rotation takes place, the head is twisted a little on the shoulders. As soon as the head is completely born it resumes its natural position with regard to the shoulders, the occiput turning towards the mother's left or right thigh. This movement, which sometimes occurs almost with a jerk, is called restitution. Once this occurs external rotation of the head occurs and the shoulders may be delivered.

Symptoms And Signs Of Labour/diagnosis

The symptoms and signs of the onset of labour are:

1. The 'show'
2. Regular, painful uterine contractions
3. Cervical effacement and dilatation
4. Rupture of the membranes.

Management Of Labour

The aims of successful labour management are to ensure the safe delivery of a healthy baby to a fit, satisfied mother with minimum interference. At the same time it is important to provide appropriate choices for analgesia, position in labour and a pleasant environment in which to give birth.

Majority of women giving birth are confined to hospitals today simply because obstetric emergencies such as fetal distress or postpartum haemorrhage may occur suddenly in apparently normal cases. The expertise and facilities to deal with an emergency are immediately available in the hospital whereas there is inevitable delay if the crisis occurs in the patient's home, a delay that may be the deciding factor between life and death for the woman and/or her baby.

Modern management of labour is carried out by a team consisting of obstetrician, senior registrar (resident) and or registrar and a midwife. The role of the midwife is of utmost importance. At present the midwife is the senior person present at over 70% of normal deliveries.

The first imperative in the conduct of labour is to determine whether labour has in fact started, the accurate diagnosis of labour is essential because so much depends on defining starting points.

First Stage Of Labour

On admission the woman's antenatal record is reviewed to determine whether there have been any abnormalities during her pregnancy. History of labour and a complete physical examination and obstetric examination are then carried out to ascertain her general health status, vital signs, and characteristics of uterine contractions, fetal presentation, level and position of the presenting part as well as cervical effacement and dilatation. Her urine is next checked for the presence of albumin or sugar.

It is important to establish a good channel of communication at this stage and to allay the patient's anxiety and fears. If there is adequate

communication, there is likely to be better cooperation and better outcome.

✓ If the presenting part is engaged there is no need for the woman to remain in bed during early labour. If the presenting part is not engaged the woman is kept in bed to reduce the likelihood of prolapse of the cord when the membranes rupture.

During labour there is a delay in gastric emptying and food or fluids may remain there for several hours. If general anaesthesia is to be administered for any reason there is a risk of vomiting and inhalation of acid contents of the stomach, which can cause bronchiolar spasm (Mendelson's syndrome). It is better to withhold solid food during labour, and if fluid or glucose is to be given this is done intravenously.

Particular attention should be paid to the condition of the fetal membranes. If the history suggests that they have ruptured before admission it is important to look for confirmatory evidence of this especially during the initial vaginal examination. Adequate clear liquor draining is generally a reassuring sign that the fetus is in good condition to withstand the rigors of labour.

The timing of amniotomy is critical. The optimum time for amniotomy lies at the point of transition from latent phase to active phase, when the contractions are well in train and the cervix is fully effaced and at least 3 - 4cm dilated. If labour is progressing well and the mother and fetus seem well, there is no compelling need for amniotomy.

Monitoring of the fetal and maternal condition and progress of labour is vital to a successful management of labour.

Partograph

This is a graphical representation of the events and progress of labour. Routine observations of the mother's pulse rate and blood pressure, with an assessment of the strength and duration of uterine contractions are entered on it. Records of findings at successive vaginal examinations are plotted on a graph, showing the dilatation of the cervix in centimetres against the time in hours. The curve obtained is compared with an average curve. If the patient's progress is normal her curve will correspond with the normal curve or lie to the left of it. If labour is not progressing well in the active phase, the patient's partograph will be to the right of the normal.

Fetal Monitoring

This is vital to successful management of labour. The different methods of fetal monitoring are: Intermittent auscultation of fetal heart tones using the foetal stethoscope (Pinard's stethoscope)

- ii. External electronic fetal monitoring with Doppler.
- iii. Fetal Scalp Electrode
- iv. Fetal blood sampling for pH

Intermittent auscultation of fetal heart tones using Pinard stethoscope is mostly done in this environment. It involves auscultating for fetal heart tones every 15 minutes. Normal fetal heart rate during labour is 120-160 beats per minute.

The external electronic fetal monitoring gives a recording of the uterine contractions and fetal electrical heart activity simultaneously on the same tracing.

Fetal scalp electrode is used in patients that are difficult to monitor externally with Doppler or where there is repetitive decelerations on the external electronic monitor tracing. The scalp electrode is applied after the membranes have ruptured.

Fetal blood sampling for pH is used when the fetal heart tracing is not reassuring, as it helps to assess fetal hypoxia.

Maternal Monitoring

Labour as the word connotes involves a lot of work on the part of the mother and puts a lot of stress on her. Complications can also crop up at any time so the mother should be monitored regularly in order to avoid or detect them for early management. The vital signs, urine output, urine protein and ketones are measured regularly and charted on the partograph.

Analgesia

The amount of pain experienced during labour varies enormously from woman to woman. Patients should be told about the various means of relieving pain. The ideal analgesic for labour should neither interfere with uterine action nor depress the respiratory centre of the newborn. It should also be effective and easy to administer. There are various methods of pain relieve in labour.

They include:

1. Injectable analgesics - opioids and opiates: morphine sulphate, pethidine, and pentazocine.
2. Inhalational analgesia: Nitrous oxide 50% premixed with 50% oxygen (Entonox).
3. Transcutaneous electrical nerve stimulation (TENS)
4. Epidural block
5. Caudal block

Management Of Second Stage Of Labour

The aim of management in the second stage of labour is to ensure smooth delivery and prevent maternal and fetal injuries. Delivery is usually accomplished with the patient in the dorsal or lateral position.

As the fetal head begins to crown for a vaginal delivery, the accoucher must control the head to prevent its being born suddenly, and it is delivered by extension until the largest diameter has passed the vulva outlet except for face presentation which is delivered by flexion.

Episiotomy, or incision of the perineal body, may be necessary in some cases and a clean incision is preferable to an irregular laceration, or even to a grossly overstretched perineal skin.

Once the head of the infant is delivered the mouth and upper airways are sucked clear of mucus. After suction, the infant's neck is checked for umbilical cord. If a nuchal cord exists, an attempt is made to reduce the cord over the infant's head; if the cord is tight, it is clamped in two places and cut then the rest of the delivery follows, first with delivery of the anterior shoulder by exerting direct downward pressure on the infant's head.

Once the anterior shoulder is visualised, a direct upward pressure is exerted to deliver the posterior shoulder. After this, exertion of gentle traction will deliver the rest of the infant. Care is taken not to raise the infant high above the mother to prevent fetal to placental blood shift. The cord should not be clamped immediately to allow for transfer of blood from the placental pool to the fetal circulation. However, in infants, suspected of having haemolytic disease of the newborn, late clamping results in the serum bilirubin levels being higher.

Management Of Third Stage Of Labour

Placental separation usually occurs within 5 to 10 minutes of delivery of the infant. However, up to 30 minutes is usually designated as within normal limits. The three signs of placental separation include cord lengthening, a gush of blood, and uterine fundal rebound as the placenta is detaching from the uterine wall.

Until these signs are noted no attempt should be made to deliver the placenta.

Current management of the third stage of labour is to make it active. It involves use of oxytocic drugs and active delivery of the placenta by controlled traction on the cord with elevation of the uterus (Brandt - Andrew's technique). These help to reduce blood loss to a minimum.

Following delivery of the placenta and membranes, a close inspection of the tissues must be done to ensure that they are complete.

Complications Of Labour

First Stage of Labour

- (i) Cord prolapse
- (ii) Fetal distress
- (iii) Maternal Dehydration
- (iv) Maternal exhaustion
- (v) Poor progress in labour
- (vi) Obstructed labour
- (vii) Ruptured uterus

Second Stage of Labour

- (i) Perineal lacerations
- (ii) Fetal distress
- (iii) Shoulder dystocia

Third Stage of Labour

- (i) Post partum haemorrhage
- (ii) Retained placenta

Controversies In Physiology And Management Of Labour

In spite of long years of research there still remains a lot of unanswered questions about the exact trigger and mechanism of initiation of natural labour. There are still unanswered questions about the exact roles of all the putative biological compounds implicated in labour. The primary stimulus to increase prostaglandin synthesis is not settled. The exact role of the fetal brain is not also settled especially since labour can still start spontaneously in pregnancies with anencephalic fetuses.

There are also controversies surrounding the active management of labour vis a vis the benefits to the mother and fetus. Some workers have also questioned the use of episiotomies routinely in primigravidae.

The exact significance of fetal heart tracing abnormalities in labour is still intensely being debated.

Conclusion

Labour is the whole process whereby the products of conception are expelled from the mother. It is an important human experience, which could give intense joy or sorrow depending on the outcome. A good understanding of the basic physiologic principles underlying labour is imperative in order to be able to make appropriate interventions where necessary. This will help in the drive towards making the experience of labour safe.

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The Partograph

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Introduction

Maternal mortality remains a major problem in the developing countries. It is estimated that about 600,000 women die through childbirth every year and about 99% of them occur in developing countries. Unfortunately the women are dying from causes that are to a large extent preventable and not by the use of any advanced technology. The means by which maternal mortality was reduced in the developed countries are to a large extent available to us in developing countries, such as, antenatal care, antibiotics and blood transfusion and safe anaesthesia. What is not available is widespread education and improved socio-economic conditions. So why are maternal mortality figures so high?

The major causes of maternal deaths are:

- Haemorrhage
- Sepsis
- Eclampsia
- Obstructed labour
- Sickle cell disease
- Complications of induced abortion

Prolonged and obstructed labour contributes to maternal death in different ways. Prolonged labour may lead to obstructed labour; obstructed labour if unrelieved will lead to maternal exhaustion, dehydration, ruptured uterus, postpartum haemorrhage, sepsis, and vesico-vaginal fistula. Thus a death that is recorded under haemorrhage, sepsis, and rupture of uterus may have prolonged labour as the underlying factor.

Prolonged labour in the developing world is commonly due to cephalo-pelvic disproportion (CPD), which may result in obstructed labour. Protracted labour is more common in primigravid women than in multipara and the complications and effects of CPD differ between them. In countries where CPD is not prevalent, abnormal progress of labour is often due to inefficient uterine action. Universally, less direct consequences of prolonged labour include maternal sepsis, post-partum haemorrhage and neonatal infection.

Early detection of abnormal progress of labour and

prevention of prolonged labour would significantly reduce the risk of postpartum haemorrhage and sepsis and eliminate obstructed labour, uterine rupture and its sequelae.

The partograph, a graphic recording of progress of labour and salient features in the mother and fetus has been used since 1970 to detect labour that is not progressing normally, to indicate when augmentation of labour is appropriate and to recognize cephalo-pelvic disproportion long before labour, becomes obstructed.¹

Friedman in 1954 described a normal cervical dilation pattern: he divided labour into (early) **latent phase** extending over 8-10 hours and up to 3cm dilatation and an **active phase** characterized by acceleration from 3-10cm at the end of which deceleration occurred.²

Hendricks et al demonstrated that in the active phase the rate of dilatation in primigravidae and multigravidae varies little and that there was no deceleration at the end of the first stage of labour.³

Philpot in extensive studies of primigravidae in Central and Southern Africa constructed a normogram for cervical dilation and was able to identify deviations from the normal and provide a sound scientific basis for early intervention leading to prevention of prolonged labour.⁴

The partograph serves as an "early warning system" and assists in early decision on transfer, augmentation or termination of labour. It also increases the quality and regularity of observations on the fetus and mother in labour, and aids early recognition of problems with either.

The partograph has been in use in a number of countries, and used extensively in a few.⁵⁻¹⁵ It has been found to be inexpensive, effective and pragmatic in a variety of different settings including developed and developing countries. It has shown to be effective in preventing prolonged labour, in reducing operative intervention and in improving the neonatal outcome

The partograph has been in use at Korle-Bu Teaching Hospital for 12 to 15 years and has been

such an effective tool in the management of labour. The World Health Organization (WHO) advocated the partograph, which is currently in use in most developing countries. This partograph like the others is basically a graphical presentation of the events of labour plotted

against time. Measurements that can be recorded graphically give the clearest picture of the first and second stages of labour. With all the main features in a simple uncluttered graph it is easier to make decisions based on all the relevant factors in the labour.

WHO/MCH/88.3

PARTOGRAPH

Name Gravida Para Hospital No.

Date of admission Time of admission Ruptured membranes hrs

180																								
170																								
160																								
150																								
140																								
130																								
120																								
110																								
100																								
FETAL HEART RATE																								
LIQUOR MOULDING																								
9	Latent Phase							Active Phase																
8																								
7																								
6																								
5																								
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2																								
1																								
0																								
TIME	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
5																								
4																								
3																								
2																								
1																								
CONTRACTIONS PER 10 MINS																								
Oxytocin 10U drops/min																								
DRUGS GIVEN AND I.V. FLUIDS																								
180																								
170																								
160																								
150																								
140																								
130																								
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PULSE AND S.R.																								
TEMP °C																								
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URINE ACET																								
VOL																								

Principles of the Partograph

1. The active phase of the labour commences at 3cm dilatation.
2. The latent phase should not last longer than 8 hours
3. During the active phase, the rate of cervical dilatation should not be slower than 1cm/ hour
4. A lag time of 4 hours between the slowing of labour and the need for intervention is unlikely to compromise the fetus or mother and avoids unnecessary intervention.
5. Vaginal examination should be performed as infrequently as is compatible with safe practice
6. Midwives and other personnel may have difficulty in constructing alert and action lines and it is better to use a partograph with preset lines.

Patient information: Fill out name, gravidity, parity, hospital number, date and time of admission and time of ruptured membranes.

Fetal heart rate: Record every half hour.

Amniotic fluid: Record the colour of amniotic fluid at every vaginal examination:

- * I: membranes intact;
- * C: membranes ruptured, clear fluid;
- * M: meconium-stained fluid;
- * B: blood-stained fluid.

Moulding:

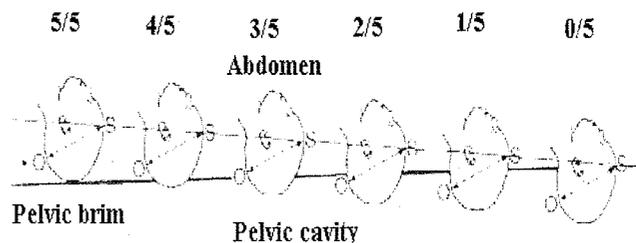
- 1: sutures apposed;
- 2: sutures overlapped but reducible;
- 3: sutures overlapped and not reducible.

Cervical dilatation: Assessed at every vaginal examination and marked with a cross (X). Begin plotting on the partograph at 3cm.

Alert line: A line starts at 3 cm of cervical dilatation to the point of expected full dilatation at the rate of 1 cm per hour.

Action line: Parallel and 4 hours to the right of the alert line.

Descent assessed by abdominal palpation: Refers to the part of the head (divided into 5 parts) palpable above the symphysis pubis; recorded as a circle (O) at every vaginal examination. At 0/5, the sinciput (S) is at the level of the symphysis pubis.



Completely above	Sinciput high, Occiput easily felt	Sinciput easily felt	Sinciput felt, Occiput just felt	Sinciput felt, Occiput Not felt	None of head Palpable
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Time: Record actual time.

Contractions: chart every half hour; palpate the number of contractions in 10 minutes and their duration in seconds.

- * Less than 20 seconds: [Small black square]
- * Between 20 and 40 seconds: [Diagonal lines square]
- * More than 40 seconds: [Large black square]

Oxytocin: Record the amount of oxytocin per volume IV of fluids in drops per minute every 30 minutes when used.

Drugs given: Record any additional drugs given.

Pulse: Record every 30 minutes and mark with a dot (·).

Blood pressure: Record every 4 hours and mark with arrows.

Temperature: Record every 2 hours.

Protein, Acetone and volume: Record every time urine is passed.

The partograph consist of three components. (Figure 1)

- 1 The fetal condition.
- 2 The progress of labour.
- 3 The maternal condition.

T can be used for all labours. High-risk patients are transferred to hospital immediately and in the periphery it is used only for low risk labours where spontaneous vaginal delivery is anticipated.

The partograph does not replace adequate screening of women on arrival in labour to exclude high-risk conditions that need urgent attention or immediate referral. The major role of the partograph is to detect deviations that develop as the labour progresses.

The partograph should only be started if the woman is in labour. She must be contracting enough to start a partograph and in the latent phase contractions must be two or more in 10 minutes each lasting 20 seconds or more. In the active phase the contractions must be one or more in 10 min. each lasting 20secs or more. The partograph must not be used on patients who arrive in second stage or are for immediate caesarean section.

The Fetal Condition

The fetus is monitored closely on the partograph by regular observation of fetal heart rate, liquor and moulding of the fetal skull bones.

The base line fetal heart rate is between 120 and 160 beats per minutes and the darker lines indicate this range. It is recorded every half hour and it is important to listen for one minute with the woman in the left lateral position. If possible under conditions of fetal bradycardia (<120) or tachycardia (>160) the fetal heart must be listened to every 15 minutes for at least 1 minute immediately after a contraction. If the heart rate remains abnormal over three observations, action should be taken unless delivery is imminent. A heart rate of 100 or less indicates very severe distress and action should be taken immediately especially if liquor is meconium stained.

In the column of liquor observation, record:

- **I:** if the membranes are intact
- **C:** if the membranes are ruptured and draining clear liquor
- **M:** if the liquor is meconium stained.
- **A:** is recorded if the membranes are ruptured and the liquor is absent.

These observations are made at each vaginal examination. Thick fresh meconium staining of the liquor may be an indication of fetal distress and necessitate more frequent fetal heart rate monitoring.

Moulding is an important indication of how adequately the pelvis can accommodate the fetal head. Increasing moulding with the head high in the

pelvis is an ominous sign of CPD. Because moulding occurs at the junction of the occipito-parietal bone and later at the parieto-parietal junction it is of value to score the degree of moulding at the junctions separately. Degrees of moulding can be scored by recording the following:

- **Zero:** if the bones are apart
- * **1+:** if they are touching
- * **2+:** if they overlap but can be reduced by digital pressure
- * **3+:** if they overlap and cannot be reduced by pressing.

Moulding may be difficult to assess in the presence of a large caput succedaneum but that in itself should alert the attendant to possible CPD. The presence of caput alone provides less information than the amount of moulding and is more difficult to measure and score. The evidence of a progressively increasing amount of caput is of more significance than a single observation.

The Progress Of Labour

The most important measure of the progress of labour are the rate of dilatation of the cervix and the rate of descent of the fetal presenting part. The cervicograph is divided into a latent phase and an active phase.

The latent phase

The latent phase of labour is from the onset of labour until the cervix reaches 3cm dilatation. If this phase is delayed for longer than 8 hours in the presence of at least 2 contractions in 10 minutes, the labour is likely to be problematic and therefore if the woman is in a health centre she should be transferred to hospital. In the hospital a critical assessment is made to determine subsequent management. The options are:

1. No action (woman not in labour, abandon partograph).
2. Delivery by caesarean section (if fetal distress or factors likely to lead to obstruction or other medical complications necessitating termination of labour).
3. Artificial rupture of membranes and oxytocin (if contraction pattern and or cervical assessment suggests continuing labour).

Further review (in cases of continuing labour):

- a. Continue vaginal examinations once every 4 hours, up to 12 hours.

- b. If not in active phase after 8 hours of oxytocin, delivery by caesarean section is done.
- c. If active phase is reached within or by eight hours but progress in active phase is <1cm per hour, delivery by caesarean section may be considered.
- d. Monitor fetal heart rate every half-hour while on oxytocin.

The active phase

Once the cervical dilatation reaches 3cm the labour enters the active phase. In the primigravidae, the cervix dilates at a rate of at least 1cm/hour in the active phase. **The alert** line drawn from 3 to 10cm represents this rate of dilatation. Therefore if cervical dilatation moves to the right of the alert line, it is slow and an indication of delay in labour. If the woman is in a health centre she should be transferred to the hospital; if in hospital she needs more frequent observation.

The action line is drawn 4 hours to the right of the alert line. Once cervical dilatation reaches the action line there should be critical assessment of the cause of the delay and decision taken to overcome the delay. The WHO model of the partograph is designed for use in all maternity settings.

At the health centre, the central function is to give early warning that labour is likely to be prolonged and that the woman should be transferred to hospital (alert line function). There is 4 hours to the action line and it gives ample time to arrange and transport the patient to a facility where either augmentation with oxytocin or caesarean section can be done.

In the hospital setting, moving to the right of the alert line serves as a warning for extra vigilance but the action line is the critical point at which specific decisions must be made including oxytocin augmentation and caesarean section.

Cervical dilatation should be measured 4 hourly when the patient is in established labour and more frequently when problems require an early decision.

When the action line is reached or crossed then one should consider intravenous infusion, bladder catheterization and or analgesia.

The options are:

1. Delivery (usually by caesarean section), if fetal distress or obstructed labour.
2. Oxytocin augmentation by intravenous infusion if there are no contraindications.
3. Supportive therapy only (if satisfactory progress is now established and dilatation is at 1cm/hour or faster).
4. In cases continuing in labour, vaginal examinations after 3 hours; then in two more hours. Failure to make satisfactory progress, measured as cervical dilatation rate of less than 1cm/hour between any of these examinations means delivery is indicated. Fetal heart rate whiles on oxytocin infusion must be checked at least every half-hour.

Descent of the fetal head

The level of the head is best measured by abdominal palpation as the number of 5th above the pelvic brim. The number of 5th is plotted on the cervico-graph with an O using the lines zero to 5. It is important to observe the rate of the descent of head in relation to any increasing moulding as these two features in combination give an index of the degree of CPD present. It is a more reliable way of gauging the descent than vaginal examination where large caput succedaneum formation often leads the inexperienced to confuse scalp descent with skull decent. Immediately before vaginal examination an abdominal examination must always be done. It should be noted that there is minimal descent in the Latent phase or even in the early active phase.

Descent might be completed in the second stage of labour. Half hourly recordings of the duration and frequency of uterine contraction are plotted immediately below the cervicograph. The frequency is assessed by counting the number of contractions occurring in ten minutes of each half hour period. The duration of contraction is measured in seconds and the numbers of blocks representing frequency are filled in dots if the duration of contraction is less than 20 seconds. The blocks are crosshatched if less than 40 seconds and blacked-out if more than 40 seconds.

Findings	Diagnosis
Cervix not dilated No palpable contractions/infrequent contractions	False labour
Cervix not dilated beyond 3 cm after 8 hours of Regular contractions	Prolonged latent phase
Cervical dilatation to the right of the alert line on the Partograph	Prolonged active phase
* Secondary arrest of cervical dilatation and descent of presenting part in presence of good contractions	* Cephalopelvic disproportion
* Secondary arrest of cervical dilatation and descent of presenting part with large caput, third degree moulding, cervix poorly applied to presenting part, oedematous cervix, ballooning of lower uterine segment, formation of retraction band, maternal and fetal distress.	* Obstruction
* Less than three contractions in 10 minutes, each lasting less than 40 seconds.	* Inadequate uterine activity
* Presentation other than vertex with occiput	* Malpresentation or anterior malposition

The maternal condition

The **maternal pulse** is measured every half hour and the BP and temperature every 4 hours or more frequently if indicated. The woman is encouraged to pass urine every two to four hours and the volume measured. The urine is also tested for protein and acetone.

Drugs and intravenous fluids are charted in the appropriate column just below the area for oxytocin regime.

Discussions And Controversies

There are many different types of partographs in use all over the world but essentially they all enable recordings of the three main determinants in the management of labour, mainly, fetal condition, progress of labour and maternal condition.

Fetal condition can be assessed with different degrees of sophistication, depending on facilities at the various centres. While cardiotocography and fetal scalp blood sampling enable the observation of fetal asphyxia with greater degrees of accuracy, the Pinard stethoscope can be equally effective if used properly. Secondly, midwives and obstetricians might lose their skills if they are over dependant on the few monitors at health centres and subsequently find it difficult to manage larger

number of patients for whom monitors are not available in developing countries.

In the assessment of the progress of labour cervical dilatation and the descent of the presenting part are important and therefore, the first stage of labour needs to be studied probably separately in its latent and active phases.

Should there be a Latent Phase?

In most health centres in the developing world the active phase begins when the cervix is 3cm dilated and fully effaced. In this way progress of labour can be seen in the latent phase when there is cervical effacement at a time that there is minimal progress in cervical dilatation. These are guidelines and flexibility is necessary during the latent phase as some countries do not use the latent phase and rather begin the active phase from 4cm dilatation of the cervix. It is believed that such modifications make reading of the partograph simpler and easier to use.

It is most times difficult to determine the onset of labour in a patient who arrives with uterine contractions, which started in the house. The cervix should be assessed for dilatation and effacement and recorded at zero time on the left-hand side of the labour graph in the latent phase if she is less

than 3cm dilated. The cervix should be reassessed after 4 hours and if there is no change in the cervix but there is uterine contraction 100 mg of pethidine is administered to test whether this is false labour.

The patient should be examined four hours later, that is, eight hours after admission. If contractions cease it is regarded as false labour and the patient is transferred to the antenatal ward to await the onset of true labour or she is discharged home. If however, cervical dilatation reaches 3cm or more her recordings are transferred from the latent phase to the active phase section of the partograph and the cervical dilatation is plotted on the Alert line. If after eight hours in hospital she is still in labour but no change in cervical dilatation then it is regarded as delay in the latent phase.

The woman should be reassessed probably she is not in labour. If there has been a change of cervical effacement or dilatation, and no evidence of CPD the membranes should be ruptured and oxytocin or prostaglandins administered.

Misdiagnosing false labour or prolonged latent phase might lead to unnecessary induction or

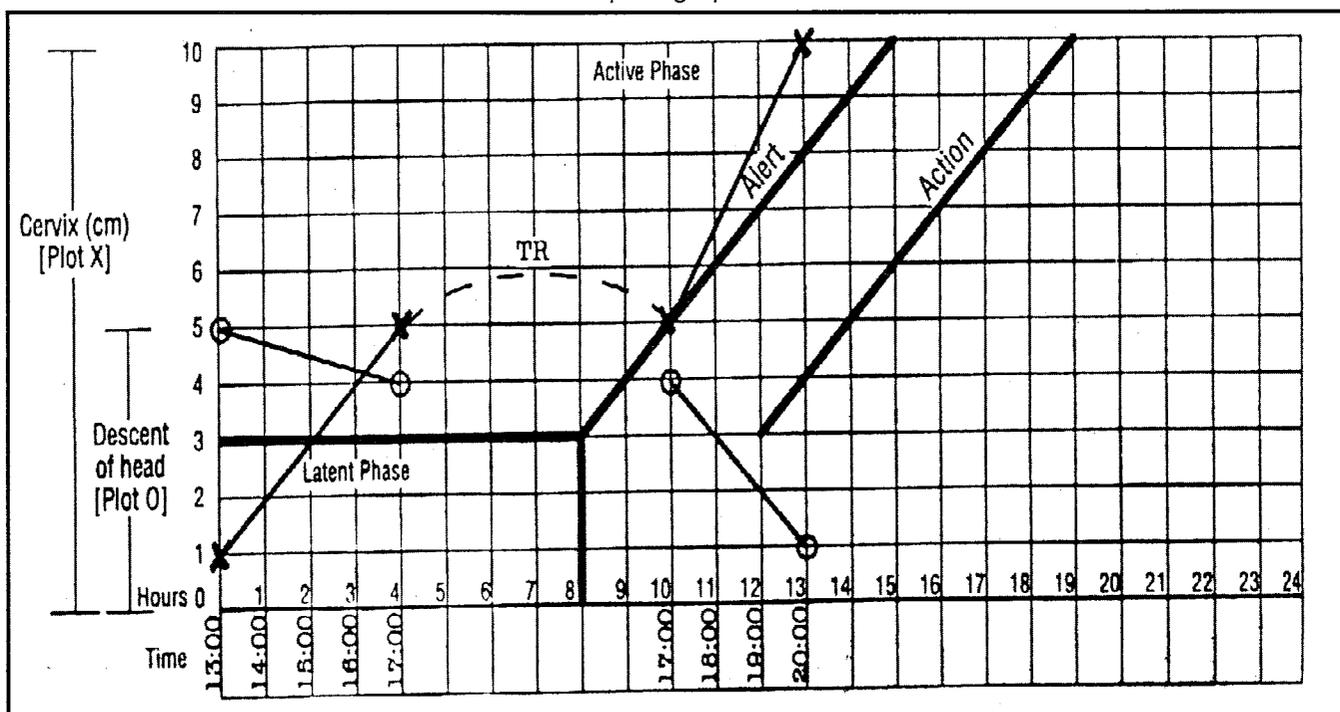
augmentation, which may fail. This may lead to unnecessary caesarean section and amnionitis. Probably this might have contributed to other health centres not using the latent phase but certainly proper management of the latent phase has advantages and will help prevent prolonged labour, fetal and maternal morbidity and mortality.

If cervical dilatation is between the Alert line and the Action line without much progress in labour early augmentation should be considered and artificial rupture of membranes is done with administration of oxytocin. The use of oxytocin has complications and therefore the patient should be assessed properly before setting up oxytocin infusion.

Another controversy is augmentation of breech presentation at the Action line. It is true that there should be adequate contractions for progress of labour but if breech presentation reaches action line the labour should be carefully reassessed to rule out disproportion, big baby, malpresentation and deflexed head. It is always better to consider caesarean section if breech labour reaches the action line

The following are examples showing plottings on the Partograph ⁽²²⁾
Example1: Plotting descent of the fetal head.

On the left side of the graph is the word "descent" with lines going from 5-0. Descent is plotted with an "O" on the partograph.



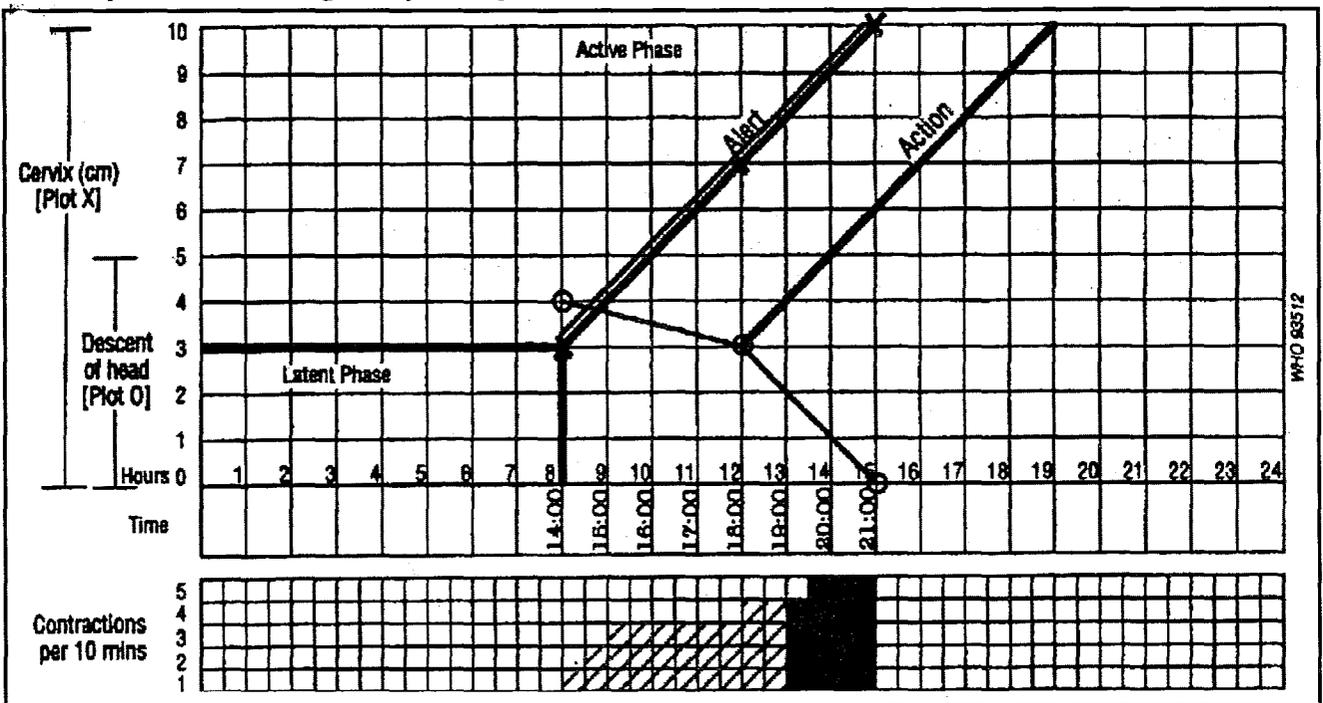
Observations on the above figure.

- * On admission at 13:00, the head was 5/5 above the pelvic brim and the cervix was 1 cm dilated.
- * After 4 hours at 17:00, the head was 4/5 above the brim and the cervix was 5 cm dilated.
- * Labour is now in the active phase. Cervical dilatation is transferred to the alert line;

descent of the head and time are transferred to the vertical line downwards from 6 cm.

- * After 3 hours, the head was only 1/5 above the pelvic brim and the cervix was 10cm dilatation.
- * The length of the first stage of labour observed in the unit was 7 hours.

Example 2: Plotting frequency and duration of contractions



Observations on the above figure.

- * The woman was admitted at 14:00 in the active phase of labour.
- * The cervix was 3 cm dilated; the head was 4/5 above the pelvic brim.
- * Contractions: there was 1 contraction in 10 minutes, lasting 20-40 seconds. After 30 minutes, there were 2 contractions in 10 minutes, each lasting 20-40 seconds.

At 18:00 the cervix was 7 cm dilated, the head 3/5 above the pelvic Brim and there were 4 contractions in 10 minutes, each lasting between 20 and 40 seconds.

At 21:00 the cervix was 10 cm, the head 0/5 above the pelvic brim and there were 5 contractions in 10 minutes, each lasting over 40seconds.

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Obstructed Labour

SA Obed

Introduction

Obstructed labour is almost non-existent in the developed countries where good antenatal care and supervised delivery are the norm. In contrast, antenatal coverage and supervised delivery in developing countries are still far from satisfactory levels. In Ghana for instance antenatal coverage in formal health institutions was about 58.3% and health care providers supervised only 40.8% of all deliveries in 1998⁽¹⁾.

The unsupervised deliveries are often prolonged due to failure to recognise mechanical disorders that are more likely to lead to obstructed labour.

Definition

Obstructed labour occurs when there is lack of progress in spite of good adequate uterine contractions. Progress here refers to cervical dilatation and descent of the presenting part. The prolongation of labour from obstruction could lead to maternal exhaustion and keto-acidosis.

Causes

The commonest cause of obstructed labour is cephalo-pelvic disproportion. General pelvic contraction may be closely related to impairment of growth by ill health or malnutrition during childhood and adolescence. Minor disproportion may be combined with occipito-posterior position when the fetus is in deflexed attitude and can constitute a major mechanical difficulty.

Impacted transverse lie and breech presentation of a large fetus can also lead to obstruction. The after coming head of average sized baby presenting with breech and especially with extended arms may be obstructed.

This often occurs when the body of the baby is pushed out before full dilatation of the cervix.

Other malpresentations like brow, mento-posterioface and compound presentation may cause obstruction. Macrosomic babies especially when the mother is a diabetic and other fetal anomalies like hydrocephalus and fetal ascites may constitute mechanical barriers even in a capacious maternal pelvis.

Soft tissue abnormalities such as fibroids, impacted ovarian tumours and stenosis of the cervix or the vagina could obstruct labour. Rarely locked twins and double monsters cause obstruction.

Clinical Features

Patients who have obstructed labour are usually non-attendants or poor attendants at the antenatal clinic. Quite often they are grandmultiparas with little or no formal education. They have had prolonged labour with most of it being unsupervised or at a spiritual home with an untrained birth-attendant^(2,3,4,5). Some of them also present with retained second twin^(6,6).

Occasionally they are seen after failed attempts at an operative vaginal delivery. Most of these are due to failure to recognise cephalo-pelvic disproportion.

General examination reveals a woman exhausted from lack of sleep and severe unremitting pain. They are usually very anxious and sometimes terrified to an almost uncontrollable state.

There is dehydration from inadequate fluid intake and muscular activity. The skin is hot, dry and inelastic, the tongue dry and furred and the lips are cracked.

The urine is scanty and highly concentrated.

Accumulation of lactic acid from contracting uterine and skeletal muscles leads to metabolic acidosis. Catabolism of fat in the absence of carbohydrates also leads to increased acidosis from the production of ketone bodies.

The patients thus present with rapid pulse, deep and rapid respiration and the breath smells of acetone. They are also pyrexia as a result of dehydration and birth canal infection from multiple unsterile vaginal examinations. Vaginal examination reveals oedema of the vulva and lower vagina with thick offensive vaginal discharge. Bleeding per vaginam usually indicates uterine rupture. The upper part of the vagina is hot and dry. The cervix is puffy and poorly applied to the presenting part.

In cephalic presentation, there is extreme moulding and marked caput succedaneum formation. Abdominal palpation usually shows that most of the fetal head is still above the pelvic brim.

Management

The diagnosis of obstructed labour is clinical. Before embarking on procedures to relieve the mechanical obstruction, the effects of the prolonged labour should be rectified at least partially.

An intravenous line with a wide bore canula is established for fluid and electrolyte imbalance correction. Blood is taken for full blood count, grouping and cross matching against at least 4 units of blood, blood urea, electrolytes and creatinine, and culture and sensitivity if septicaemia is suspected. Rapid infusion with normal saline or Ringers lactate is started.

Endocervical swab for culture and sensitivity is done after which an in-dwelling Foley's urethral catheter is passed for continuous drainage of the urine. The passage of the Foley's catheter may be difficult due to compression of the urethra by the presenting part. Insertion of two fingers on either side of the urethra between the presenting part and the symphysis pubis aids in the passage of the catheter. Naso-gastric tube is passed to empty the stomach as these patients might have eaten or given herbal concoction at home or at a spiritual temple.

Parenteral broad-spectrum antibiotics are administered to control infection. In some communities' herbal concoction, cow dung and mud are applied into the vagina when labour is prolonged. These may contain tetanus spores. Prophylactic anti-tetanus serum should be given in such cases.

Below is a brief outline of the initial steps taken in management of obstructed labour:

1. Intravenous fluids
2. Full blood count
3. Group & X-matching of 4 units blood
4. Blood urea and electrolytes
5. Serum creatinine
6. Endocervical swab for C/S
7. Blood for C/S
8. Catheter
9. NG tube
10. Antibiotics/and antitetanus serum/vaccine

Some of the women who present with obstructed labour are told that the misery they are going through is due to marital infidelity or other antisocial activities. It is important to allay the anxieties of such patients and win their co-operation for an effective management.

Mode Of Delivery

A balanced decision must be taken on the best method of delivering the patient after initiating the resuscitation measures and the patient is in a stable condition. The obstruction must be relieved by operative means. The options are abdominal delivery or extraction of the fetus per vaginam.

Abdominal Delivery

The main indications for abdominal delivery are uterine rupture, live fetus and a surgeon unskilled in operative vaginal techniques. The surgical team should prepare for a possible caesarean hysterectomy when undertaking abdominal delivery in obstructed labour. Occasionally repair of the torn bladder will also be undertaken. It is important to involve a senior obstetrician as early as possible in cases of obstructed labour. The lower uterine segment caesarean delivery is recommended in obstructed labour, as it is less likely to rupture in subsequent pregnancies. Other advantages include less likely intestinal obstruction from adhesions and in infected cases risk of general peritonitis is considerably reduced compared to a classical section. However, classical section may be done when the lower uterine segment is not accessible on account of lowly placed uterine fibroids or scarring from previous operations.

Operative Vaginal Delivery

Operative vaginal deliveries in obstructed labour include forceps delivery, vacuum extraction, symphysiotomy and destructive operations.

Symphysiotomy

Symphysiotomy is performed in cases of cephalopelvic disproportion with a vertex presentation in a live fetus^(7,8). It is performed when the descent of the head is such that at least one third or more of the fetal head has entered the pelvic brim and the cervical dilatation is more than 7cm. It can be done after a failed vacuum extraction in areas without theatre facilities. This procedure is obsolete but may be life saving.

The technique of symphysiotomy

The symphysiotomy instrument tray

The tray itself does not need to be sterilized. Keep the symphysiotomy tray separate from other delivery trays, so that it is always ready in an emergency.

Place on the tray the following:

- small bottle of iodine or spirit

- swabs, sterile gauze in packets
- 10 ml syringe, various needles and a bottle of local anaesthetic
- sterile, wrapped (No4) large-size scalpel handle with size 22 or 24 scalpel blades
- Foley's catheter and Nelathon catheter.
- Other instruments as for 'delivery tray' and vacuum extraction
- Your notes or text on how to perform symphysiotomy.

The woman is placed in lithotomy position and two assistants position a leg each as shown in Figure 1, instead of placing them in stirrups. It is very important that the assistants are very careful not to abduct the thighs. Make sure that they understand that there must always be less than 90 degrees of angle maintained between the thighs. Turn off any oxytocin drip for the time being if labour is being augmented.

Have an assistant shave the pubic hair for 3-4 centimetres square just above the clitoris. Swab the shaved area with iodine or spirit. Inject 5-8 ml of local anaesthetic down to the symphysis. Insert a firm catheter of medium to large size (Nelathon) into the bladder.

Put on new sterile gloves, and put the index and middle fingers of your left hand into the vagina. (The following notes on technique refer to a right-handed operator.) Push the catheter (and thereby the urethra) laterally to the patient's right. Keep the catheter running along the outer border of your index finger. Hook the top of your middle finger over the top of the symphysis to protect the bladder neck (Figure2).

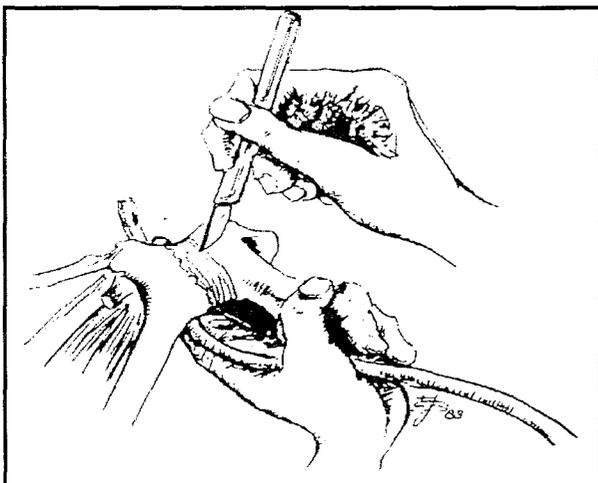


Fig 2. Position the urinary catheter and index finger during the procedure of symphysiotomy

Make a stab incision about 3-4cm above the base of the clitoris down to the symphysis. Cut down through the symphysis until you can feel the scalpel exerting pressure on your middle finger in the vagina. Make sure you cut in the midline. If you are in doubt where the midline is, palpate the midline groove in the symphysis with your right hand before you begin cutting. If you cut into bone, you are not in the midline.

Cut downward, i.e. towards yourself, and then rotate the blade 180 degrees to cut upwards towards the patient's abdomen. When you have completely divided the symphysis you should be able to tap the blade through the length of the divided symphysis on to your finger in the vagina. There is virtually no danger of cutting your finger, as there are still bands of ligaments across the back surface of the symphysis as well as subcutaneous tissues and vaginal skin between the blade and your finger. The common part of the symphysis to miss is the upper anterior border. To make sure that you have divided the whole symphysis, palpate the divided symphysis through the skin incision with a finger of your right hand. If there is still a bridge of undivided symphysis, continue to complete the division before going on.

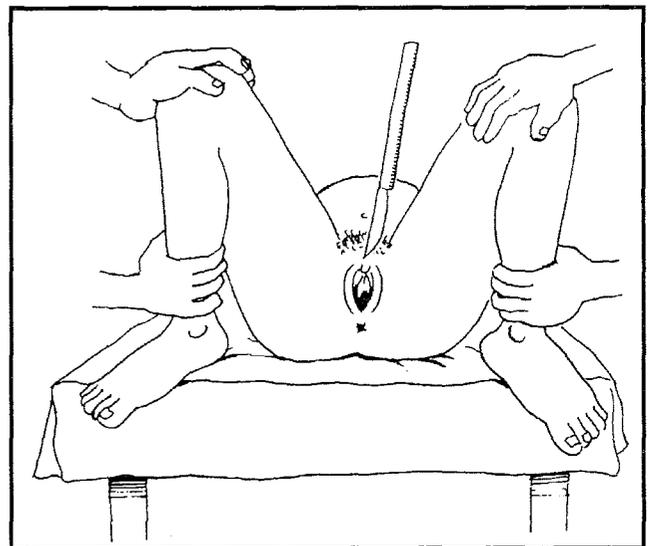


Fig 1. Supporting the patient for a symphysiotomy operation.

If you have not cut an episiotomy do so now, even if the mother has a lax perineum or is a grandmultipara. It is very important that you have as much room posteriorly for the head to be pulled down into.

Ask an assistant to turn the oxytocin drip back on and run it at about 60 drops per minute. Inject oxytocin 5IU into the IV bag if you do not have this in place already.

Place the vacuum extractor cup on the baby's head posterior to the caput if possible, and tell the mother to push hard with the next contraction. Never apply the forceps after symphysiotomy, as you are very likely to cause serious maternal injury. When she does have a contraction, pull downward away from the symphysis: this minimizes the possibility of dislocating the urethra from its supports. You will probably hear a clunk as the posterior symphyseal ligaments tear apart and the head drops in the pelvis.

After the delivery of the baby, tell the two assistants who are holding the thighs to bring the knees together and hold them there whilst you deliver the placenta and suture the episiotomy. This minimizes the blood loss from the symphysiotomy wound site. Suture the symphysis skin wound with one mattress suture going down deep to the level of the symphysis.

It is kind to give your patient a dose of IV pethidine for the suturing: by this time she is in need of rest.

Insert a Foley's catheter into the bladder (to replace the Nelathon one) and allow continuous bladder drainage for 24-48 hours. It is usually safe to remove the catheter as soon as the urine in the bag is clear of blood. Put a loose bandage around the thighs to keep them together for 24 hours. Do not bind them tightly together as this may lead to deep venous thrombosis.

Postoperative orders

- i. Broad-spectrum antibiotics
- ii. Remove the Foley's catheter when the urine is clear, usually in 24-48 hours. The only reason for leaving the catheter in for longer is when you have accidentally cut into the bladder or urethra.
- iii. Remove the bandage around the thighs after 24 hours and tell the patient to move her legs around in the bed, but not to try and get out of bed for another day.
- iv. Allow normal diet, and for 48-72 hours provide pain relief with pethidine or a similar opioid drug.

- v. Prepare a walking frame (any table, cot or other trolley with wheels will do) for the patient to hold on to and walk with when she first gets out of bed. Allow her to get out of bed and try to walk with the walking frame after 2 days. It will be a painful experience and she will feel very insecure and unstable at first. Reassure her.

Common problems associated with the symphysiotomy procedure

1. You cannot get the Nelathon catheter into the bladder. This is because the head is so jammed down in the pelvis that it has squashed the urethra. Try to dislodge the head a little upwards. Try to guide the catheter up the urethra with a finger in the vagina.
2. You hit bone with your scalpel. You are not in the midline. Check for the midline with a finger of your right hand, and begin cutting again in the midline.
3. The symphysis does not separate when you have finished cutting it. You have not completely divided the symphysis. The common place to miss is the top anterior (front) part of the symphysis. Palpate the divided symphysis through the skin incision with a finger of your right hand to check for the part, which you have missed.
4. You still cannot deliver the baby. This should never happen as long as you have followed the rules for trials of vacuum extraction, and you have in fact divided the symphysis. As long as the baby is still alive, there is no alternative but to do a caesarean section.

Problems in the immediate postoperative period

There is no doubt that the immediate postoperative period for symphysiotomy patients is just as uncomfortable as for those who have had caesarean section, if not more so. Always give adequate analgesia after symphysiotomy operations: do not allow these patients to be labelled as another vaginal delivery. They need much more nursing assistance postoperatively than the average caesarean section patient. Because of the instability of the pelvis, symphysiotomy patients also take longer to walk well postoperatively.

1. The patient feels too unstable in the pelvis to walk by the second day after symphysiotomy. She usually just needs reassurance and support from a walking frame; however, a broad belt pulled tight around her hips may provide added support for a couple of days.
2. Postpartum haemorrhage is common after symphysiotomy because most patients are primigravidae who have had a prolonged and possibly augmented first stage, and a prolonged second stage too. Use 0.5mg IV ergometrine after the baby is delivered and put up oxytocin 20IU in the IV bag to run at 30 drops per minute postoperatively. Massage the uterus firm if it is not well contracted.
3. The patient is not able to control her micturition when you take the catheter out, i.e. there is severe stress incontinence. This is more common the longer you leave the catheter in situ. Remove the catheter within 48 hours or earlier if the urine is clear. Teach your patient to do pelvic floor exercises. The problem usually resolves with time and exercises. Give her a course of Cotrimoxazole in case she has developed cystitis.

Problems of a more long-term nature
Maternal death is far less commonly associated with symphysiotomy than with caesarean section in the comparable situation. Maternal morbidity then is the main reason for objection to the use of the operation by some obstetricians. Why is it that doctors are more afraid of morbidity than mortality? When your caesarean patient dies, you grieve about it for a short while, and usually find reasons to justify your actions, and the problems, which led to her demise. However, with serious morbidity, the patient often keeps coming back to clinic, and does not let your conscience so easily forget your involvement in the case.

The only major serious complications of symphysiotomy are pelvic joint pain and ambulatory difficulty, and intractable urinary incontinence. Both these complications should be extremely rare when care is taken with technique and selection of cases.

To prevent excessive separation of the symphysis after it has been divided and undue stress on the sacroiliac joints, the two assistants holding the legs

must be very careful to keep the knees pointing directly at the ceiling, and the angle between the thighs less than 90 degrees.

When urinary incontinence is persistent, examination under anaesthesia should be performed to exclude a fistula. The most likely cause is avascular necrosis from the prolonged obstructed labour rather than the symphysiotomy operation. Precipitate delivery after division of the symphysis can cause detachment of the urethra from the back of the pubic arch, and this can lead to severe stress incontinence or complete urethral incontinence. To prevent this from happening the delivery of the head should be controlled carefully after division of the symphysis. This problem is more likely to occur in multiparas.

On discharge, the patient is advised to refrain from lifting heavy weights and from hard physical work for three months. Hospital delivery is indicated in subsequent pregnancies.

Destructive Operations

Destructive operations are not common nowadays but are indicated when the fetus is confirmed to be dead and the operator is competent to perform the procedure. Ruptured uterus should also be excluded. These procedures are safely performed when cervix is fully dilated although in the hands of an experienced operator it could be done at cervical dilatations of 7 cm or more. General anaesthesia or regional analgesia is required.

Destructive operations usually performed include craniotomy, decapitation, cleidotomy, embryotomy and decompression of a hydrocephalic head.

Craniotomy

Craniotomy is the commonest performed destructive operation. It is indicated in a neglected and obstructed labour with a dead fetus. If the head is more than three-fifths palpable above the pelvic brim or if it is mobile, craniotomy may be difficult and dangerous. In such situations caesarean section should be done. The procedure is done by first emptying the bladder by urethral catheterisation. After exposing the large caput succedaneum of the fetus by parting the labia, an incision of about 3cm is made on the posterior aspect of the scalp with a Mayo Scissors. The index finger of the non-dominant hand is inserted into the incision and moved around so as to identify the suture and trace the fontanelle lying posteriorly.

With the index finger resting on the fontanelle, and the palmar surface facing upwards, the Mayo

Scissors is then carefully directed towards the palm of the non-dominant hand and index finger, and made to rest with the tip of scissors touching the fontanelle. The scissors is steadied and pushed through the fontanelle into the cavity of the skull. The scissors is then opened out in a cruciate direction and the brain is evacuated with the fingers.

A Kocher's forceps is now clamped on each lip of the incised scalp, making sure that a good bite is taken. The patient's legs are then removed from the lithotomy stirrups and placed on two stools. Two layers of bandage are passed through the handles of the two Kocher's forceps, with the other end attached to a chosen weight. The weight is left to hang down gently. The body is delivered by the traction of the weight. In some cases the use of the weights may not be necessary. After delivery, the bladder is drained continuously for 7-10 days.

Decapitation

Decapitation is indicated in obstructed labour when the fetus is dead with a shoulder presentation. Careful vaginal palpation will determine the exact position of the fetal neck, and an assistant may have to pull down an arm to give a better exposure of the fetal neck. The decapitation can be done with Blond-Heidler decapitation saw or in smaller fetuses the neck can be severed with stout scissors. After decapitation, the trunk of the fetus is delivered by traction on the arm. The after-coming head is then rotated in the uterus until the stump of the neck is pointing down the birth canal. The stump is then grasped with a heavy volsellum and the head delivered. In cases of a suspected gross disproportion, a Caesarean section should be performed.

Cleidotomy

Cleidotomy is indicated when the shoulders are impacted in a dead fetus. A pair of stout scissors is used to cut the clavicles, starting with the more accessible one. The reduction in size of the shoulder girdles following division of the clavicles facilitates delivery.

Embryotomy

Embryotomy is rarely done for an abdominal tumour or a very large fetus following craniotomy and cleidotomy. An incision is made into the abdomen or thorax. The viscera are then manually evacuated. It is most easily performed if the breech is presenting.

Decompression

Decompression of hydrocephalic head can be done

by draining the CSP of the fetus before full dilatation of the cervix. This can be achieved vaginally by perforating the skull with a sharp instrument like Simpson's perforator, Drews-Smythe catheter, a spinal needle or a pair of scissors. The hydrocephalic head can also be decompressed trans-abdominally using a spinal needle. The collapsed head is easily delivered vaginally. It can also be done for an after coming hydrocephalic head in breech presentation.

The use of symphysiotomy and destructive operations in obstructed labour is declining in West Africa. Caesarean Sections have become quite safe in the sub-region with decreasing morbidity and mortality. Many Obstetricians are also expressing reservations on the use of symphysiotomy and destructive operations even though studies have shown that long-term disabling sequelae of symphysiotomy^(13,14) and destructive operations⁽⁹⁾ are over emphasised. Medical Officers learn how to perform Caesarean section early in their career and thus may feel more confident in deciding to do a Caesarean operation rather than a destructive operation. Destructive operations should not be disregarded, as post-operative infective morbidity tends to be lower than pertains in a Caesarean section for obstructed labour in the face of overwhelming genital tract infection.⁽¹⁵⁾

Complications Of Obstructed Labour

Uterine Rupture

The likelihood of the uterus to rupture in obstructed labour is influenced by parity. It is more common in multiparous women. It is rare for a nulliparous woman to rupture her uterus in labour. Most of these few nulliparous women who rupture may have a scarred uterus from a silent uterine perforation in a previous surgical termination of pregnancy.

Rupture of the uterus may be spontaneous from over stretched lower segment, or induced by oxytocics or by operations for the relief of obstruction, which further distend the vulnerable lower segment.

The anterior wall is most commonly involved in uterine rupture and the tears are more commonly transverse than longitudinal.^(10,11,12) Occasionally the tears are multiple. Uterine tears may involve the bladder anteriorly or laterally into the broad ligaments with massive bleeding from the uterine arteries.

Lower Genital Tract Injuries and Fistulae

Unrelieved obstructed labour may cause severe damage to the lower genital tract. Necrosis of the anterior vaginal wall may involve the posterior wall of the bladder and urethra leading to a vesico-vaginal fistula. Posteriorly, sloughing may result in a recto-vaginal fistula from compression between the presenting part and the sacrum.

Peripheral Nerve Injuries

Prolonged and difficult labour may injure the nerves supplying the lower limbs. These may be due to prolonged pressure on the lumbo-sacral trunk by the fetal head impacted in the pelvis and stretch injury of the sciatic nerve by prolonged and extreme hyperflexion of the thighs on the trunk. The commonest presentation is foot drop.

The plantar-flexors and gluteal muscles may also be involved. Occasionally sensory loss may accompany the paralysis.

Osteitis Pubis

Infection of the pubic bone after damage to the periosteum and superficial cortex by pressure necrosis, which has extended unusually deep, can cause osteitis pubis.

Pressure Sores

Obstructed labour may lead to a chronically ill patient who may be immobile in bed. If the patient does not have frequent turning and treatment of the pressure points, sores tend to develop. The common sites affected are the sacrum, scapulae, heels and elbows.

Amenorrhoea

Some patients have amenorrhoea after surviving a prolonged and traumatic labour. This is probably due to severe endocrine upset.⁽³⁾ The menstrual cycle returns after a year or more sometimes after the improvement in general health, which follows the repair of associated vesico-vaginal fistulae.

Discussions And Controversies

The efficient management of obstructed labour demands the full range of nursing, medical and surgical skills, which are often lacking in most parts of rural areas in developing countries. Obstructed labour is a preventable calamity in obstetric practice.

Good antenatal care can select at-risk patients for supervised delivery by physicians or elective

Caesarean Section. The use of the Partograph to monitor labour can also help to identify patients whose labour may be difficult and thus facilitate early referral refer to centres with facilities to handle them.

Anticipation During Antenatal Period

Most of the causes of obstructed labour can be detected during the antenatal period. Most short women less than 1.5metres tend to have small pelves.⁽²⁾ Measurement of maternal height at the antenatal clinic is useful to select women at risk of pelvic contraction for clinical pelvimetry. Women who have had prolonged difficult deliveries usually with unfavourable outcomes would require referral for special obstetric supervision.

During the third trimester, examination will reveal fetal macrosomia, abnormal lie, malpresentation and malposition. Ultrasound scan can also detect these abnormalities as well as pelvic tumours.

An elective Caesarean Section can be planned to overcome mechanical impediments to delivery.

Anticipation In Labour

Competent observation in early labour can detect the predisposing factors of obstructed labour even if they were not recognised during the antenatal period. The use of the Partograph has been proven to prevent prolonged labour.⁽³⁾ The graphical notation makes it easy to detect abnormal labour. Referral of patients when the cervical dilatation crosses the alert line in peripheral centres and re-assessment, when the action line is crossed at the referral centres, when the WHO Partograph is used, goes a long way to help take early decisions to prevent obstructed labour.⁽⁴⁾

In multiparous women, the uterus reacts to obstruction by increasingly frequent and more violent contractions of the upper segment, which is subsequently followed, by tonic contraction.

Retraction continues and the lower uterine segment thinned by circumferential dilatation in the first stage of labour, elongates for the first time and thus becomes progressively thinner. Retraction of the lower segment proceeds until it ruptures.

In nulliparous women, further retraction ultimately ceases and labour usually comes to a standstill with the uterus in firm spasm. In the early stages of obstruction palpation of the uterus reveals very strong frequent contractions with little relaxation in between.

The uterus later becomes hard, uniformly convex and tender to pressure, particularly over the distended lower uterine segment. When the uterus ruptures, the patient's condition deteriorates with collapse and severe abdominal pain. In obstructed labour extreme compression between the back of the symphysis pubis and the presenting part prevents the patient from emptying her bladder. Urethral catheterization may be impossible.

The bladder thus forms a tender palpable swelling above the symphysis pubis. Prolonged compression traumatises the bladder leading to bloodstained urine without necessarily uterine rupture. The fetus invariably dies in obstructed labour from asphyxia due to interference with placental exchange by prolonged frequent strong uterine contractions.

In vertex presentation excessive moulding can lead to tentorial tears and intracranial haemorrhage. Asphyxia from umbilical cord prolapse may occur in shoulder presentation. Most of the fetuses, which do not die in-utero usually, succumb in the early neonatal period from a combination of asphyxia, birth trauma and intra-uterine infection.

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Ruptured Uterus

K.A Danso

Introduction

Ruptured uterus is one of the major life threatening complications in obstetrics. It occurs during labour and delivery and to a lesser extent during pregnancy. Irrespective of its time of occurrence, however, ruptured uterus is an obstetric catastrophe [1,2].

Definitions

Ruptured uterus is defined as the total disruption of the wall of the pregnant uterus with or without extrusion of its contents either the baby or the placenta. The rupture is complete when the visceral peritoneum overlying the uterus is also disrupted. It is incomplete when the overlying peritoneum is intact. In such case, there is usually haematoma in the broad ligament without blood or liquor in the peritoneal cavity.

Uterine scar dehiscence is used to describe the herniation of intact amniotic membrane into an existing uterine scar. Uterine scar rupture, however, is the separation of the scar along its entire length often with involvement of the unscarred area and rupture of the amniotic membranes.

Uterine rupture is regarded as traumatic when it is preceded by some form of intervention such as manipulation, obstetric instrumentation, pharmacological stimulation or external physical force. Otherwise, it is referred to as spontaneous.

Aetiology and predisposing factors of ruptured uterus

Uterine rupture complicates labour and delivery in either the scarred, unscarred or congenitally malformed uterus. It may also occur during pregnancy in the scarred or congenitally malformed uterus. In many instances, two or more factors operate to cause the rupture even though each factor on its own can also cause the uterus to rupture.

The causes of uterine rupture are:-

- * Obstructed labour in the parous and, especially, the grand multiparous woman.

The obstruction may be due to fetopelvic disproportion from a malpresentation and malposition and from a contracted pelvis, a big baby or both.

- * High parity. Spontaneous rupture is very rare in first labours without other predisposing factors but increasing parity, particularly grandmultiparity is a risk factor for uterine rupture.
- * Uterine hyperstimulation with oxytocics particularly in the presence of disproportion, malposition or high parity.
- * Previously scarred uterus. This is found in caesarean section, hysterotomy, repaired uterine rupture, extensive myomectomy, other uterine incisions reaching into the endometrium, cornual resection or repair following ectopic pregnancy, uterine perforation, excessive curettage of the uterus and metroplastic procedures.
- * Intrauterine manipulations during labour and delivery such as internal podalic version, breech extraction and manual removal of retained placenta.
- * Operative vaginal delivery such as forceps and embryotomy
- * Congenitally malformed uterus with pregnancy especially in an undeveloped uterine horn.
- * Placental growth abnormalities such as placenta increta or percreta
Trophoblastic diseases such as invasive mole or choriocarcinoma.

Pathological Anatomy

Rupture of a scarred uterus is usually along the axis of the scar. If the rupture occurs in the lower segment, there are distinct edges with minimal bleeding due to limitation by the scar tissue. With the unscarred uterus majority of the ruptures occur in the anterior wall of the uterus transversely or

obliquely in the overstretched lower segment. The edges are uneven, bruised and bleed significantly. Rarely posterior or combined anterior and posterior ruptures are found. There may be a vertical extension of the transverse rupture on one side upwards into the upper segment or downwards into the vagina. Sometimes only vertical ruptures occur laterally or the uterine rupture results from an upward extension into the lower segment of a lateral cervical tear.

Lateral uterine ruptures or the lateral extensions of transverse and oblique ruptures open into the broad ligament and may involve the uterine artery and its branches resulting in massive haemorrhage and maternal shock. It also puts the ureter at risk of damage during surgery. The bladder may become involved in uterine rupture through laceration of its posterior wall. This occurs commonly in situations where the bladder is adherent to the uterus at the site of a scar as in a previous lower segment caesarean section or myomectomy. When uterine rupture is complete, the fetus and placenta may be wholly or partially expelled into the peritoneal cavity, which then becomes soiled with liquor and meconium.

Clinical Epidemiology

Ruptured uterus is relatively uncommon in developed countries although it is still a common obstetric complication in developing countries. The incidence shows appreciable variation in different parts of the same country. Incidence rates of 1 in 6,673 deliveries and 1 in 1,650 deliveries have been reported in the United States of America [3, 4]. An incidence of 1 in 2,500 deliveries was found in Great Britain in 1979 [5]. In parts of Nigeria in 1974, 1 rupture in 112 deliveries with a maternal case fatality of 7.6% was found [6]. An incidence of 1 in 93 deliveries was found in Uganda in 1960 [7], 1 in 225 deliveries in India in 1975 [8] and 1 in 96 deliveries in Turkey in 1990 [9].

Between 1995 and 1999, 47 uterine ruptures were recorded among 46,078 deliveries at the Komfo Anokye Teaching Hospital in Kumasi, Ghana: an incidence of 1 uterine rupture in 980 deliveries [10]. In a review of uterine ruptures which occurred between 1990 and 1992 at a district hospital in the Ashanti Region of Ghana, 33 cases were found among 3,780 deliveries, giving an incidence of 1 in 115 deliveries with a maternal case fatality rate of 24.2% and perinatal mortality of 84.8% [11]

In developing countries ruptured uterus is one of the five major causes of maternal deaths and in some

areas its incidence is rising [12]. Moreover, apart from the direct clinical or immediate causes of uterine rupture, there are many indirect, non-clinical or remote factors which combine to influence its occurrence and cause deterioration in the clinical states of the patients. Such factors include widespread poverty in the rural areas, childhood and adolescent infections and malnutrition which compromise pelvic growth, unhealthy sociocultural practices some of which encourage low status for women, dislike for abdominal delivery, inaccessible or even complete lack of health facilities and unskilled manipulation by untrained or inexperienced attendants during labour. Consequently, in the developing countries, the majority of uterine rupture occur in the unscarred uterus as a sequelae of neglected obstructed and/or prolonged labour resulting from disproportion, malpresentation and malposition in the multipara, especially the grandmultipara [12, 13, 14], and also following misuse of oxytocics [15]. However, with increasing caesarean section rates reaching up to 20% in some centres in developing countries, the contribution from the scarred uterus will rise in these areas in the near future. In the primigravid unscarred uterus spontaneous rupture following prolonged or obstructed labour is uncommon [13, 14]; the usual outcome in such cases is the development of the obstetric fistula [16, 17, 18].

In developed countries, the most common cause of uterine rupture is the scarred uterus, especially a previous caesarean section scar. Scar rupture rates of 0.2- 0.5% have been found with lower segment scars [19, 20] as compared with 3-4% for vertical scars in the upper segment ("classical scars").

Clinical Features

The clinical features and presentation of uterine rupture are quite variable. Spontaneous uterine rupture occurring during pregnancy is insidious and produces vague abdominal discomfort and pain. It is accompanied by abdominal tenderness and, if significant haemoperitoneum has occurred, anaemia, shock, fetal distress or even fetal death. It is therefore useful to have a high index of suspicion for uterine rupture in pregnancy if high risk factors such as classical uterine scar, previous uterine rupture or extensive uterine scars are encountered in the history.

When uterine rupture occurs during labour, the early features may be minimal or even silent with no obvious symptoms although cases seen late may

may become complicated by dehydration, peritonitis and electrolyte imbalance making the clinical diagnosis difficult. In rupture of a scarred uterus, the woman may initially describe a tearing sensation in the lower abdomen. In the unscarred uterus, however, there may be an initial period of calm and temporary pain relief following relentless uterine contractions following obstructed labour. In such a case, a Bandl's retraction ring, formed at the junction between the thickened upper and the thinned lower segments and showing as a transverse depression dividing the abdomen into two, is found. However such classic features as described above are often absent and so for the woman in labour the finding of any of the following features should raise a suspicion of uterine rupture:

- * Cessation of the uterine contractions with or without vaginal bleeding in the multipara.
- * The presence of severe abdominal discomfort, pain or tenderness with distortion of the shape, contour or position of the uterus.
Easily palpable fetal parts with displacement of the presenting part from the pelvis
- * Demonstration of free fluid in the abdomen.
- * Fetal distress or fetal death
- * Vomiting, ileus, shock or maternal distress
- * Blood or meconium staining of the urine

The above symptoms and signs may also characterize abruptio placenta and advanced abdominal pregnancy and should be differentiated from ruptured uterus in labour. The non-dilatation of the cervix in abdominal pregnancy and the tonically contracted uterus in abruptio placenta help in differentiating these other conditions from ruptured uterus.

Uterine rupture occurring in the terminal stages of the second and third stages of labour becomes evident after the delivery of the baby and placenta or even days into the puerperium. In these cases, uncontrollable primary postpartum haemorrhage when atony and lower genital lacerations have been excluded or taken care of, a lateral pelvic swelling and tenderness which may become generalised, sepsis, abdominal distension and retention of urine are the salient clinical features.

Management

It is important to consider measures to prevent uterine rupture or reduce its incidence in the management of this obstetric catastrophe. Efforts must therefore be directed towards improving the overall geographic, socio-economic and living conditions particularly in those areas where incidence rates are high. Trained attendants must attend to all women during pregnancy and labour. Accessibility to health facilities, availability of essential obstetric care at all first referral centres coupled with the promotion of community awareness to utilise these facilities should become health care priorities. Indeed these preventive or public health measures whilst positively resolving the indirect contributory factors of ruptured uterus also enhance obstetric care in general and thus ensure the safety of motherhood.

The principles for the treatment of ruptured uterus once diagnosed are as follows:

- * intensive resuscitation
- * emergency laparotomy
- * broad spectrum antibiotics
- * adequate post operative care
- * A surgical team made up of the obstetrician, anaesthetic personnel and nurse-midwives or health personnel trained to perform these functions is required to effectively treat a case of ruptured uterus.

Uncorrected hypovolaemia from haemorrhage, sepsis or dehydration can cause death of the patient before, during or even after the surgery. Consequently resuscitation must begin with securing venous access with large bore intravenous cannulas to liberally and rapidly give crystalloid infusions and compatible screened blood. The patient should be monitored to ensure adequate fluid and blood replacement. Intravenous broad spectrum antibiotics such as a cephalosporin-metronidazole combination should be started as early as possible. Blood volume expansion may in itself worsen bleeding from vessels damaged in the uterine rupture and so the laparotomy should not be delayed once the patient's condition has improved, however slight, with the resuscitation.

On opening the abdomen a careful inspection is initially carried out of the undisturbed anatomy of

the uterine rupture. If the fetus and placenta have been extruded from the uterus and lying free in the abdomen they are removed, otherwise they are delivered through the rent. If more room is required for the delivery, the tear is extended towards the midline. This avoids potential damage to the ureter and uterine artery if the tear is extended laterally. The uterus is then exteriorised and further assessment is made to identify other possible injuries such as bladder and posterior uterine wall lacerations. Bleeding vessels are located and carefully ligated.

The surgical options at laparotomy are subtotal / total *hysterectomy* or *repair of the uterus*. The latter option is followed by a bilateral tubal ligation if retention of reproductive function is not desired. The procedure chosen should depend on the anatomy of the rupture, the condition of the patient and the skill of the surgeon. It should be the one that is short, safe and yet feasible in that particular case. In general, however, and unless the rupture is very extensive or of the combined anterior and posterior type, repairing the tear using continuous through and through suturing is faster than a hysterectomy. To avoid catching the posterior wall of the bladder into the uterine sutures when repairing a lower uterine rupture or closing the vaginal stump following hysterectomy, the bladder should be adequately reflected downwards. Similarly, to avoid ligating the ureter when dealing with lateral tears the ureter should be identified along its course to the bladder.

The resuscitation is continued well into the post-operative period and until the patient is stable. Close monitoring of the vital signs and urine output is maintained for the next 24 hours whilst analgesics and antibiotics are continued appropriately. With early administration and maintenance of effective antibiotics, the insertion of abdominal drain is not necessary. A nasogastric tube is passed after the operation when peritonitis and ileus are anticipated. If the rupture was preceded by prolonged obstructed labour or the urine was blood stained preoperatively or a bladder repair was carried out, a Foley's catheter is maintained for 10-12 days. As soon as bowel sounds are detected, usually by the 2nd or 3rd day, graded oral feeding is started.

Complications

The immediate maternal complications of ruptured uterus are hypovolaemic shock, infection and death whilst that of the fetus is hypoxia, shock, anaemia and death. Death is always more common for the fetus than the mother in ruptured uterus. Of those

mothers surviving after surgery, further complications are pyrexia, anaemia, genital tract and wound sepsis, obstetric palsy, urogenital fistula, and, in the longterm, intestinal obstruction from adhesions and repeat uterine rupture in a subsequent pregnancy if uterine repair without bilateral tubal ligation had been done.

In the analysis of the 33 cases of ruptured uterus at a district hospital in Ghana, 8 women died: 3 intra-operatively and 5 post-operatively within 72 hours. Only 5 babies were delivered alive: two by mothers who ruptured following failed vacuum and three by mothers with a previous lower segment caesarean section. Furthermore 3 babies out of 6 cases (50%) of previous lower segment caesarean section survived, while only 2 babies out of 27 cases (7.7%) from rupture of the unscarred uterus survived. The post-operative complications among the surviving mothers were: pyrexia 8 patients (32%), anaemia 6 (24%), genital tract sepsis 6 (24%), wound sepsis 4 (16%), vesico-vaginal fistula 3 (12%) and foot drop 3 (12%). Twenty out of the twenty-five surviving women developed more than one complication.

Discussion and Controversies

Like other obstetric complications such as haemorrhage, eclampsia, puerperal sepsis, anaemia and unsafe abortion, ruptured uterus is a major problem for the obstetrician practising in developing countries. It is a 'near-death' event for the childbearing woman and often actually contributes significantly to the over half-a-million maternal deaths occurring annually in these parts of the world [21, 22, 23]. Majority of uterine rupture found in this context is the spontaneous rupture of the unscarred uterus following obstructed labour. Since this is preventable, the continued occurrence of this type of uterine rupture, which in some areas is even assuming increasing incidence, reveals the inadequacy of maternal health care in general and the poor intrapartum care in particular. The ideal approach to uterine rupture therefore lies in its prevention through a positive influence on the non-clinical factors and making available, accessible and affordable essential obstetric services in the community and motivating the people to utilise the services.

The need to eliminate or minimize the non-clinical causes of uterine rupture, which, incidentally, are often beyond the control of the clinician, cannot be overemphasized. The increasing caesarean section rate also occurring in these areas is bound to increase the proportion of scarred uterus in subsequent labours. With the scarred uterus prone

to rupture earlier than the unscarred uterus in the face of obstructed labour, any delay in seeking, reaching or actually obtaining relief of the obstruction arising from persistence of the non-clinical causes will ultimately increase the burden of ruptured uterus. Furthermore, it is important to mention that successful vaginal delivery after caesarean section does not eliminate the risk of uterine rupture in a subsequent labour. In fact the risk increases since the uterus becomes weaker and the babies may get bigger making obstructed labour and uterine rupture more likely.

There exists some variation in the aetiological classification of ruptured uterus by different authors. Some authors categorise ruptured uterus into *rupture of previous uterine scar* and *traumatic rupture* [24], considering rupture from obstructed labour and that from iatrogenic intervention as traumatic rupture. Others classify into *spontaneous* and *traumatic* rupture [25]. It is however obvious that rupture of the scarred uterus for instance can occur unprovoked or following actual injury [2]. A classification into spontaneous and traumatic rupture has therefore been adopted in this chapter. Uterine rupture from prolonged obstructed labour or rupture in the scarred uterus without any intervention is therefore a spontaneous rupture. Traumatic rupture is that preceded by interventions. These interventions may be obstetric manipulations such as internal podalic version, breech extraction and manual removal of retained placenta; obstetric instrumentation such as forceps or vacuum delivery and destructive operations; pharmacological stimulation such as syntocinon augmentation of labour; or external physical force such as in a road traffic accident [26] and the discredited fundal pressure employed by unskilled attendants to assist delivery.

The most controversial issue in ruptured uterus is the choice of surgical procedure: *whether to perform a hysterectomy or repair the uterus*. Hysterectomy removes an organ that is damaged and has the potential of becoming infected. Repairing the uterus saves the uterus. It has been suggested that in communities where women think of losing the uterus as loss of body image or where the subsequent loss of menstruation is a psychological burden, uterine repair should be preferred [3, 27]. However, the decision to take or leave the uterus should depend on the in situ anatomy of the rupture, the condition of the patient and the skill of the surgeon so that a short, safe and feasible procedure is done for each patient. Indeed, as Lawson emphasised, 'repairing the uterus

should not be influenced by a desire to conserve childbearing function or menstruation, nor should hysterectomy be performed because it removes a damaged and infected organ' [13].

Whether, a total or subtotal hysterectomy is performed when hysterectomy is indicated is another controversial issue. In line with its preference in elective gynaecological surgery, total hysterectomy is performed for most patients in some centres [28, 29], although subtotal hysterectomy for most patients has also been found in other centres [9, 27, 30]. Clearly, the experience of the surgeon and the condition of the patient should dictate the choice for total or subtotal hysterectomy. In the already bloody operative field of ruptured uterus, a total hysterectomy may lengthen operation time, induce more bleeding and pose the risk of damage to the lower ends of the ureters and the bladder. A subtotal hysterectomy should therefore not be looked down upon if it is found to be easier or the better choice in a particular case.

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Obstetric Anaesthesia And Analgesia.

Frank Boni

Introduction

Obstetric Anaesthesia has seen great strides over the past few decades in some parts of the world. Mothers in developed countries do not dread the pain of labour as before, and the maternal mortality and morbidity have dropped dramatically from the 1940's in these countries to very low levels now. ⁽¹⁾ It is however generally accepted in these countries that more can still be done to eliminate preventable causes of death and morbidity. Maternal mortality is not the only area where great improvements have occurred.

- * Regional anesthesia, especially epidural anesthesia and analgesia is now routine in many hospitals in developed countries.
- * The welfare of the fetus is now considered vital in all aspects of Obstetric Anaesthetic practice and is only compromised in extreme situations when the life of the mother is at stake.
- * The concept of pain free delivery is a right and not a privilege.
- * Because of public interest and medico-legal reasons, most hospitals have dedicated and experienced Anaesthetists who cover maternity units 24 hours a day. There are however some very good reasons for paying special attention to Obstetric Anaesthesia in all parts of the world.
- * We are dealing with 2 individuals a mother and a baby at the same time.
- * There are many anatomical and physiological changes in pregnancy which make anesthesia hazardous.
- * The fetus changing from intra-uterine to extra-uterine life is very vulnerable.
- * The sheer numbers and ratios of Obstetric patients in any community pose logistic problems

- * There are many life threatening medical conditions peculiar to pregnancy e.g. eclampsia, and HELLP syndrome
- * The general population do not take perinatal mortality as lightly as other surgical deaths. ⁽²⁾

What Is The Situation In Africa?

Despite our high birth rates and the poor condition of the patients when they come to hospital, there are very poorly developed Anaesthetic services to cope adequately with the situation. ⁽⁴⁾

Some Causes Of High Morbidity And Mortality In Africa

- * Poor general state of patients when they present to hospital.
- * Poor theatre and postoperative conditions.
- * Lack of some basic equipment and drugs.
- * Difficulties with staff development and retention.
- * Poorly developed critical care and resuscitation facilities for high-risk patients. Inadequate support services like Blood banks, X-rays.
- * Poorly developed and ineffective administrative structures.
- * High prevalence of infections and infestations.
- * Inadequate financial support.
- * Unhelpful local beliefs and practices.
- * Low public awareness of good Anaesthetic and Surgical practice.
- * Lack of medico-legal considerations in our practices and many others.

* Many of these are not directly under the control of the Obstetrician and the Anaesthetists but they can greatly influence them in various ways.

Pain Relief In Labour.- Definitions

Pain

Pain is an unpleasant sensory or emotional experience due to actual tissue damage or potential damage or described in terms of such damage. It is always subjective and is influenced by one's experiences, culture and other factors, of which some cannot easily be explained.

Analgesia

Analgesia is the absence of pain.

Labour

Labour can be described as regular, forceful, painful uterine contractions, which bring about cervical dilatation and expulsion of the fetus. Labour is usually very painful. This type of pain is intermittent and gradually increases in both duration and intensity until it reaches its peak in the second stage of labour. It continues into the third stage when the placenta is being expelled and some intermittent after-pains may occur due to uterine contractions long after the main bulk of the placenta has been expelled.

Other pains like headaches, muscle and joint pains can also occur during labour.

When we talk about pain relief in labour we normally refer to relief of pain due to uterine contractions but the Anaesthetist or Anaesthesia may be required for any other Obstetric, Surgical or Medical conditions occurring during labour.

Is Pain Relief In Labour Really Necessary?

Pain during labour has been recorded since the beginning of mankind. It is mentioned in the book of Genesis as the punishment for Eve's disobedience to God. Many consider it as a normal thing, which should not be treated. There are no doubts about some detrimental effects of pain relief especially when not done properly. There are however many advantages which by far out-weigh the negative ones. One just has to go into a labour ward to hear women screaming at the top of their voices, rolling and some falling off beds, many cursing and

refusing to lay their eyes on their husbands. There is a lot of stress on doctors and nurses. Pain relief can therefore be justified purely on humanitarian and social grounds.

Severe pain causes a lot of neuro-endocrine effects, which can have profound effects on all major organs and systems in the body. These are even worse in high-risk patients like those with Pregnancy Induced Hypertension and Heart disease.

Maternal exhaustion due to pain can lead to inability to cooperate during the second stage and this leads to increased rate of complications to the mother and the baby. Some women can actually die of pain for example from severe Vaso-vagal attacks

Hyperventilation and excessive breath holding especially if it is a Valsava type can cause electrolyte problems and cardiac arrhythmias including arrest in high-risk patients. It is therefore very difficult not to recommend pain relief to any patient in labour unless there are serious contra-indications.

Many women especially multi-parous women may not need any form of pain relief during delivery. Since pain relief with drugs invariably has some unwanted effects on the mother or the baby, any mother who can do without drugs should not be forced to accept them.

Types Of Pain Relief In Labour

1. Pharmacological. Oral and parenteral drugs.

Oral, IM, IV analgesics and sedatives can be given to relieve pain in labour. Nearly all sedatives and analgesics have been tried in labour but few have been found suitable. They not only affect the level of consciousness of the mother but cross the placental barrier very easily to depress the baby leading to increased mortality and morbidity of the newborn. Oral analgesics and sedatives are not very suitable for pain relief during labour. Intra-muscular barbiturates and other sedatives are best avoided except if there is a specific indication. IM diazepam is painful, an irritant and its effect unpredictable. It is not recommended and if it is indicated in cases like eclampsia, it has to be given intravenously. Barbiturates have also been used but can cause even more problems. Phenergan is sometimes used for its anti-emetic properties with

narcotics but sometimes it is also given alone for its sedative effects. It is usually given with a narcotic like Pethidine but when given alone, it can be anti-analgesic. Intra-muscular narcotics like Pethidine are still the most widely used forms of pain relief in Africa. Its advantages include ease of administration, some sedation and euphoria for the agitated mother. Its onset of action may be 15-30 minutes depending on the circulation with a peak effect at about 30 minutes to 1 hour. Duration of action is up to about 3 hours. It however causes drowsiness, severe nausea and vomiting, blurred vision but more importantly severe respiratory depression in both the mother and the baby. It should not be given at about 1-2 hours before the estimated time of delivery. Appropriate anti-emetics should be prescribed and a narcotic antagonist Naloxone together with equipment for intubation and resuscitation of the newborn should be available when narcotics are prescribed in labour. Recommended regime for Pethidine is a dose 1-1.5mg/kg or 75-100 mg for the adult in the first few hours of labour. Phenergan 25mg IM or Metoclopramide 10mg IM can be given for nausea and vomiting. Respiratory and central nervous system monitoring is vital. Avoid Pethidine if there is any history of adverse effects or if the patients are on drugs like MAOI. Fear of addiction to narcotics is not a problem in the use of Pethidine in acute situations like labour and it should not be withheld on those grounds. Partial agonist/antagonists like Pentazocine are not very popular because of the possible dysphoria and other side effects. Intravenous narcotics and sedatives should not be used except under close supervision of a Doctor capable of intubating and ventilating the patient and adequately trained nurses to monitor and look after such patients.

2. Inhalational Anesthetic agents

Low doses of most Inhalational anesthetic agents have analgesic properties. This fits into the first stage of Anaesthesia as classified by Geudel⁽⁵⁾. Thus ether, methoxyflorane, tri-chloroethylene have all been used with special vaporizers to deliver low doses for pain relief in labour. Most of these have become obsolete and Nitrous oxide is now the main inhalational agent used in most labour wards. It is used in a 50% mixture with Oxygen as Entonox. Nitrous oxide is a potent analgesic and a weak anesthetic. 25 % inspired Nitrous oxide concentration can be as effective as 10mg of morphine. It is delivered to the patient via a 2-stage pressure reducing valve facemask or mouthpiece, which the patient breathes through at the onset of painful uterine contractions. It is very quick acting

and quite effective at all stages of labour. It is however expensive and not readily available in developing countries. It can also cause patients to be light headed and confused especially if they are hyperventilating. Pollution of the labour wards with low doses of Nitrous Oxide (N₂O) can also be a problem to staff working in labour wards. Nitrous oxide inhalation to supplement IM Pethidine in the first and second stages of labour is the most widely used form of analgesia in labour now in areas where epidurals are not possible.

3. Epidural and Spinal Analgesia

Introduction of a local anesthetic drug with or without narcotics into the Epidural space in a controlled manner by a competent Anesthetist is now the best form of pain relief in labour. It can provide excellent analgesia for all stages of labour without the associated problems of IM narcotics like nausea, vomiting, altered sensorium and respiratory depression of both mother and baby. The associated drop in blood pressure can be very beneficial if it is not severe. Epidural or spinal anesthesia can also be used for surgical procedures to completely replace General Anaesthesia.

Epidural anesthesia is not widely used in Africa now because it requires specially trained Anaesthetists and Midwives to give round the clock service, which is not available in most African hospitals. It is however a technique which should be available in all Teaching Hospitals for Primi-gravidae who are distressed, for patients with PIH without altered blood clotting, for twin deliveries where the 2nd twin can be handled better and quicker and many more. It should also be available for those who can afford to pay for having a pain free labour but have no specific medical indications.

When conducted properly, the complications are very few and minor. The most common complication is hypotension and so precautions are taken against it. Drugs and equipment for monitoring, and resuscitation and medical and nursing staff that can detect problems with both the mother and the baby should be available 24 hour before epidurals are given. Dural punctures and spinal headaches, which are feared by all mothers, can also be reduced to a minimum if training and supervision of newly qualified and inexperienced Anaesthetists are done before they start performing this procedure.

Non-Pharmacological methods of pain relief.

Psychological methods including Hypnosis and relaxation exercises fall in this group. There are many women who may not want any drugs during labour. Hypnosis in medical terms means sleep but the term is also used to describe 'programmed automated responses to a particular situation.' The Obstetric patient can be hypnotized by a trained person or can be taught to hypnotize herself so as to feel less pain in labour. It is however time consuming and requires trained tutors to work for hours in the antenatal period. It does not have the same effect in all patients and is not widely practiced. It could however be useful in certain group of patients if a qualified person organizes it.

Transcutaneous Electrical Nerve Stimulation (tens)

Trans-cutaneous Electrical Nerve Stimulation is a simple way of relieving pain without drugs. Low currents are passed into electrodes and skin pads attached to the back of the patient. This seems to alter pain modulation. It is however not very helpful in the late first stage and the second stage of labour when most of the pains are sacral in origin and also due to dilatation and stretching of the perineal structures.

Acupuncture

Acupuncture is not used much in labour because it is not very useful in this type of severe acute intermittent pain. Professor Dundee during a visit to China was surprised to find that the Chinese regarded painful labour as normal and were not too keen on treatment. Acupressure at a particular point on the wrist may however be used to reduce the incidence of nausea and vomiting due to narcotics given during labour.^(7,8)

Breathing And Relaxation Exercises

These have been used by traditional and hospital midwives for ages and is very useful if they are taught well and by experts in the antenatal period. Husbands could help and should form part of the classes in the antenatal period. Some centers supplement these by allowing mothers to deliver in special positions and sometimes under water.

Advantages Of Pain Relief In Labour

Mother

A calm, cooperative mother with no psychological injuries. The incidence of hypertension and other stress responses are minimized. The mother is not unduly exhausted before the second stage of labour when she needs to use all her reserve energies to push the baby out.

Baby

The baby will come out with better Apgar scores provided the timing and technique of pain relief is sound.

Staff

The working environment for all staff in the labour ward is better with good pain relief and everyone can give off his or her best. Hypertensives, twin pregnancies and many other problems in labour are more easily managed.

Relatives

Husband and relatives can see and communicate with a smiling happy mother.

Problems And Complications With Pain Relief In Labour

Mother

Nausea and vomiting, altered level of consciousness, Respiratory depression, blurred vision when narcotics, are used. Dizziness and feeling of suffocation is sometimes felt in addition to the above when inhalation agents are used. Theatre pollution is another problem with inhalation agents.

Baby

If narcotics are not timed properly, the baby may be born with low Apgar scores and may need to be intubated and ventilated due to respiratory depression.

Staff

Extra effort and work may be needed to prevent and treat complications due to pain relief. More staff at all levels will be needed.

Costs

There are increased costs involved in providing good and safe pain relief in labour. Extra equipment and facilities will be needed and all

these cost money, which may not be readily available in the developing countries.

Anaesthesia For Obstetrics

It is generally accepted that absolute pain relief is required for surgical procedures. Even when God, again in the book of Genesis was going to remove the rib of Adam to make Eve, Adam was put into a deep sleep. This was the first recorded Anaesthesia given. What is most surprising is that it was not until after 1846 that the practice of Anaesthesia became a reality. It then quickly became a necessary requirement for all types of surgery including Obstetrics. There are however many who take Obstetric Anaesthesia as simply "putting a mother to sleep". This is a gross understatement. One has to abolish pain and induce sleep but at the same time keep the mother and baby alive and well. Anaesthesia however comes at a price and it is important to know the possible complications and how to avoid or treat them when they occur.

Types Of Anaesthesia And Their Indications

General Anaesthesia

This involves rendering the patient reversibly and safely unconscious using Intravenous and/or Inhalation drugs and sometimes Intra-muscular drugs like Ketamine. General Anaesthesia aims at providing ideal conditions for operations. These can be summarized as

1. Providing adequate analgesia or suppression of reflexes. Narcotics and Nitrous Oxide (N_2O) can achieve this.
2. Providing unconsciousness- Intravenous and Inhalation Anesthetic drugs can be used.
3. Providing adequate muscular relaxation- Deep anesthesia or Neuromuscular blocking agents is used.
4. Ensuring adequate gaseous exchange by delivering adequate amount of Oxide (O_2) and eliminating Carbon Dioxide (CO_2) whiles at the same time preventing and treating any unwanted effects due to the General Anaesthesia.
5. There are many general Anesthetic techniques and drugs available as at now.

The technique used will depend on:

1. The patient,
2. The operation
3. The facilities available
4. The Surgeon
5. The Anaesthetist.

There are now some minimum standards set up in many developed countries for various procedures based on mortality and morbidity data. Unfortunately this is not happening in the less developed countries. Limited financial resources, misplaced priorities and the lack of awareness and litigation from the general public are some of the reasons for the present situation. These are bound to change. It is therefore imperative that all doctors should be familiar with proven safe methods of general Anaesthesia instead of continuing to justify the use of outdated methods without creditable independent review of their outcome.

Anaesthetic Considerations In The Pregnant Patient

- * The effects of anaesthesia on both the mother and the baby
- * Cardio-Pulmonary resuscitation of the pregnant woman and neonate are difficult.
- * The ever-present danger of the Mendelson's syndrome.
- * The transfer of premedicant and anaesthetic drugs across the placental barrier.
- * The supine hypotensive syndrome and the effect of maternal positioning.
- * Operations are usually emergencies and one may not be adequately prepared for problems.
- * Bleeding, coagulation and transfusion related problems cause very high mortality.
- * The public does not easily accept obstetric complications and mortalities.
- * Obstetrics forms the bulk of the hospital workload especially in developing countries.

Other Drugs Used Peri-operatively

Muscle relaxants: Depolarisers like Suxamethonium and non-depolarisers like Vecuronium Cholinergics like atropine and Anti-cholinergics like neostigmine for reversal of muscle relaxants. **Sympathomimetics:** e.g. Ephedrine for hypotension in regional anaesthesia

COMMON DRUGS USED IN OBSTETRIC ANAESTHESIA.

Drugs	Types	Indications	Precautions
Thiopentone	Intravenous	Induction of anaesthesia	Hypotension, allergies
Propofol	Intravenous	Induction of anaesthesia	Hypotension, sepsis
Fentanyl	Narcotic	GA and epidural	Respiratory depression etc
Halothane	Volatile agent	Maintain anaesthesia	uterine atony, hepatitis
Isoflorane	Volatile agent	Maintain anaesthesia	uterine atony in high doses
Ether	Volatile agent	Maintain anaesthesia	Fires and explosions
Nitrous Oxide	Gas	Analgesia, GA	
Ketamine	IM/IV agent	Induction/ maintenance	Hypertensives, CNS diseases
Oxygen	Gas	All anaesthesia	Neonates
Lignocaine	LA agent	Regional/ local anaesthesia	Allergies, IV injection Overdose
Bupivacaine	LA agent	Regional/local anaesthetic	IV injection, overdose

Resuscitation drugs: Adrenaline, NaHCO₃, Naloxone, Calcium chloride, anti-emetics etc.

Medical Drugs: used for all medical emergencies e.g. Insulin

Antacids: e.g. H₂ blockers like Ranitidine and soluble oral anti acids like sodium citrate,

Equipment Required For General Anaesthesia

- * Anaesthetic machine and gas supplies. Draw-over apparatus like the EMO, or the Continuous Flow or plenum systems like the Boyle's Machine are used to deliver Anaesthetic gases and volatile inhalation agents to the patient.
- * Monitoring equipment for continuous Blood pressure, pulse, O₂ saturation, Cardiac activity (ECG) etc.
- * Resuscitation equipment and trolley for both the mother and the newborn
- * Intubation equipment and trolley for both the Mother and the newborn
- * Suitable operating tables and trolleys

2. Regional Anaesthesia

(a) Spinal Anaesthesia

This is a reversible blockade of nerve conduction after introduction of a local Anaesthetic drug at the appropriate level into the subarachnoid space usually below the second Lumbar vertebra. It provides Anaesthesia i.e. absence of all sensation,

in the region below the block without unconsciousness.

It has many advantages in the Obstetric patient.

Local Anaesthetic drugs used for spinal anaesthesia in Africa are: Lignocaine (Lidocaine) and Bupivacaine.

(B) Epidural Anaesthesia

This produces the same conditions as described above with the introduction of a local Anaesthetic drug into the epidural space. Drugs used for epidural and their doses

Para-cervical block and Nerve blocks like Pudendal blocks.

Local Infiltration techniques

Sometimes aqueous solutions of local Anaesthetic drugs are injected directly near nerves where they reversibly block nerve conduction at the terminal nerve endings. They may look deceptively simple to do. One has to know the safe methods of injecting the drugs, the safe doses and when to use various drugs and additives like adrenaline. In some cases like Caesarean Sections, large areas will have to be infiltrated and it may be difficult to use effective concentrations and volumes without exceeding the maximum doses.

Range Of Operations In Typical Tropical Obstetric Practice

- * Caesarean Section
- * Examinations under Anaesthesia (EUA)
- * Manual removal of placenta (MRP)
- * Cervical cerclage (Shirodkar)

- * Laparoscopy
- * Ruptured Uterus repair or removal
- * Ruptured Ectopic gestation
- * Suture of Episiotomies and tears
- * Evacuation of Uterus (EOU)
- * Sterilization

General Anaesthesia For Caesarean Section

Pre-operative management

It is necessary for both the Obstetrician and the Anesthetist to see all patients and to confer and decide on the management of the patient before, during and after the operation. This is mandatory in both elective and emergency Caesarean sections. A thorough history and examination of the mother together with details about the progress of labour, the indications for the section and the state of the fetus are all crucial in planning and preparing the patient. Past and present history of medications, medical diseases, allergies, past operations and also social and economic status of the patient should all be taken.

Examination of the mother should include assessment for intubation and all the vital systems of the body. The appropriate investigations should be done. Hemoglobin levels, Sickle cell status, Urine for sugar and proteins as well as Blood grouping and cross matching are vital. Blood coagulation studies are vital in severe PIH and eclampsia as well as long standing Intra-uterine deaths to rule out DIC and treatment initiated before operations. If lack of time and money make this impossible to get, a rough estimate of the clotting time can be of value. A blood sample is taken into a syringe or test-tube and this is rotated slowly until clots form. The time taken for this to happen should not normally be more than 8-12 minutes. X-rays are best avoided but may be unavoidable. Emergency Caesarean sections should however not be unduly delayed for the sake of non-essential investigations. Routine anxiolytics and sedative pre-medication are best avoided before C/S because of the effects on the baby after delivery. Anti-acid treatment is however essential. In elective sections, H2 blockers like Ranitidine 150mg given the night before operations and repeated 2 hours before the operation will reduce the volume of acid and increase the pH. of gastric contents. About 30 minutes before the operation, a non-particulate anti-acid like sodium citrate is given to neutralize the acid in the stomach. 30 mls of 0.3M sodium Citrate is used in many centers. A large bore IV cannula, 16G or bigger is inserted into a suitable vein. Ordinary hypodermic needles and butterfly-

needles are best avoided. IV fluids preferably Ringers lactate or Normal saline should be set up prior to the induction of Anaesthesia. In many cases in our environment, prophylactic antibiotics will be required. It is better to give the antibiotics some minutes before the induction of Anaesthesia so that any problems like allergies will not be confused with those caused by anesthetic drugs. The peak effect will also occur during the actual operation. The patient should be positioned supine with a lateral tilt on the bed to prevent the Supine Hypotensive Syndrome. This can be achieved with a pillow or wedge if the operating table cannot be tilted laterally.

Straps are applied to the patient to prevent her from falling off the bed. All drugs and equipment including resuscitation equipment for both mother and the newborn are checked and made ready before Anesthesia is induced. The patient is then hooked on to monitors for continuous monitoring of at least the Blood pressure, pulse and respiration. A pulse oximeter is now considered essential and may be more useful than an Electrocardiograph machine (ECG). The Surgeons, Scrub nurses, Pediatrician or Midwife receiving the baby should all be ready. The fetal heart rate is checked for the last time before induction of Anaesthesia to rule out intra-uterine death.

Intra-operative management

Pre-oxygenation with at least 6L/minute of 100% oxygen for 5-10 minutes is vital for the mother and baby. A trained assistant helps the Anesthetist to induce Anaesthesia by identifying the cricoid cartilage and applying pressure before muscle relaxants are given and the trachea intubated.

A sleep dose of Thiopentone 3-5 mg/kg of 2.5% solution is administered into a large vein. Very reduced doses for the sake of the baby should be avoided because they cause more harm than good and the normal doses given do not unduly affect the baby because of the distribution pattern of the drug. In the absence of a high blood pressure, 0.25-0.5mg of Ketamine a minute or two before the Thiopentone can greatly smoothen the induction and decrease the incidence of awareness.

As soon as the patient starts becoming unresponsive, the assistant applies cricoid pressure before the Suxamethonium is given for rapid muscle relaxation and intubation is carried out. There should be no attempt to ventilate the patient by squeezing the re-breathing bag until the endotracheal tube is in place, the cuff inflated and its position confirmed by auscultation of both lungs.

This technique of induction is frequently referred to as "Crash Induction" or "Rapid sequence induction" and the application of the cricoid pressure is sometimes called the Sellick' manoeuvre. Anaesthesia is maintained by 50% Nitrous oxide in 50% Oxygen, not more than 0.5% Halothane, and Intermittent Positive Pressure Ventilation after paralysing with reduced doses of non-depolarizing muscle relaxants like Tubocurarine or Vecuronium.

Spontaneous respiration using Ether and a Draw-Over machine like the EMO can be used if there are no contra-indications. Its use without Oxygen supplementation should be discouraged because of the high incidence of Sickle cell disease in our environment and the high Oxygen requirements of both the mother and the baby. Extubation is done preferably with the mother awake, breathing very well and on the side.

Techniques To Avoid And Precautions.

Some District hospitals use intermittent injections of Thiopentone as the sole Anaesthetic for Caesarean Sections and other operations. This should never be done. Thiopentone is an anti-analgesic and repeated or large doses can be fatal to both the mother and the baby or cause gross morbidity. Large doses of Ketamine as the sole agent may not be very good especially for the baby but is superior to Thiopentone in cases of high-risk patients like shocked patients. One should note that Ketamine does not provide adequate protection for the airway as was previously thought. It should also be avoided in hypertensive, epileptic and psychiatric patients. If there is inability to intubate the mother for a Caesarean section, facemasks or Laryngeal Mask airways (LMA) can be used only if adequate precautions are taken to avoid gaseous insufflation of the stomach, vomiting or regurgitation. A failed intubation drill suitable for the Obstetric unit is vital.

Post-operative management

This should be done in a properly set area with staff and equipment to monitor all vital signs so as to detect and manage all the expected and unexpected complications that may arise in the post-operative period. A recovery ward or post-anaesthetic room with well trained staff is recommended at all times until there is full recovery from anaesthesia. Clear instructions on Pain relief, positioning of patient, drugs to be administered, and other instructions to patients and staff should be written down in the patients notes. Facilities to manage airway and breathing problems, nausea vomiting and, cardiovascular emergencies should

be readily available. The area should be close to the theatre so that surgeons can be readily contacted and patient can be sent back to theatre with minimum delay when necessary.

General Anaesthesia For Other Obstetric Procedures

1. Retained placenta:

Uterine relaxation may be required with Halothane and steps should be taken to treat severe haemorrhage. Arrangements should be made for blood transfusion.

2. Examination under Anaesthesia (EUA) and Evacuation of Uterus (Eou):

Although these are minor surgical procedures, endotracheal intubation may be required for even the shortest of procedures and full preparations should be made for major operations if these are being done before or after delivery because of the danger of Mendelson's syndrome.

3. Ruptured Ectopic gestation:

Hypovolaemic shock with its associated problems has to be dealt with.

Ketamine is the induction agent of choice. Auto-transfusion of blood in the peritoneal cavity may be necessary and facilities should be available for these especially in the remote areas with limited blood transfusion services⁽⁶⁾

The techniques and drugs employed for these may differ from what has been described for Caesarean section and one is advised to know the details of the expected problems in all these procedures before giving the simplest types of Anaesthesia for these procedures.

Regional Anaesthesia For Caesarean Section

Spinal Anaesthesia is the main technique used although Epidural can be used in larger obstetric centres. Regional Anaesthesia has a lot of advantages over General Anaesthesia when practiced properly.

The mother is awake and retains nearly all her protective reflexes. Aspiration of gastric contents and airway obstruction are thus avoided. The

mother can cooperate with the Surgeon and Anaesthetist and even warn or give vital information during operations.

Cadio-respiratory depression is avoided.

The husband can be present during the operation and bonding of the parents with the baby can start immediately after delivery of the baby rather than hours or days after general anaesthesia.

The baby comes out without asphyxia or the depressant effects of anaesthetic drugs.

Post operatively, there is pain relief without the nausea and vomiting and drowsiness of the narcotics.

One has to perform the technique properly and take certain precautions to give safe and pleasant regional anaesthesia.

Pre-operative management

The patient's consent is required and the procedure explained to the patient. The amount of detail depends on the circumstances. But the patient should have a clear idea of what is going to be done before her cooperation can be guaranteed. All other contra-indications should be ruled out⁽⁷⁾. At least infections at the puncture site, gross abnormalities of the spine, allergies to local anesthetics, bleeding abnormalities, hypovolaemia and septicaemia should be ruled out. The notion that a spinal can be done in poorly prepared patients with full stomachs should be discouraged.

Premedication with sedatives and CNS depressants should be avoided since they will affect the baby. Anti-acids and anti-emetics can however be given. The patients should be well hydrated with IV fluids before regional anaesthesia but not necessarily over-hydrated. One may give prophylactic vasopressors like Ephedrine before the procedure.

Intra-operative management

One can consult the standard textbooks about the details of the procedure but there are some points worth stressing.

One should have all facilities to give General Anaesthesia should there be any problem with the regional technique. The procedure should be done under fully sterile conditions with gown, masks, drapes etc. There should be continuous monitoring of the patient's blood pressure, pulse, oxygenation and level of consciousness. Oxygen, Intravenous fluids and other drugs are given as required. The patient should not be left on her own at anytime.

If a Doctor has to perform a spinal and operate on the patient as is done in some remote areas with no

Anaesthetist, he should make sure the patient's vital signs are stable, and the level of block is checked.

Finally there should be a dedicated nurse who should stay and monitor the patient throughout the operation. Steps are taken at all times to prevent, detect and treat hypotension, which is the commonest and most troublesome complication of spinal Anaesthesia. This is due to the inevitable vasodilatation as a result of sympathetic blockade. Readers are again advised to consult the standard textbooks for complications associated with Spinal and Epidural Anaesthesia.

Post-operative management

Monitoring of patients should continue into the post-operative period until the return of sensation and motor function. If a small bore needle like pencil tip size G26 is used and multiple dural punctures are avoided, it is not necessary to keep patients lying flat for 24 hours to avoid post spinal headaches.

Spinal Anaesthesia is especially suitable for sickle cell disease and those with difficult airways even in some emergency situations.

Other Local Anaesthetic Techniques

These include Nerve blocks like the Pudendal block, Para-cervical block or infiltration blocks for episiotomies. One should know the safe techniques for injections and also the safe doses and possible side effects and complications of local anaesthetics before using them.

Special Problems In Obstetric Anaesthesia

APH/PPH management will require good blood transfusion services. PIH/Eclampsia will require a High Dependency Unit (HDU). Sickle Cell Disease management may have to involve Haematologists or Physicians. Infections and Infestations, including HIV, Hepatitis, Malaria are common.

Disseminated Intra-vascular Coagulation (DIC), Acute Respiratory Distress syndrome (ARDS), THE Syndrome of Haemolysis, Elevated Liver Enzymes and Low Platelets (HELLP), Amniotic Fluid Embolism and many others exist and can be diagnosed and managed if properly trained staff are available and have the facilities provided for them.

Resuscitation And Critical Care

Definitions

Resuscitation is derived from the Latin word "Rhesus" which means, "to bring back to life". It therefore implies any action, which is aimed at correcting or preventing a life-threatening situation.

More specifically, some may refer to cardiopulmonary resuscitation (CPR) as reversal of clinical death before permanent death or biological death sets in by restoring cardiopulmonary function as quickly as possible. Cardiac arrest can be defined as the sudden cessation of the heart in a person who is not expected to die. It can be caused by many factors. Respiratory arrest is defined as cessation of respiratory function. Cardiac arrest will invariably be followed by respiratory arrest and vice versa because once there is lack of blood flow or oxygen supply to the brain, these sensitive structures are quickly affected. The aim of cardiopulmonary resuscitation or more correctly cardiopulmonary cerebral resuscitation is to ensure adequate blood flow and oxygen to the brain and other vital organs in the absence of normal cardiac and pulmonary function.

Basic Life Support (BLS)

Defined as resuscitation using virtually no equipment. All you need are your mouth, hands, brains and the necessary training.

Advanced Cardiac Life Support

requires, one to have additional training and equipment together with drugs listed below.

The ultimate place to treat the critically ill patient be it Obstetric or otherwise is in an Intensive care unit where staff and equipment for handling multi-organ failure are found. Thus every busy hospital handling acute cases with an annual turnover of a few thousand cases should have an intensive care unit or at least a High Dependency Unit. In an Obstetric Unit we need facilities to resuscitate both the mother and the newborn.

Equipment

Every Obstetric centre should have the basic equipment for CPR within easy reach. One should know and have all the Basic Resuscitation equipment and Drugs in all Obstetric units.

Personnel

Everyone working in a hospital should be able to perform Basic life support. In fact, even laymen should be taught BLS so that they can handle situations in the house or in public until help arrives. Advanced cardiac life support should be taught to all nurses, doctors, and labouratory X-ray and other ancillary workers. Cardiac arrest teams with cardiac arrest codes and drills are vital to ensure prompt and precise response to calls at all times. The procedures will depend on the layout and facilities like communication and staff available. It is thus

important for every hospital to devise its own procedure for handling cardiac arrests and other forms of resuscitation.

CPR in The Obstetric Patient

CPR is particularly difficult to perform on the Obstetric patient and requires additional expertise and equipment. Even in the best of hands the outcome after cardiac arrests are not very good. One should therefore aim at correcting any derangement before they lead to cardiac or respiratory arrest. Reasons are anatomical, physiological, supine hypotensive syndrome, individuals, etc

Some Common causes of cardiopulmonary arrest

1. Shock (Hypovolaemic, Cardiogenic, Anaphylactic, Neurogenic)
2. Medical Emergencies in Obstetric practice, e.g. neuromuscular disease
3. Cardiopulmonary arrest can occur during and after Anaesthesia because of many reason like drugs used peri-operatively or and the numerous possible causes of hypoxia and hypercapnoea.
4. Certain simple diagnostic and therapeutic Obstetric procedures like External version of a fetus can also result in cardiopulmonary arrest.

The Newborn

Post delivery care

The newborn must have the nose and pharynx sucked immediately after the delivery of the head before they take their first breath and aspirate liquor and blood especially during Caesarean sections. They are then quickly dried to prevent hypothermia and handed over to a trained midwife or Doctor as the case may be for resuscitation.

The Apgar score at one and five minutes is used to assess the degree of asphyxia, the need of active resuscitation and other treatment.

Condition of Baby

Score at 1 minute and 5 minutes Action

0-4 poor Start active CPR to treat possible causes.

5-7 fair Suction, oxygen and tactile stimulation may be enough

APGAR SCORE			
	2	1	0
RESPIRATION	regular	gasping	absent
HEART RATE	>100	<100	absent
COLOUR	pink	blue extremities	blue or white
TONE	good flexion	average	limp
REFLEX IRRITABILITY	good cry	some response	absent

8-10 good, specific treatment not usually required

Neonatal emergencies are more likely to require resuscitation i.e.

- * All Caesarean Section babies
- * Babies of mothers with PIH and Eclampsics
- * Babies of Mothers with medical conditions like cardiac, pulmonary, endocrine and renal diseases
- * Premature babies
- * Babies with congenital abnormalities
- * Multiple pregnancies¹³²
- * All babies born after difficult labour and delivery, including unusual presentations
- * Babies of mothers who have had sedation including narcotics in the last few hours before delivery.

Things to avoid during resuscitation of the newborn.

1. Too much suction with high negative pressures with stiff catheters.
2. Too vigorous stimulation like spanking or holding upside down and shaking vigorously.
3. Avoid niketamide and respiratory stimulants. (Do give opioid antagonists if required).
4. Avoid artificially ventilating without first sucking the airway and maintaining a patent airway.

Some General Measures To Improve Obstetric Anaesthetic Services

1. There should be a global approach to the planning and running of Anaesthetic services. Obstetricians, Nurses, Anaesthetists, Laboratory and other health workers as well as politicians, opinion leaders in the community and the general public should be involved.
2. Good theatre design with provision for a post Anaesthetic recovery ward as well as an intensive care or a high Dependency Unit with qualified Anaesthetists available at all times
3. Properly trained staff, doctors, nurses, technicians, at all levels with good working conditions are required to produce good results. Refresher courses and regular assessments are necessary.
4. Well laid down guidelines for antenatal care, peri-operative care, pain relief and other aspects of obstetric care should be adopted and adequately funded.
5. Africans should no longer accept sub-standard conditions with the excuse that we are poor. We should not only improve the present conditions but aim for the maximum conditions possible. A healthy population will in the long run improve the economy of any nation and therefore we should set our priorities right when sharing the national cake.
6. External funding and help are necessary in any developing country but there should not be over-reliance on them. Local personnel do best at

planning and implementation of policies. They should therefore be properly trained and supported. The human resource pool in Africa can be enormous if priorities are set right.

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Fetal Distress

E Y Kwawukume and O Fakeye

Introduction

Fetal distress is one of the acute obstetric emergencies seen in our labour wards and expedient action must be taken to deliver the baby as quickly as possible to prevent fetal death.

Fetal distress can present in subtle forms, which are difficult to diagnose and as a result, they are overlooked. Some of these are a rising base-line fetal heart rate, changes in the grade of meconium in the amniotic fluid, the absence of accelerations, the presence of unusual variable decelerations or a combination of the above. In the presence of late decelerations or increased fetal heart rate above 160 beats per minute the diagnosis of fetal distress becomes obvious.



It is not easy to determine which fetus is likely to develop reduction in oxygenation and therefore distress in labour. Several indicators have been looked at and some of them are:

- * Fetal heart rate
- * Meconium in the amniotic fluid
- * Fetal acid-base balance
- * Fetal movement
- * Fetal respiration



In our sub-region the two most important factors are variability of the fetal heart rate pattern and meconium stained liquor. Either of these in isolation might not predict fetal distress. However, they are important signals for the Obstetrician and the Midwife.

Many times when the diagnosis of fetal distress is suspected and the quickest and the most efficient methods are resorted to, to deliver the woman, the baby comes out as a healthy screaming baby without any sign of distress. It is important to critically evaluate the diagnosis and take the appropriate interventional steps to salvage the baby.

Meconium and fetal distress

Meconium is the intestinal contents of the fetus in utero consisting predominantly of water (75%), proteins (mucous glycoproteins and plasma proteins), mucopolysaccharides and digestive

enzymes. Meconium is present in the fetal gut from the 10th week of gestation ⁽¹⁾ and the incidence of meconium in the amniotic fluid increases with gestational age. ^(2,3,4) It is not common to find meconium-stained liquor before 34 weeks' gestation. The incidence reaches about 30% at 40 weeks' gestation and 50% at 42 weeks' gestation. ^(2,3,4)

Meconium aspiration is dangerous and can lead to neonatal mortality. Meconium aspiration refers to the presence of meconium below the vocal cords and this happens in about 21% to 58% of births in the presence of meconium stained liquor. ⁽⁵⁾

Hypoxia causes the aspiration of meconium and might also play a part in the release of meconium into the amniotic fluid. It is known that babies who aspirate meconium but are not hypoxaemic during labour are unlikely to suffer any serious consequences, and 90% will be asymptomatic ^(5,6,7) unlike babies who are already hypoxaemic and inhale meconium during labour. The mechanism within the fetus that causes relaxation of the sphincters for the passage of meconium is not known but it has been demonstrated that the passage of meconium is a very late phenomenon after hypoxia has occurred ⁽⁸⁾

It is very common to observe the presence of meconium in the absence of hypoxia. While hypoxia may play a part in the release of meconium into the amniotic fluid, meconium is not always present with hypoxia but hypoxia is well established as causing meconium aspiration.

Meconium staining of the liquor could be mild, moderate or severe. Mild or grade 1 looks like yellowish-green staining and severe or grade 3 is thick green meconium of pea soup consistency. The presence of meconium alone is not specific for diagnosis of fetal distress unless there is an associated fetal heart abnormality. Old meconium is not diagnostic of distress but fresh meconium might be. Similarly, mild meconium stained-liquor is often found in post-term pregnancies after artificial rupture of the membranes. Its presence in isolation is not associated with fetal hypoxia or distress. Fetal heart rate above 160 or below 120 beats per minute results in increased perinatal mortality and

morbidity.⁽⁵⁾ Also fetuses with thick meconium have increased neonatal morbidity when compared with fetuses without meconium staining. A combination of meconium stained liquor and fetal heart rate abnormalities are associated with greater mortality than when taken alone in isolation.^(3,5,6)

It should also be noted that fetuses that start labour with clear amniotic fluid and develop meconium stained liquor during labour constitute a high-risk group of fetuses. They might not be coping well with the stress of labour and closer surveillance should be instituted. In these cases, the presence of meconium might be a significant sign of possible fetal distress.

Fetal Heart Rate and Fetal Distress.

Cardiotocography (GTG) remains the cornerstone of making diagnosis in fetal distress but it is not available at many hospitals in our sub-region. In the absence of CTG, which utilises continuous electronic fetal monitoring (EFM), the intermittent auscultation used in many centres in Africa gives equally good results. Studies have demonstrated that the long-term results are as good as those seen in pregnancies managed by electronic FHR monitoring, with fewer caesarean sections.^(9,10)

Good results can be obtained with intermittent auscultation if auscultation is performed diligently at regular intervals using the fetal recordings on the partograph. The most useful time for auscultation are during contraction and at the end of every contraction in order to detect the simultaneous deceleration in the FHR and to assess the recovery of lag time of the fetal heart rate. The lag time lengthens with increasing fetal compromise.^(11,12)

Cardiotocography and Fetal Distress

Although the cardiotocograph has a high sensitivity the specificity is low.⁽⁷⁾ The CTG might alert the obstetrician about fetal well-being and therefore prevent intrapartum fetal mortality but not the long-term neonatal morbidity such as cerebral palsy or Erb's palsy.

Base line fetal heart rate

This is the rate recorded between contractions and the normal range is 120-160 bpm. Isolated cases of fetal heart rate beyond the normal range are occasionally seen and can be regarded as normal if they are transient. Fetal heart rate persisting above 160 bpm or below 120 bpm are classified as tachycardia and bradycardia respectively and they are more associated with hypoxia than is the normal heart rate

Causes of fetal tachycardia

- * Maternal fever such as malaria infection or chorioamnionitis
- * Fetal infection
- * Maternal dehydration and anxiety
- * Maternal thyrotoxicosis
- * Fetal anaemia
- * Fetal tachyarrhythmias
- * β -sympathomimetic drugs given to the mother
- * Parasympatholytic agents such as atropine given to the mother

Causes of bradycardia

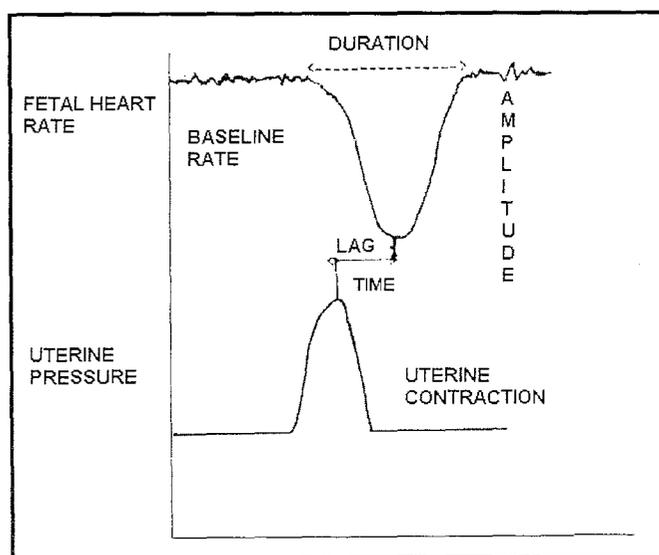
- * Maternal medications such as sedatives or opiates
- * β -blockers such as propranolol
- * Congenital heart block
- * Uterine hypertonicity

Heart Rate Variability

This is the interval between R waves of continuous ECG complexes or short-term variability on a beat-to-beat basis, usually ranging from 3 to 5 bpm around an imaginary average heart rate. Long-term variability occurs as well with amplitude of 10 to 20 bpm.

It is the most reliable indicator of fetal wellbeing and represents the interplay between the cardioinhibitory and cardioaccelerator centres in the fetal brain stem.

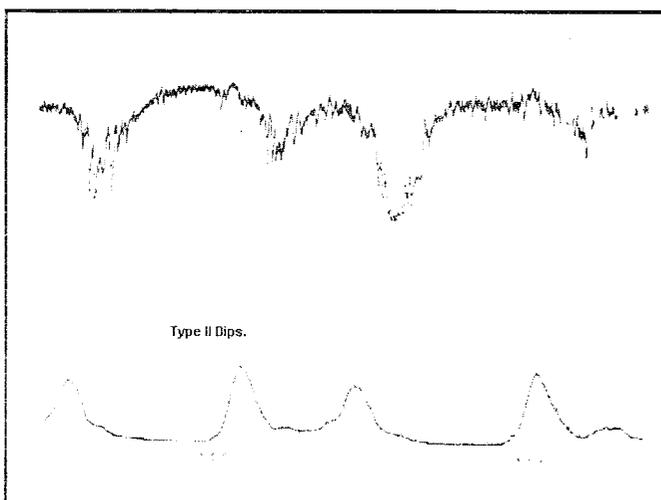
Lag time is the time interval between the peak of uterine contraction and the lowest point of the fetal heart deceleration



Early Deceleration

This begins with onset of uterine contraction and returns to the baseline levels just as the contraction is finished. It is called early deceleration because it starts early in the contraction. It is usually present at the later stage of the first stage of labour and is due to head compression as the fetal head moves down the birth canal.

The mechanism is a reflex slowing mediated by the vagal nerve with release of acetylcholine at the sinoatrial node. The degree of this action depends on the pressure applied to the fetal vertex. Early decelerations are rarely sinister. But if the decelerations are below 100 bpm and become increasingly frequent and present in the early stages of labour, the fetus should be closely monitored.



CTG tracing showing late decelerations

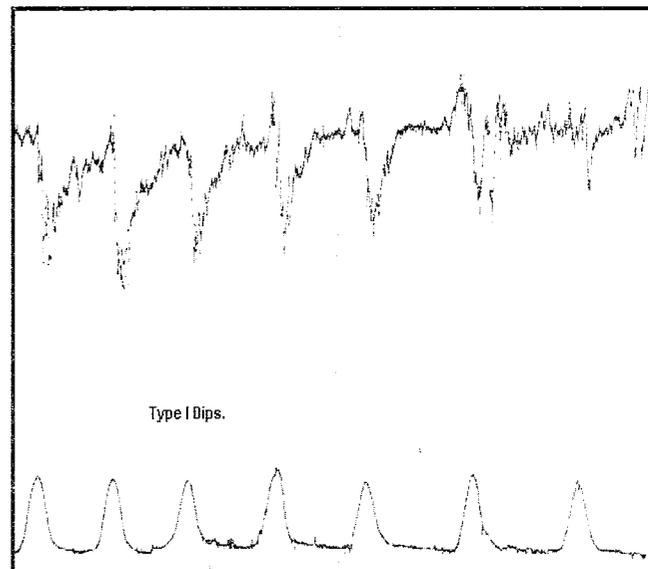
Late Deceleration

Late deceleration starts after onset of uterine contractions and does not return to the baseline rate until after the contraction is over. It is almost always indicative of uteroplacental insufficiency and decreased intervillous exchange between the mother and the fetus resulting in fetal hypoxia. During contraction the intervillous blood flow is limited and fetal oxygenation is impaired resulting in late timing of the deceleration. Late deceleration is associated with intrauterine growth restriction (IUGR), oligohydramnios, placental abruption, excessive uterine activity maternal hypotension, anaemia or ketoacidosis

Variable deceleration

There is no constant relationship to the onset of a uterine contraction. They vary in shape and sometimes in timing with respect to each other.

They are the commonest type of deceleration seen during labour and are generally caused by partial or complete cord compression. It is a reflex-mediated change in fetal heart rate mediated by the vagal nerve¹³. It is corrected by changing maternal position to relieve cord compression



CTG tracing of variable deceleration

Mixed Pattern

This is a combination of two decelerative fetal heart rate patterns such as a mixed variable-late deceleration. The potential fetal compromise associated with the fetal heart rate pattern should be judged by the worst component of the pattern, that is, late deceleration, which should be corrected

Acceleration

There is increase in fetal heart rate associated with uterine contraction or fetal movement and this is the main reassuring characteristic of the 'reactive' CTG. It shows that the fetus is adequately oxygenated

Diagnosis of fetal distress

1. Fetal heart rate of less than 120 bpm or more than 160 bpm. Note that the normal heart rate is between 120 and 160 bpm
2. Irregular FH with loss of beat to beat variation
3. Late decelerations
4. Variable decelerations
5. Meconium stained liquor.
6. Fetal scalp blood pH, if available, might be useful to confirm fetal asphyxia.

Management

1. Stop oxytocin infusion if on.
2. Take blood for grouping and cross-matching
3. Start IV normal saline infusion.
4. Turn patient to left lateral position to prevent vena caval compression (orthostatic hypotension)
5. Administer oxygen (6L per minute) by facemask or intranasally.
6. Perform vaginal examination to exclude cord prolapse or cord presentation and note cervical dilatation.
7. If the fetus recovers from the above resuscitative measures, continue labour with the partograph and aim at vaginal delivery.
8. Monitor labour more closely.
9. If fetal distress persists prepare the patient for instrumental delivery if the cervix is fully dilated or caesarean section.
10. Inform Paediatric Resident and theatre staff.

Why is Normal saline infusion preferred to dextrose in fetal distress? In fetal distress there is decrease in fetal oxygenation and by biochemical principles there is diminished production of ATP by the Krebs cycle. The main pathway then would be the Embden-Meyerhoff glycolytic pathway. In this regard, lactate is produced from glucose phosphorylation and the H⁺ ion from lactic acid would lead to a further drop in pH of the blood of the fetus if dextrose were administered in the absence of oxygen thus worsening the fetal acidosis caused by the hypoxia. Normal saline also increases the blood volume and therefore the blood flow to the placenta leading to increase in placental perfusion.

The Role Of Fetal Scalp Blood Sampling

The severity of fetal heart rate pattern has a linear correlation with the degree of acidosis present in the fetus. During labour there may be fetal acidosis probably from impaired feto-maternal exchange at the intervillous space. A scalp pH measurement is therefore necessary to support or refute a CTG diagnosis of abnormal fetal heart rate before intervention. The main role of the fetal scalp blood sampling is:¹⁴



1. To provide a measure of the extent of acidosis (pH) and the degree of fetal reserve present (base excess) where fetal compromise is confirmed

2. To provide reassurance to the obstetric team and the labouring woman that time is available to continue with expectant management if fetal compromise is ruled out

Blood Gases

Fetal acidosis during labour may result from impaired feto-maternal exchange. If not detected early, sufficient hypoxia and acidosis may develop and cause brain damage or death from asphyxia.

Collection of fetal blood for pH and respiratory gases evaluation at the appropriate time may alert the obstetrician to impending fetal distress and therefore correction of the underlying problem or delivery by the safest route for the mother and the fetus.

A pH value greater than 7.25 is reassuring for the fetus during labour. The pH range of 7.20-7.25 is regarded as borderline and should be repeated in 15 to 30 minutes. A pH below 7.20 should be regarded as acidosis and a value of 7.00 is critical and potentially damaging to the fetus.

Fetal Pulse Oximetry

Pulse oximetry measures the colour of pulsatile blood and calculates the oxygen saturation ("SaO₂") of the blood. It is non-invasive. In the past fetal pulse oximetry was not reliable and not widely used because in the presence of caput, meconium-stained liquor and fetal hair, oximetry monitoring from the fetal scalp was difficult. But at present, improved technology, better sensors and new wavelength systems have been developed. These are placed against the fetal cheek or temple to reduce the physiological and environmental errors.¹⁵ Multicentre studies in Europe have now shown a good correlation with FHR patterns and fetal scalp blood values. The normal SaO₂ is 30 to 70 %. Values below 30 % are suggestive of fetal hypoxia and distress. Fetal pulse oximetry combined with CTG may in future be reassuring and reduce the need for fetal blood sampling.

Other newer methods being developed to measure the oxygen status of the fetus are Continuous Tissue pH Measurements, Fetal ECG and Near Infra-Red Spectroscopy. The Near Infra-red spectroscopy works on the principle that biological tissue contains chromophores whose light-absorbing properties vary with levels of oxygenation. This instrument has the potential to provide continuous data on brain oxygenation during labour and will help to reduce fetal distress and unnecessary fetal loss.

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Shoulder Dystocia

Sarah Baffoe and G A Arthur

Introduction

Shoulder dystocia is a serious obstetric emergency. Although uncommon, its occurrence is unpredictable, and is associated with a high risk of fetal injury and death, if not properly managed.

Definition

The definition of shoulder dystocia is problematic. A functional definition describes shoulder dystocia as any documented difficulty in extracting the shoulders of the fetus after delivery of the head.

This definition obviously has a highly **subjective** component, leading to overestimation of the incidence of shoulder dystocia from centre to centre. It may also lead to a higher incidence of reported cases and lower complication rates.

For a more objective comparison, researchers prefer a more 'technical definition', which considers true shoulder dystocia to have happened when there is documentation of specific release manoeuvres to deliver the shoulders, in addition to an episiotomy and gentle downward traction⁽¹⁾. Significant fetal trauma only occurs in true shoulder dystocia. This definition however, may overlook milder forms of this disorder, underestimate incidence rates, and skew complication rates upward.

Incidence

Incidence figures vary over a wide range depending on the definition used. Although the actual incidence is unknown, quoted figures range between 0.15 to 1.7 percent of vaginal deliveries^(2,3).

The incidence is increasing due to increasing incidence of maternal obesity, increasing birth weight, improved perinatal care, better reporting and documentation. Increased perinatal morbidity and mortality are also reported^(2,3).

Aetiology

Shoulder dystocia occurs when the anterior shoulder fails to pass below the maternal symphysis pubis after delivery of the head. This leads to impaction of the shoulder girdle within the maternal pelvis.

It occurs due to:

- (i) Disproportion between the fetal chest and maternal pelvis
- (ii) Malrotation of the fetal shoulder at the pelvic inlet
- (iii) A maternal pelvis flattened anteroposteriorly or a platypelloid pelvis

Risk Factors

Although single risk factors are of little value in predicting shoulder dystocia, a combination of risk factors may increase the risk. Traditionally, risk factors have included macrosomia, maternal diabetes mellitus, maternal obesity, prolonged pregnancy, dysfunctional labour, previous history of shoulder dystocia, prolonged second stage and mid-pelvic instrumental delivery.

Macrosomia

Excessive birth weight is associated with increased risk of various morbidity including shoulder dystocia. Macrosomia has been defined as a birth weight greater than 4,000g. Others use 4500g for the definition of macrosomia. Whilst the term large-for-gestational-age is defined in relation to maturity, macrosomia refers to an absolute birth weight regardless of gestational age or other demographic variables⁽⁴⁾.

Shoulder dystocia is 11 times more common in babies weighing more than 4,000g and 22 times more common in babies weighing 4,500g than the average⁽⁵⁾. Although shoulder dystocia is traditionally associated with macrosomia, macrosomia as a single risk factor might not be predictive of this complication. Many labours with babies weighing 4.0 to 4.5kg do not suffer shoulder dystocia, and many cases of dystocia occur with infants weighing less than 4.0kg. In every case risk factors should be assessed and selected cases should be offered caesarean section especially patients who are diabetic.

Maternal Diabetes Mellitus

Maternal diabetes mellitus increases the risk of fetal macrosomia and shoulder dystocia. Infants of

diabetic mothers may not only be macrosomic, but may also have greater shoulder-to-head and chest-to-head ratios than do fetuses of similar weights born to non-diabetic mothers⁽⁴⁾. Thus the macrosomia of Insulin Dependent Diabetes (IDM) is characterised by selective organomegaly. At fetal weights greater than 3500g, the risk of shoulder dystocia is more than doubled in diabetic women (6). Both poorly controlled gestational diabetes mellitus and pregestational diabetes mellitus without vasculopathy may give rise to macrosomia and infants with truncal obesity. Intrauterine growth retardation and low birth weight infants are more likely to result in the presence of vasculopathy.

Prolonged Pregnancy

Although prolonged pregnancy has been associated with uteroplacental insufficiency and growth failure, a number of infants, particularly male fetuses, will continue growing and exceed 4,000g. In contrast to the classical withered, fragile, dysmature infant, over 25% of postdate infants weigh over 4,000g⁽⁷⁾.

Indeed it is practical to suspect macrosomia, and anticipate shoulder dystocia, in all prolonged gestations except in the obvious case of clinically demonstrable intrauterine growth retardation. Freeman et al⁽⁸⁾ documented that shoulder dystocia occurred in two percent of patients with prolonged pregnancies.

Maternal Obesity

Macrosomia is common among obese women. Johnson et al reported that shoulder dystocia occurred in 5.1% of obese labouring women weighing more than 250 pounds compared with 0.6% for a control group who weighed less than 200 pounds (9).

The macrosomic infant of obese, non-diabetic woman is symmetric, characterised by increase in both chest circumference and head circumference.

Miscellaneous

Other antepartum factors that may increase the risk of shoulder dystocia include advanced maternal age, multiparity, previous macrosomic birth, previous shoulder dystocia, large maternal habitus and excessive maternal weight gain. The male fetus has consistently been shown to predominate in cases of shoulder dystocia.

Intrapartum Risk Factors

Certain intrapartum events may herald shoulder dystocia. Primary dysfunctional labour and arrest

disorders, including a prolonged deceleration phase during the first stage of labour have been associated with shoulder dystocia. This is not surprising as macrosomia itself is associated with dysfunctional labour.

Prolonged second stage and midcavity instrumental delivery have also been associated with a high incidence of shoulder dystocia. Although not considered a risk factor in the real sense, oxytocin augmentation of labour may overcome mild cephalopelvic disproportion and rotational abnormalities and lead to shoulder dystocia.

Prediction and Prevention

The best management strategy for this infrequent but often catastrophic emergency is prevention. Unfortunately, predicting its occurrence is fraught with inaccuracies despite the numerous predisposing risk and associated factors. In a series by Beneditti and Gabbe⁽²⁾, over 95% of infants of midpelvic instrumental deliveries after a prolonged second stage were delivered without shoulder dystocia. Even in the presence of an additional risk factor, 79% of infants weighing more than 4000g were delivered without this complication. The positive predictive value of each of the listed factors and even in combination is indeed low. However, macrosomia and maternal diabetes consistently appear across many studies as the two risk factors most strongly associated with shoulder dystocia.

Conversely, shoulder dystocia may unexpectedly follow a fairly short labour, a brief second stage, or spontaneous delivery of the vertex. In the confidential Enquiries into Still births and Deaths in Infancy (CESDI) report of 1994 and 1995, 25% of babies who died as a result of shoulder dystocia weighed less than 4kg.

Other Preventive Measures

Other preventive strategies include:

- * Avoidance of mid cavity instrumental deliveries particularly for macrosomic infants following a delayed second stage.
- * Appropriate management of prolonged pregnancy
- * Tight control of diabetic pregnancy would certainly prevent macrosomia, and therefore shoulder dystocia.
- * Pre-pregnancy weight control

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- * Pre-pregnancy weight control

* Proper diet in early pregnancy
The last three measures may all help reduce the incidence of macrosomia.

Since the unexpected case will still occur in the face of optimal risk assessment by even the most experienced obstetrician, it is imperative that all labour ward staff are conversant with its management in order to minimise injury to the infant and mother. Good communication between staff and patients is important. If there is a suggestion of a large baby antenatally, either clinically or by ultrasound scan findings; this information must be communicated to those who will be caring for the woman in labour by indicating the fact boldly on her records.

Finally, all labour ward staff should anticipate shoulder dystocia if there are risk factors, and an attitude of low threshold for caesarean section should be adopted in the presence of multiple risk factors.

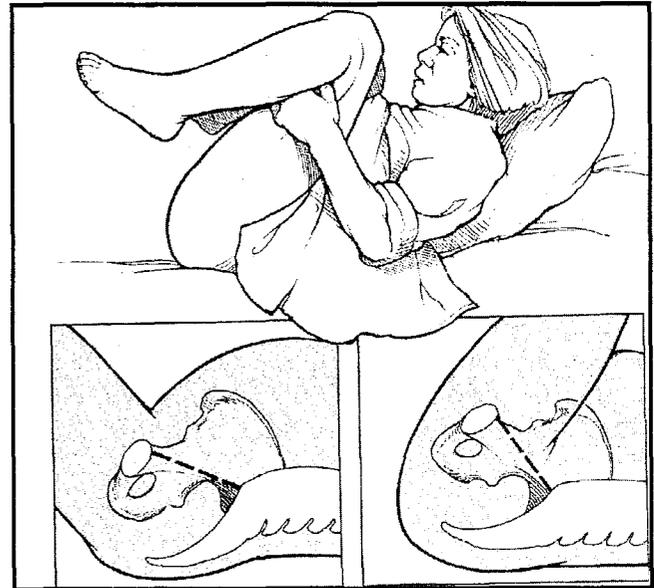
Management of the established Shoulder Dystocia

Shoulder dystocia is a grave emergency that requires prompt, intelligent and logical action to prevent injury to the mother and her infant. It is important that adequate help is mobilised immediately including a senior obstetrician skilled in dealing with shoulder dystocia, two or more midwives, other senior labour ward staff, an anaesthetist and a paediatrician. Best results are achieved when all attending staff are conversant with the pathophysiology and management of this condition.

A paediatrician skilled in neonatal resuscitation must be called at once, and equipment for resuscitation made available. Management requires initiation of a deliberate and planned sequence of activities. Maternal pushing or fundal pressure will only worsen impaction. The worst attitude is to panic.

Various Manoeuvres for Delivery of Shoulder Dystocia

1. **McRoberts manoeuvre.** A generous episiotomy is mandatory before disimpacting the shoulders. The McRoberts manoeuvre should be employed as a primary procedure.



McRoberts manoeuvre. Sharp ventral rotation of both maternal hips brings the pelvic inlet and outlet into a more vertical alignment, facilitating delivery of the fetal shoulders.

This procedure first described by Gonik et al and popularised by William McRoberts Jnr at the University of Texas Medical School at Houston, is successful in 85 to 90% of cases⁽¹⁰⁾ and is the procedure of choice among several other manoeuvres.

The manoeuvre involves hyperflexion of maternal thighs over the maternal abdomen by a midwife. A second obstetrician applies gentle suprapubic pressure in such a way as to "hunch" the shoulders, thereby reducing the bisacromial diameter of the fetus. This manoeuvre also rotates the pelvis and the symphysis cephalad; align the axis of the pelvis to that of the lumbar spine, which gets flattened thereby facilitating disimpaction. The fact that the manoeuvre involves maternal manipulation reduces forces on the fetus and thereby the risk of fetal injury.

All other manoeuvres involve direct manipulation of the fetus with an associated increase in fetal injuries. Besides, general anaesthesia may be needed to relax maternal tissues.

2. **The Wood's cockscrew** manoeuvre involves rotating the fetus through 180° arc as in turning a screw so that the posterior shoulder, which usually is lower, appears anterior and below the symphysis. As the posterior shoulder rotates anteriorly, it will often deliver.

3. Delivery of the posterior arm and shoulder is another technique that creates room in the sacral hollow allowing easier disimpaction of the anterior shoulder. The obstetrician introduces his hand into the vagina, following the posterior arm of the fetus to the elbow. The arm is flexed and swept out over the chest and the perineum.

4. Deliberately fracturing one or both fetal clavicles is a popular technique when the fetus is dead. It is done by pressing the anterior clavicle against the pubic ramus. In the live fetus the fracture subsequently heals quickly. These manoeuvres may be used after a failed Mc Roberts manoeuvre.

5. Zavanelli procedure. Sandberg⁽¹¹⁾ presented a manoeuvre involving replacement of the fetal head and abdominal delivery when all other manoeuvres have failed. Although OLeary and Gunn⁽¹²⁾ reported successful use of this so-called Zavanelli procedure, several other workers have been unsuccessful. This procedure is not used in modern obstetric practice.

6. Cleidotomy with scissors or other sharp instruments are normally used on dead fetuses but may cause dangerous maternal injuries.

7. Symphysiotomy has also been applied successfully in certain settings⁽¹³⁾

Several other techniques have been introduced. However, there is no evidence that any one manoeuvre is superior to another in relieving the impacted shoulder. The McRoberts manoeuvre is easy, has high success rates without associated increase in risk of injury to the fetus. It is definitely the primary procedure of choice in our centre.

Complications of Shoulder Dystocia

Newborns experiencing shoulder dystocia are at risk for serious immediate and long-term morbidity and death. Immediate injury occurs in 20% of surviving infants and late neuropsychiatric abnormalities were present in 30% of surviving infants⁽¹⁴⁾. Perinatal mortality has been reported to range from 21-290 per 1000 cases depending on the definition⁽²⁾.

The cause of death is normally from asphyxia or its consequences. The commonest injuries include asphyxia with its consequences and brachial plexus palsy. These injuries may still occur despite use of appropriate obstetric manoeuvres. When

C5 and C6 segments of the Brachial plexus are injured, the result is Erb's (or Duchenne's) palsy. Klumpke's palsy occurs when the T1 segment is injured. Klumpke's palsy is less common and has a less favourable prognosis with 40% recovery at 1 year compared with 72-92% rate with Erb's palsy⁽¹⁵⁾.

Other injuries include fractures of the clavicle and humerus, and diaphragmatic paralysis.

Maternal Complications

Immediate complications include genital tract haematomas, lacerations, uterine atony with postpartum bleeding, and rupture of the uterus.

Late complications. In the tropics where shoulder dystocia may have been referred over a long distance, sepsis, genital fistulas and problems with continence may follow shoulder dystocia.

Discussion And Controversies

The role of caesarean section

The role of caesarean section in the presence of single prepartum and intrapartum risk factors as a means of pre-empting shoulder dystocia remains controversial. Shoulder dystocia is a problem of labour, and perhaps, sidestepping labour and performing caesarean section in the presence of recognised risk factors will reduce the risk. However, this argument merely exchanges one kind of risk for another; the risks associated with caesarean section. Various studies could not predict elective caesarean section to prevent shoulder dystocia. In a series by Delpapa et al, fetuses with estimated birth weights of 4,000g or more an additional 76 caesarean deliveries prevented only 5 cases of shoulder dystocia, none of which resulted in permanent injury. A recently published decision analysis estimated an additional 2,345 caesarean deliveries would be required to prevent one permanent injury resulting from shoulder dystocia if all fetuses suspected of weighing 4,000g or more underwent caesarean section⁽¹⁶⁾. It is always difficult to say whether the benefits outweigh the risks. Performing elective caesarean section would prevent more cases of shoulder dystocia but the caesarean section rate would also be greater. If all the predisposing factors are taken into consideration, probably planned caesarean section may provide more benefit than risks.

Nevertheless, caesarean section has a preventive role in many situations after careful assessment in selected cases. In these situations, a higher risk of shoulder dystocia is predictable. When macrosomia, diabetes mellitus, and previous shoulder dystocia is found in combination with one other risk factor, the predictive value markedly increases. Approximately one third of babies weighing more than 4000g born to diabetic mothers suffered shoulder dystocia, as did 35% of babies weighing more than 4,500g with delayed second stage.

Although all cases should be individualised, it is reasonable to perform caesarean section for diabetic mothers with estimated fetal weights over 4000g.

Other situations that may indicate elective delivery include macrosomia in a parturient with a previous history of shoulder dystocia, post-term pregnancy with dysfunctional labour.

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Postpartum Haemorrhage (PPH)

E Y Kwawukume

Introduction

Postpartum haemorrhage is the most common cause of obstetric haemorrhage and is the number one cause of maternal mortality in the hospitals in developing countries.^(1,2) It is therefore prudent to find the quickest means of stopping postpartum haemorrhage both conservatively and/or surgically to prevent irreversible shock and death.⁽³⁾

Death can almost always be avoidable if adequate preventive and therapeutic measures are utilized. Normally after the third stage of labour the uterine muscles contract to occlude the open sinuses that previously fed blood into the placenta. Therefore, any event that interferes with contraction of the uterus contributes to postpartum haemorrhage.

Excessive bleeding can be expected to occur whenever uterine muscle activity is decreased. Failure of the uterus to contract effectively usually results from dysfunctional labour or overdistention of the uterus. Excessive bleeding in the third stage of labour usually originates at the placental site rather than from an injury and can therefore be controlled if the obstetrician and the midwife take control of the situation and act quickly and precisely by stimulating uterine contraction.

Definition of Postpartum Haemorrhage

Primary PPH is defined as blood loss of 500ml or more or any amount likely to compromise the patient's haemodynamics, for example in sickle cell disease and anaemia. Massive primary postpartum haemorrhage is one of the most feared and potentially dangerous problems in obstetrics.

Secondary PPH occurs when there is excessive bleeding from the genital tract after the first 24 hours of delivery up to 6 weeks post delivery

Causes of PPH

There are many causes of postpartum haemorrhage, singly or in combination. The most common ones are uterine atony and laceration of the genital tract. Uterine atony may be caused by retained placental tissue, either whole or partial, succenturiate lobe of placenta, a morbidly adherent placenta and uterine fibroids.

Episiotomy alone causing severe postpartum

haemorrhage is uncommon but episiotomy and other genital tract lacerations, notably, cervical laceration or tear, pelvic or broad ligament haematoma, vulva haematoma and vaginal or paravaginal haematoma might cause significant bleeding. Lower genital tract lacerations characteristically manifest immediately after delivery of the infant and before expulsion of the placenta. Upper genital tract lacerations may not be noticed quickly because of bleeding into the abdominal cavity. Operative vaginal delivery, especially forceps delivery with rotation; precipitate labour and delivery; macrosomia with or without shoulder dystocia are associated with an increased risk of laceration of the genital tract. Other causes of haemorrhage are ruptured uterus, uterine inversion, coagulopathies (DIC), amniotic fluid embolism and intraperitoneal or retroperitoneal haematoma.

Causes of PPH

1. Uterine atony
2. Genital tract lacerations
3. Rupture of uterus
4. Uterine inversion
5. Retained products of conception
6. Coagulopathies (DIC)
7. Amniotic fluid embolism
8. Intraperitoneal/Retroperitoneal haematoma

Predisposing factors

Mismanagement of the second and third stages of labour can cause PPH. Mismanagement can occur in the second stage if oxytocic medication is not given on time, at the delivery of the anterior shoulder. Mismanagement of the third stage is more common especially when excessive traction is applied on the unseparated placenta to hasten the third stage. In addition, squeezing and

kneading the already contracted uterus is likely to impede the physiological mechanism of placental detachment causing incomplete separation and increased bleeding.

An overdistended uterus is very likely to be hypotonic after delivery as seen in multiple pregnancy, polyhydramnios and a big baby. Similarly, labour either induced or augmented with oxytocin is more likely to be followed by post-delivery uterine atony and haemorrhage. Uterine atony causing haemorrhage can be anticipated whenever excessive concentration of halothane is used for general anaesthesia or to relax the uterus. The woman whose labour is characterised by uterine activity that is either remarkably vigorous or barely effective and prolonged with maternal and uterine exhaustion is also likely to bleed excessively after delivery. The grand multiparous woman is at increased risk of haemorrhage as well as the woman who has previously suffered postpartum haemorrhage.

Antepartum haemorrhage, from either abruptio placenta or placenta previa predisposes to further bleeding after delivery. In the case of placenta previa the lower uterine segment has fewer muscular fibres and as a result does not contract as efficiently as the upper uterine segment. Furthermore, implantation of the placenta in the lower segment predisposes to PPH.

In abruptio placenta there may be coagulation failure leading to bleeding. In addition there might be Couvelaire uterus, which does not allow for efficient uterine contraction. Excessive postpartum bleeding can result in disseminated intravascular coagulation (DIC)⁽¹¹⁾. Similarly, any patient receiving large quantity of intravenous infusion especially crystalloids in such situations is at risk for dilutional coagulopathy and requires adequate coagulation factor replacement.

Ideally prothrombin time, partial thromboplastin time, platelet count, and fibrin degradation products should be performed but might be difficult in our sub-region. In such a situation, about 5 ml of blood is drawn into a test tube and watched for whole blood clotting within 10 minutes. If there is no clotting or partial clotting then DIC should be suspected and about 2 to 4 units of fresh frozen plasma (FFP) should be obtained for transfusion.

Predisposing Factors to PPH

1. Prolonged labour.
2. Overdistended Uterus
 - * Multiple pregnancy
 - * Polyhydramnios
 - * Big baby
3. Oxytocin stimulated labour.
 - * Induction
 - * Augmentation
4. General anaesthesia with halothane
5. Amnionitis
6. Instrumental delivery
7. Grand multiparity
8. Past obstetric history of PPH
9. APH, from either abruptio placenta or placenta praevia

Prevention of PPH can be categorised into 3 broad groups. Management of PPH can be stormy. The adage that prevention is better than cure could not be more apt than in the consideration of postpartum haemorrhage⁽⁵⁾

Postpartum haemorrhage tests the vigilance and the capability of the obstetric team. Standard protocols should be followed to prevent this emergency. Mismanagement of the first and second stages of labour can cause PPH but more often inappropriate management after the baby is delivered results in postpartum haemorrhage.⁽⁶⁾

Postpartum haemorrhage can be prevented if there is proper management of the second and third stages of labour, routine use of oxytocic drugs in the management of the second and third stages of labour and identification of high-risk patients

Management

Early diagnosis of PPH is crucial. An intravenous line should be secured and at least two units of blood products or whole blood should be grouped in readiness of any eventuality if the patient has known risk factors for PPH. If the patient has no known risk factors, intravenous line should be obtained before the second stage of labour. Always inform a superior from the start and reassure the patient to allay her anxiety.

If PPH occurs rub up uterine contraction and set up an intravenous line if it has not been set already. In addition, take blood for grouping and cross match 2 to 4 units of blood. Full blood count and platelet count are obtained including blood clotting profile. Give intravenous ergometrine (0.5mg) if she is not a known hypertensive or give 10 units of oxytocin intravenously. Intravenous infusion with crystalloid products should be continued and blood added as the situation dictates. Ensure that the bladder is empty by passing a self-retaining catheter. The timing of the onset of haemorrhage is important. Bleeding that begins before delivery of the placenta might be due to laceration of the lower genital tract or coagulopathy. Bleeding that occurs after delivery of the placenta might be uterine atony, retained placenta, ruptured uterus or uterine inversion. Further management depends on whether the **"Placenta is Out or Placenta is In"**. If the **Placenta is out** check for completeness of the membrane and lobes and rub up contraction. Intravenous oxytocin or ergometrine, 0.5 mg should be repeated if necessary. Add 10 units of oxytocin to 500 ml of the intravenous infusion.

If the **uterus is not well contracted** perform bimanual compression of uterus and inject prostaglandin if available into the myometrium or intramuscularly. If all the conservative measures fail to control bleeding prepare for exploration of the uterine cavity under anaesthesia (EUA) or perform emergency laparotomy. At laparotomy, check for uterine rupture and repair or perform hysterectomy if there is extensive rupture. If the uterus contracts and relaxes and there is no rupture perform hypogastric artery ligation or hysterectomy.

If the **uterus is well contracted and the patient is still bleeding** examine the genital tract for trauma especially cervical laceration or tear, vaginal and perineal tears. Remember to check the episiotomy site and suture any vaginal or cervical lacerations or tears. Explore the uterine cavity. Gentle curettage is performed to ensure that no products of conception are retained and that the uterus is intact and not ruptured. If a defect in the uterine wall is detected, prepare and perform laparotomy. At laparotomy, inspect the uterus first for haematoma or rupture. If none of the above are present and the patient is still bleeding perform bilateral hypogastric artery ligation or in extreme cases a hysterectomy is done to preserve the patient's life. **If the placenta is in-situ** perform Control Cord Traction (CCT) and if this fails perform manual removal of retained placenta. For appropriate delivery of the placenta, watch for an increase in lengthening of the

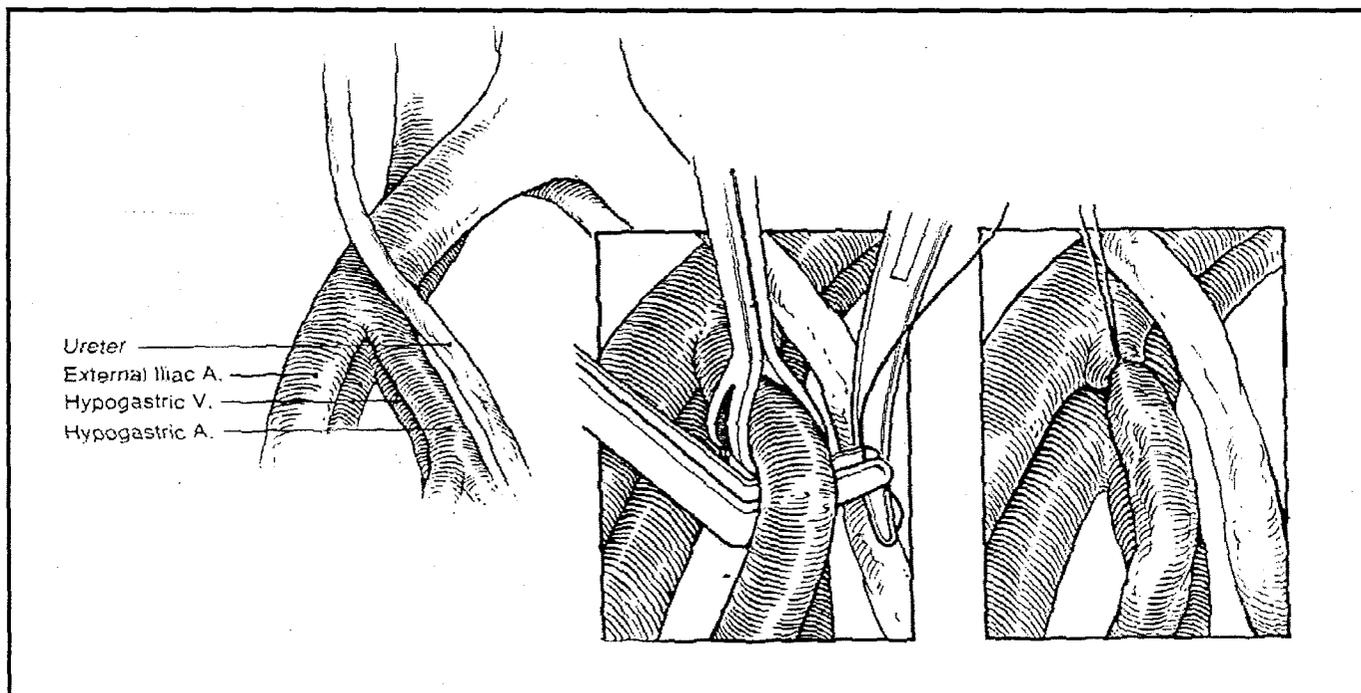
umbilical cord and vaginal bleeding as signs of placental separation. Apply gentle pressure on the umbilical cord with counter traction upwards and backwards on the uterus to deliver the placenta and membranes. Most placentas are separated within 10-15 minutes of delivery of the infant but after waiting for 30 minutes perform manual removal under anaesthesia. Abnormally adherent placenta though not common in our hospitals must be watched for. It is usually due to abnormal development of the fibrinoid layer of the placenta leading to loss of natural cleavage plane for placental separation. Depending on the degree of trophoblastic invasion through the myometrium it may be accreta, increta or percreta. If one of the placental abnormalities is suspected or a lobe of placental tissue is retained there is a need for manual removal under anaesthesia or curettage to remove the tissue under oxytocic infusion. Difficulty in removal must be followed by laparotomy. ^(8,9,10)

Surgical management of PPH

1. Uterine artery ligation
 - * Vaginal
 - * Abdominal
2. Hysterectomy
 - * Supracervical (sub-total)
 - * Total
3. Hypogastric artery ligation
4. Interventional radiography
 - * Selective transarterial embolization (gel foam)
 - * Selective arterial infusion of vasopressin

Procedure for manual removal of placenta ⁽⁴⁾

The patient should be put in the lithotomy position with adequate anaesthesia. After gowning and wearing sterile gloves empty the bladder by catheterisation, if necessary. Hold the cord taut and insert the other hand into the vagina with the thumb in the palm and the hand shape like a cone: Follow the cord to its insertion on the placenta and identify the edge of the placenta. Place one hand on the abdomen to locate and steady the uterine fundus. With Counter-pressure from the abdominal hand separate the placenta from uterine wall with sideways slicing movements and remove the placenta when it is completely separated.



An illustration of hypogastric artery ligation

Ligation of Anterior Division of the Hypogastric artery Operative Technique.

On entering the abdominal cavity, the common iliac artery is palpated at its bifurcation to the hypogastric and external iliac arteries. The posterior parietal peritoneum is lifted and opened on the lateral side of the common iliac artery near its bifurcation, with the ureter still attached to the medial peritoneal reflection. This would prevent the disturbance of the blood supply to the ureter.

The adventitia over the hypogastric artery is cleared up to about 2-3 cm below its origin to avoid ligating the posterior. Division of the hypogastric artery and a right-angle clamp is passed around the anterior division of the hypogastric artery, care being taken to avoid the adjacent vein. Double ligate the artery with No. 0 silk and repeat the procedure on the other side of the uterus, thus ligating both hypogastric arteries.

Hypogastric artery ligation was introduced into obstetric practice in 1990 at Korle-Bu Teaching Hospital, Ghana and this has diminished abdominal hysterectomy as treatment for PPH secondary to atonic uterus.^(12,13) Hypogastric ligation has a number of advantages:

- It takes a short period to complete
- The uterus is conserved and the woman has a chance of future child-bearing
- It is safe and easy to perform

Some physicians feel reluctant to attempt hypogastric artery ligation in primiparae even though it allows conservation of the uterus. This is because of a fear of failure and the natural disinclination to perform a relatively unknown procedure in an emergency. Others fear that ligation might render the patient a "pelvic cripple" from ischemic changes distal to the ligation but it must be noted that the pelvis has a lot of collateral circulation.

It has been documented that^(14,15) there was no circulatory embarrassment in the first 82 patients and other researchers also documented one patient who had hypogastric artery ligation got pregnant after twelve months of ligation⁽¹²⁾. She had spontaneous vaginal delivery at term without postpartum haemorrhage

The mechanism of the ligated anterior division of the hypogastric artery stopping PPH lies in the reduction of the pulse pressure on the bleeding vessels and the isolation of collateral uterine circulation from the pelvis.^(16,17,18) The reduction in pulse pressure permits clotting to develop and to persist. Ligation therefore virtually transforms the affected pelvic circulation into a venous system with decreased arterial pressure in the vessels.

While hypogastric artery ligation is relatively easier to perform than hysterectomy, one must not perform blind dissections and should be extremely careful not to damage the adjacent internal iliac vein. If the hypogastric vein is damaged inadvertently, pressure by an assistant must be applied both proximally and distally to the vein and

a round body needle can be used to repair the rent. Another surgical technique to control massive postpartum haemorrhage and preserve the uterus is the **B-Lynch suturing technique (Brace suture)**¹⁸. It is a compression suturing technique with sutures passed around the uterus both anteriorly and posteriorly after exteriorising the uterus. The test of potential efficacy is simple bi-manual compression of the uterus.

Management of other causes of PPH

1. Genital tract lacerations

a. Cervical laceration - Is a common cause of PPH. Clean the vaginal canal properly and make sure the blood is not trickling down from the uterus. Sometimes the cervix might look ragged but on close inspection you would realise that there is no actual laceration. This would help avoid unnecessary sutures on the cervix and other causes should then be sorted out to prevent time wasting.

If the cervix is lacerated apply sponge-holding forceps to the two torn edges and suture starting from above the base. Either running or interrupted 3-0 absorbable sutures can be used.

b. Pelvic haematomas

Vulva haematoma (Intrafascial haematoma)

The Colles' fascia and the urogenital diaphragm limit blood loss anteriorly and the anal fascia limits it posteriorly resulting in a visible vulva haematoma. The vulva is incised and the blood and clot is evacuated. The dead space is closed with sutures as this condition is often the result of bleeding from small vessels and the lacerated vessels may not usually be identified easily. Compress the dead space with sterile dressing and leave an in-dwelling corrugated drainage or Foley catheter for 24 to 36 hours.

3. Vaginal haematoma (Suprafascial haematoma)

In this case, the blood accumulates above the level of the pelvic diaphragm and the most frequent complaint is severe rectal pressure. The blood loss is usually limited by the small space and treatment is incision and evacuation of the blood clot. The cavity is packed and the pack removed after 24 hours. There is no need to close the incision. Leave a corrugated drain or Foley catheter.

(iii) Retroperitoneal haematoma

This results from laceration of a branch of the hypogastric artery and the symptoms may not be impressive until shock intervenes. Ligate both hypogastric vessels if unilateral ligation on the lacerated side is ineffective. Occasionally, the surgeon should open the haematoma and ligate the bleeding vessel and drain the site with corrugated drain or Foley catheter. Packing of the haematoma site is advisable in the presence of persistent bleeding.

Uterine Inversion

Uterine inversion can be described as the fundus of the uterus turning "*inside out*". This is one of the obstetric emergencies that can produce profound postpartum shock. It can be caused by undue traction on the umbilical cord and/or fundal pressure⁽⁷⁾ before placental separation, hypotonic uterus and placenta accreta, increta or percreta. It can present with haemorrhage, shock, pain or a protruding mass.

Treatment. One should remember that this could happen after delivery and prompt recognition is essential. If steps are not taken quickly the cervix will close down and trap the uterine fundus in the vagina or at the introitus. The fundus should be replaced quickly mainly by manual replacement. Tocolysis such as MgSO₄, halothane, ritodine or terbutaline can also be used to relax the uterus.

After administering one of these uterine-relaxing drugs pressure should be applied around the edges of the inversion to gently replace the fundus to its normal anatomic position. Once it has been replaced, oxytocic agents should be administered to maintain pressure within the fundus until uterine tone returns. Perform laparotomy if vaginal replacement is not successful. Once the uterus is replaced oxytocin infusion is set up to produce uterine contraction.

Postpartum haemorrhage can lead to Sheehan's syndrome. The syndrome occurs because of severe hypotension from blood loss resulting in ischemic necrosis of the pituitary. Sheehan's syndrome can present in varying degrees and presenting as panhypopituitarism with onset of symptoms beginning during the postpartum period. Symptoms might present as weakness, nausea and fatigue which if not critically appreciated might be attributed to the postpartum state. Later, decrease in the secretion of prolactin, which is usually the first recognised pituitary deficiency might present as lack of lactation. Hypoprolactinaemia is followed by the development of hypothyroidism, hypoandrenalism, and hypogonadotropism.

Secondary Postpartum Haemorrhage

There is no quantitative analysis of the amount of blood lost in secondary postpartum haemorrhage unlike primary postpartum haemorrhage. Blood loss is a subjective impression of an increase in the amount of bleeding after the first 24 hours of delivery and this may occur any time up to the end of the puerperium.

Causes

1. Retained products of conception
2. Infection
3. Submucous fibroids
4. Polyps
5. Dehiscence of a caesarean section scar
6. Rupture of a vulval haematoma
7. Development of choriocarcinoma following a normal delivery. This is however very rare.

Subinvolution caused by infection is treated with broad-spectrum and potent antibiotics. There might be retained products and the patient should be resuscitated and dilatation and curettage should be performed. The curetting should be sent for histological diagnosis to exclude choriocarcinoma.

In summary the following points should be noted concerning deaths from massive haemorrhage.

1. Failure to anticipate the high-risk patients
2. Criticism of excessive use of Crystalloid and failure to compensate for blood loss resulting in under-transfusion
3. Failure to detect early stage DIC
4. Failure to use CVP monitoring
5. Blood unavailable in adequate amounts
6. Lack of involvement of Senior Staff at an early stage
7. Resort to surgical haemostasis (e.g. hypogastric artery ligation or hysterectomy) too late.
8. Always remember to act fast and know when to call for help without procrastination.

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Vaginal Birth After Caesarean Section

Akinyinka O. Omigbodun

Introduction

The natural route for childbirth is through the lower female genital tract. Unfortunately, a sizeable proportion of parturient women are unable to deliver their babies via this route for a variety of reasons and the accoucheur is left with no other option but the delivery of the fetus, placenta and membranes through an incision in the abdominal and uterine walls. This alternative route is known as caesarean section. The origin of the term is shrouded in some controversy. While some believe that its origin is in Roman law, *lex caesarea*, from the time of the rule of dynasties of Caesars from about 700 BC^{1,2}, others believe that the term originated from the Latin words *caedere* [to cut] or *caesum* [cut]³. Whatever its origin, there are many who now believe that the term is obsolete and should be replaced with the more etymologically appropriate term "hysterotomy"². If that suggestion were to be adopted, then the appropriate title for this chapter should be "vaginal birth after hysterotomy" and not the current "vaginal birth after caesarean section". However, since caesarean section is still the term most widely used among obstetricians worldwide, the term hysterotomy being restricted to extraction of the conceptus by the abdominal route prior to the 24th week of gestation, we will stick to using caesarean section here. In a large number of patients, the factors leading to the decision to have a caesarean section do not recur in subsequent pregnancies and the natural assumption is that obstetricians would revert to allowing birth by the natural route. However, there is the new factor of the presence of a scar on the uterine wall that has to be taken into consideration. For a long time the presence of 'the scar' seemed to override all else and Craigin's dictum⁴ shaped obstetric practice: "once a caesarean, always a caesarean". As the procedure was developed further and various modifications were introduced, people felt bolder to try vaginal birth after caesarean (VBAC) and the obstetric literature is replete with articles that have reported the experience with this approach in several parts of the world. Yet the mindset of 'the scar' being the all-important factor still dominates the thinking of many obstetricians and an attempt at VBAC is often referred to as a 'trial of scar'^{1,5,6}. The objectives here

are to review the available evidence on VBAC, to outline the factors that favour its practice and those that do not; and also to describe how it can be properly conducted, with the safety of the mother and the neonate being paramount.

Types Of Caesarean Sections

In order to be able to discuss the selection of patients that are suitable for an attempt at VBAC, it is essential to describe the various types of uterine incisions that can be made at caesarean section and the impact of this on the management of the patient in subsequent pregnancies.

The four main types of uterine incisions that are still being used in obstetric practice are:

- * The longitudinal incision made on the anterior part of the uterine fundus, also known as "classical caesarean section".
- * The transverse incision made over the lower uterine segment
- * The longitudinal incision made on the lower uterine segment, also known as DeLee incision.
- * The transverse lower uterine segment incision that is elongated into the upper segment at one end, also known as the 'J-incision'.

Many would disagree that there is ever a need to have a J-incision on the uterine wall at caesarean section, contending that a standard lower segment incision should suffice in all such situations.

The lower segment transverse incision is the most common type used. Its advantages include the fact that it follows the predominant direction of the muscle fibres in that location, permitting easy stretching of the incision to the desired length. It is also in an area of less dynamic activity during the puerperium, allowing the scar to heal relatively undisturbed. Its main disadvantages are the tendency towards severe haemorrhage, particularly when the incision extends to the large

vessels in the broad ligament; and the limited access it provides in cases where the babies are macrosomic or are lying abnormally in the uterine cavity. The other lower segment incision, the vertical incision, is seldom used. Although it is associated with less bleeding and it can be easily extended to the upper segment when necessary, it is seldom possible to make an incision of adequate length unless the lower segment has been stretched by labour; and there is always the danger of the incision extending into the bladder or the cervix and vagina. Where a true vertical lower segment incision is made, the scar formed is just as strong at withstanding dehiscence as those from a lower segment transverse incision^{7,8}.

The 'classical' incision was the first type of incision to gain wide acceptance among obstetricians, but it has virtually been abandoned except in a few instances when the lower segment incision proves to be impracticable. The major reasons for this development are the notorious weakness of classical scars due to the constant motion that takes place in the upper uterine segment in the early puerperium; and due to the propensity of such scars to be associated with intra-abdominal adhesions and intestinal obstruction. Upper segment scars are also more liable to rupture in subsequent labours, and sometimes, even before the onset of labour^{3,5,9}.

Factors Favouring VBAC

Caesarean section has become a much safer procedure due to the advent of modern blood transfusion services, safer anaesthesia and powerful antibiotics, removing the spectre of the high maternal mortality rates from massive haemorrhage, anaesthetic complications and overwhelming infection that were hitherto associated with the procedure. Coupled with the assumed benefits to the fetus from choosing this route of delivery, many obstetricians often resort to caesarean births simply because a patient had undergone a caesarean section before. Hence, nearly one third of all caesarean sections being done in the developed parts of the world now are repeat sections¹⁰. Nevertheless, it is important to bear in mind that the rates of maternal mortality associated with caesarean sections can vary from 4 to 26 times the rates found in association with vaginal delivery^{3,5,10}. This is incentive enough to seek out those women who can safely deliver by the vaginal route, but there are other incentives as well.

In addition to a risk of dying, several other

complications attend abdominal deliveries that are much commoner than would be found after vaginal delivery. Puerperal endometritis can be up to ten times more common after caesarean section than after a vaginal delivery^{5,7}. Other complications that occur exclusively or are more frequent after caesarean births are bladder injury, bowel injury, ureteric injury, urinary tract infections, aspiration pneumonitis [Mendelson's syndrome], deep vein thrombosis and pulmonary embolism. Caesarean sections cost much more, result in a longer stay in hospital for the patient and creates a delay in mother-baby bonding³. In view of the foregoing, it is essential to have ways of identifying those patients that are likely to be able to avoid repeat caesarean sections.

The factors that have been identified as favouring successful VBAC include the following^{3,6,11,12,13}:

- * A history of a previous vaginal birth *before or after* the caesarean section
- * When the previous caesarean birth was for malpresentation, pregnancy induced hypertension or some other non-recurrent cause
- * When a lower segment uterine incision was used in the previous caesarean section
- * When there was no additional morbidity following the previous caesarean section
- * When the Bishop score in the patient is 4 or more at the time of assessment at term

Indeed methods of predictive scoring based on these criteria have been devised^{11,14,15} and the proper application of these scores can help in selecting patients who are likely to achieve VBAC successfully. Unfortunately, these scores may not be directly applicable in a developing country and modifications may have to be made to these scoring systems or new criteria may have to be developed and validated for the tropical African environment.

Predictors Of Failed VBAC

The major factors that impede the success of an attempt at VBAC are fetal macrosomia, intrauterine growth restriction and fetal asphyxia^{6,11}. A short birth interval, especially when the interpregnancy interval is less than six months, can also diminish a woman's chances of successful VBAC because of the substantially increased risk of scar disruption in

such women¹⁶. Maternal age does not seem to affect a woman's chances of having a VBAC and even women having twin gestations can have a safe VBAC^{1,17}. However, grandmultipara who are attempting VBAC tend to have a higher frequency of complications¹⁸ even if their chances of success [about 60%] compares favourably with the overall success rates for VBAC which ranges from about 52%¹⁹ to more than 80%^{6,20}. Success rates in the developed world seem to hover around the 80% range^{6,11,20}, but in West Africa, the success rates seem to be closer to about 60%^{21,22,23}. This difference probably results from patient selection in that there is greater readiness to resort to caesarean sections in the developed world than in the developing countries because of the more abundant and better facilities available in the richer countries. In 1993, only a quarter of parturient women who had a previous caesarean section were allowed an attempt at VBAC¹⁰. Patients in many parts of West Africa tend to place a huge cultural premium on the ability to deliver vaginally, hence they are less willing to consent to repeat caesarean sections²⁴. Rather surprisingly, a diagnosis of cephalopelvic disproportion in the previous pregnancy did not seem to be an impediment to successful VBAC in many of the studies reported. In fact more than 60% of patients in this group had successful VBAC in some studies¹¹.

The only absolute contraindication to an attempt at VBAC that most obstetricians subscribe to is the presence of a classical caesarean section scar on the uterus. Previously, many would also consider a history of two or more previous caesarean sections as an absolute contraindication, but evidence accumulated over time suggests that this needs not be so and there are now some reports that suggest that safe VBAC can be achieved in a carefully selected few of such patients^{3,21,25,26}.

Preconception Care Of Women With Previous Caesarean Births

Although it is not yet routine practice, the care of women who have uterine scars should begin in the preconception period. Several reports have now been published on the value of hysterosalpingography, ultrasonography and sonohysterography in the assessment of the integrity of uterine scars^{27,28,29,30}. While ultrasonographic assessment may be feasible during pregnancy, the other methods are applicable only to the non-pregnant woman and would fit quite well into a preconception care

programme. The other important investigation that is also appropriate at this period is an erect lateral pelvimetry or, where the facilities are available, a computer tomographic pelvimetry to get exact measurements of the pelvic dimensions. The thickness of the scar area and the measurements of the bony pelvis can provide valuable information that will enable the obstetrician accurately assess the chances of a safe VBAC.

Antenatal Management Of Patients With A Uterine Scar

The antenatal supervision required by this category of patients does not differ substantially from what should be offered to most pregnant patients, especially where appropriate preconception care had been offered and the scar thickness and bony pelvic measurements are known.

However, the past obstetric history must include as much detail as the woman can recollect about the circumstances that led to the previous caesarian birth(s) and what transpired in the puerperium, especially when the previous medical records are unavailable.

The other additional measure required in such women will be placental localization and a reassessment of lower segment thickness by ultrasonography at 36 weeks of gestation. Various cut-off points of what could be regarded as safe have been proposed, ranging from 2mm to 3.5mm of lower uterine segment thickness at its thinnest point^{28,29}. This is an area requiring further study before a consensus can be reached.

Where exact pelvic measurements are unknown prior to conception, it becomes necessary to do a clinical pelvic assessment at the 36th week of gestation and, where the adequacy of the bony pelvis is in doubt, to resort to radiological pelvimetry. In this regard, CT pelvimetry is the safer option because the fetal exposure to ionizing radiation is just about 20% of what could be expected with standard erect lateral pelvimetry.

Magnetic resonance imaging is even better in that the fetus is not exposed to any ionizing radiation at all, but the equipment is very expensive and beyond the reach of most healthcare institutions in the developing world. An attempt should also be made to estimate the size of the fetus at this stage of gestation to ascertain whether there is macrosomia or intrauterine growth restriction, known risk factors for failed attempts at VBAC.

Whatever be the case, the obstetrician must assess all the information available to him just before the patient reaches term at 37 weeks and decide whether an attempt should be made at VBAC or the patient should be offered an elective repeat caesarean section.

Conduct Of Labour In Patients After Previous Caesarean Birth

Women making an attempt at VBAC should always be looked after in a fully equipped labour ward with facilities for a caesarean section. Anaesthesiology and neonatology coverage must be available. On admission, the patient should have a sample of blood sent for grouping and cross matching and a wide-bore canula should be inserted, with intravenous fluids being infused slowly throughout labour and for at least one hour afterward. The management of all stages of labour should proceed in an active manner. Where possible, the patient should be on continuous cardiotocographic monitoring and once membranes have ruptured, there should be an intrauterine pressure catheter. These measures have their inherent risks and there is some evidence that they are not absolutely necessary for safe VBAC¹. In any case, these facilities are seldom available in tropical health facilities and other means for monitoring maternal and fetal condition must be devised.

The most important indices to look out for are evidence of vaginal bleeding, urine colour, maternal pulse and blood pressure, and the fetal heart rate. All of these can be done with simple items of equipment that are available in virtually every healthcare facility worth that name. When these indices are recorded, together with the cervicograph on the partograph, careful monitoring of the emerging pattern should enhance optimal management of the patient. Some have suggested that in patients attempting VBAC, the zone two to three hours after crossing the alert line on the partograph is that of highest danger of scar disruption and an action line in this time zone would probably help reduce the rupture rate, without an unacceptable increase in the rate of caesarean section³¹. Evidence of scar disruption includes a continuous abdominal pain rather than the intermittent pain associated with uterine contractions, cessation of uterine contractions, vaginal bleeding, maternal tachycardia, fetal heart rate abnormalities and haematuria. Vigilant healthcare providers can detect all these signs with relative ease.

Complications

The commonest major complication of an attempt at VBAC is failure to achieve vaginal delivery, compelling recourse to a repeat caesarean section. The main factors precipitating such a decision are fetal distress, slow progress in labour and cephalopelvic disproportion^{9,19}. The other fairly common problems that may confront the obstetrician conducting the delivery are a delay in the third stage of labour, a slightly increased risk of postpartum haemorrhage and a higher frequency of retained placenta³³. There is also an increased risk of abnormal uterine bleeding after the puerperium³⁰.

The most dreaded complication of attempting vaginal birth in a woman with a uterine scar is uterine rupture. In this regard, caesarean section scars are much more liable to rupture than myomectomy scars³⁴. Attempts are often made to classify scar disruptions into those described as 'dehiscence' and those called 'rupture'. Dehiscence refers to disruptions in which there is an intact peritoneum over the separated scar while rupture implies that there is a communication between the peritoneal and uterine cavities⁵. The distinction seems unwarranted because the approach to management is the same and there is no evidence that the prognosis differs between the two groups. Where the peritoneum is still intact, the separation may be painless, but once the serosa is breached, there is usually an acute onset of abdominal pain that is continuous in nature. The other features of scar disruption have been highlighted earlier.

Scar disruption rates vary from place to place, depending on the quality of maternity care available to the women at risk. The rates that have been reported vary from about 8 per thousand⁶ to about 41 per thousand³⁴. In most places, the rates are between 20 and 30 per thousand^{12,31,32}. It is important to institute resuscitative measures as soon as possible after the diagnosis is made. A wide-bore intravenous canula should be inserted and an infusion set up. Blood samples should be sent for haematocrit estimation and blood typing. Crosshatched blood must be available when needed. An indwelling urethral catheter should be inserted to monitor urinary output. The patient should be taken to the operating room once resuscitation has been established. The choice of procedure will depend on the degree of damage. The choices are between uterine repair (with or without tubal sterilization) and hysterectomy (subtotal or total). The individual characteristics of the patient, the level of experience of the surgeon

and the kind of resuscitative support available in the institution all have to be factored in when the decision is made.

Controversial Issues in Care:

The major areas of controversy in the conduct of labour in this group of patients are the use of oxytocics for induction or augmentation of labour, the use of epidural analgesia in labour and whether or not to do digital exploration of the lower uterine segment after completing the third stage of labour.

Many obstetricians now agree that it is safe to use oxytocin for the augmentation of labour in patients attempting VBAC^{1,3,5,9}. Safeguards must however be in place to ensure that its infusion is carefully controlled and that maternal and fetal well-being is carefully monitored. Induction of labour is slightly more controversial. It is sometimes argued that when labour has to be induced in a patient who already has a uterine scar that is a juxtaposition of two risk factors, which should lower the threshold for opting for a repeat caesarean birth. The available evidence however suggests that induction of labour can be carried out safely, using prostaglandins or oxytocin^{1,12}. The success rate of VBAC after labour induction, the rate of scar disruption and the neonatal outcome are similar to those following spontaneous onset of labour. The important point to note is that parturients for induction of labour should be properly selected to remove those with other risk factors for failure. It is also essential that the Bishop score should be 6 or more at the time labour is induced.

There was a time when it was suggested that epidural analgesia could potentially mask scar rupture because the patient will not be able to feel the pain associated with the event. However, pain itself may not be such a reliable feature of scar disruption and epidural analgesia may not completely block the pain associated with uterine rupture because the pain often goes above the level of the block³. Evidence now abounds to support the safety of epidural analgesia in these women^{1,5,9}. Many of these patients are having their first vaginal deliveries and they often require more pain relief than other parturient women and it seems cruel to deny them this facility when it can be safely administered.

For several decades after the introduction of the lower uterine segment incision, the standard practice was to do a digital exploration of the lower uterine segment through the cervix in a bid to determine whether scar disruption had occurred or

not. Although many obstetricians still engage in this practice, current evidence suggests that this practice confers no benefits on the patient and may in fact be harmful^{5,32}. Patients should undergo digital exploration only if they have suprapubic pain, placental retention or primary post-partum haemorrhage³². Digital exploration in the asymptomatic patient may indeed lead to the creation of a rent where there was none or the enlargement of a small one by the examining fingers.

Conclusion

As the relative safety of the procedure is an important factor in choosing whether or not to have a caesarean birth, the fact that optimal facilities are available only in the developed parts of the world should restrict the indications to perform the operation in developing countries. In 1993, only a quarter of parturient women who had a previous caesarean section were allowed an attempt at VBAC. This is an indication that far too many avoidable caesarean sections are still being done. Efforts are needed to improve the proportion of women allowed to try VBAC without compromising on safety.

Summary

The management of a patient who has had a caesarean section in the past should ideally begin in the preconception period when accurate pelvic bone measurement and ultrasound assessment of scar thickness should be done. The patient should also be encouraged to observe a minimum birth interval of two years (inter-pregnancy interval of at least 15 months). Careful assessment should be made during the antenatal period to determine the feasibility of a VBAC. Patients with more than one previous caesarean section should also be assessed individually to seek those who can safely make an attempt at VBAC. Intrapartum care should be meticulous, with facilities made available for caesarean section if the expected level of progress is not attained. There is no evidence that digital exploration of the lower uterine segment after birth confers any benefit and the practice may indeed be harmful.

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Induction and Augmentation Of Labour

E Y Kwawukume

Introduction

Induction of labour is one of the most important interventions in obstetric practice. About 5-15% of patients in our unit have an unfavourable cervix requiring ripening by prostaglandin E2. Pessaries or its analogue, Foley's catheter or other agents to allow for a more natural progression of labour and delivery. ⁽¹⁾ Although the biochemical events associated with labour have been studied the actual trigger mechanism that initiates labour in humans is not known.

Alterations in the levels of estrogen and progesterone have been proposed without firm evidence. Prostaglandins (PG), which are long-chain hydroxy fatty acids, now remain an important mediator and possible initiator of uterine contractions. Because of this, the various methods used for ripening the cervix before induction of labour are either prostaglandins or agents leading to the release of PG. Prostaglandins are synthesised by the decidua, amnion, chorion and the myometrium.

Definition of Induction of Labour

Induction of labour is the initiation of uterine contractions after the 28th week of gestation and before the onset of natural labour by medical and/or surgical means for the purpose of normal delivery.

The indication for induction of labour may be due to maternal or fetal conditions and it depends on the potential risks or benefits to the fetus and the mother in continuing the pregnancy or terminating it by delivery.

Indications

The indication for induction should be specific and justifiable so that the process is not started and repeatedly postponed. The idea of induction is to deliver a potentially at risk baby that is better managed out of the uterus than inside and for better maternal survival. If the baby has to be delivered it may be necessary to combine both medical and surgical methods for a more effective result. A delicate balance between uterine activity, cervical dilatation rate, and response of the fetus should be taken into consideration to achieve this

goal. Once induction is started there is the potential risk of ending with Caesarean section therefore pelvic adequacy and underlying maternal and fetal conditions should be thoroughly evaluated beforehand.

Selected patients with previous low transverse caesarean section can have a trial of labour. The risk of uterine-scar dehiscence, risks of instrumental vaginal delivery, birth trauma or poor neonatal outcome are no greater with induced than with spontaneous labour as long as uterine contractions are monitored closely ⁽²⁾.

Fetal Indication

1. Post-term
2. Haemolytic disease
3. Intrauterine growth retardation
4. Unstable lie
5. Fetal death
6. Fetal abnormality

Maternal Indication for Induction

1. Preeclampsia.
2. Eclampsia
3. Antepartum haemorrhage (APH) from placental abruption and some cases of placental praevia at term.
4. Premature rupture of membranes
5. Chorioamnionitis
6. Polyhydramnios
7. Diabetes mellitus
8. Chronic hypertension
9. Renal disease

Contraindications of Induction

1. Cephalopelvic Disproportion (CPD) due to abnormalities of pelvic bones
2. Extensive myomectomy
3. Two or more previous caesarean section
4. Central or total placenta praevia
5. Active genital herpes
6. Grand multiparity
7. Malpresentation of fetus (shoulder, footling breech, transverse lie.)
8. Invasive carcinoma of cervix

1. Bishop Scoring System				
CERVIX	0	1	2	3
1. Dilatation	Closed	1-2 cm	3-4 cm	> 5 cm
2. Consistency	Firm	Medium	Soft	
3. Length	>2 cm	1-2 cm	0.5-1 cm	<0.5cm
4. Position	Posterior	Mid	Anterior	
5. Station	-3	-2	-1	+1

NOTE: Effacement and length of the cervix are used interchangeably.

Interpretation of Bishop's score

- * 0-4: Cervix is unfavourable
- * 5-8: Cervix is moderately favourable
- * 9-13: Cervix is "ripe" and is predictive of successful induction.

Methods For Pre-induction Cervical Ripening

In cervical ripening the state of the cervix and the parity of the patient influence the duration of labour induction. The more favourable the cervix the greater the chances of successful induction of labour. This was established by Bishop⁽³⁾ who designed a scoring system for induction of labour. This scoring system provides valid parameters for evaluating patients prior to induction of labour and it predicts the ease of induction especially in the multiparous patient.

A Bishop's score between 9 to 13 indicates favourable cervix and is predictive of successful induction of labour. Between 5 and 8 indicates a moderately favourable cervix. The cervix is unfavourable if the score is less than 5. This is associated with failure to induce labour, prolonged labour and a high incidence of caesarean section.⁽⁴⁾ Ideally, the agents used for cervical ripening should be effective within 24 hours, simple, non-invasive, must not stimulate labour excessively and must not compromise the mother or the fetus. Prostaglandin or its analogues are currently most used followed by osmotic dilators and Foley's catheter.

1. Prostaglandin (PGE2)

Prostaglandin E2 is used either as the pessary or as the gel. The gel is applied locally on the cervix and the 3mg pessary, is inserted into the posterior fornix and the cervix is assessed after 6 hours. If the cervix is not ripe, another 3 mg is again inserted before setting up the oxytocic infusion. Since the effects of PGE2 may be exaggerated by oxytocin, at least a period of 4 to 6 hours is needed to observe the patient before setting up the oxytocic infusion.^(4,5) At least 6 hours is needed to clear the principal metabolite of PGE2 from the blood therefore oxytocin should be used with caution and in low doses if there is still uterine activity from the prostaglandin. The mechanism of action of prostaglandin is to cause dissolution of collagen bundles with increase in submucosal oedema as seen histologically.⁽⁶⁾ At term the connective tissue changes observed are similar to those seen in early labour.

2. Misoprostol (CYTOTEC. PGE1)

Cytotec is an analogue of PGE1 and is the most common agent used in induction of labour in many places. Fifty micrograms is inserted into the posterior fornix every 4 hours and the cervix assessed for ripening. About 2 to 4 doses are needed for cervical ripening and in some cases labour might be initiated without further intervention. Cytotec is effective, less expensive than PGE2 and offers better results at less cost.

The use of prostaglandin has been shown to increase the chance of successful initiation of labour and decrease the incidence of prolonged labour.^(4,7) These agents also mimic spontaneous labour and enhance the myometrial sensitivity to oxytocin.⁽⁸⁾ In more than 70 prospective clinical trials in which more than 5,000 pregnant women were studied using intracervical or intravaginal prostaglandin preparation and compared with placebo or no therapy, it was realised that cervical effacement and dilatation was enhanced using prostaglandin preparations.⁽⁸⁾

Prostaglandins should however be used with caution in patients with severe hepatic dysfunction, glaucoma, renal impairment or asthma.

3. Cervical Dilators

Some of these agents are Foley's catheter with inflated balloon, Laminaria tents and Dilapan or osmotic dilators. Foley's catheter can be introduced through the cervical canal into the internal os and the balloon should be inflated with 30ml of sterile water to lie between the membrane and the internal os. This procedure enhances prostaglandin release from the membranes and adjacent decidua to initiate cervical softening and dilatation. The osmotic dilators are placed in the endocervix and care should be taken not to rupture the membranes. They reduce cost and it is convenient to hospital staff because of less need for subsequent monitoring of the uterus and the fetus.

4. Sweeping Of Membranes

The chorioamniotic membrane is digitally separated from the wall by the action of sweeping of the cervix and the lower uterine segment to release prostaglandin locally^(9,10) and thus cause ripening of the cervix.

Stripping of the membranes will also excite autonomic neural reflex and/or cause the release of maternal oxytocin from the posterior pituitary.⁽¹¹⁾ There is the danger of uterine infection, rupture of membranes and bleeding from unsuspected placenta praevia. This method can be painful and must be used cautiously. Secondly, the vertex should be well applied to the cervix for maximum success.

Methods Of Induction

1. Surgical

Amniotomy:

Before amniotomy is done the gestational age and the indication for the procedure should be confirmed. In addition, abdominal examination

should be performed to confirm the lie, presentation and fetal heart rate.

Pelvic examination to determine cervical dilatation, cervical consistency, length of the cervix and the station of the presenting part should also be done before amniotomy. This finding should be related to the modified Bishop score as stated above.

Labour usually starts within 12 to 24 hours after amniotomy. Amniotomy alone is often not adequate for effective uterine contraction and it is usually combined with oxytocic infusion to achieve delivery within a reasonable time.

Procedure

After putting on High Level Disinfectant (HDL) gloves, two fingers are introduced into the cervix and the membranes are swept away from the cervix. An Amniotome or Kocher's forceps is introduced through the cervical canal across the examining hand to lie beside the examining finger or the groove formed between the two examining fingers. The amniotome is applied gently to the bulging membranes and turned, either hooking and/or scratching the membranes to release liquor. The fetal heart tones should be auscultated with the Pinard stethoscope or continuous CTG before and after amniotomy.

Advantages

Amniotomy has a lot of advantages, for example, it would reveal blood or meconium stained amniotic fluid, which would help in the further management of the labour. It has 88% success rate⁽¹²⁾ and intrauterine catheter can be inserted for uterine pressure readings as well as insertion of fetal scalp electrode. There is easy access to fetal scalp blood sampling to determine the acid/base status of the fetus.

Disadvantages

Include umbilical cord prolapse, therefore, before amniotomy cord presentation should be noted to prevent this potential danger. Prolonged rupture of membranes could lead to chorioamnionitis. There could also be accidental fetal injuries, vasa praevia rupture and fetal distress. In these days of HIV/AIDS infection, the evidence of mother to child transmission is more if the membranes are ruptured early.

2. Medical

Oxytocin infusion

Oxytocin is an octapeptide that is widely used all over the world to effect adequate uterine contraction with cervical dilatation and descent of

the fetal presenting part without compromising the safety of the fetus and the mother. The more advanced the gestational age the more the sensitivity of the uterus to oxytocin. It is known that the uterus responds increasingly to oxytocin slowly from 20 to 30 weeks of gestation and the sensitivity increases rapidly from 35 weeks until term.⁽¹³⁾ Important predictors of required oxytocin dosage include gestational age, parity and cervical dilatation. The goal of oxytocin administration is to stimulate uterine activity that is sufficient to produce cervical change and fetal descent while avoiding uterine hyperstimulation and fetal distress. Ideally, uterine contractions should be 3 contractions in 10 minutes lasting about 45 to 60 seconds. If contractions exceed 5 in a 10-minute period or last longer than 60 seconds or if there is significant fetal heart rate deceleration, the oxytocin infusion should be discontinued.

Administration of oxytocin

- Add 5 units of oxytocin to 500 ml of 5% dextrose in water, Ringers lactate or Normal Saline
- Set up an intravenous line and connect to the oxytocin solution
- Start with 15 drops of the oxytocin solution per minute
- Monitor uterine activity and fetal heart tones every 15 minutes in the absence of CTG
- Increase drip rate to 30 and 60 drops/min every 30 minutes till effective uterine contraction of 3 in 10 minutes lasting 40-60 seconds is achieved
- Maintain drip rate at the rate producing the effective uterine contractions
- Observe for any hyperstimulation and decrease accordingly

If uterine contractions are inadequate at 60 dpm, increase dose of oxytocin:

- Add 10 units of oxytocin to fresh 500 ml of dextrose in water or ringers lactate
- Start with 30 drops per minute
- Increase drip rate to 40, 50 and up to 60 drops every 30 minutes till effective uterine contractions are established.⁽¹⁴⁾

Complications

The main complication of oxytocic infusion is mainly related to the dose causing hypercontraction and hyperstimulation leading to uterine rupture and death of the fetus. To avoid bolus infusion, oxytocin should be administered

with an infusion pump. In the absence of an infusion pump, there should be proper monitoring by the labour ward staff. Infusions should be gradually increased especially in grand multiparous patients to prevent uterine rupture.⁽¹⁵⁾ The half-life of oxytocin in the blood stream should be taken into consideration before increasing the infusion. The time needed to reach a steady-state concentration in plasma is 40 to 60 minutes after infusion.⁽¹⁶⁾ Increasing the rate of infusion at less than 30-minute intervals could cause hyperstimulation and therefore abnormal fetal heart rate pattern.⁽¹⁶⁾

Oxytocin does not cross the placenta and has no direct effect on the fetus but uterine hyperstimulation with a high resting tone between contractions can lead to uteroplacental hypoperfusion and fetal distress from hypoxia.

Water intoxication can occur because oxytocin is structurally and functionally related to antidiuretic hormone (ADH). Antidiuretic effect may be observed after prolonged induction with more than 40 Units of oxytocin.⁽¹⁷⁾

Augmentation of Labour

Implies that labour has already begun but the quality or progress is unsatisfactory. In deciding to augment labour, the diagnosis of labour must first be made to prevent false labour, which in most instances do not progress to established labour.

When false labour occurs at term many obstetricians fearing that such conditions may be associated with unrecognised fetal compromise, decide to intervene. Others simply observe and await the definite onset of labour. Whatever the case might be, diagnosis must be established so that the patients are not left alone without the necessary care. One must remember that there are many causes of unsatisfactory progress of labour and poor uterine contraction is just but one of these. Cephalopelvic disproportion, which is very common in our sub-region, may be responsible for poor cervical dilatation. Augmenting the labour of such patients leads to obstructed labour, uterine rupture and fetal demise.

Discussions And Controversies

One controversy is whether to rupture membranes before setting oxytocin or setting oxytocin before rupturing the membranes. Practically, both give good results. Sometimes, it is difficult to rupture membranes especially in nulliparous patients and constant infusion of dilute intravenous oxytocin and artificial rupture of the membranes after the initiation of labour appears to be a successful technique. Some obstetricians prefer to rupture the

membranes before starting oxytocic infusion because there is the theoretical risk of amniotic fluid embolism. This is not common but amniotic fluid embolism has been seen in some patients undergoing oxytocin-induced labour in the presence of fetal demise.

The timing of amniotomy during induction of labour is also controversial. Amniotomy could be reasonably performed when the cervix is about 3 to 4 cm dilated and the presenting part is engaged to prevent cord prolapse. Early amniotomy might reveal meconium-stained liquor but care should be taken to note umbilical cord presentation and also avoid dislodging the fetal head.

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**SECTION 3
COMPLICATED
PREGNANCIES
(MATERNAL COMPLICATIONS)**

Prolonged Pregnancy

E Ejiro Emuveyan

Definition

Prolonged pregnancy is pregnancy that exceeds 40 weeks (280 days) from known time of ovulation or conception. The expression post-dates and post-term are being used synonymously to describe a pregnancy, which exceeds 294 days (42 weeks) from the last menstrual period assuming a 28-day cycle (FIGO-1980).

Post-maturity

Post-maturity is another confusing term being used synonymously with prolonged pregnancy. This is a clinical syndrome in infants with clinical signs of intrauterine malnutrition, which may occur at any gestation. These signs include loss of subcutaneous fat, dry and cracked desquamated skin, meconium-stained skin and membranes, long fingernails, absence of lanugo hairs, respiratory distress, alert and apprehensive facies and increased body length in relation to body weight. Although the majority of infants delivered after 42 weeks are healthy, the increased morbidity and mortality in a small percentage of cases create significant stress for the patient, her family and those caring for her. Therefore, the obstetrician should reassure the patient and discuss the various options of management with her.

Incidence

Prolonged pregnancy occurs in 8-10% of pregnancies when based on the first day of the last menstrual period. It has however been shown that accurate dating in early pregnancy can reduce the incidence to as low as 1%.

Predisposing Factors/Aetiology

The actual cause of prolonged pregnancy is not known. However, several factors have however been associated, mainly:

Hormonal Influence: Fetal anencephaly has been found to often lead to prolonged pregnancy. It is therefore hypothesized that there may be a dysfunction in the fetal pituitary-adrenal axis that is needed for initiation of normal labour. There is however little evidence of endocrinological defects in the majority of prolonged pregnancies.

Uterine Factors: Uterine Contractility has equally been shown to be diminished in some cases of prolonged pregnancy suggesting a cause intrinsic to the myometrium

Seasonal Variation: Pregnancy is said to be longer in the summer than the winter months, with an average difference of 2.5 4days (Boe 1951).

Living Standard: Prolonged pregnancy has been associated with improved living standard (Boe 1951). There is however no concrete evidence to justify this.

Hereditary/Racial Factors: Prolonged pregnancy tends to recur in successive pregnancies in the same woman and often runs in families. About 50% of patients having one prolonged pregnancy will experience it with their next gestation.

Black women have been found to be less predisposed to prolonged pregnancy than their caucasian counterparts.

Placental Ageing: This has been implicated though no concrete evidence has been found.

Diagnosis of Prolonged Pregnancy

In order to avoid unnecessary intervention, accurate dating of pregnancy is essential. This can be established from the history, physical examination and investigations.

History: A woman's last menstrual period (LMP) is a good predictor of the date of confinement (Anderson et al 1981). As useful as this is, a large proportion of women are uncertain of their date of the last menstrual period. Basal body temperature charts or other symptoms and signs of ovulation, including Mittelschmerz pain may also be of help.

In a woman with regular menstrual cycle, a history of one or more missed periods especially if associated with fatigue, nausea, vomiting and breast tenderness strongly suggests pregnancy. Urinary frequency caused by the enlarging uterus is another common symptom.

Furthermore, seeking information on menstrual pattern, the use of ovulation-induction agents or recent discontinuation of hormonal contraception may be beneficial.

Quickening defined, as the date when the patient first feels fetal movements for three consecutive days is useful though of less value in dating. This is because it has a wide range and differs from primigravidae to multigravidae 15-22 weeks and 14-22 weeks respectively.

Early pregnancy is associated with dietary cravings in some women. Some may be due to the patient's perception that a particular food may alleviate nausea and heartburn.

Physical Examination

On physical examination, softening and enlargement of the pregnant uterus become apparent 6 or more weeks after the last normal menstrual period. A pelvic examination in early pregnancy is one of the better ways to date pregnancy.

Beginning at about 12-14 weeks gestation, the uterus is enlarged sufficiently to be palpable in the lower abdomen. Until approximately 18-20 weeks, the uterine size is generally stated as 'week size' such as 12 week size, 15-week size etc. Beginning at 18-20 weeks gestation when the fundus is palpable at or near the umbilicus, the uterine size can be assessed with the use of tape measure fundal height measurement. Up to 36 weeks, the number of weeks of gestation corresponds closely with the fundal height in centimetres in the normal singleton pregnancy. Thereafter, the fetus moves downward into the pelvic beneath the symphysis ("lightening") and fundal height measurement is less reliable.

Other genital tract findings early in pregnancy include congestion and bluish discolouration of the vaginal CHADWICK'S sign and softening of the cervix HEGAR's sign.

Investigations

(i) **Laboratory:** Measurement of human Chorionic Gonadotrophin (hCG) (A hormone produced by the syncytiotrophoblast of the growing placenta) in the serum and urine can be employed in detection of early pregnancy. Standard laboratory urine pregnancy test becomes positive 4-6 weeks following the 1st day of the LMP.

Serum pregnancy tests are more specific and sensitive because they test for the unique α subunit of hCG. This allows detection of pregnancy very early in gestation even before the patient has missed a period. Further information about a pregnancy may be obtained by the quantification of hCG. Curves of normal quantitative values of α hCG and the expected rate of production can help differentiate normal from abnormal pregnancies. After 12 weeks the rate of α hCG changes no longer correlate with advancing gestational age.

(ii) **Ultrasound:** Ultrasound scanning is now the best method available for dating a pregnancy. With transabdominal scan, a gestational sac is initially seen 5-6 weeks after the beginning of the last normal menstrual period (corresponding to α hCG concentration of 5,000-6,000 IU/ml).

The newer transvaginal technique can detect intrauterine gestational sac at 3-4 weeks (corresponding to α hCG concentration of 1,500-2,500 mIU/ml). By 6-7 weeks, the embryo should be visualised by all techniques, and cardiac activity detected. From around 9 weeks onward transabdominal ultrasonic measurement of the crown-rump length can be used to predict accurately the expected date of delivery.

At around 12 weeks, the fetus develops a kyphosis and the crown-rump length measurement loses its accuracy. The biparietal diameter, femur length, head circumference and abdominal circumference all become more relevant. The accuracy of pregnancy dating from 12-20 weeks by ultrasound is probably almost as good as that carried out earlier.

(iii) **Roentgenography:** The appearance of ossification centres in fetal bones may be used to date pregnancy. The distal femoral epiphysis can first be detected between the 35th and 40th week and by term is present in 95% of fetuses. The proximal tibial epiphysis appears between the 37th and 42nd week and by term is present in 75% of fetuses. In the third trimester, prediction of gestational age without prior information is more difficult. Radiological detection of fetal femoral and tibia epiphyses have been used in the past.

Roentgenography is no longer used for dating pregnancy because of the risks associated with it the fetus is exposed to total body radiation and also because more accurate and safer alternatives are available.

(iv) **Miscellaneous Investigations** Amniotic fluid sampling has equally been used to confirm fetal maturity. Measurement includes fetal fat filled cells, amniotic creatinine and lecithin/sphingomyelin ratio. These methods however are now less useful because of their invasive nature and the availability of more reliable and safer tests.

Implications Of Prolonged Pregnancy

Prolonged pregnancy poses a number of risks to the fetus and is associated with increased fetal and neonatal morbidity and mortality. It also has implications for the mother.

1. Fetal Implications

(i) Antepartum

- * Oligohydramnios: Amniotic fluid volume is said to decrease by about 33% each week past the expected date of delivery. Oligohydramnios has been implicated in the aetiology of fetal jeopardy in cases of prolonged pregnancy. This is partly because of the increased likelihood of cord compression.
- * Meconium stained amniotic fluid. The incidence of meconium staining of the amniotic fluid is higher in prolonged pregnancy than normal term pregnancy.
- * Fetal macrosomia There is the likelihood that the more prolonged the pregnancy the larger the weight of the baby.
- * Fetal dysmaturity syndrome - There is an association between the features of dysmaturity syndrome and increased perinatal morbidity and mortality as seen especially in meconium aspiration syndrome and dystocic labour.
- * Fetal death (stillbirth): The perinatal mortality rate falls starting from the 37th week and reaches the lowest levels just before 41 weeks. Thereafter, it rises sharply again to attain a peak at 43 weeks.
- * Studies on evaluation of gestation specific risks of fetal and infant mortality have shown a direct proportional relationship between gestational age and stillbirth, neonatal death and post-

neonatal death rates. These rates rise at 37 weeks to attain a peak at 43 weeks.

(ii) Intrapartum

- * Increased incidence of meconium staining of liquor
- * Increased incidence of meconium aspiration
- * Birth injury from shoulder dystocia or fetal macrosomia.
- * Intrapartum fetal death (stillbirth).

2. Neonatal And Early Childhood Implications

- (i) Nerve palsies e.g. Erb's palsy from birth trauma
- (ii) Neonatal death
- (iii) Post neonatal death: It has been found that childhood mortality is increased for up to two years after delivery in prolonged pregnancies.

3. Maternal Implications

- (i) Maternal anxiety.
- (ii) Maternal trauma from difficult delivery (e.g. with a macrosomic infant).
- (iii) Increased caesarean section rate, which carries with it, morbidity, not only for the index pregnancy, but also for subsequent pregnancy.

4. Legal Implications

- (i) Paternity disputes: Prolonged pregnancy may give rise to doubts about paternity of a child especially if the couple did not cohabit for period deemed longer than the normal duration of pregnancy.
- (ii) Malpractice suits.
- (iii) Prolonged pregnancy is one of the obstetric problems commonly associated with legal suits for perceived malpractice.

Management

The first step in management of prolonged pregnancy is a careful review of the information used to establish the gestational age to be as certain as possible that this is correct. Once the diagnosis of prolonged pregnancy is established, the management options are either to induce or to continue surveillance of fetal well being until spontaneous labour occurs or there is need for intervention.

If gestational age is felt to be firmly established, factors that influence the decision of whether to deliver or not rest with the patient's concerns and desires, the assessment of fetal well being and the state of the cervix. Induction of labour is

appropriate if the cervix is favourable softened, effaced and somewhat dilated with the patient's informed consent. If the cervix is not favourable, it should be ripened prior to induction of labour.

Antenatal Surveillance

Antenatal assessment of fetal well-being can be carried out using several methods but none seems to be wholly satisfactory.

Clinical assessment based on palpation of the abdomen, symphysis - fundal height measurement and the impression of Oligohydramnios gives a poor prediction of fetal well being in utero.

Fetal movement count: The recognition that reduction in maternally perceived fetal movement may proceed fetal death by a day or longer led to the development of the Cardiff count to ten, (fetal kick chart). Though it is a popular and inexpensive method, it does not always predict fetal compromise in time for appropriate action to be taken and maternal compliance may be poor.

Antenatal Cardiotocography: is needed for a more specific assessment of the fetal state.

Biophysical Profile: A biophysical profile done twice weekly is also widely used to assess fetal well being in utero in prolonged pregnancies. Five parameters are assessed Quantitative amniotic fluid volume, gross fetal movement, fetal breathing movements, fetal tone and fetal heart reactivity. Twice weekly scores will differentiate precisely between normal fetuses and those at risk of intra-uterine hypoxia. Although, the biophysical profile has been shown to have a low false negative rate, fetal death, do still occur within one week of a normal test.

Doppler ultrasound measurement of umbilical artery flow velocity waveform differentiates between normal and high-risk pregnancies. Doppler ultrasound showing absence of end-diastolic wave seems to be the most sensitive predictor of intra-uterine compromise.

Intrapartum Management

Patients with prolonged pregnancies should be advised to come promptly to the hospital when labour pains commence. Once the onset of regular contractions occur, careful electronic fetal monitoring should be employed throughout labour because of the risk of fetal compromise. Intrapartum management include artificial rupture of membranes, where indicated, to allow detection

of meconium and to allow the placement of a fetal scalp electrode and intrauterine pressure catheter. Due to the increased risk of caesarean delivery for fetal distress and because of the potential problems associated with meconium, both anaesthetist and neonatologist should be alerted.

At the time of delivery, special precautions are taken. If meconium passage has been noted, the infant naso and oropharynx should be suctioned prior to delivery of the shoulders, since compression of the chest has forced fluid within the chest upward to the pharynx.

Neonatologist should be present at delivery. This mode of management significantly decreases but does not eliminate the likelihood of meconium aspiration syndrome.

In addition, because of the risk of macrosomia, a skilled obstetrician should undertake the delivery.

Conclusion

In most cases, prolonged pregnancy probably represents a variant of normal and is associated with a fair outcome, regardless of the form of care given. In a minority of cases there is an increased risk of perinatal death and early neonatal convulsions.

Induction of labour at less than 41 weeks gestation is not associated with any advantage apart from a small reduction in meconium staining of the amniotic fluid. The reduction in perinatal death appears to be confined to pregnancies of 41+ weeks' duration. A policy of routine induction at 40-41 weeks in normal pregnancies cannot be justified in the light of the evidence from controlled trials, and is unacceptable to many mothers.

Induction of labour after 41+ weeks' gestation is not associated with any major disadvantage and reduces the risk of perinatal death in women with prolonged pregnancies. Provided that appropriate induction methods are used, it may also result in a small reduction in the high risk of caesarean section run by women with prolonged pregnancies.

In the light of the available evidence, the best policy is to offer women a choice of induction of labour by the best method available once the duration of pregnancy has with certainty attained 41 weeks or more.

Obstetricians and parents should also be aware of the poor quality of the evidence available to support the use of all methods of fetal surveillance commonly offered to women with prolonged pregnancies.

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Antepartum Haemorrhage (APH)

E Y Kwawukume

Introduction

Any bleeding through the genital tract is alarming to the pregnant woman and needs immediate evaluation. Sometimes bleeding might be inconsequential but most often it signals a serious condition.

Antepartum haemorrhage, just like any other obstetric emergency carries an increased risk of maternal and perinatal morbidity and mortality. The risk increases more so because the obstetrician is managing at least two individuals, the fetus(es) and the mother. An attempt is made to optimise the outcome for both of them.

What is best for the mother might not be best for the fetus. Preterm delivery might result in prematurity with great increases in perinatal morbidity and mortality whereas continuing the pregnancy might put the mother at risk from bleeding. On balance, the decision to continue the pregnancy or to terminate it requires a fine judgement of the risks and benefits of all the management options for both the mother and the baby.

The placenta contains maternal blood within the intervillous space, which is from the uterine and ovarian arteries. There is close adherence of the placenta to the maternal decidua and the open arterioles discharge into the choriodecidual space, which remains intact until the delivery of the fetus, so long as the placenta is inserted into the upper segment of the uterus. During effacement and dilatation of the cervix there is separation of the chorion, from its attachment to the myometrium. This might cause a small amount of bleeding or "a show". However if the placenta is inserted either wholly or partly into the lower segment (placenta praevia) bleeding may be heavy. If the placenta is normally situated then separation from the decidual bed may lead to massive haemorrhage and further separation (placental abruption).

Definition

Antepartum haemorrhage can be defined as bleeding from the genital tract after the 28th week of gestation until the delivery of the baby. However there are advances in paediatric care of the very low birth weight infants so that even infants born as early as 24 weeks' gestation now have a better chance of survival. This might mean that

intervention in pregnancy for the sake of the infant could now be undertaken before the 28 weeks of gestation.

The 28 weeks gestation is convenient probably because of viability and registration of stillbirths. The International Federation of Gynaecology and Obstetrics (FIGO) has recommended that perinatal death statistics should include any fetus born after 22 weeks or weighing 500 g or more. It might therefore be considered that 22-24 weeks should form the limit between the definition of APH and early bleeding in pregnancy. Taking weeks of gestation into consideration depends on the facilities at the Neonatal Intensive Care Unit (NICU) at various centres and countries. In Africa 28 weeks is generally accepted as the landmark between early pregnancy bleeding and antepartum haemorrhage.

The incidence of Antepartum haemorrhage (mainly placenta praevia and abruptio placenta) in Korle Bu Teaching Hospital, Accra is about 1.2 to 1.8 % of total births. However, this figure may be influenced by the number of patients referred to this hospital since it serves as a referral hospital for the southern part of Ghana and beyond.

Causes

1. Placenta praevia
2. Placenta abruption or abruptio placenta
3. Local causes
 - * Polyps
 - * Friable condyloma acuminata
 - * Cervicitis
 - * Carcinoma of cervix
4. Vasa praevia
5. Circumvallate placenta
6. Unknown causes

The most common causes of APH are placenta praevia and placental abruption, which are potentially fatal leading to maternal/fetal morbidity and mortality. The condition requires prompt assessment to determine between conservative management and immediate delivery.

Although lesions of the vagina and cervix are rare causes of heavy bleeding, cervical carcinoma is a serious condition that should be excluded.

Placenta Praevia

Is defined as placenta that is wholly or partially located in the lower uterine segment. The extent of attachment to the lower uterine segment determines both the management and prognosis.

Placenta praevia can be classified into 4 types, mainly:

Type 1. Placenta is partially located in the lower segment. Part of the placenta can be in the upper segment but the part in the lower segment does not reach the internal Os.

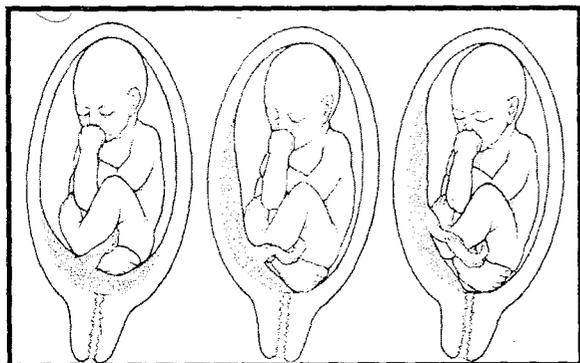
Type 11. Placenta is attached to the lower segment with its lower margin reaching the Internal Os but does not cross it to the opposite side. Type 11 is divided into a and b, that is, anterior and posterior respectively

Type 111. The placenta eccentrically reaches the Internal Os and crosses to the other side but does not completely cover the Os. In this case the placenta covers the undilated Internal Os but not necessarily during cervical dilatation.

Type 1V. The placenta completely covers the Internal Os when the cervix is dilated or undilated. This is complete or central placenta praevia.

The above classification is only descriptive and might give the impression that placenta praevia is diagnosed by vaginal examination. This is not true since vaginal examination is contraindicated in placenta praevia. Others might classify placenta praevia as follows:

- Complete or total implantation of the placenta across the cervical os
- Partial placenta covers part of the internal os
- Marginal placenta just reaches the edges of the internal os



The fig. above shows the three main types of placenta praevia.

Low-lying placenta is placenta implanted in the lower uterine segment but not reaching the cervical os, and it has similar clinical manifestations as the above classification. In general, these terms have limited prognostic value and do not necessarily predict the different bleeding patterns. All are associated with potential life-threatening haemorrhage during labour, and classifications have become less important as Caesarean section has become the preferred method of delivery for most patients with any degree of praevia with bleeding episodes.

A total placenta praevia is usually associated with a greater blood loss than either partial or marginal praevia^(1,2)

Predisposing factors^(3,4,5,6)

1. Advanced maternal age
2. Increased parity
3. Previous uterine surgery including caesarean section.
4. Intrauterine synechiae
5. Multiple pregnancies
6. Abnormality of endometrial vascularisation
7. Prior trauma to the endometrium or myometrium including DD&C.

It has also been postulated that smoking increases the risk of placenta praevia about two-fold.^(7,8,9) Reports of the relationship between risk, quantity and duration of smoking vary. The relative carbon monoxide hypoxaemia associated with smokers, may result in compensatory placental hypertrophy with increased placental surface area that might cover a greater area in the lower segment and thus cover the internal os

Presentation

Placenta praevia is the leading cause of third-trimester bleeding and classically presents as painless, bright red vaginal bleeding in about 70 % of cases. Fifteen percent have bleeding associated with uterine activity and the remaining patients are diagnosed incidentally at the time of Caesarean section or at ultrasound examination performed for another indication.^(10,11) As pregnancy advances, Braxton Hicks contractions may cause the lower segment to thin and even result in dilatation of the cervix. Bleeding might be recurrent and unprovoked and the patient might feel wet and wake up to discover that she has been bleeding. Occasionally, bleeding might occur in the second trimester and may be minor, but since the lower uterine segment has poor contractile ability, the bleeding from placenta praevia might be torrential. Bleeding is usually severe after 34 weeks. With

routine ultrasonography in early pregnancy, many cases of placenta praevia might be suspected before any bleeding occurs.

The mechanism of bleeding involves progressive stretching of the lower segment, which normally occurs during the 3rd trimester and labour but the inelastic placenta cannot stretch with it. This leads to inevitable separation of part of the placenta with unavoidable bleeding. The closer to term, the greater is the amount of bleeding.

Diagnosis

Placenta praevia may be asymptomatic but more often there is recurrent painless bleeding associated with a soft abdomen with no tenderness. The fetal heart is usually audible. The presenting part of the fetus may be high and freely mobile since the placenta is in the lower segment and acting as a pelvic tumour. A deeply engaged presenting part is reliable evidence of minor degree placenta praevia.

In addition, there might be abnormal lie and malpresentation, such as breech, oblique, transverse and unstable lie, which might even be detected before any bleeding episode.⁽¹²⁾

Vaginal examination is contraindicated immediately when a patient presents with antepartum haemorrhage since it might provoke serious bleeding. Vaginal examination if necessary should be done in theatre where preparations are made for Caesarean section. This might be done if there is suspicion of placenta praevia or placental abruption. But if facilities are available for ultrasonography, and placenta praevia and placental abruption, which are serious causes of APH are eliminated then a gentle speculum examination should be performed to detect local causes such as carcinoma of the cervix.

Placental localisation Ultrasonography

Prior to the introduction of Ultrasound in Obstetric practice, placenta localisation was done by radiography and isotope techniques. These are now outmoded because of the accuracy, safety, and ease with which placenta is now located by ultrasonography. Posterior placenta praevia might be difficult to identify due to shadowing from the presenting part of the fetus. This can be overcome by head-down tilt of the patient or displacing the presenting part manually. If difficulty is still present, the distance between the presenting part and the promontory of the sacrum could be measured. If this exceeds 1.5 cm, it might mean that there is soft tissue or placenta interposed in-between.

The diagnosis of placenta praevia in the 1st and 2nd trimesters could be misleading because of the theory of placental migration. It has been observed that the placenta was low-lying in 28 % of women who were scanned before 24 weeks of gestation; this figure dropped to 18 % after 24 weeks reaching 3 % at term.⁽¹⁵⁾ Placental migration could be due to 3 factors:

1. Difficulty in delineating the cervix and the cervical canal especially if the bladder is not adequately filled.
2. The lower segment elongates during pregnancy, carrying with it an attached placenta.
3. Placentation is a dynamic process with growth on the decidua side and atrophy on the membranous areas, such that the placenta actually might "migrate" to some degree of the sonographer cannot clearly delineate the cervix then placenta in the lower segment should be reported as "low-lying placenta, possibly marginal or partial praevia;" But if the placenta is absolutely central covering the cervical canal, it might not migrate and praevia can be diagnosed.

Ultrasonography is now the commonly used method of choice for localisation of the placenta. The other methods that were formally used have disadvantages. Some of them are:

- Pelvic arteriography: This gives the best outline of the placenta but is painful and potentially dangerous
- Using counters such as Geiger Muller counter, involves the injection of radio-isotope but the picture does not outline the lower edge of the placenta clearly
- Soft-tissue placentography: It involves lateral radiograph of the soft tissues of the pelvis taken while the patient is standing. The radiologist looks for any displacement of the fetal head away from the promontory or the symphysis pubis. Significant displacement of the head indicates that placenta is interposed between it and the bony pelvic inlet.

2. Magnetic resonance imaging (MRI)

This may be the most precise method of diagnosing placenta praevia but it is an expensive method of tissue imaging. It does not use ionising radiation and it is safe. Placenta localisation is perfect since both the placental edge and the cervical canal can be readily identified. Unfortunately, in using MRI,

the tissues need to be still for some seconds while the imaging is performed therefore clear fetal images are difficult to delineate. At present MRI equipment is available in very few centres in Africa.

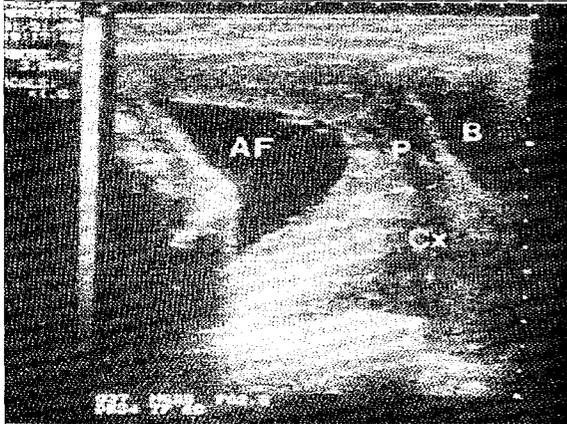


Fig 19.2
Major placenta praevia, AF: Amniotic fluid
P: Placental, B: Bladder, Cx: Cervix

Vasa Praevia

This is bleeding from fetal vessels. It often results from velamentous insertion of the umbilical cord. The cord inserts at a distance from the placenta and it is not protected by Wharton's jelly. The umbilical cord vessels traverse between the chorion and amnion without protection and might cross the os. Spontaneous or artificial rupture of membranes or dilation of the os might lead to rupture of these fetal vessels.

Bleeding from the fetal vessel is usually associated with abnormal fetal heart pattern and delivery should be rapid by emergency caesarean section. The total blood volume of a term fetus is approximately 375 ml. Shock in the fetus will occur with blood loss of approximately 72 ml. ⁽¹³⁾ This amount of blood loss can occur within minutes of ruptured vasa praevia. A high index of suspicion is required to diagnose and rapidly manage ruptured vasa praevia.

The incidence is approximately 1 per 5,000-singleton deliveries. ⁽¹⁴⁾ Fetal mortality is very high about 75 to 100 % of cases of rupture of these vessels ⁽¹³⁾ **The Apt test** is used in the diagnosis of vasa praevia by mixing suspected bloody vaginal fluid with water and after centrifuging, the supernatant is mixed with 1% NaOH. A pink colour after another centrifuge indicates the presence of fetal blood. The physiology behind this test is that fetal oxyhaemoglobin is more resistant to alkali than adult oxyhaemoglobin and adult haemoglobin is converted to alkaline globin haematin (denatured).

Expectant Management

This is for fetuses remote from term. If the bleeding is slight and the gestation is less than 37 weeks, conservative treatment is indicated till the end of 37 weeks. The patient is kept hospitalised with bed rest and observed till delivery after 37 completed weeks of gestation. It is essential to correct anaemia if present. The haemoglobin, haematocrit and the sickling status of the patient should be done. There should be blood transfusion if the patient bleeds significantly.

Haematinics

This will maintain the maternal haemoglobin above 10.0 gm/dl and provide a margin of safety in the event of heavy bleeding. At least 2 units of cross-matched blood should be saved for emergency situations. The essence of this approach is to maintain the fetus in a healthy intrauterine environment without jeopardising maternal condition.

In placenta praevia, maternal haemorrhage is most feared but fetal blood can also be lost during the process of placental separation. Blood group and Rhesus factor (Rh) should be checked and if the mother is Rh negative, anti-D immune globulin should be administered.

Use of tocolysis

In some patients with placenta praevia there will be evidence of preterm contraction or labour. This is difficult to diagnose because vaginal examination to document cervical dilatation is absolutely contraindicated. Tocolysis could be given. They are safe and cost effective if contractions are present. ^(16,17) Uterine contractions in patients with placenta praevia may trigger bleeding, which might compromise the life of the mother and the baby. The danger of using tocolytic agents is that they might produce tachycardia and palpitations, neither of which is desirable in the presence of maternal hypovolaemia. Furthermore, they are contraindicated in placental abruption therefore the diagnosis of placenta praevia must always be established.

If there is intermittent bleeding remote from term and threatening delivery, steroids should be given for lung maturity. Tocolytic agents can also be given to allow steroids to take effect before delivery. The use of antenatal corticosteroids to accelerate fetal pulmonary maturity is effective. ^(18,19,20,21)

Conservative management with bed rest preferably in hospital is beneficial if there is a major placenta praevia or the mother is not haemodynamically stable. The fetal heart tones should be checked at least twice daily and fetal kick count instituted if

there are no facilities for nonstress testing. The sanitary pad should be inspected often to assess bleeding and blood should always be available for maternal transfusion in the event of sudden haemorrhage.

If the patient is in labour, **vaginal delivery** is allowed if the placenta is type 1 or 11a, vertex presentation and adequate pelvis with no soft tissue obstruction. If the cervix is dilated amniotomy should be done. Amniotomy has two benefits mainly because it allows the descent of the head to compress the placental site and thus prevent further bleeding. Secondly, it abolishes the shearing movement of the placenta during uterine contraction. In addition the bulging of the forebag of water during contractions with intact membranes will drag the edge of the placenta evoking more bleeding.

Caesarean section is indicated when:

- Placenta partially (type 111) or completely covers the internal os (type 1V) even if the fetus is dead
- Posterior placenta attached to the lower segment so that its lower margin reaches down to the internal os but not crossing it. (type 11 posterior). This is because the placenta encroaches on the true conjugate diameter delaying engagement of the head and engagement of the head will compress the placenta or the umbilical cord against the sacrum, causing fetal asphyxia.
- Any placenta in the lower segment with repeated bleeding episodes.
- Severe bleeding
- Presentation other than vertex
- Other obstetric indications such as contracted pelvis and cord prolapse.

Although upper segment Caesarean section is sometimes advocated in order to avoid the placenta, lower segment Caesarean section is preferable because it allows better control of bleeding from the placental site and also leaves a stronger scar that can withstand subsequent vaginal delivery. If the placenta praevia was anteriorly implanted it is gently displaced laterally to reach the membrane, which is ruptured to deliver the baby. Cutting through the placenta may exsanguinate the fetus resulting in fetal mortality if the fetus is not delivered quickly.

Double set-up examination

Double set-up is performed to rule out placental implantation in the lower segment. Many times, there is no ultrasound facility or trained personnel to localise the placenta or there are blood clots at the end of the placenta with poor visualisation of the edge of the placenta.

Double set-up is performed at term (thirty-seven completed weeks of gestation) where there is suspicion of placenta praevia. It is done in the operating theatre in the presence of two obstetricians. One obstetrician performs the digital examination and the other is scrubbed and gowned in preparation for Caesarean section if excessive vaginal bleeding occurs. The anaesthetist and the operating room should be set-up for Caesarean section. Blood should be taken for grouping, cross matching, clotting factors and intravenous infusion should be commenced.

Double set up is less commonly used in modern obstetric practice because of improvements in ultrasound resolution, which can localise the lower margins of the placenta

Procedure for Double set-up

After general anaesthesia the patient is positioned in the dorsal position and the surgeon ensures that the patient's bladder is empty.

The fore and middle fingers are inserted into the vagina and the fornices palpated gently. Fullness in the fornices suggests the presence of placenta extending down toward the cervix. If the fornices are free, the index finger is carefully introduced into the cervical os to explore the lower segment. The procedure should be abandoned if placental tissue is felt.

If no placenta is felt and no significant bleeding occurs and the cervix is favourable, amniotomy is performed and oxytocin infusion is commenced. The patient is transferred to the first stage room to await labour. The reason being that the baby is mature and there is no point continuing intrauterine management. The main disadvantage is that induction carries the risk of caesarean section with its associated complications.

If the cervix is not favourable for induction of labour, the patient should be sent to the ward for ripening of the cervix and induction of labour. In sending the patient to the ward, the chances of the cervix getting ripe are increased for vaginal delivery. This last measure would reduce the risk of failed induction and the resultant caesarean section

Placenta membranacea. This is a rare abnormal placental development in which there is

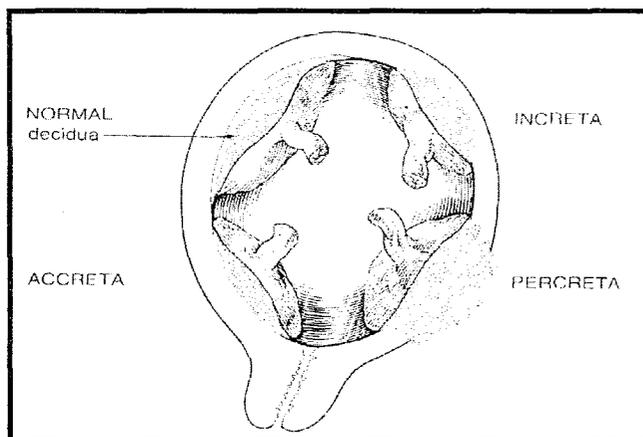
no differentiation of placenta into chorion laeve and frondosum. The chorion remains covered with villi and is sometimes in the lower segment causing vaginal haemorrhage and postpartum haemorrhage.

The complications associated with this condition are life threatening maternal haemorrhage, increased Caesarean delivery, and increased risk of placenta accreta, maternal/fetal morbidity and mortality. It can also result in increased risk of postpartum haemorrhage

Abnormal Placental Adherence

Morbidly adherent placenta in the form of placenta accreta, placenta increta and percreta are sometimes encountered. Though they are very rare in our sub region we do experience some of them and one should be aware to institute management as early as possible.

1. Placenta accreta. This is when the placenta is directly attached to the uterine muscle without intervening decidua. Diagnosis is finally histological where specifically there is absence of the decidua basalis.
2. Increta - placenta penetrates into the myometrium
3. Percreta - placenta penetrates through the uterine wall to the serosa.



Uteroplacental relationship in abnormal placentation

Some of the **predisposing factors** for abnormal placental adherence are uterine scar, placenta praevia, prior caesarean section, submucous fibroid, intrauterine synechiae and chronic endometritis. This abnormal placental adherence

can lead to complications such as uterine inversion and rupture, during placental removal and hysterectomy for incompletely removed placenta with maternal haemorrhage.

In the presence of a scarred uterus, such as previous caesarean section or previous myomectomy, one has to be careful in the evaluation of suspected placenta praevia. This is because the risk of morbidly adherent placenta or placenta accreta increases above that for placenta praevia alone. In the absence of a scarred uterus the risk of accreta at the time of delivery with placenta praevia is 1-5 %, which increases to 25 % if there has been one previous caesarean section and as high as 45 % when there have been two or more previous uterine surgeries ⁽²²⁾.

Placental Abruption (abruptio Placentae)

Placental abruption is premature separation of a normally situated placenta before the delivery of the fetus. Bleeding could be external or concealed. The incidence of abruptio placentae is approximately 1.1 % in Korle Bu Teaching hospital, Accra and nearly 95 percent result in perinatal deaths.

Placental abruption can occur with slight vaginal bleeding and some uterine irritability without any change in maternal blood pressure. This is mild abruptio and maternal fibrinogen levels and fetal heart pattern might be normal. In severe cases bleeding will be severe and could be concealed or revealed.

The uterus is tetanic and painful with maternal hypotension and frequently fetal death. Fibrinogen levels are often reduced to less than 150mg percent with thrombocytopenia and other coagulation abnormalities

Pathophysiology

Abruptio placentae could be either concealed or revealed after spontaneous rupture of the blood vessels at the placental bed with haematoma formation. In the concealed type there is a lot of pressure at the placental bed because the blood does not decompress through the vagina and the adjacent myometrium is unable to contract around the torn vessels.

Bleeding therefore continues with progressive placental separation. Some blood might dissect into the myometrium causing the **Couvelaire uterus**.

With the disrupted placental site there is deranged metabolic exchange resulting in fetal hypoxia and probable death. This process might continue with the release of tissue thromboplastin into maternal circulation and microvasculature. Disseminated Intravascular Coagulation (DIC) sets in, which might trigger the coagulation cascade system with depletion of fibrinogen, platelets and other clotting factors. Consumptive coagulopathy continues and finally results in inappropriate bleeding, which would further compromise the maternal condition.

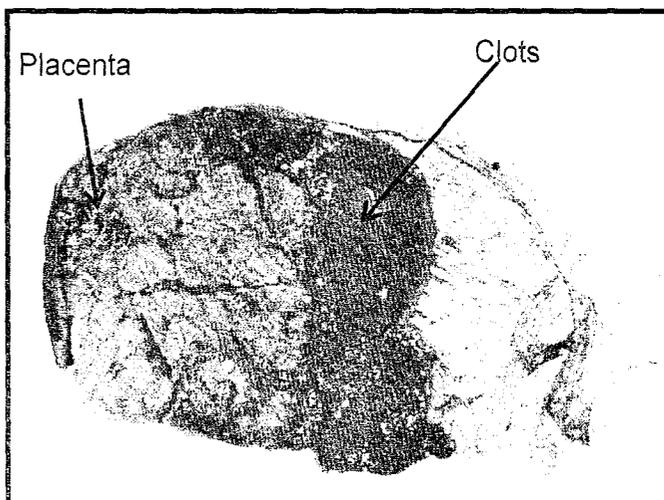
Aetiology

The primary aetiology of abruptio placentae remains unknown but maternal hypertension seems to be the most consistent predisposing factor.⁽²³⁾ Intrapartum hypertension significantly increases the risk of abruption and this is most marked in severe abruption. Among patients with abruption as many as 44% have hypertension.⁽²⁴⁾ Over 50% of severe abruptions fatal to the fetus are associated with maternal hypertension.

Both chronic hypertension and pregnancy-induced hypertension contribute to the incidence of abruption. Others have associated abruption with abnormalities of the uterine blood vessels⁽²⁵⁾ and unexplained increases in the maternal serum alpha-feto protein (AFP) concentration. This might suggest the possibility of a defect at the decidual-placental interface.⁽²⁶⁾ Other contributing factors are maternal trauma, which happens in about 1.5 to 9.4 percent of abruptions.^(27,28) Advanced parity and maternal age have been implicated and probably age could be a reflection of the effect of parity.

Other conditions that have been associated with placental abruption include rupture of fetal membranes, which might be related to rapid decompression of uterus. The two clinical conditions that can cause abruption are multiple gestations and polyhydramnios.

Rapid decompression of the uterus should be avoided in a patient with polyhydramnios by slowly releasing amniotic fluid by amniocentesis before the induction of labour or when spontaneous labour has been established. Uterine fibroids, especially if retroplacental, and smoking, which is not common in our environment, are thought to cause abruption because of decidual necrosis. Severe trauma in the presence of major maternal injuries can cause abruption in about 35% of cases⁽²⁹⁾.



Abruptio Placenta Showing Retroplacental Clots

Diagnosis

The initial diagnosis of abruptio placenta is usually based on the clinical presentation. Vaginal bleeding in the third trimester of pregnancy with painful uterine contractions is the hallmark of placental abruption. Bleeding could be concealed or revealed and about 80 percent of patients who eventually have abruptio exhibit some degree of vaginal bleeding. The remaining 20% have a concealed abruption and are commonly diagnosed as having preterm labour.⁽³⁰⁾

Ultrasonographic diagnosis of abruptio placenta can be disappointing because fresh clot might not show the typical sonographic picture of retroplacental clot. Retroplacental haematomas are most likely to be hyperechoic or isoechoic compared with the placenta. The haematoma becomes hypoechoic within a week and sonolucent within 2 weeks.⁽³¹⁾

These haematomas may be confused with uterine myomas, chorioangioma, or succenturiate placental lobe. In addition, if the blood lost leaves the uterus through the cervical canal, there will be no sonographically detectable haematoma.⁽³²⁾ Studies have shown that retroplacental clot was found in only 1 of 59 patients. Negative ultrasonographic finding does not exclude abruptio placenta, therefore⁽³³⁾ ultrasound might be more useful in excluding placenta praevia, which is a potentially life-threatening cause of APH. If ultrasonography fails to show placenta praevia and if other local causes of vaginal bleeding such as, cervical or vaginal trauma, labour, malignancy, have been ruled out, placental abruption becomes a more likely diagnosis.

In severe abruption, the patient looks distressed and the abdomen is tender and the uterus is woody hard. The fetal parts are usually difficult to palpate with fetal bradycardia or absent fetal heartbeat. If the placenta is situated posteriorly, tenderness is less marked and the patient complains of backache, there might be maternal hypotension associated with haemorrhage,

which might confuse the coexistence of hypertension with abruptio, whether it is cause or effect. Silent abruptio placentae are unsuspected and might be diagnosed in retrospect. With such a variation in clinical presentation, placental abruption can be classified depending on the clinical and labouratory findings as either mild (grade 1), moderate (grade 2), or severe (grade 3).

(34)

Management

The initial assessment of placental abruption should be swift and decisive since the prognosis for the fetus and the mother is worsened by delay. An intravenous line is set up and the infusion of a crystalloid solution begun. Blood should be taken for haemoglobin estimation, coagulation studies (platelet count, fibrin degradation products, PT, PTT) and grouping and cross matching. At the same time, 5ml of blood is drawn into a test tube and observed for whole blood clotting time. If the clot does not form within 10 to 12 minutes or forms and lyses within 30 minutes, a coagulation defect is probably present and FFP should be started before the coagulation results are received. At least 2 units of packed cells and 2 units of FFP should be made available to treat or guard against DIC. Blood loss

Grade of Abruptio Placenta	Clinical/Labouratory Findings
Mild	Slight vaginal bleeding Minimal uterine irritability Normal maternal blood pressure and heart rate No evidence of coagulopathy Normal fetal heart rate pattern
Moderate	Mild to moderate vaginal bleeding Tetanic contractions may be present Maintenance of maternal blood pressure Maternal tachycardia Orthostatic changes in blood pressure and heart rate Hypofibrinogenemia (150-250 mg%) Fetal distress
Severe	Usually heavy vaginal bleeding, but may be concealed Tetanic, painful uterus Maternal hypotension Hypofibrinogenemia (<150 mg%) Other coagulation abnormalities Fetal death

should be corrected as soon as possible. If facilities are available, central venous catheter is inserted to prevent under-transfusion before delivery and over-transfusion during the vital hours following delivery, especially in pre-eclamptic patients who have reduced circulating blood volume.

Oxygen should be given if necessary and an indwelling self-retaining catheter (Foley catheter) passed to monitor the urinary output, which should be at least 30 ml/hr.

If the pregnancy is at term and both the mother and fetus are stable, cautious induction with vaginal delivery may be attempted. Amniotomy might

hasten the onset of labour and, by encouraging uterine contractions, reduce uterine bleeding. If there is intrauterine fetal death (IUFD), the mother should be stabilised before vaginal delivery. If there is fetal distress and cervix is not fully dilated or there is severe maternal bleeding, Caesarean section must be performed.

If the fetus is preterm, the mother is haemodynamically stable, blood loss is minimal, and there is no evidence of fetal distress, careful expectant management with corticosteroids to accelerate fetal lung maturity can be initiated. The fetus should be continuously monitored till it is safe to deliver.

Complications

1. Maternal

- Life-threatening maternal haemorrhage and shock.
- DIC resulting from release of thromboplastin from the placental site and fibrinogen is converted to fibrin by activation of the extrinsic clotting cascade system.
- Increased caesarean delivery
- Increased risk of postpartum haemorrhage.
- Ischaemic necrosis of distal organs especially the kidneys, resulting in acute tubular necrosis (ATN) or bilateral cortical necrosis.
- Oliguria
- Uraemia
- Maternal death

2. Fetal

- Hypoxia
- Anaemia
- Growth retardation from conservative management
- Fetal death

Controversies

Preterm infant with placental abruption

A fetus remote from term with mild abruption could be monitored continuously aiming for lung maturity before delivery but it should be noted that a small placental abruption could stimulate uterine irritability and further separate the placenta. This is a challenging situation and there could be fetal demise.

Tocolytic agents such as magnesium sulphate or beta-adrenoreceptor agonists could be used to inhibit uterine contractions so long as there are no signs of fetal distress, but should be weighed against the likelihood of survival and mortality if the infant were delivered. If true preterm labour is encountered, tocolytics of any kind are unlikely to prolong pregnancy to any clinically significant extent.⁽³⁴⁾ Generally, sacrificing the fetus against maternal survival should be considered as against prolonging fetal survival in the presence of abruptio placenta. Considerable controversy still remains regarding the appropriate time of delivery. A number of patients present with a live fetus, only to have that fetus die undelivered while awaiting vaginal delivery. It is therefore suggested that once the maternal condition has been stabilised, the patient should be delivered by Caesarean section for increased fetal survival.

Caesarean delivery with a dead fetus

There is a place for Caesarean delivery when there is:

- * Severe uncontrollable maternal bleeding.
- * If blood cannot be replaced as rapidly as the loss because of lack of blood in the blood bank.
- * Prolonged labour with prolonged period of ruptured membranes.

If delivery is delayed the patient might bleed and lose all the clotting factors until she develops DIC with resultant fetal and maternal demise. If caesarean section is needed, then it must be performed before irreversible shock is established. However, if the patient is stable, vaginal delivery is the method of choice.

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Premature Rupture Of Membranes

E Y Kwawukume

Introduction

Premature rupture of membranes (PROM) still features in the majority of cases of neonatal morbidity and mortality and accounts for a great number of patient-days in infant care nursery. The reported incidence of PROM varies between 3 and 18.5 per cent of all deliveries. ⁽¹⁾ In the Korle Bu Teaching Hospital the incidence is about 3.7 % of total deliveries. There is the need to confirm this because of the increased risk of maternal and fetal infection and risk of cord prolapse if the presenting fetal part is not fixed in the pelvis. Management of PROM has long been controversial and obstetricians are faced with simply awaiting spontaneous onset of labour, induction of labour, and delivery by caesarean section when induction failed or delivery by primary caesarean section.

Definition

Premature rupture of membrane is defined as the spontaneous rupture of membranes prior to the onset of labour at any gestational age. It can be Term Premature Rupture of Membranes (TPROM) or Preterm Premature Rupture of Membranes (PPROM). Term PROM is rupture of membranes after 37 completed weeks of gestation and before onset of labour. Preterm PROM is rupture of membranes before 37 completed weeks of gestation. The latent period is the interval between rupture of membranes and the onset of labour. Prolonged rupture of membranes is rupture of membranes for more than 24 hours before onset of labour.

Aetiology

The aetiology of preterm PROM is not completely understood. Infection has been implicated in many cases. Indirect evidence showing that infection antedates preterm rupture of the membranes is provided by the observation that histologically apparent amniotic fluid infections are two-to-three fold more common when the fetal membranes rupture just before labour starts than when they rupture just after the onset of labour. ⁽²⁾ It has also been shown that amniotic fluid samples from patients with PPRM are more frequently

colonised with pathogens than are samples from patients without preterm rupture of the membranes. ⁽²⁾ In preterm, PROM infection of the choriodecidual interface often precedes rupture of the membranes while infection of the chorioamnion more often follows membrane rupture at term.

Other factors proposed include physical stress, bacteria and macrophage producing proteases, elastases, phospholipases and cytokines. Also implicated are eicosanoids that produce uterine irritability, cervical ripening, and membrane weakness and rupture.

The fetal membrane has a thin layer of amnion on the fetal part and a thicker outer layer of chorion on the maternal part. Interspersed between the amnion and chorion is a collagen-rich connective tissue. The two membrane layers together withstand greater bursting pressures than they do separately, though the amnion has greater tensile strength than the chorion.

As pregnancy progresses, the physical stress tolerated by the membrane decreases as well as the relative concentration of collagen. ⁽³⁾ Studies have suggested that some membranes rupture early because of local defects within the membranes ^(3,4,5,6,7) and the site of rupture is usually immediately above the cervix, an area of membranes that is poorly supported physically and nutritionally. Histology on intact membranes excised at the time of caesarean section at term show localised areas of weakness over the internal os and also in the area directly across from the placental site. ⁽⁸⁾ Organisms associated with PROM are usually polymicrobial such as *Neisseria gonorrhoea*; group B *Streptococci*, *Peptostreptococci*, *Trichomonas vaginalis*, *Chlamydia trachomatis*, *Escherichia coli*, *Bacteroides fragilis* and other anaerobes. Most of these genital tract organisms have been isolated twice as often in women with PROM. Reduced bursting pressure of membranes has also been demonstrated when membranes grown in tissue cultures were exposed to some of these organisms such as *E coli* and group B *Streptococcus*. ^(9,10)

RISK FACTORS IN PRETERM PROM INCLUDE:

1. Previous history of PROM
2. Preterm labour
3. Bacteria infection: cervicovaginitis and chorioamnionitis
4. Incompetent cervix
5. Uterine distension eg. Polyhydramnios and multiple pregnancy
6. Trauma
7. Amniocentesis
8. Prior cervical surgery e.g. cervical conization
9. Vaginal bleeding in the first or second trimester
10. Low socio-economic status
11. Poor nutrition
12. Weakening of the membranes may result from physiological changes combined with shearing forces created by uterine contractions
13. Emergency cervical cerclage
14. Connective tissue disorders such as Ehlers-Danlos syndrome

In the past several factors had been implicated as risk factors for PROM. Over the years with increasing researches some factors are no longer believed to be risk factors. Some of these are:

- Cervical examination
- Coitus
- Maternal exercise
- Parity
- Vitamin or mineral deficiencies
- Changes in barometric pressure
- Male fetal sex

Diagnosis

Most of the patients present with leakage of fluid or watery discharge from the vagina. This could present as a sudden gush or a persistent increase in vaginal moisture. It is important to note the time of rupture of membranes presence of fetal movements, and colour of fluid, whether clear, thin or thickly meconium stained. The amount of discharge and continued leakage of fluid should also be noted. Calculate the gestational age from the last menstrual period (LMP) or early ultrasound dating.

General examination should be performed and

fever and maternal tachycardia documented. In addition, examine the abdomen determine symphysis-fundal height, lie, presentation and level of presenting part. The uterus should be examined for tenderness and fetal tachycardia.

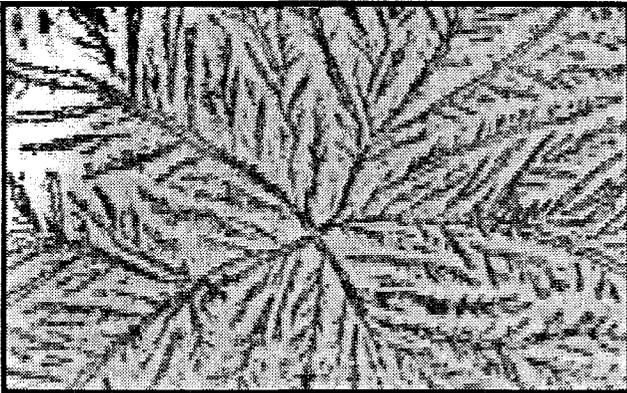
Never perform a digital vaginal examination because it would increase the chance of carrying microorganisms from the vagina into the endocervix and uterus, that is, ascending intraamniotic infection. Digital examination can, however, be done, if delivery is anticipated within 24 hours. Sterile speculum examination should be performed and amniotic fluid might be seen trickling from the cervix or pooling of amniotic fluid in the posterior vaginal fornix. If there is no fluid present encourage the woman to perform the Valsalva manoeuvre, which might allow visualisation of amniotic fluid flowing from the cervix.

Test the pH of the amniotic fluid with litmus or nitrazine paper if PROM is still in doubt. Litmus paper will turn blue and nitrazine changes from yellow to blue at a pH above 6.0. The pH of the normal vaginal fluid is 4.5-5.5 and the pH of amniotic fluid is about 7.0 to 7.5. False positive results may occur from semen, blood, serum, trichomonas infection, alkaline urine, soap, and bacterial vaginosis. In the presence of one of these substances, the presence of ferning is more reliable.

During the speculum examination the dilatation and effacement (length) of the cervix are also noted and a high vaginal swab (HVS) and endocervical swab should be performed for culture and sensitivity. Amniotic fluid from the vagina should also be aspirated for lung maturity testing and the colour and odour of the liquor should be accessed. Finally, the presence or absence of an umbilical cord prolapse should be noted

Ferning

Demonstration of amniotic fluid arborisation pattern or the fern test is another useful examination to confirm the presence of amniotic fluid. Place a little amount of amniotic fluid from the posterior fornix on a slide and allow to air dry for at least 10 minutes. Watch for ferning under the microscope, which is crystallisation of the amniotic fluid when it dries. This is due to the interaction of amniotic fluid salts and proteins. **False positive** results might be due to fingerprints on the slide, cervical mucus, talc and semen. **False negative** tests occur with a dry swab or in the presence of blood.



Picture of Ferning showing arborisation pattern

Ultrasonography should be performed. This will show decreased amniotic fluid volume, give the gestational age if there was no previous scan, localise the placenta, show the fetal lie, presentation, and fetal heart tone and the fetal weight can be estimated. Cardiotocography (CTG) evaluation for fetal well-being should be performed and particular attention paid to the occurrence of variable decelerations that might suggest compression of the umbilical cord as a result of reduced amniotic fluid volume. The baseline full blood count and sickling should be performed including C-reactive protein if possible and urine for microscopy.

Differential diagnoses:

- Vaginal discharge
- Urinary incontinence
- Mucous discharge.

Further Management options

- Expectant management with immediate delivery if infection, labour or fetal compromise occurs.
- Deliver when fetal lung is matured or the gestational age of the fetus is 34 weeks and above, taking into consideration the neonatal facilities to manage a preterm infant.
- Deliver when the risks of prematurity are small compared with the risk of infection.

In managing preterm PROM the likely course of pregnancy in the absence of medical intervention should be taken into consideration. At 26 weeks of gestation about 30 to 40 percent of patients will gain at least one week and 20 percent at least 4 weeks before delivery.⁽¹¹⁾

Within 24-48 hours of ruptured membranes, about 80-90 % of term patients are in labour while only about 50% of preterm patients experience labour over the same time period. Generally, about 70% of preterm patients are in labour within 7 days.^(12, 8, 4)

It is difficult to predict the clinical outcome of an individual with preterm PROM and while most Obstetricians would induce labour in term PROM patients after 48 to 72 hours,⁽¹⁵⁾ as Term PROM does not pose so much of a problem as preterm PROM. Decision to deliver a case of Preterm PROM should be considered at 34 weeks of gestation since the complications of prematurity are low and delivery would prevent the potential problems of sepsis or cord prolapse. Patients with PROM prior to 34 weeks of gestation should be managed conservatively if no maternal or fetal contraindications exist.

Expectant Management

PROM presents the obstetrician with numerous dilemmas but these can be limited to two major risks, which are infection and prematurity. Management decisions depend on the relative risk of these two problems. Generally, the main risk of term PROM is infection while that of preterm PROM is prematurity.

At Term

Following appropriate confirmation of the diagnosis, a period of at least 24 hours from time of PROM can be allowed for, during which time, the majority of these patients will go into labour. At the end of this time, induction of labour is commenced. Vaginal prostaglandin is administered (up to two doses 6 hours apart) or cytotec (PGE1 analogue) 50 microgram 4 hourly for a maximum of four doses. The cervix is assessed and IV oxytocin administered if there are no spontaneous contractions.

Pre-Term:

In the management of preterm PROM there are three options that should be considered. **First**, is conservative or expectant management in which the patient is kept in hospital and monitored for infection or change in fetal parameters at which time labour is allowed or induced at the appropriate gestation. This option will allow prolongation of

pregnancy and possibly reduce caesarean section rate. **Secondly**, the patient could be delivered when the gestation is beyond 34 weeks. With this strategy, it is assumed that fetal lung maturity is adequate and there is no need for continuous fetal monitoring for infection. The main disadvantage is that the fetus would have gained additional weeks for further development to reduce neonatal morbidity if it had been allowed a few more weeks in utero. This strategy might increase caesarean section rate because the cervix might not be favourable and there may be prolonged labour with resultant increased operative deliveries. The **third** strategy is to delay delivery and administer antibiotics and steroids to prevent infection and accelerate fetal lung maturity respectively. In addition tocolytics are given to allow the antibiotics and the steroids have a therapeutic effect. All three strategies are appropriate and satisfy the individual differences of tolerance to risks of infection and immaturity.⁽¹⁶⁾

In labour PROM might be associated with a high incidence of nonreassuring fetal heart tracing because of umbilical cord compression secondary to oligohydramnios. To prevent or decrease the number and severity of variable decelerations on the fetal heart rate tracing amnioinfusion with saline should be performed.

Management Of Preterm

1. Admit to lying-in ward
2. Bed rest
3. Check vital signs i.e., 4 hourly BP, Pulse, and Temperature.
4. Fetal Kick Count
5. Ask for abdominal pain and examine for abdominal tenderness daily
6. FH twice a day.
7. Inspect sanitary pad at vulva twice a day
8. Measure abdominal (umbilical) girth daily if <29 week.
9. CTG and Ultrasound scan twice weekly
10. Doppler with biophysical profile at least once a week
11. Repeat WBC & C-reactive Protein every 48 hours.

COMPLICATIONS

1. Prematurity
2. Chorioamnionitis
3. Respiratory distress syndrome
4. Cord prolapse.
5. Intraventricular haemorrhage
6. Necrotising enterocolitis
7. Increase in caesarean sections because of nonvertex presentations
8. Fetal pulmonary hypoplasia and orthopaedic deformations
9. Oligohydramnios
10. Placental abruption
11. Maternal morbidity and mortality
12. Neonatal morbidity and mortality

Discussions And Controversies

Antibiotics

Use of *Antibiotics* is now generally accepted in PROM. Swabs should be taken before starting antibiotics. Several meta-analyses have shown that in the presence of preterm PROM antibiotics are beneficial in terms of pregnancy prolongation and/or fetal morbidity⁽¹⁷⁾

It has been demonstrated that levels of antibiotics, such as, ampicillin and gentamicin can be achieved in the amniotic fluid and fetal circulation after maternal intravenous administration, which are adequate to be effective against a high proportion of infecting pathogens.⁽²⁾ Antibiotics used include amoxyl, erythromycin and augmentin.

Corticosteroids

Use of corticosteroids in Preterm Prom is controversial since, unlike the situation in preterm labour where there is general agreement that steroids reduce the risk of RDS, with preterm PROM there are almost as many studies reporting no benefit from steroid administration as there are describing improvement in outcome.

An explanation for the less pronounced effect of steroids in this situation is that preterm PROM itself provides a stress that stimulates production of endogenous steroids causing accelerated fetal lung maturation. An increased L/S ratio has been found in fluids from patients with preterm rupture of the membranes for greater than 24 hours.^(18, 9)

Although there are conflicting reports, the preponderance of evidence supports the view that premature rupture of the membrane promotes pulmonary maturity but it does not guarantee protection from RDS.

The use of *Corticosteroids* in *PROM* appears to accelerate fetal lung maturation. Meta analyses from published randomised trials suggest overall benefit for the neonate. They do not increase the risk of chorioamnionitis or neonatal sepsis. Corticosteroids have been shown to clearly reduce the incidence of respiratory distress syndrome (RDS) and overall mortality. It is the policy in the Korle Bu Teaching Hospital, Accra to administer these agents to women with *PROM* less than 34 weeks gestation. There is no increased risk of infections for the neonate.⁽²⁰⁾

Tocolytics

The use of *Tocolytics* in patients with *PROM* is also controversial. In general, tocolytics are not used but might be beneficial for 24-48 hours to allow steroids have an effect on the lungs if there is no evidence of chorioamnionitis. It should be noted that infrequently, spontaneous onset of labour might be followed shortly by increase in maternal temperature, other signs of infection and increased pulse rate. In such cases, labour could be an early sign of chorioamnionitis and continued use of tocolytics might prevent recognition of infection until it becomes serious. Common tocolytics used are MgSO₄, Ritodrine, or Salbutamol.

Resealing of ruptured membranes can happen but is rather rare. It is most common after a high leak, especially after amniocentesis. In our experience, some seal after spontaneous rupture and it is essential that patients are watched and evaluated continuously after ruptured membranes.

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Breech Presentation

C. T. John and A.O.U Okpani

Definition

Breech presentation is defined as the entrance of the fetal buttocks or lower extremities into the maternal pelvic inlet.

Types of Breech Presentation

Three types are described:

1. The complete or flexed at the knees. This type is commoner in the multiparous gravida.
2. The Frank Breech: This refers to a fetus with both thighs flexed on the abdomen and both lower limbs extended at the knees. This is the commonest type of breech, occurring in almost all primigravidae and in some multiparous women.
3. The footling Breech: This refers to a fetus with one or both lower extremities extended below the level of the fetal buttocks. The knee presentation, a very rare type in which one or both knees are flexed below the extended hip joints is usually classified with the footling breech.

Incidence

Frank breech presentations occur in 50 per cent of all breech deliveries, whereas footling and complete breech presentations occur in 35 and 15 per cent respectively. Together the three types of breech presentations account for approximately 3 per cent of all deliveries at term.

Aetiology

Factors thought to contribute to the incidence of breech presentation include prematurity, multiple pregnancy, uterine abnormalities, abnormal placental position (cornual or praevia), oligofetal umbilical cord and maternal pelvic abnormalities. Large pelvic tumours that obstruct the birth canal may contribute to breech presentation by preventing fetal version from the breech to the cephalic presentation in late pregnancy. In many cases the aetiology of the breech presentation is not clear, as none of the above associations may be present. The mal-presentation has been noted in about 25 per cent of gravidae examined between

28 to 30 weeks gestation but by the 34th week spontaneous version has occurred in most of these fetuses such that the vertex presents. Thus prematurity is a contributory factor in many cases as pre-term labour is associated with a high incidence of breech presentation.

Diagnoses

The diagnosis is made initially by abdominal examination. Though inspection of the abdomen reveals nothing unusual, on palpation the presenting part feels firm but not bony hard, and less rounded than the head. The fetal head, hard, round and ballotable is felt in the uterine fundus occasionally to one side of the midline. The Pawlick grip is usually more suitable than bimanual examination for differentiating the breech at the pelvic inlet from the fetal head.

Difficulties in making a diagnosis by palpation arise when the anterior abdominal wall is obese, when extended legs obscure ballotment of the fetal head, with the fetus is in dorso-anterior position, and when polyhydramnios is present.

On auscultation with the Pinard stethoscope, the area of greatest intensity of the fetal heart sounds is above the level of the maternal umbilicus although, if the legs are extended, the sounds tend to be heard at a lower level. Extended legs, therefore should always be suspected if the breech feels small, is sunk well into the pelvic brim, and the fetal heart sounds are heard best at the level of the umbilicus.

On vaginal examination, the fetal buttocks are noted to be less hard and less rounded than the head. With a dilated cervix and ruptured membranes the natal cleft is felt. Only if the presenting part is high (and the abdominal findings inconclusive) might the fetal anal orifice be mistaken for the mouth in a face presentation. The fetal sacrum may be palpable, the spines feeling rather like a string of pearls. The genitalia are soft and not easily recognized.

The feet may be felt close to the buttocks or may be the sole presenting part in a footling breech. The fetal umbilical cord may be felt in cases

complicated by cord prolapse. Confirmation of a diagnosis of breech presentation is easily accomplished by ultrasonography. In addition, exclusion of associated aetiological factors like prematurity, placenta praevia, multiple pregnancy, uterine abnormalities, pelvic tumours, oligohydramnios, fetal congenital abnormalities and reliable estimation of fetal weight are other information that ultrasound scanning for suspected cases of breech presentation provides.

Complications of Breech Presentation

1. Fetal Complications

Compared to the cephalic presentation at term, the fetus presenting by the breech is at greater risk of perinatal and neonatal mortality and neonatal morbidity.¹⁻⁵ Intracranial haemorrhage from rupture of the flax cerebral or tentorium cerebelli may occur due to rapid moulding of the fetal skull. Dislocation of the neck, Erb-Duchene and Klumpke's paralyses, and damage to the sternomastoid muscles due to traction can occur during a vaginal breech delivery.

Excessive pressure on the fetal trunk or faulty handling can lead to rupture of a viscus-usually the liver or kidney. Genital oedema or ecchymoses may develop due to caput formation.

Dislocation of the shoulder, clavicular fracture and fracture of the humerus may follow attempts to deliver the arms especially if extension or nuchal displacement has occurred. Prolapsed of the fetal umbilical cord occurs occasionally especially with a footling breech or knee presentation.

Dislocation of the hip joint may occur following traction. Femoral fractures can occur while attempting to flex extended legs. Inadvertent hyperextension of the knee instead of flexion when delivering the legs can result in disruption of the knee joint.

The risk score for abnormal fetal outcomes is also increased by congenital malformations, and birth asphyxia, the latter often caused by prematurity and the wide spectrum of birth trauma mentioned above.⁶

Maternal Complication

In terms of maternal outcomes, vaginal breech delivery is associated with less risk to the mother compared to caesarean section. Formerly,

perineal tears, vaginal lacerations, paravaginal haematomata, and cervical laceration, occurred occasionally during difficult assisted vaginal breech deliveries and breech extractions especially in unbooked emergencies. These maternal injuries are now very rare because of careful selection of patients for vaginal breech delivery and increased resort to caesarean section. Caesarean section is associated with anaesthetic risks, haemorrhage and increased need for blood transfusion, as well as postpartum morbidity and other complications, infection and intestinal obstruction.⁽⁷⁾

Management of Breech Presentation

Antenatal care: Early booking, a feature seen infrequently in obstetric practice in West Africa, affords the opportunity to evaluate the previous obstetric performance of the gravida. Breech presentation is seen frequently before the 30th week of pregnancy and provided known aetiological associations have been excluded, interventions are unnecessary up to 37 completed weeks as spontaneous version to cephalic presentation will occur in many cases if multiple pregnancy, placenta praevia and uterine/gross fetal abnormalities are absent. Where the breech persists beyond 37 completed weeks, external cephalic version should be considered.

External Cephalic Version (ECV)

This procedure is the manipulative trans-abdominal conversion of the breech to a cephalic presentation and has been practiced since ancient times. ECV is easier to perform and more likely to be successful if the gravida is multiparous, adequate amounts of liquor are present and the station of the breech is above the pelvic brim. Anaesthesia is usually not necessary for the procedure but a tocolytic to facilitate uterine relaxation may be necessary. Ultrasound scanning prior to the procedure should be carried out to exclude placenta praevia and multiple pregnancy and fetal heart monitoring after the procedure should be undertaken for a short period of time.

The procedure is contraindicated if placenta praevia, hypertension or previous episodes of antepartum haemorrhage in the index pregnancy are present.

Previous caesarean section, multiple pregnancy, pelvic contracture (and other contraindications to vaginal delivery) are other contraindications to ECV.

After successful ECV there is no special risk to the fetuses with regard to labour and delivery and no need to carry out an induction of labour without obstetric indications. ECV has been shown in some centres to reduce the incidence of breech presentation in labour and the fetal complications of vaginal breech delivery as well as avoiding maternal risks i.e. maternal mortality and morbidity associated with vaginal breech delivery and the need for caesarean section.⁽⁸⁻¹¹⁾

Decision on Mode of Delivery

Elective Caesarean Section Versus Planned Vaginal Assisted Breech Delivery

Perinatal mortality, neonatal mortality and morbidity, and maternal morbidity are associated with vaginal breech delivery if judicious antenatal criteria for a planned vaginal delivery of a term breech presentation are not met. Lack of experience with assisted vaginal breech delivery, so common recently especially among younger obstetricians also add to the risk. Even with experienced personnel, unforeseen complications like extended arms, nuchal displacement of the arms, entrapment of the fetal head by the cervix and obstruction due to undiagnosed hydrocephalic after coming head and soft tissue tumours may present suddenly during the procedure.

Adding these multiple potentials for poor obstetric outcomes to the fact that dysfunctional labour, early rupture of the membranes with the risk of cord prolapse, and insufficient data on the merits and demerits of the use of oxytocin in labour when a breech presents, has led to a consensus presently among obstetricians and midwives that, there should be more resort to elective caesarean section for term breech presentation.

Elective caesarean section is indicated for term breech if there are additional obstetric complications like a previous caesarean section, hypertension, bad obstetric history, previous infertility and pelvic contracture or asymmetry. Most primigravidae are now being delivered by elective caesarean section for term breech presentation even in the absence of any complications. Clinically demonstrable fetopelvic disproportion, footling breech and estimated fetal weight greater than 3500 grams are other indications.

An exclusion of these multiple contraindications by history, examination, estimation of fetal weight by ultrasonography and clinical/x-ray pelvimetry is recognized as essential in the selection of gravidae

who should undergo labour. Close monitoring in labour preferably with cardiotocography should be undertaken. Indications for terminating the labour include early rupture of the membranes, when contractions are still weak, poor state of cervical dilatation, fetal distress and cord presentation or prolapse.

Vaginal Breech Delivery Techniques

Assisted Vaginal Breech Delivery: This is the procedure of choice and should be undertaken by the most senior obstetrician present.

Epidural block is the best anaesthetic method but this may not be practicable for many obstetric units in developing countries. Pudendal block and local anaesthetic infiltration of the perineum can be used when an epidural block is not possible. Blood should be grouped and cross-matched and arrangements completed for immediate emergency caesarean should the need arise. The most important aspect of vaginal breech delivery is the delivery of the after coming fetal head. This is best accomplished by the use of obstetric forceps. The Piper's forceps is ideal but other forceps can be used. Forceps when used to deliver the after coming head protects the unmoulded head. Jaw Flexion and shoulder traction (the Mauriceau - Smellie - Veit Manoeuvre) and the Burns - Marshall methods are alternatives if forceps delivery is not possible.

Oxytocics should be given only after the after-coming head is delivered and the placenta is delivered by controlled cord traction.

2. Breech Extraction

Breech extraction implies a total absence of maternal effort and the gravidae being under general anaesthesia. Indications for breech extraction are now very few. There may be a place for the procedure in cases of retained second twin with breech presentation or those in transverse lie. In the latter situation a prior internal podalic version is carried out.

Breech presentation with intrauterine fetal death or gross hydrocephalus may be managed by breech extraction with craniotomy to the after-coming head to avoid caesarean section. In such cases the uterine cavity should be explored after the delivery. Details of caesarean and vaginal breech delivery techniques are available in standard midwifery, and obstetrics and gynaecology textbooks/atlasses. However the techniques are best learnt in the labour ward.

Delivery of the preterm breech

Delivery of the preterm breech is an area of controversy⁽¹²⁾. Mortality and morbidity in the fetus are apparently related to the condition causing the preterm delivery, and complications of prematurity⁽¹³⁾. In this group of babies especially very low birth weight babies, intracranial haemorrhage occurs very often when there is birth asphyxia, infection and even spontaneously after vaginal or abdominal delivery of the after coming head.⁽¹⁴⁾ The circumstances of each case have to be taken into consideration including the level of sophistication of the neonatal unit and especially in developing countries where there is a psychological aversion to caesarean section; a policy for the routine use of caesarean section for the preterm breech is not justified.⁽¹⁵⁾

Breech presentation in the unbooked obstetric patient

Unbooked gravidae presenting as emergencies that have had no prior clinical assessment, usually have multiple complications. Prior unskilled interference usually compounds the clinical problem.

In this group of gravidae with breech presentation, referral to a tertiary unit is mandatory. Careful clinical assessment by the most experienced member of the team is usually necessary before decisions on management can be taken.⁽¹⁶⁻¹⁷⁾

Controversies in the management of breech presentation

Breech presentation is an abnormal presentation and associated with maternal and fetal complications. ECV assisted vaginal breech delivery and even caesarean section is each associated with some complications. The timing of ECV and the route of delivery continue to plague the obstetrician. Certain cultural taboos associated with delivery by caesarean section in parts of West Africa Sub-Region complicate matters further. Breech presentation must be diagnosed on time and at term all the clinical expertise of the obstetrician and the facilities necessary to determine safe passage either vaginally or abdominally of the baby are advocated. Even in places and situations where caesarean section is the norm for term breech presentation, strong advocacy is made for the obstetrician to be well versed in the art of assisted vaginal breech delivery. One can never know when this expertise will become necessary to save a life.

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Multiple Pregnancy

K Nkyekyer

Introduction

Multiple pregnancies continue to intrigue both the medical profession and the lay public. In the past 15-20 years the incidence of multiple pregnancies in developed countries has increased dramatically, mainly as a result of increasing use of assisted reproduction technologies (with the resultant increase in numbers of higher order multiple pregnancies) and older age of mothers.

Multiple pregnancies is associated with increased risk of obstetric complications as well as increase in perinatal and neonatal mortality rate; the risk of perinatal mortality is 5-10 times that for singleton pregnancy. There is also increased risk of infant mortality. It is not until the second year of life that mortality rates for twins are the same as those for singletons.

Since twin pregnancy is the commonest form of multiple pregnancies it will form the main subject of this chapter with references being made to higher order multiple pregnancies as appropriate.

Types of Twinning and Placentation

There are two types of twinning: dizygotic and monozygotic. In dizygotic twinning two separate ova are fertilised by two separate spermatozoa; it accounts for about two-thirds of all twin gestations. Monozygotic twinning results from fertilisation of one ovum, by one spermatozoon, which subsequently divides into two separate embryos. Higher order multiple pregnancies (triplets, quadruplets, etc) may be monozygotic, multizygotic or a combination of the two.

In dizygotic twinning, because there are two separate blastocysts implanted, each will develop its own chorion and amnion and the placentation will therefore be dichorionic diamniotic.

The type of placentation present in monozygotic twinning depends on when the division into two embryos occurs. Division within the first 72 hours after fertilisation, by which time the inner cell mass will not have formed and cells destined to become the chorion will not have been determined, results in two separate blastocysts eventually being

implanted. The placentation in this case will therefore be dichorionic diamniotic, the same as that for dizygotic twinning. This type of placentation accounts for about 30% of all monozygotic twinning.

Sometimes with dichorionic diamniotic placentation the sites of implantation of the placentas may be so close as to give rise to "fusion" resulting in the appearance of a single placenta. Closer examination, however, will reveal that they are separate placentas.

If division into two embryos occurs between days 4 to 8 after fertilisation, the embryos will be enclosed in the same chorionic sac but each will later develop its own amniotic sac. By this time the inner cell mass will have formed and cells destined to become the chorion differentiated (i.e. the blastocysts will have been formed). The placentation will therefore be monochorionic diamniotic and it is the commonest type seen in monozygotic twins, accounting for just a little less than 70% of that type.

When division occurs between days 8 to 13, by which time the amnion will have formed; both embryos will be enclosed in the same amniotic and chorionic sac, giving rise to monochorionic monoamniotic placentation. This is the least common type and occurs in about 1% of monozygotic twins; it carries a high perinatal mortality rate primarily due to cord entanglement and twin-to-twin transfusions.

Attempts at division after 13 days, when the embryonic disc will have formed, results in incomplete division and the formation of conjoint twins and monsters. The incidence of conjoint twins is about 1 in 50,000-100,000 births.

Incidence Of Twinning

The incidence of monozygotic twinning is known to be the same all over the world, 3-5 per 1000 births. It is thought to be a random event, which is not affected by the other factors, which influence the rate of dizygotic twinning. It may be mentioned, however, that for as yet inexplicable reasons

monozygotic twins seem to occur at approximately 2-3 times the normal rate in pregnancies resulting from assisted reproductive technologies.⁽¹⁾

Differences in twinning rates between various ethnic groups and geographical areas are due mainly to differences in dizygotic twinning rates. The rate of spontaneously conceived dizygotic twins per 1000 births is highest in black Africans (the highest reported, 45 per 1000 births, is among the Yorubas of Nigeria), median among the Caucasian populations of Europe and North America and lowest in Asians.

- * Apart from race other factors associated with increased incidence of twins are:
- * Increasing maternal age, independence of parity; the incidence increases up to about 40 years and then declines.
- * Increasing parity, independent of age.
- * Previous multiple pregnancy.
- * Family history of multiple pregnancies, especially first-degree relation on the woman's side. Male contribution is questionable.
- * Large and tall women have increased incidence as opposed to small women; this is most likely related to nutrition rather than just body size.
- * Use of ovulation induction agents: clomiphene is associated with about 10% incidence of multiple pregnancies while the incidence with gonadotrophins is 30-50%.
- * Use of Assisted Reproductive Technologies, especially IVF.

There is some suggestion that those who have increased incidence of spontaneous twin pregnancies may have higher levels of circulating gonadotrophins.

It is now abundantly clear, with the use of ultrasonography in early pregnancy that the incidence of twinning is much higher than is suggested by figures based on findings in late pregnancy. There have been several reports in which first trimester ultrasonography has revealed twin gestational sacs only for the pregnancy to proceed later as a singleton pregnancy; the loss of

one twin may or may not be preceded by an episode of bleeding. This phenomenon is referred to as "vanishing twin"⁽²⁾ and has been estimated to occur in 20%-25% of early twin pregnancies.

Complications of Twin pregnancy

- * Multiple pregnancies are associated with increased incidence of certain maternal and fetal complications.
- * Maternal complications include:
- * Increased severity of early pregnancy symptoms, especially vomiting; indeed in a woman who is diagnosed, as having hyperemesis gravidarum multiple pregnancies must be excluded.
- * Later on in pregnancy the grossly enlarged uterus may cause maternal discomfort, with breathlessness, general fatigue, peripheral dependent oedema and varicosities; marked lordosis may result in problems with backache.
- * Anaemia: this is due to the increased demands of the fetuses on maternal iron and folate stores as well as the increased haemodilutional effect of the changes of pregnancy.
- * Pregnancy Induced Hypertension (PIH): incidence in twin pregnancy is 2-3 times that in singleton pregnancy. It occurs earlier and tends to be more severe, without regard to zygosity.
- * Abruptio placentae: there is 3 times increased risk over that in singleton pregnancy. Although it most frequently occurs in the third trimester, it may also occur immediately after vaginal delivery of the first twin when reduction in intrauterine volume may lead to premature separation of the placenta.
- * Placenta praevia: Theoretically this is thought to be more likely because the large placental surface area increases the likelihood of encroachment on the lower segment.
- * Increased incidence of operative delivery (vaginal or abdominal): this is due largely to the increased incidence of fetal malpresentation and abnormal lie with

multiple pregnancies, especially with regards to the non-leading fetus.

- * Postpartum haemorrhage (PPH): over distension of the uterus results in inability of the myometrium to contract and retract effectively after delivery, resulting in uterine atony and hence excessive bleeding.

Fetal complications include:

- * Increased rates of spontaneous abortions.
- * Congenital anomalies: these are twice as common in twins and about four times in triplets as they are in singleton pregnancies. They are more common in monozygotic than dizygotic twins.
- * Preterm delivery; this is the most important factor contributing to the increased perinatal mortality and morbidity in multiple pregnancy. Preterm delivery rate in twins is about twelve times that in singletons. The average length of gestation decreases as the number of fetuses increase.
- * Twin to twin transfusion syndrome (TTTS).
- * Polyhydramnios: This is more common in twins with monochorionic placentation and may occur as an acute process, especially with severe TTTS. Polyhydramnios may predispose to preterm labour and preterm premature rupture of membranes.
- * Intrauterine growth restriction: Twins follow the same growth curves that apply to singletons until the third trimester (around 30 weeks) when their growths begin to lag behind. The incidence and degree of IUGR increases with the number of fetuses and as the pregnancy approaches term. IUGR may be due to the fact of two or more fetuses sharing the available maternally derived nutrients, reduced placental surface area for each of the two infants, or the twin-to-twin transfusion syndrome. Discordant growth (i.e. 25% difference in weight with the bigger baby's weight as denominator) occurs in about 10% of twin gestations. In TTTS hydrops of the donor twin may obscure inter-twin fetal weight differences.
- * Death of one fetus: Approximately 0.5-6.8% of twin pregnancies result in death of one

fetus in the second half of the pregnancy. In monochorionic placentation this could adversely affect the surviving twin, with damage such as ischaemic necrosis of the brain (with multicystic encephalomalacia) and of the kidneys. Two theories are put forward to account for this. One is the embolic theory in which thromboplastin-like material is transfused through the vascular connections into the survivor's circulation resulting in end-organ damage. The other is the ischaemic theory; in this case blood is shunted via an open anastomosis into the low-resistance vascular system of the dead fetus, resulting in acute hypovolaemia, ischaemia and end-organ injury, or even death in the other fetus. The latter is thought to be the more likely mechanism.

- * There is increased incidence of single umbilical artery (about 3-4% as opposed to 0.5-1% in singletons); the significance of this lies in the fact that about 30% of babies with single umbilical artery have other congenital anomalies. There is also increased incidence of marginal and velamentous insertion of the cord in twin as compared to singleton pregnancies.
- * Intrapartum complications: Malpresentation, cord prolapse, dysfunctional uterine action and operative delivery (both vaginal and abdominal) are more common in twins than in singleton pregnancy. Cord entanglement is a very real risk in monoamniotic twins. Locked twins are extremely rare.

Diagnosis

- * Personal or family history of twin pregnancies
- * Use of ovulation induction drugs
- * Exaggerated early pregnancy symptoms, especially excessive vomiting.
- * Uterine size larger than expected for gestational age
- * Palpation of more than two fetal poles
- * Multiple fetal parts
- * Simultaneous detection of two different fetal heart beats (with the use of the *sonicaid* or the electronic fetal monitor).

The diagnosis will always have to be confirmed by ultrasound examination. It is useful to diagnose twin pregnancy as early as possible. Early diagnosis is associated with an improvement in the perinatal mortality, probably due to the improved

antenatal care and the planned delivery of the second twin. In this regard there is everything to say for routine ultrasound examination between 16-18 weeks gestation; this is likely to detect almost all cases of ongoing twin pregnancies.

Where two fetuses are identified at ultrasonography thorough examination must be carried out to exclude the possibility of three or more fetuses. It is necessary to determine the chorionicity and amnionicity of the twin pregnancy. If there are two separate placentae, the placentation is dichorionic, diamniotic. If there is only one placenta further examination includes:

- * Assessing the sex of the fetuses; if they are of different sexes then they are dichorionic diamniotic.
- * Assessing the thickness of the membrane separating the two sacs; if it is <2mm then it is likely to be monochorionic diamniotic whilst if it is 2mm it is most likely to be dichorionic diamniotic. There has been a report in which with high frequency ultrasonography it was possible to count the layers in the membrane separating the two sacs and thus determine the chorionicity; two layers suggested monochorionic diamniotic whilst three or more was diagnostic of dichorionic diamniotic placentation.⁽³⁾
- * The presence of the "twin peak" or "lambda" sign is indicative of dichorionic diamniotic placentation.⁽⁴⁾

If no separating membrane can be identified between the two fetuses, that suggests they are monochorionic monoamniotic and in that case every effort must be made to exclude conjoint twins.

In general, in view of the increased incidence of congenital anomalies in twin pregnancy, it is important to perform detailed ultrasound evaluation of each fetus to rule out any anatomical abnormalities.

Management

Antenatal

Once the diagnosis of ongoing twin pregnancy has been confirmed, it is necessary to inform the woman of the diagnosis and of the increased risks associated with it. Discussion may be carried out on adequate diet as well as on the probable need for adjustments in life-style, and the early warning

signs of preterm labour. An additional 300kcal/day is needed over and above that for the woman carrying a singleton pregnancy; it is necessary to provide iron and folate supplementation. Pregnancy weight gain of 16-20kgs is considered appropriate. Modifications in life-style may include reduction in workload in paid employment outside the home, avoidance of carrying of heavy loads and of any chores that involve a lot of physical exertion.

In addition to the detailed ultrasound evaluation of the anatomy of the fetuses, other prenatal diagnostic procedures may be carried out as indicated. Concerning maternal serum alpha-fetoprotein, the cut-off point for twins is 2.5 multiples of the median for singleton pregnancy. Chorionic villus sampling (CVS) may be performed at 10-12 weeks gestation and it must be kept in mind that the sample from one twin may be contaminated with tissue from the other. When amniocentesis is performed it must be ensured that both sacs are sampled. A small volume, about 1-2mls of dilute indigo-carmin solution (approximately 0.08%) may be injected into the first sac so that unstained amniotic fluid with the second tap will confirm sampling from the other twin. Methylene blue must not be used because it causes fetal bowel atresia.

It is important that when CVS or amniocentesis is performed, detailed documentation is made of the location of the fetuses and the separating membrane so that if there should be any abnormal results the affected twin can be easily located. If the result of one twin is abnormal, selective termination/feticide of that twin may be carried out, although it must be remembered that the procedure could result in the loss of both fetuses.

Antenatal clinic visits may need to be more frequent in order to detect any complications early; prompt admission into hospital is necessary if any signs of complications, especially preterm labour, should develop. Serial ultrasound scans are performed to assess fetal growth and detect any discordance in growth between the two fetuses; these may be performed at 3-4 week intervals up to about 28-30 weeks and thereafter fortnightly. Other tests of fetal surveillance (non-stress test, biophysical profile, umbilical artery Doppler blood flow studies) are not routinely used unless there is IUGR, abnormal liquor volumes, growth discordance, fetal anomalies, TTTS, monoamnionicity or maternal complications (e.g. Pre-eclampsia).

Preterm labour:

Various methods have been tried to prevent preterm labour in twin pregnancy. These include bed rest, prophylactic tocolysis with beta-mimetics (oral and intravenous), and prophylactic cervical cerclage, but none of them has been found useful. As far as progestins are concerned, there is no consensus in available data for its use in the prevention of preterm labour. There is some evidence that life-style modifications as described above may be useful in reducing the preterm delivery rate.

With regards to prediction of preterm labour home uterine activity monitoring, cervical status measurement by ultrasound and estimation of fetal fibronectin in cervicovaginal secretions may be helpful.

In established preterm labour management is very much along the same lines as that for singleton pregnancy. There is however increased risk of pulmonary oedema with the use of beta-mimetics and corticosteroids.

Labour And Delivery

There is some controversy over the optimal route of delivery in twin pregnancy. This will have to be individualised, with the management depending on presentation, gestational age, presence or absence of maternal or fetal complications, experience of the obstetrician and availability of anaesthesia and neonatal intensive care facilities.

Several studies have shown that minimal perinatal mortality was observed for twins at 37-38 weeks as opposed to 39-40 weeks for singleton gestations.⁽⁷⁾ It has also been recognised that twins are more mature than singletons at the same gestational age. These observations support the hypothesis that optimal length of gestation differs between twins and singletons. To avoid perinatal mortality and other adverse perinatal outcomes some have suggested induction of labour at 38 weeks gestation for twins.

The various combinations of presentations and their relative frequencies have been reported as follows:

- * Vertex - Vertex 40.9%
- * Vertex - Non-vertex 35.7%
- * Non-vertex Vertex or Non vertex 23.4%⁽⁸⁾

With vertex-vertex presentations there is no increase in neonatal morbidity or mortality when the twins are delivered vaginally as opposed to delivery by Caesarean section, regardless of gestational age. Vaginal delivery is therefore preferred unless some other obstetric complicating factor is present.

The management of vertex-nonvertex combination is a major source of controversy. In general if the gestational age is such that chances of survival are very low (for example less than 28 weeks in our environment) then vaginal delivery must be aimed at.

Between 28 and 32 weeks gestation (1000-1500gm) external cephalic version of twin II may be attempted after delivery of twin I and if successful may be followed with delivery by the vertex. If unsuccessful, Caesarean section should be performed. Internal podalic version with breech extraction in this situation is not advised since it is associated with a poorer outcome. Indeed most authors agree that the very low birth weight infant (<1500gm) in non-vertex presentation should be delivered abdominally.

Where the estimated fetal weight is more than 1500 gm (>32 weeks) there is no increase in perinatal morbidity or mortality in the non-vertex second twin being delivered vaginally. If external cephalic version fails with the fetus in transverse lie or is accompanied by fetal distress or inadvertent rupture of the membranes, internal podalic version with breech extraction may be performed. On the other hand one may proceed to Caesarean section if the skill to perform internal podalic version is not available. Vaginal delivery of the non-vertex twin II should be considered in the light of standard criteria for singleton vaginal breech delivery: adequate maternal pelvis, flexed fetal head, and estimated fetal weight less than 3500gm.

When it comes to nonvertex-vertex/nonvertex-nonvertex most authors agree that Caesarean section be recommended for this group where the pregnancy has reached viable gestational age. The potentially disastrous possibility of locked twins with breech-cephalic presentations must be kept in mind, although this is extremely rare. However, in a report from South Africa 35 women with breech-breech or breech-transverse presentations were delivered successfully vaginally without any increase in adverse neonatal outcome compared to 27 women who were delivered electively by Caesarean section. The authors suggested that vaginal delivery might be a reasonable option in

such situations provided the criteria for breech delivery are adhered to.⁽⁹⁾ If twin I is in transverse lie delivery must be by Caesarean section.

Management of labour

The latent phase of labour is shorter in twins than in singletons; the active phase and the second stage, however, are longer and hence overall duration of labour is similar to that of singleton pregnancy. Similar to the situation with singletons, the average duration of labour with twins is longer in the nulliparous woman than that in the multiparous. One reason for the prolonged active phase is the increased incidence of dysfunctional labour; it may therefore be necessary to augment labour with oxytocin infusion.

In order to achieve optimal outcome careful intrapartum management of twin gestation is essential. When a woman carrying a twin pregnancy is admitted in labour, after the routine examinations, blood should be taken for grouping and cross matching of at least two units of blood and an intravenous access secured with gauge 14 or 16 canula. This is in anticipation of a possible postpartum haemorrhage, which is a very real risk in this situation.

Both twins have to be monitored in labour, and this is best done continuously with the electronic fetal monitor; where the membranes are ruptured fetal scalp electrode may be applied to the leading twin and the second twin monitored externally.

As regards analgesia, epidural is generally, considered most desirable. This is because not only does it provide adequate analgesia during labour, it can also be used for manipulative procedures or Caesarean section. Moreover its use avoids the neonatal depressant effect associated with narcotic analgesia.

In the second stage, ideally there should be present in the delivery room two paediatric teams each with resuscitating equipment (one team for each baby), an anaesthetist and an obstetrician. The delivery of twin I may be conducted, as in singleton delivery; after delivery of the baby the cord is clamped in two places and cut between the clamps.

Examination of the abdomen is then performed to determine the lie and presentation of the second twin; ultrasound scan may be used if there are any difficulties. If twin II is in transverse lie external cephalic version is attempted. If successful, delivery is continued; if not then internal podalic version with breech extraction (in cases of

gestational age >32 weeks) or Caesarean section (in cases of gestational age =32 weeks) is performed as the case may be. If twin II is in longitudinal lie resumption of uterine contractions is awaited and as the presenting part descends into the pelvis the membranes are ruptured and delivery completed. If uterine contractions do not resume after 5-10 minutes oxytocin infusion may be started.

The delivery interval between twins has been limited to 30 minutes in an effort to avoid adverse perinatal outcome in twin II. This still applies in centres without electronic fetal heart rate monitoring facilities. Where such facilities are available, provided that the second stage is progressing and there is no significant vaginal bleeding or evidence of fetal distress an extended delivery interval is acceptable.

After delivery of twin II the cord may be clamped in three places and cut such that two clamps are left on the placental end of the cord; this is to enable easy identification of each baby's cord (and placenta). Oxytocic drug administration for the management of the third stage is given with the delivery of the anterior shoulder (in vertex presentation) or after delivery (in breech presentation) of the second twin. The placenta is delivered as in singleton pregnancy. To reduce the risk of postpartum haemorrhage it may be useful to set up an intravenous infusion of 20 units oxytocin in 500 mls of normal saline and run this over a period of about four hours.

After delivery an attempt should be made to determine zygosity. If the fetuses are of different sexes then they are dizygotic. In like-sexed twins with single placenta examination of the separating membrane will reveal four layers in dichorionic and two layers in monochorionic placentation; in addition, in the case of monochorionic placentation vessels may be seen crossing from one side of the placenta to the other on the fetal surface. If there is any doubt a specimen of the separating membrane may be sent for confirmatory histological examination. About 25% of like-sexed dichorionic twins will be monozygotic.

Other laboratory tests (e.g. blood group typing, DNA analysis) may be necessary to establish zygosity. The importance of zygosity determination lies in the fact that if they are monozygous then they may have the same predisposition to certain medical conditions (diabetes mellitus, asthma, depression), and if cancer should develop in one

then careful assessment on an ongoing preventive basis in the other must be instituted. Most importantly when it comes to the issue of organ transplantation there are unlikely to be problems with rejection in monozygous twins.

It is important to appreciate that the emotional and physical demands of caring for twin infants are potentially overwhelming. Every effort must therefore be made to provide physical and emotional support for the parents.

Twin-to-twin Transfusion Syndrome

(TTTS): This occurs in monochorionic placentation; because they share one placenta there is invariably intercommunication between the circulations of the two fetuses. Although some cases of vascular intercommunication have been reported in fused dichorionic placentas with the occurrence of twin-to-twin transfusion syndrome this is so rare that the phenomenon may be considered to be almost exclusive to monochorionic placentas. Vascular communications that may occur include arterio-arterial, arterio-venous and veno-venous; these shunts may occur in such a manner that they are balanced, with no net loss or gain to either fetus. This is likely to explain the fact that despite the high incidence of transplacental vascular communications (more than 90%) the incidence of TTTS in monochorionic placentation is low (about 15%).

It is thought that where TTTS occurs there is unbalanced shunting in which an artery from one twin supplies a cotyledon only for the vein from that cotyledon to drain to the other twin. TTTS may be chronic or acute. In chronic TTTS one twin (the donor) chronically loses blood to the other (the recipient). The donor gradually becomes anaemic and the recipient polycythaemic. The recipient, because of hypervolemia, has increased urine output resulting in polyhydramnios; it grows much bigger than the donor because of the shunting of nutrients to it. The hypervolaemia and hyperviscosity lead to cardiomegaly, and in severe cases cardiac failure with hydrops may ensue. The donor is hypovolaemic and therefore does not pass much urine, giving rise to oligohydramnios, which may be so marked as to give rise to what is referred to as the "stuck twin" sign. There is also fetal growth restriction. The donor may adapt to chronic anaemia by increased haematopoiesis; cardiovascular adaptation to longstanding anaemia may also cause hydrops.

Acute TTTS usually occurs during labour in the interval between the clamping of the cords of the first and second born twins. During that period the second twin has the only connection to the entire placenta and may receive significant transfusion. In cases of antepartum TTTS the larger, recipient twin is usually born first; therefore the second born antenatal donor may paradoxically be born plethoric because of considerable acute perinatal transfusion.

On the other hand, some monochorionic twins without antenatal TTTS may undergo acute perinatal TTTS so that while there may not be growth discordance, one twin is plethoric and the other anaemic; this may be so severe as to require treatment.

Antenatal TTTS:

This may be diagnosed when in confirmed monochorionic placentation there is discordant fetal growth, discordant amniotic sacs (polyhydramnios and oligohydramnios) and evidence of hydrops in one twin. The prognosis without treatment depends on the gestational age at diagnosis; the most severe forms of TTTS are diagnosed at less than 25-26 weeks, and the mortality without treatment is more than 90%, gestational age at delivery (greater than 28 weeks is desirable) and presence or absence of hydrops.

Treatment modalities have included

- * Termination of the entire pregnancy.
- * Maternal digoxin therapy.
- * Selective termination of one fetus by fetoscopic ligation of the cord.
- * Septostomy of the interfetal membrane to equilibrate the amniotic fluid volumes of the twins.
- * Bed rest with tocolysis alone or in combination with some of the other mentioned alternatives.
- * Aggressive repeated therapeutic amniocentesis
- * Fetoscopic laser occlusion of chorangiopagous vessels (FLOC procedure). (Chorangiopagus is the term for interfetal vascular anastomosis.

The last two seem to hold the greatest promise for improving the outcome in this condition.^(5,6)

Discordant fetal growth:

This is diagnosed antenatally if there is a 5mm difference in BPD, 5% difference in HC or 20mm difference in AC. Close fetal surveillance (NST, BPP and UAD studies) is essential. If the tests are satisfactory and both fetuses show proportional growth in serial ultrasound evaluations, conservative management may be continued.⁽⁷⁾

37-38 weeks gestation. If one fetus shows signs of being in jeopardy and the gestational age is 34 weeks or above, the babies should be delivered. At less than 34 weeks gestation, fetal lung maturity needs to be established; if the amniotic fluid from the bigger twin shows mature lungs then the smaller one is also likely to be mature. Where lung maturity is not demonstrated, corticosteroids may be administered and delivery planned. In situations of extreme preterm gestation, it may not be possible to save the compromised twin and the pregnancy would need to continue for the sake of the other twin, especially in dichorionic placentation.

Intrauterine death of one twin: The management of this situation depends on gestational age as well as the chorionicity. In monochorionic twins, there is a real risk of the surviving twin suffering ischaemic injury to vital organs (especially the brain). This therefore underscores the importance of early confirmation of chorionicity.

If death occurs preterm in dichorionic placentation management should be conservative, with close fetal surveillance and weekly monitoring of maternal coagulation profile. If no abnormalities develop delivery may be affected at 37-38 weeks; vaginal delivery should be aimed at unless this is contraindicated by some other factors.

In monochorionic twins if death should occur after 34 weeks gestation prompt delivery of the surviving twin should be affected. Before 34 weeks it is necessary to confirm fetal lung maturity before delivery. If the gestational age is such that the chances of extra uterine survival of the other twin are remote then conservative management may need to be resorted to, in spite of the risks of continued existence in utero.

Previous Caesarean Section.

Multiple pregnancies have been cited as a contraindication to trial of scar with the argument that over distension of the uterus would weaken the scar; no evidence, however, was produced to support this assertion.⁽¹⁰⁾

There have been reports that have documented the safety of trial of scar in twin pregnancy. Successful delivery of both twins in at least 70% of cases has been achieved without any increase in maternal or neonatal morbidity or mortality. In the absence of contraindications to vaginal birth, a trial of labour after a previous Caesarean section is a safe and effective alternative; the same strict criteria used to

govern the safe conduct of trial of scar in singleton pregnancy should be applied.^(11, 12, 13)

Monoamniotic Twins

These are especially at risk of antepartum and intrapartum fetal demise due to cord entanglement and fetal interlocking. Elective Caesarean section is recommended after documentation of pulmonary maturity because these complications can occur before onset of labour.

Conjoint twins should be evaluated with the paediatric surgical team to assess the chances of saving one or both infants after delivery. In most cases elective Caesarean section is indicated to optimise fetal outcome or avoid maternal injury.

In other situations in which there is congenital anomaly, compatible with life, of one or both twins and which increases neonatal morbidity or mortality with delivery by the vaginal route Caesarean section may be performed.

Undiagnosed twins

In spite of the availability of ultrasound there are still cases in which twin pregnancy may not be diagnosed antenatally, especially in women who are non-attendants or poor attendants. It is therefore necessary for labour ward staff to be alert to this possibility in situations where the abdomen is grossly enlarged or much bigger than expected for gestational age, or the fetal presenting part is much smaller than expected for the size of the uterus. An ultrasound scan may then be performed in the labour ward to confirm the diagnosis; if this is not available administration of the oxytocic for the management of the third stage should be delayed till after delivery when clinical examination has excluded the presence of another fetus in utero.

If twin pregnancy is detected after the oxytocic has been administered during delivery of the first twin then internal podalic version with breech extraction must be carried out fairly rapidly unless delivery of the second twin follows quickly.

There have been reports documenting the successful use of intravenous nitroglycerin (up to 1 mg) to achieve uterine relaxation in order to carry out various intrauterine manipulations, including internal podalic version for the delivery of the second twin.⁽¹⁴⁾ Uterine relaxation has occurred within 35-95 seconds and lasted about one minute. It may be worthwhile, in the situation under consideration, to apply this treatment.

In the meantime, preparations can be made for urgent Caesarean section should the attempt at

vaginal delivery fail. The blood pressure and pulse must be checked every 1-2 minutes after intravenous administration of nitroglycerin for the first 15-20 minutes. Halothane administration as part of general anaesthetic for Caesarean section may also be used to achieve uterine relaxation until the baby has been delivered.

Delivery in Triplet pregnancy

This is another area of controversy. In view of the high rates of prematurity and malpresentation, difficulties in continuous monitoring of all fetuses during labour and delivery, and the potential increased perinatal morbidity and mortality in the second and third triplets, routine delivery by Caesarean section has been recommended by most practitioners.^(7, 15) Others have reported no significant difference in outcome between those delivered vaginally and those delivered abdominally and have reaffirmed the place of vaginal delivery in appropriately selected cases. Criteria that have been used for attempting vaginal delivery include: non-contracted pelvis, vertex presentation of the first triplet, absence of maternal contraindication to vaginal delivery and ability to monitor all fetuses appropriately. It appears that limiting the time interval between deliveries of each of the triplets maximises the chance of successful and uncomplicated vaginal delivery.⁽¹⁶⁾

Higher order multiple pregnancies

As a result of the increasing use of ovulation induction drugs and assisted reproductive technology there has been increased incidence of higher order multiple pregnancies (triplets and higher). These have been associated with adverse perinatal outcomes and over the past ten years or so the practice of multifetal pregnancy reduction (MFPR) has become established. This is the deliberate reduction of the number of viable fetuses in a given multifetal pregnancy. The idea is to sacrifice some of the fetuses in order to allow the remainder to be carried to viability with increased gestational age, increased birth weight and decreased perinatal morbidity and mortality. The procedure is performed in the first trimester and there are three main methods:

- Transcervical aspiration
- Transvaginal needle injection of KCl into the fetal thorax and
- Transabdominal needle injection of KCl into the fetal thorax.

The first two are used at earlier gestational ages (9-10 weeks) than the third (about 10-12 weeks). The

third method is the most commonly used currently. In most cases the number of fetuses is reduced to two, except in circumstances such as prior poor outcome in twin pregnancy or reason to believe that twins would be significantly compromised in a particular case, in which case only one fetus may be left. Depending upon the method used and experience of the provider pregnancy loss rates range from 8.8% to 16.2%.^{20, 21} The loss rates increase with the number of fetuses to start with. Needless to say, this procedure raises a lot of ethical, moral and psychological issues. Every effort must therefore be made to prevent these iatrogenic higher order multifetal pregnancies from occurring.

Delayed-interval delivery

There have been several reports of twin or higher order multiple pregnancies in which delivery of one or more fetuses at very early gestational ages has been followed by measures to delay delivery of the remaining fetus (es) in order to increase their chances of survival²¹⁻²⁴. The following conditions must be present:

- The pregnancy should be between 18 and 28 weeks gestation at the time of the expulsion of the first fetus.
- The chorionicity must be dichorionic between the first fetus and the fetus (es) still in utero.
- The membranes in the remaining gestational sac(s) must be intact.
- There must be no fetal distress, congenital anomalies, abruptio placentae, intra-amniotic infection or maternal indication for delivery (for example severe pre-eclampsia).

The cord of the delivered sibling is ligated as close to the cervix as possible under aseptic conditions, using appropriately sized absorbable suture, and the placenta left in situ. Parenteral broad-spectrum antibiotics are given and in some cases tocolytics and insertion of cervical cerclage suture have been used. While some have performed amniocentesis of the retained sibling(s) to exclude incipient infection, others have depended on clinical signs to make a diagnosis of intra-amniotic infection. Corticosteroids have been administered where appropriate. Delays of between 3-143 days, with a mean of 44.8 days and median of 35 days, have been documented with significant improvement in perinatal outcome but no marked increase in maternal complications.

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Hypertension In Pregnancy

EY Kwawukume

Introduction

Hypertension complicates about 7% of all pregnancies and in Africa pre-eclampsia/eclampsia is a major cause of maternal mortality. ⁽¹⁾ In Ghana it accounts for about 15-25% of maternal deaths. The incidence varies among different districts, regions, countries and hospitals. The term ***hypertensive disorders of pregnancy*** is used for all forms of hypertension occurring during pregnancy. This disorder could be mild or produce serious complications for both the mother and the baby. Pre-eclampsia could occur in the early half of pregnancy or during labour. Severe pre-eclampsia during the early half of pregnancy (after 20 weeks gestation) might suggest that the woman is at risk of serious complications in future pregnancies.

The terminology of **PET, PIH and Transient Hypertension** can be confusing. Clinicians often use the term PIH to refer to patients with pre-eclampsia, patients in whom they suspect pre-eclampsia will develop, acute hypertension in pregnancy especially after the second half of gestation or patients with transient hypertension. Once there is understanding of any terminology used, treatment can be initiated accordingly to prevent maternal and fetal mortality. In general, **PET or pre-eclampsia** is used to denote increase in blood pressure, proteinuria and/or oedema after the second half of pregnancy. Most people refer to PET as pre-eclampsia and not as Pre-eclamptic Toxaemia because multiple factors are implicated in the etiological of PET and the "T" in Toxaemia is still maintained probably because of old terminology.

Classification Of Hypertensive Disorders Of Pregnancy

Hypertension: Hypertension occurs if the systolic pressure is more than 140 mm Hg or the diastolic is more than 90 mm Hg measured on two separate occasions more than 6 hours apart or there is an increase of at least 30 mm Hg systolic or 15 mm Hg diastolic over the booking blood pressure in the first half of pregnancy. An increase in mean arterial pressure of 20 mm Hg or a mean arterial pressure of

105 mm Hg, if the prior blood pressure is unknown also shows an increase in blood pressure. The mean arterial pressure is one-third the pulse pressure plus the diastolic pressure.

Proteinuria is significant if the protein in the urine is more than 300mg in 24 hours or above 1g/L on dipstick testing in at least two random urine specimens collected 6 or more hours apart.

Pre-eclampsia Elevated blood pressure accompanied by proteinuria with or without oedema, occurring after 20 weeks of gestation in a known normotensive non-proteinuric woman.

Eclampsia: Is the occurrence of convulsion in a pre-eclamptic patient in the absence of coincidental neurological disease.

Chronic Hypertension. This is hypertension occurring in pregnancy with or without proteinuria in a woman known to have hypertension or chronic renal disease before or persisting after pregnancy. It occurs before 20 weeks' gestation in the absence of neoplastic trophoblastic disease

Chronic hypertension could be Primary Essential hypertension usually without proteinuria or Secondary hypertension involving renal, endocrine and neurological causes. Renal causes include acute glomerulonephritis, chronic nephritis, lupus nephritis and diabetic nephropathy. Some of the endocrine causes are, Cushing syndrome, primary aldosteronism, phaeochromocytoma and thyrotoxicosis.

Neurologic disorders such as quadriplegia might also be implicated.

Other known hypertensive diseases are aortic coarctation, phaeochromocytoma and Cushing's disease.

Chronic Hypertension with Superimposed Pre-eclampsia:

This is the condition in which a known chronic hypertensive patient develops proteinuria and /or oedema during pregnancy.

Gestational hypertension:

This is the development of hypertension in the latter half of pregnancy without other evidence of pre-

eclampsia or chronic hypertensive vascular disease.

Pre-eclampsia

Pre-eclampsia can be classified as severe or mild. In mild disease, blood pressure rises above 140/90 mm Hg on two measurements 6 or more hours apart or an increase of 30 mm Hg systolic and 15 mm Hg diastolic from first-trimester values if pre-pregnant value is not available. In severe pre-eclampsia the systolic and diastolic blood pressures should be more than 160 mm Hg and 110 mm Hg respectively, recorded on at least two occasions at least 6 hours apart with the patient at bed rest. There should be proteinuria of more than 5g in 24 hour urine collection. In addition, there should be oliguria less than 400 ml in 24hrs, cerebral or persistent visual symptoms, epigastric pain or upper quadrant pain, pulmonary oedema or cyanoses, impaired liver function or hepatocellular damage, thrombocytopenia or overt haemolysis and sometimes intrauterine growth retardation.

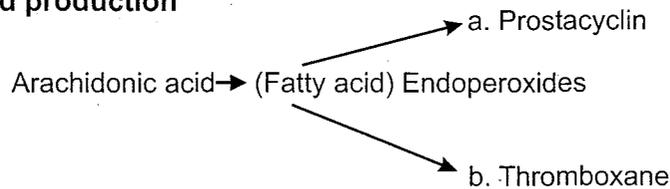
Pre-eclampsia involves multisystem decompensation, cardiovascular collapse and death of the mother and fetus in severe cases. Majority of the cases occur in the young, nulliparous

patients but the condition is becoming prevalent in multiparous women.⁽²⁾

The change in biologic actions of Prostacyclin and thromboxane also influence the severity of pre-eclampsia. In normal pregnancy prostacyclin decreases vasoconstriction and also decrease platelet aggregation. It also decreases uterine activity and increases uteroplacental blood flow. On the other hand thromboxane does the opposite by causing vasoconstriction and platelet aggregation. It also increases uterine activity and decreases uteroplacental blood flow.

Many researchers reported abnormal prostanoid production in either fetal or placental tissues.^(3,4) It was suggested that umbilical artery production of prostaglandin I₂ (PGI₂) is reduced and the capacity of umbilical vessels to synthesize PGI₂ and thromboxane A₂ (TXA₂) is impaired. In addition there is general agreement in the literature that in pre-eclampsia, placental production of PGI₂ is reduced while that of TXA₂ is increased, leading to an increase TXA₂/PGI₂ ratio. When production rates of PGI₂ and TXA₂ were measured simultaneously in normal and preeclamptic patients it was found that the production of TXA₂ by placentas from preeclamptic patients was three times as high as that in placentas from

Pathway of prostanoid production



normotensive pregnancies, whereas PGI₂ production was less than half. Furthermore, the ratio of the placental production rates of TXA₂ to PGI₂ was seven times higher in preeclamptic than normotensive pregnancy^(5,6). It was therefore suggested that this imbalance of increase thromboxane and decrease prostacyclin in PET would account for the major clinical symptoms seen in pre-eclampsia^(4,9)

Theories Of The Cause Of Preeclampsia

Multiple theories have been proposed as causes for pre-eclampsia but to date there is no single, satisfactory unifying explanation. Although increased blood pressure is an essential component of pre-eclampsia, numerous other pathophysiologic events contribute to its

development. Pre-eclampsia is associated with vasospasm, vasoconstriction, activation of the coagulation system, alterations in prostanoid metabolism and abnormal haemostasis. Several studies showed that there is endothelial injury, increased platelet activation with platelet consumption in the microvasculature; capillary leak syndrome and excessive clotting activity.^(7,8,9) Some of the theories proposed are:

- **Immunologic response.** There is inadequate maternal antibody response to the fetal allograft resulting in vascular damage from the circulating immune complexes in some pregnant women. Examples of support for this theory are nulliparous women or women who have changed their partners in subsequent pregnancies. These women have limited antigen exposure resulting in PET.

- **Excessive placental mass.** In some situations there is increased fetal antigen as in twins, molar pregnancies, diabetics with large placentas and hydropic pregnancies.
- **Circulating toxins.** This theory became known when some women with pre-eclampsia had vasoconstrictive substances in their blood, placenta and amniotic fluid.
- **Disseminated intravascular Coagulation (DIC)** causing vascular damage in the kidney and placenta from microvascular thrombin formation and deposition.
- **Endothelial damage.** It has been noted that there is decrease in prostacyclin production (vasodilator) and a relative increase in thromboxane A₂ (vasoconstrictor) resulting in vascular damage. Aspirin therapy is somewhat protective.
- **Endogenous vasoconstrictors.** There is increase sensitivity to vasoconstrictors i.e., vasopressin, norepinephrine and epinephrine.
- **Placental ischaemia.** There is increased incidence of fetal growth retardation

Risk Factors For The Development Of Pre-eclampsia

Clinicians always face the problem of preventing pre-eclampsia. Most times the disease process is quite advanced by the time it is clinically evident and primary prevention would have been ideal with reasonable non-invasive, reliable screening test but this is not available. In the absence of an easily applied screening test the following risk factors should be assessed and taken into consideration in the management of the pregnant women. Examples of such risk factors include nulliparity, extremes of age, family history of pre-eclampsia, chronic hypertension, chronic renal disease, African race, antiphospholipid syndrome, diabetes, twin gestation and angiotensinogen gene T235.

Signs and Symptoms of Pre-eclampsia

In mild Pre-eclampsia there are no symptoms. In moderate to severe disease, there are often symptoms and fulminating or imminent eclampsia present usually with symptoms. Cerebral manifestation includes headache, dizziness, tinnitus, drowsiness, and change in respiration, tachycardia and fever. There are visual changes

such as diplopia, blurred vision, scotomata and amaurosis. Some patients would exhibit gastrointestinal symptoms of epigastric pain, nausea, vomiting and haematemesis. The renal presentation are seen as oliguria, anuria, haematuria and haemoglobinuria

HELLP Syndrome has unique features involving **Haemolysis, Elevated Liver Enzymes, and Low Platelets.** Patients may or may not have other signs of pre-eclampsia. HELLP syndrome most often presents with rapid deteriorating disease and delivery should be considered.

The liver enzymes that are notably increased are SGOT (serum glutamic oxaloacetic transaminase) and SGPT (serum glutamic pyruvic transaminase). Antenatal suspicion depends on clinical evidence of pre-eclampsia though severe hypertension or proteinuria might not be present. Epigastric pain especially a right upper quadrant pain with vomiting and blurred vision. A combination of the above symptoms may indicate liver involvement, which might result in liver rupture⁽¹⁰⁾

The following management protocol is suggested to guide against liver rupture.

- Any patient with pre-eclampsia especially severe pre-eclampsia should be routinely investigated for HELLP Syndrome - as first sign of hepatic rupture.
- Ultrasonogram might show haemorrhage or haematoma
- If HELLP is confirmed, then delivery is considered, either vaginally or by caesarean section after correcting coagulation defect.
- If there is rupture of the liver perform laparotomy and evacuate and drain the haematoma. The laceration should be sutured with a round body needle if possible.

Investigations

Proteinuria as mentioned earlier should always be established but a more realistic value is 24-hour protein collection. Increasing proteinuria shows the severity of the disease.

Uric acid elevated levels are particularly useful in diagnosing and monitoring the pre-eclamptic patient, because increased levels often occur early in pre-eclampsia and reflect the severity of the disease.

Uric acid is filtered by the glomerulus with other electrolytes and reabsorbed at the proximal convoluted tubules and secreted at the distal

convoluted tubules. If there is damage to the tubules then the uric acid levels rise quickly in pre-eclampsia. On the other hand 50 % of the glomerulus can be damaged and still filter Blood Urea and Electrolytes (BUE) without causing severe increases in serum levels. When BUE rises then there is extensive damage to the glomerulus. It is therefore essential that uric acid levels are routinely checked in Pre-eclampsia and monitored as a prognostic factor.

Serum uric acid levels decrease 25% during early pregnancy because of the increased renal clearance, reaching levels of 2.5 to 4 mg/dl, and increasing towards nonpregnant values late in pregnancy. If therefore uric acid rises to or above nonpregnant values in early pregnancy then such levels should be considered high and necessary steps are taken to arrest the situation.

Other chemistry evaluation should include **Blood Urea and Electrolyte** and serum **creatinine**. Complete **blood count** should be done and occasionally elevated haematocrit might be due to haemoconcentration.

Platelet levels are also measured and values less than 150,000/L or falling platelet levels should draw attention to the severity of the disease. In addition prothrombin time and partial thromboplastin time should be measured. Increased **aspartate transaminase (AST)** and **alanin transaminase (ALT)** over 1000 IU/L are significant. In general any rise in liver enzymes suggests liver involvement is beginning, and prompt action should be taken. Clotting profile should be assessed early to guide against intravascular coagulation

Management

In the management of pre-eclampsia the following principles are applied

- Control hypertension
- Prevent fits
- Safe mother followed by delivery of a live matured baby.

The ultimate aim of management of pre-eclampsia is safety of the mother first, followed by a live baby. In severe cases the fetus is sacrificed in the interest of the mother. When one considers expectant management as against immediate delivery, the severity of the disease process, maternal condition, gestational age, fetal condition and cervical effacement using the modified Bishop score should be assessed. Ideally, the patient should be hospitalised for bed rest, which is good therapy for

fetal survival by decreasing progression of disease to severe state. Bed rest may also arrest the clinical course of the disease, improve fetal maturity without compromising maternal condition and allows the fetus to be matured enough to decrease prolonged neonatal intensive care admission. The condition should be explained to the patients and relatives so that in spite of the economic constraints in our sub-region the mothers will be willing to rest in hospitals for delivery of healthy babies.

Pre-eclamptic patients can be followed with serial ultrasounds for fetal growth and amniotic fluid volume in the third trimester. Gestational age should be worked out from earlier ultrasound and not from late ultrasound scan. Surveillance of fetal well-being is recommended starting from the third trimester by performing fetal kick count and if possible using non-stress test (NST). Preeclampsia in the first pregnancy can recur in 25% of subsequent pregnancies and up to about 50% in subsequent pregnancies of multiparous patients who have had preeclampsia. It is not entirely a condition of nulliparous patients and can occur in multiparous women.

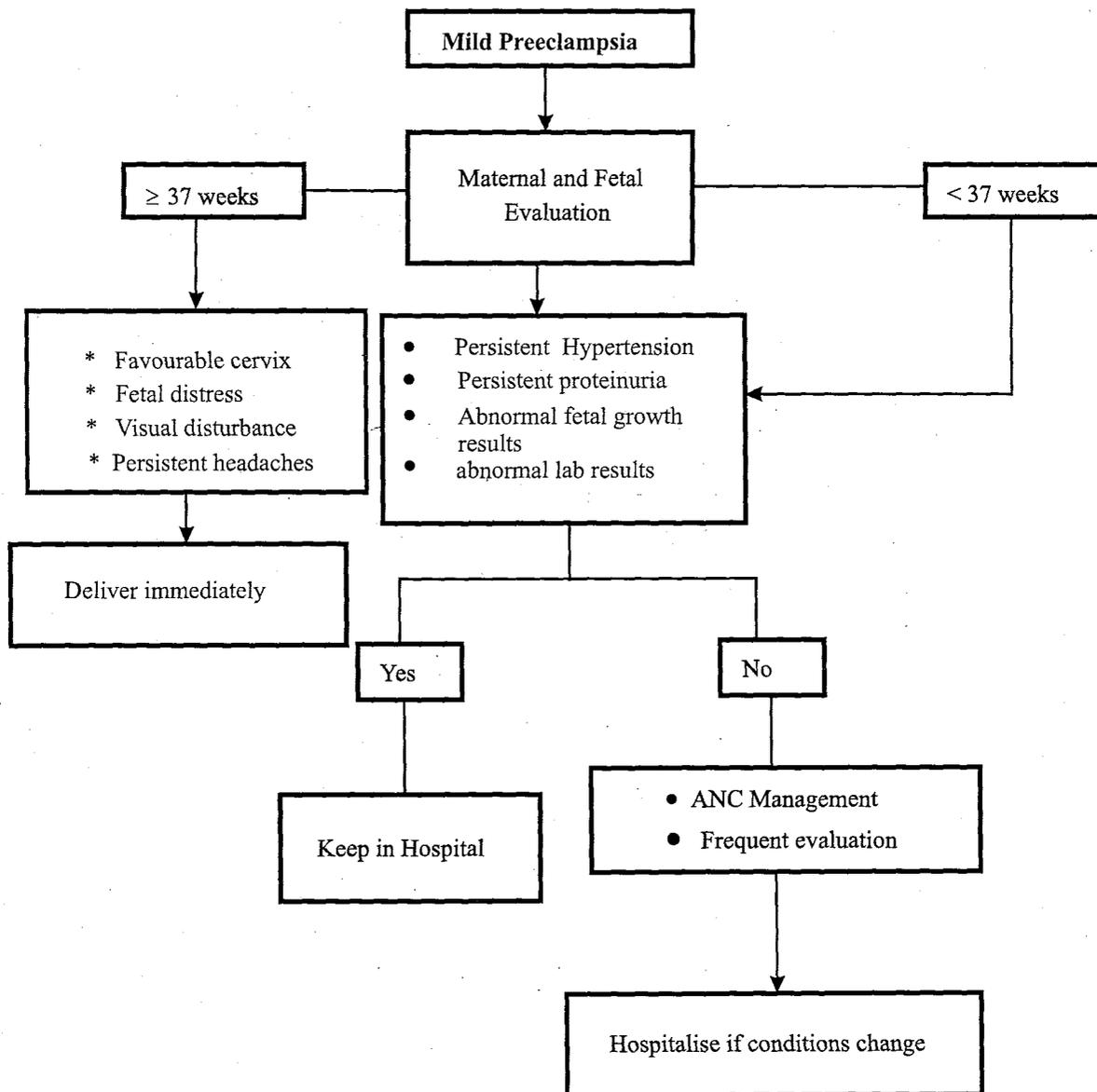
Preeclampsia does not increase the risk of hypertension later in life but the condition may persist in women who have recurrent disease. However, the history and examination should be reviewed to rule out maternal underlying causes such as chronic hypertension and other maternal diseases causing hypertension.

The cure for preeclampsia is delivery and a decision should be taken. Easily, the decision is straightforward at term whether pre-eclampsia is severe or mild because the obstetrician has to deliver the fetus. The difficulty arises when the condition is remote from term.

Bed rest with constant fetal and maternal monitoring is the key to success till pregnancy reaches term. If maternal or fetal parameters worsen, for example, the blood pressure is uncontrollable, proteinuria is increasing, there is fetal growth restriction/retardation or decreased fetal kick count then delivery must be accomplished

Choice of delivery should be based on obstetric indication and vaginal route will provide the least haemodynamically stressful outcome if there is no contraindication. If the patient is remote from term, especially nulliparous with unfavourable cervix for induction then primary caesarean section is indicated.

Algorithm for Mild Preeclampsia



Mild Pre-eclampsia

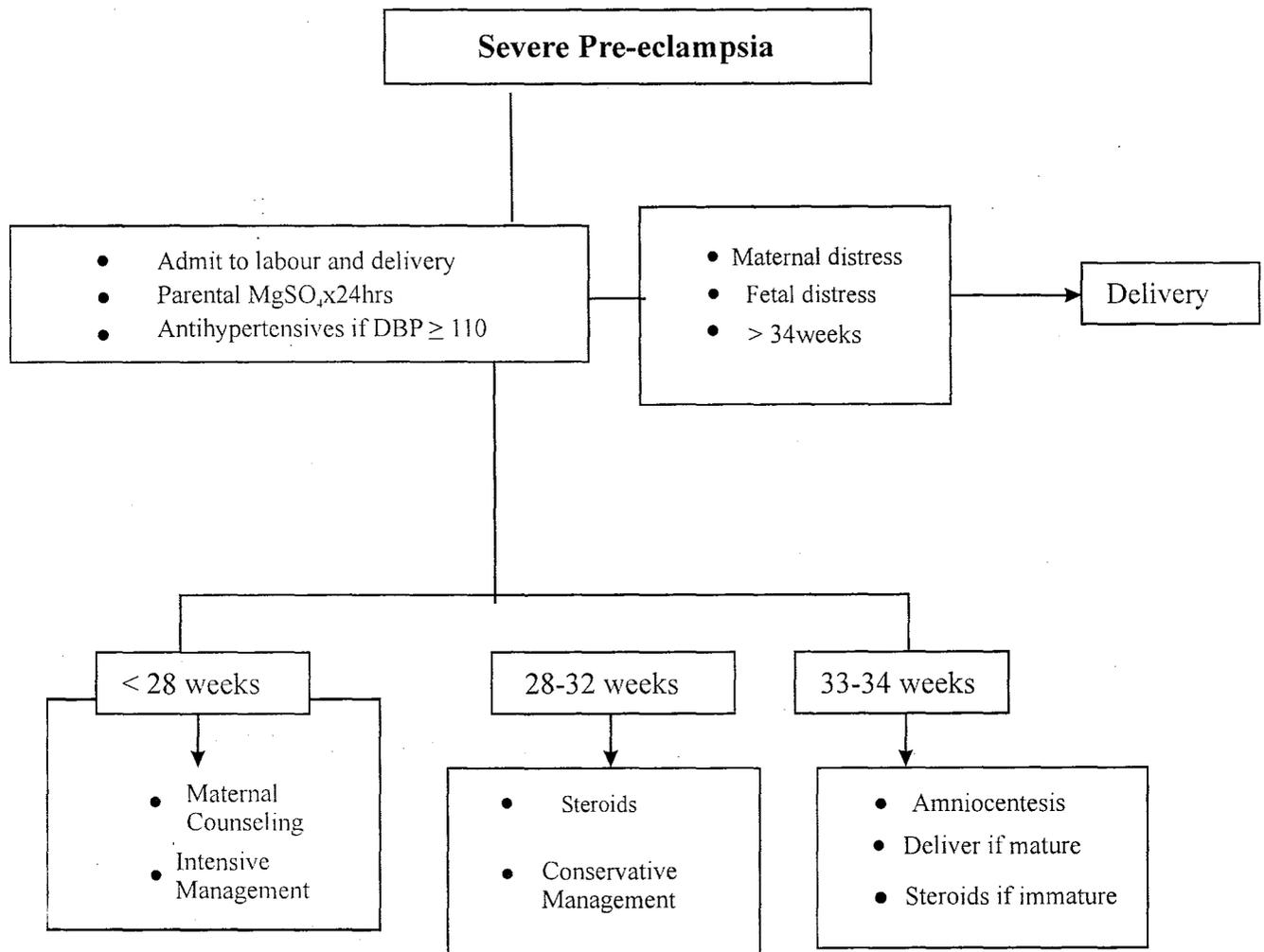
Term

Basically, term patients should be evaluated for delivery and vaginal route is preferred. Labour should be induced or augmented. In general pre-eclamptic patients whose blood pressures have been controlled should be delivered after 37 completed weeks of gestation. At this gestation, it can be assumed that there is lung maturity and the baby has attained reasonable weight to survive outside the uterine cavity.

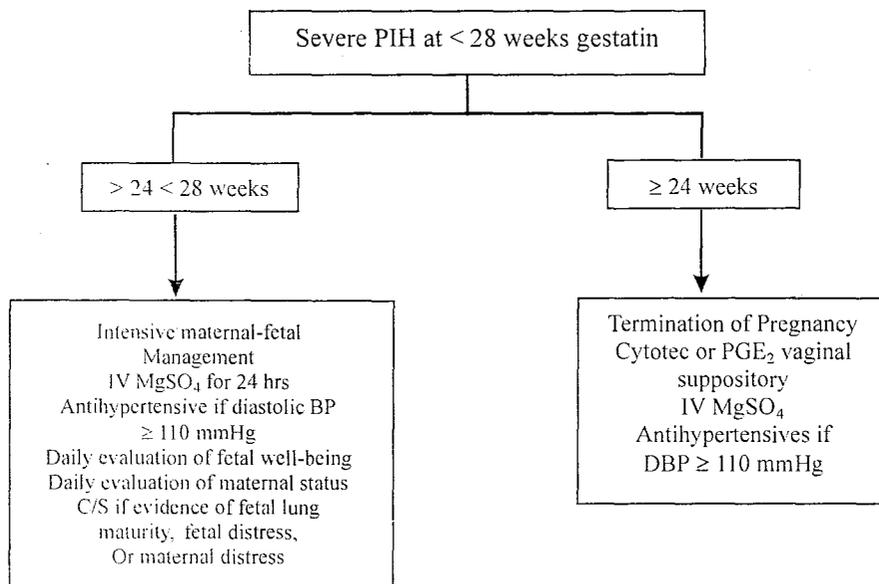
Preterm

These patients should be admitted for evaluation to minimise maternal morbidity and to achieve optimal perinatal outcome. Close observation for signs of severe disease or fetal maturity should be initiated. Traditionally, some obstetricians practise continuous observation of the preterm patient with severe pre-eclampsia. This is attractive but has been condemned by other clinicians. However, recent studies suggest that expectant management of the severe pre-eclamptic up to 32 weeks' gestation may in fact reduce neonatal complications and the neonatal stay in NICU ^(11, 2).

Algorithm for Severe Pre-eclampsia



Algorithm for severe Pre-eclampsia below 28 weeks



Management Of Severe Preeclampsia

Severe Pre-eclampsia is a deadly disease and delivery must be considered taking into account the safety of the mother first before the neonate. In general most clinicians would deliver the mother if the disease goes beyond 34 weeks where it is assumed that there is fetal lung maturity. In any

case the mother must be delivered if there is fetal compromise before 34 weeks or evidence of lung maturity.

Management of preterm Pre-eclampsia:

1. Ultrasound for growth, fetal number, fetal anatomy, fetal measurements, placental localisation and amniotic fluid volume
2. Fetal kick counts chart.
3. Non-stress test (NST). The pattern of the fetal heart rate is evaluated by external electronic fetal monitoring
4. Biophysical profile (BPP): The fetal condition is assessed by real-time ultrasound monitoring of fetal tone, movement, breathing motions, and amniotic fluid volume.
5. Vital signs: there should be 4 hourly BP and testing of the urine daily.
6. Fetal heart rate should be checked at least twice in a day
7. Daily weighing of mother
8. Betamethasone (IM 12 mg, 12 hourly) should be given if the fetal pulmonary maturity is less than 34 weeks gestation.
9. A urinary output should be monitored hourly.
10. A fluid chart should be commenced to record fluid balance on an hourly basis. The chart should include a cumulative calculation of fluid balance.
11. Sedated patients should have pulse oximetry.

Complications of Pre-eclampsia

Fetal

1. IUGR
2. Oligohydramnios
3. Abruptio
4. Placental infarcts
5. Uteroplacental insufficiency
6. Prematurity
7. Stillbirths
8. Neonatal deaths

Maternal

1. Eclampsia
2. Stroke
3. DIC
4. Increase in C/S del
5. Acute renal failure
6. Hepatic failure
7. Liver rupture
8. Maternal deaths
9. HELLP
10. Encephalopathy

Drug Treatment in Pre-eclampsia

A wide variety of antihypertensive agents are used in the antenatal management of pre-eclampsia in our sub region. Some of the drugs used are methyl dopa, hydralazine, nifedipine, propranolol and labetalol. The principal parenteral agent used is Hydralazine and sublingual Nifedipine. Hydralazine is the most widely used drug for acute management of blood pressure emergencies in the antepartum period. It is a potent vasodilator that acts directly on the vascular smooth muscle and may cause a drastic fall in blood pressure, consequently affecting placental blood flow ⁽¹³⁾. Hydralazine can also cause fluid retention, tachycardia and headache. ⁽¹⁴⁾

Nifedipine, the second commonly used first line drug in our practice for acute management of hypertensive emergencies has advantages because of oral therapy. Its most important side effect is headache. It has a negative inotropic action on the myocardium and can precipitate heart failure in combination with beta-adrenergic blocking agents. In controlling blood pressure, it is important to avoid too rapid and dramatic a fall because an extreme drop can lead to cortical blindness, myocardial infarction, renal failure and acute fetal distress. The aim should be to maintain a blood pressure below 160/100 mm. Hg.

A study comparing oral nifedipine and intravenous hydralazine ⁽²⁾ shows that nifedipine and

hydralazine achieved good blood pressure control averaging 140/96 mm Hg for nifedipine and 150/98 mm Hg for hydralazine before delivery from initial blood pressures of 190/125mmHg and 189/134mmHg respectively. The authors further showed that within the first 30-minutes, oral nifedipine and intravenous hydralazine lowered the average systolic/diastolic blood pressure by 19/17 mm Hg, and 26/28mmHg respectively. Fenakel et al ⁽¹⁵⁾ in a similar study reported a fall in blood pressures of 24/20 mm Hg and 30/34 mm Hg in the nifedipine and hydralazine groups, respectively. The authors concluded that both drugs are effective in the control of blood pressures in pre-eclampsia in our sub region.

Hydralazine is administered intravenously and needs strict monitoring. On the other hand nifedipine is administered orally and might be less demanding on hospital staff.

Hydralazine

Intravenous hydralazine is given at an initial dose of 5mg as a test dose followed by 10 mg boluses every 20-30 minute determined by the blood pressures. The blood pressure should be checked every 5-10 minute until the condition is stable. The goal of this management is to maintain the blood pressure in the range of 140 - 150/90 - 100 mm Hg to prevent uterine hypoperfusion.

In another regime intravenous hydralazine is commenced with a bolus of 5mg and followed up with an intravenous infusion of 10-15-mg of hydralazine in 500ml of normal saline. Blood pressure should be checked every 5 minutes until condition is stable with blood pressure between 140 -150 systolic and 90 - 100 mm. Hg. diastolic. Gradual reduction with intravenous infusion of hydralazine will benefit uteroplacental perfusion

Sublingual Nifedipine

In acute management of hypertension 10-mg sublingual Nifedipine is initially given. The dosage is repeated every 20-30 minutes aiming at blood pressure range of 140 to 150/90 to 100 mm Hg.

Second line drugs used in our hospitals are Aldomet, and Propanolol. Intravenous Labetalol is occasionally used for antenatal management of acute hypertension. It is both an alpha and beta-adrenergic blocking agent and has the advantage of B-blockade without side effects of tachycardia. It is however less popular than hydralazine probably because of availability.

Fluid Management

Fluid therapy is important in the management of pre-eclampsia which is characterised by vasoconstriction and intravascular volume depletion. Intravenous colloid such as Haemacel can be started initially with 500 ml over 15 minutes. This is followed by about 2 litres of crystalloids in 24 hours. The urine output should be monitored with urine production of about 30mls/hour.

Central Venous Pressure (CVP) catheter should be inserted after checking clotting profile to prevent overloading the system. If the urine output remains low, despite an adequate CVP low dose dopamine (2-3 µg/kg/minute) or 100 mls of 20% Mannitol can be administered.

Eclampsia

Eclampsia is defined as the occurrence of fit or seizure in a patient with signs and symptoms of pre-eclampsia in the absence of underlying neurologic disease. The cause of eclamptic convulsion is not known.

However, the pathophysiology of eclampsia is thought to involve cerebral vasospasm leading to ischaemia, disruption of the blood brain barrier and cerebral oedema. Most women with extreme pre-eclamptic hypertension do not have fits, conversely, however, many episodes of eclampsia occur when the blood pressure is relatively low or normal.

It often presents with few warning signs and might occur in a patient with previously mild disease and therefore predicting its occurrence is as difficult as predicting the timing.

Overt signs that might be present include headache, epigastric pain, visual disturbances, hyperreflexia and clonus and these might be present during labour, just before delivery or just after delivery.

Eclampsia is associated with organ system derangement involving the cardiovascular system with generalised vasospasm, increased peripheral vascular resistance, increased left ventricular work index and decreased pulmonary wedge pressure. There might be decreased glomerular filtration rate, decreased renal plasma flow and decreased uric acid clearance. Haematologically, there might be decreased plasma volume with associated increased blood viscosity and haemoconcentration. The obstetrician should also watch for coagulopathy.



Eclamptic Patient After Convulsion

At autopsy, there might be cerebral oedema and cerebral haemorrhage. The liver might show hepatic periportal necrosis, hepatocellular damage and subcapsular haematoma, which might lead to liver rupture

Management of Eclampsia

If the patient fits (eclampsia) she should be stabilised and the timing and route of delivery must be determined because there is generalised organ system derangement. Eclampsia usually begins as a gradual process, starting with rapid weight gain and finally generalised convulsions or coma. Excess weight gain with or without clinical oedema especially during the last trimester should be watched for because this might be the first warning sign

Hypertension is the hallmark of eclampsia and excess weight gain and/or oedema is not necessary for diagnosis. Hypertension may be relative and any rise in blood pressure that is 30 mm Hg systolic or 15 mmHg diastolic above the first half of pregnancy blood pressure reading is regarded as significant.

Eclampsia is one of the serious obstetric emergencies seen in our sub-region and requires active and proper management to prevent maternal mortality or morbidity. The maternal airway should be protected and steps taken to prevent maternal injury and aspiration. A padded tongue blade

should be inserted between the patient's teeth to prevent biting of the tongue taking care not to stimulate the gag reflex with the blade and cause vomiting and aspiration. Wait for convulsions to abate and give Magnesium Sulphate ($MgSO_4$) to control convulsions. Adequate oxygenation should be maintained and intravenous access should be secured to judiciously hydrate the patient and to administer any necessary medication.

Correct maternal acidemia and avoid polypharmacy. The mother should be turned on the left lateral position and continuous fetal heart rate monitoring should be instituted.

Do not proceed to deliver the fetus until the maternal condition is stabilised. Fetal status is usually improved as well. When the maternal condition is stable vaginal delivery should be undertaken. Abdominal delivery should be reserved for obstetrical indications. As much as possible emergency caesarean section should be avoided.

After the convulsive seizure, complete blood count, platelets, electrolytes, glucose, calcium, magnesium, blood type and antibody screen should be assessed. If there is an indication for Caesarean section the coagulation profile should be normalised first.

Careful neurologic examination should be done after seizures and the cervix should be examined to formulate delivery strategy.

Treatment of eclamptic Fits

Magnesium sulphate ($MgSO_4$) is a safe and effective agent to prevent and treat convulsions and can be given by the IM or IV route. A loading dose of 14gm of $MgSO_4$ should be given both as intravenous and intramuscular. Give IV $MgSO_4$ in doses of 20% (i.e. 4gm) slowly over 5 minutes and IM $MgSO_4$ 10mls of 50% deep into each gluteal muscle. The loading dose can always be given irrespective of the urinary output.

Maintenance Dose is given as 10 mls of 50% $MgSO_4$ intramuscular every 4 hours. Six maintenance doses are recommended. The maintenance dose should only be given if the urinary output is more than 100mls in the previous 4 hours, respiratory rate is more or equal to 12 per minute and the patellar reflexes are present.

Monitoring the Patient on Magnesium Sulphate

The patient's vital signs should be assessed as well as the urine output and reflexes every 2 to 4 hours. In addition, the patellar reflexes should be present and the respiratory rate should be above 12/min.

The pulse should be monitored for arrhythmia. Watch for oxygen desaturation, widening of QRS or prolonged Q-T interval on ECG and the urine output which should be 30 ml/hr. If any of these parameters is abnormal check the level of $MgSO_4$ and adjust the infusion.

The use of $MgSO_4$ in preventing eclamptic fits is based on empirical observation. This is because the pathogenesis of eclamptic fits and the mechanism of action of $MgSO_4$ are not completely understood. Clinically, $MgSO_4$ acts at the neuromuscular junction and abolition of patellar reflexes has been seen as an early sign of magnesium toxicity. It is therefore prudent that the patellar reflexes is carefully monitored if laboratory facilities are not readily available.

Magnesium sulphate has a wide margin of safety, but serious complications have been associated with its use.

The laboratory values quoted below can serve as a guide in the management of the patient although there is no indisputable clinical basis for choosing these levels. Laboratory values should not serve as a substitute for good clinical assessment of the patient.

Labouratory values of $MgSO_4$

- Therapeutic levels accepted are 4 to 8 mEq/L
- Signs at toxic levels
 1. Absence of deep tendon reflexes (>4 to 6 mEq/L)
 2. Somnolence (>8 mEq/L)
 3. Respiratory depression (>8 mEq/L)
 4. Cardiotoxicity (> 15 MEq/L)

If despite the regimen described above, the patient continues to fit, consider IV Phenobarbitone infusion 500mg in 500mls of Normal Saline, slowly titrated against the level of consciousness and frequency of fits.

Calcium Gluconate is the antidote to $MgSO_4$ toxicity at the dose of 1gm (i.e. 10 mls of 10%) and should be given slowly over 3 minutes.

Prevention of Eclampsia

Low Dose Aspirin Therapy prophylaxis could be given to women at high risk of pre-eclampsia, including those with history of pre-eclampsia or eclampsia, renovascular disease, diabetes mellitus prior to pregnancy and family history of pre-eclampsia. The dosage of aspirin is 60 mg/day, starting after 12 weeks of gestation. It is not useful if pre-eclampsia has already developed. Aspirin is safe for both the mother and fetus.

Calcium supplementation of 1.5-2.0g/day may be added^(19,20).

Controversies And Discussions

Thiazide Diuretics

The cause of Pre-eclampsia is not known. It is a systemic illness characterised by vasoconstriction and intravascular depletion, which are worsened by diuretics, and therefore, these agents are not used in the control of pre-eclampsia. Studies have shown that prophylactic thiazide medication does not reduce the incidence of pre-eclampsia.^(21,22) It was found that patients receiving diuretics delivered infants who weighed less than the control group. The short-term use of diuretics in pregnancy showed poor fetoplacental unit and maternal organ perfusion.⁽²³⁾ Other studies have shown that the plasma volume was significantly higher when patients discontinued diuretics but the pregnancy outcome was similar for the two groups.⁽²⁴⁾ It is also known that plasma volume depletion is associated with poor perinatal outcome⁽²⁵⁾, therefore one should use diuretics with caution in pregnancies complicated by chronic hypertension. Diuretics can however be used in the presence of pulmonary oedema.

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Intrauterine Growth Restriction

K Nkyekyer

Introduction

Birth weight is perhaps the most commonly used parameter to assess the outcome of pregnancy. It is determined to a large extent by the adequacy or otherwise of intrauterine growth. Adequate intrauterine growth, which is a most important clinical evidence of fetal well-being, is determined not only by the adequacy of nutrient availability from the mother and optimum placental transfer of these nutrients to the fetus, but also by the intrinsic potential of the fetus and its ability to utilize the nutrients supplied.

Failure of adequate intrauterine growth places the fetus at increased risk of perinatal morbidity and mortality, as well as impaired growth during childhood. It is also becoming increasingly evident that inadequate intrauterine growth is associated with an increased risk of the development of cardiovascular and metabolic diseases later in life.

It is imperative, therefore, that as much as possible measures be taken to reduce the risk of inadequate intrauterine growth and, when it occurs, to detect it early so that appropriate management measures may be instituted.

What is Intrauterine Growth Restriction (IUGR)

The commonest definition used is fetal or birth weight that is below the 10th percentile for gestational age. This presupposes an accurate determination of gestational age.

Others have used the 5th percentile of weight for gestational age, while some have suggested the use of the ponderal index (PI), which is defined by the equation $PI = \text{weight in grams} / (\text{height in cm})^3$. The baby is considered growth-restricted if the PI is less than the 10th percentile; the disadvantage with the ponderal index is the potential error introduced by cubing the height.

The word **restriction** is preferred to **retardation** (in IUGR) since the latter may connote mental retardation in the minds of the parents. Moreover, restriction makes the point with respect to pathology better than retardation, as it implies a

possibly reversible, transient condition.

The incidence of IUGR varies according to the population being studied. It is estimated to be about 4%-8% in developed and 6%-30% in developing countries. It must be mentioned that growth curves determined for one environment may not necessarily be applicable to another.

IUGR has traditionally been divided into two major types: symmetrical and asymmetrical. In symmetrical IUGR the onset is usually earlier in pregnancy (before 28 weeks) than asymmetrical and all body organs tend to be proportionately reduced. The fetus grows at a constant but slower rate than normal; head circumference and length of the fetus may therefore show no relative discrepancies and the ponderal index may be normal. This pattern is suggestive of a limited growth potential, either hereditary or acquired. It is associated with diseases that seriously affect fetal cell numbers, such as conditions with chromosomal, genetic, malformation, teratogenic, or infectious aetiologies. It is necessary in this regard to emphasise that a baby may exhibit symmetrical IUGR simply because it is constitutionally small; it is otherwise completely healthy and normal. In the strictest sense of the word that baby has not suffered intrauterine growth restriction; it should more appropriately be referred to as being small for gestational age (SGA). It follows therefore that while all IUGR infants will be SGA, not all SGA infants will be IUGR infants.

Asymmetrical IUGR, the commoner of the two, tends to occur much later on in pregnancy (after 28 weeks), so that normal growth may have been observed before the restriction becomes manifest. The rate of fetal growth slows and may even stop, and some organs are more affected than others. Brain growth is preserved, skeletal growth is only slightly affected, while growth of abdominal viscera (e.g. liver, spleen) and of somatic tissues is most affected. This phenomenon is referred to as "brain sparing". Head circumference is therefore less affected, if at all, than abdominal circumference and body weight; the ponderal index in this situation is low. Asymmetrical IUGR is usually due to impairment of oxygen and/or nutrient delivery to the

fetus during the last trimester, which could arise from a variety of causes.

The utility of this classification of IUGR has been questioned on the basis of the fact that a significant proportion of growth restricted newborns show a growth pattern intermediate between the two types, indicating a continuum of fetal growth patterns from the extreme symmetrical to the typical asymmetrical appearance. The advocates consider that there is so much overlap between the two types that distinguishing between them is artificial and without sound biological basis. ⁽²⁾

Physiology Of Fetal Growth - Some Pertinent Perspectives.

1. Maternal nutrition

There is considerable evidence that maternal nutrition has a significant bearing on fetal growth. This is not difficult to imagine since ultimately the fetus has to depend on maternal source for nutrient supply of anabolic elements and oxidizable fuel for energy. For example, in the Dutch famine of 1944-45, impaired nutrition of pregnant women was associated with a decrease in infant birth weights to a maximum of between 200 and 340gms below pre-famine levels. ⁽³⁾ It is not only the quantity but also the quality of maternal dietary intake that is important.

The major nutrient elements for the fetus that must be provided by the mother are glucose, amino acids, and lipids as well as minerals, vitamins and micronutrients.

Glucose is the major energy substrate for the fetus, and may also be stored in the form of glycogen. Amino acids are used in protein synthesis, as substrate for energy production as well as a source of carbon and nitrogen for interconversion to other substrates. Lipids are stored as energy sources in the form of triglycerides or as structural lipids in cellular membranes of various tissues. They are also used to synthesise substances like prostaglandins and leucotrienes. Polyunsaturated fatty acids are fundamental for fetal growth and development of fetal brain, to maintain the vascular system and for synthesis of eicosanoids.

2. Placental Function.

The placenta is the organ by which nutrients and oxygen from the maternal circulation are made available to the fetus. This is predicated on adequate utero-placental and feto-placental blood flow.

Glucose is transported across the placenta by facilitated diffusion. Placental glucose uptake is linearly correlated with maternal glucose concentration. It is important to appreciate that the placenta is, metabolically, a very active organ and that glucose transferred to the fetus represents about 60% of total placental glucose uptake. The remainder is used by the placenta for oxidation, stored as glycogen, or converted to lactate for use by both mother and fetus. As pregnancy advances there is increase in uterine blood flow with resultant increase in glucose delivery to the fetus. Work in pregnant sheep has shown that a greater than 50% decrease in uterine blood flow resulted in decreased fetal glucose uptake. Also, a reduction in umbilical blood flow by ligation of one umbilical artery resulted in decreased fetal glucose uptake and fetal growth restriction.

Amino acids are transported across the placenta by active transport while lipids are transported by simple diffusion.

3. Fetal Utilization

Glucose availability is the primary determinant of fetal growth, working through a glucose-insulin-insulin-like growth factor, (IGF), axis. Placental transfer of glucose stimulates fetal insulin secretion, which in turn stimulates fetal IGF secretion. Thus the primary fetal hormones involved in the regulation of fetal growth are fetal insulin and fetal insulin-like growth factors (IGFs).

There are two main types of IGFs; IGF-1 and IGF-2 both of which regulate fetal growth through the IGF-1 receptor. Both influence embryonic growth, the IGF-2 doing so earlier than IGF-1; however IGF-1 seems to be the only one influencing fetal growth. Indeed fetal size correlates well with IGF-1 concentrations. IGF-1 enhances fetal anabolism and cell replication; it also reduces placental lactate production and placental demand for amino acids, thus allowing an increased substrate delivery to the fetus. There is also evidence to suggest that IGF-1 in amniotic fluid swallowed by the fetus may promote gut maturation and influence fetal growth. ⁽⁴⁾

The primary effect of insulin is to enhance adipogenesis. For example, infants of diabetic mothers have only a small increase in lean body mass and a massive increase in adipose tissue. The effect of insulin on lean body mass is most likely mediated through IGF-1 release.

Although traditionally growth hormone (GH) has been considered not to play any role in fetal growth

It is now thought that fetal GH may have an effect on fetal growth through action independent of IGF-1 activity. (4) There is also a suggestion that leptin, an adipocyte derived peptide hormone under the control of the *ob* gene may have a role to play in the nutritional homeostasis and growth of the fetus. (5)

In summary, the major determinants of fetal tissue growth are nutrients and oxygen, and therefore of fetal growth are:

- * Maternal nutrition and health status
- * Placental blood flow
- * Placental substrate uptake
- * Placental metabolism
- * Placental transfer function
- * Umbilical blood flow
- * Fetal endocrine status.

It is estimated that, in general, about 50% of the differences on fetal growth are maternal in origin, 20% fetal and 30% unknown.

Etymology of IUGR

Considering the causes of and the factors associated with IUGR it is useful to group these into fetal, placental and maternal.

Fetal conditions associated with IUGR

Genetic:

- Constitutionally small fetus
- Chromosomal abnormalities:
 - * autosomal trisomies (18,21,13)
 - * monosomy (Turner's syndrome)
 - * deletions
 - * Inborn errors of metabolism

Infections:

- Viral:
 - * rubella
 - * cytomegalovirus
 - * herpes
- Bacterial:
 - * syphilis
 - * listeria monocytogenes
- Protozoal:
 - * malaria
 - * toxoplasma gondii

Anatomical malformations:

- * cardiovascular defects
- * gastrointestinal defects
- * genitourinary defects
- * ventral wall defects
- * skeletal dysplasias
- * central nervous system anomalies

Quantitatively, fetal conditions causing IUGR are uncommon but they are frequently associated with severe restriction of growth and a bad prognosis with respect to long-term outcome. Genetic factors are crucial in determining the rate of intrauterine growth and therefore various genetic disorders have been associated with IUGR. It is estimated that about 40% of chromosomally abnormal fetuses have IUGR. The commonest karyotypic abnormalities are the autosomal trisomies, of which trisomy 21 is the most prevalent. A recent report has documented profound IUGR and postnatal growth failure associated with deletion of the insulin-like growth factor gene. (6)

Many congenital anomalies are associated with IUGR. The rate of IUGR in infants with major structural malformations is about 22%; with increasing numbers of anomalies in any particular fetus the risk of IUGR is raised.

The agents implicated in fetal infections that lead to IUGR include viruses, bacteria and protozoa. The mechanisms by which they cause IUGR may include loss of vital cellular constituents, with their replacement by non-functioning cells, delay in cellular division and vascular insufficiency resulting from damage to endothelium of small vessels. In malaria endemic areas the incidence of umbilical cord blood parasitaemia is about 6.7%-16.9%. Cord blood parasitaemia was found to be associated with a two-fold increase in risk for IUGR, though this did not reach statistical significance after multivariate analysis. (7)

Placental conditions associated with IUGR.

- Chronic abruptio placentae
- Placenta praevia
- Velamentous insertion of the cord, Battledore
- Circumvalate placenta
- Multiple infarcts
- Chorioangioma
- Multiple pregnancy
- Malarial parasitization

Abnormalities of placentation, infarcts and chronic abruptio may result in loss of placental surface area available for exchanging vital nutrients.

Multiple pregnancy is associated with a 23-34% incidence of IUGR. It may be particularly severe in situations in which there is shared fetal circulation and therefore twin-to-twin transfusion is likely to occur. Sharing of maternal nutrients may contribute to the occurrence of IUGR.

In malaria endemic areas the rate of placental

parasitization is between 20%-45%. Placental parasitisation was found to be associated with a doubled risk of IUGR and this was significant after multivariate analysis. The effect is more pronounced in the primigravid. Antimalarial prophylaxis reduces both placental and cord blood parasitaemia and thus reduces the incidence of IUGR.⁽⁷⁾

Maternal conditions associated with IUGR.

1. Medical conditions:

- Pre-eclampsia
- Chronic hypertension
- Diabetes mellitus (especially with vasculopathy)
- Chronic renal disease
- Collagen vascular disease (antiphospholipid syndrome)
- Infections (malaria, HIV, tuberculosis)
- Haematological (sickle cell disease, anaemia)
- Hypoxic conditions (asthma, cyanotic heart disease)
- Inflammatory bowel disease

2. Environmental factors:

- Poor nutritional status (low socioeconomic status)
- Smoking
- Alcohol intake
- Drugs (antimetabolites, anticonvulsants, anticoagulants).
- Substance abuse (heroin, cocaine, methadone)
- High altitude

Others:

- Low pre-pregnancy weight (<50kg)
- Poor pregnancy weight gain
- Previous IUGR infant
- Low maternal age
- Unexplained raised maternal serum alpha-fetoprotein

Whereas in developed countries cigarette smoking is by far the most important single etiological factor for IUGR, in developing countries poor gestational nutrition, low pre-pregnancy weight and malaria top the list.

As regards physical activity moderate exercises in a healthy, well-nourished pregnant woman does not have any adverse effect on the woman or her fetus. However, heavy occupational physical activity (long hours, protracted ambulation, heavy lifting) is associated with lower birthweight. This is

especially important in developing countries in which mothers not only have compromised nutritional status but also engage in heavy physical work.

The increased risk of IUGR in women with sickle cell disease is due to chronic maternal anaemia and/or varying degrees of sickling and vaso-occlusion in the placental circulation, resulting in reduced oxygen availability to the fetus.

Low weight gain in the second and third trimester, of less than 0.1kg/week and 0.3kg/week respectively, has been reported to be associated with about two-fold increase in the risk for IUGR.⁽⁸⁾

Diagnosis of IUGR

The diagnosis of IUGR depends on an accurate estimation of gestational age. Since about 20-40% of pregnant women do not accurately know the date of their LMP, it is useful to advise pregnant women to book for antenatal care latest by early second trimester, so that gestational age may be confirmed by ultrasound scan. In the first trimester gestational age is determined using the crown-rump length (CRL). From 13 weeks gestation onwards, because of fetal flexion the CRL is not reliable and the parameters used are: biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), and femur length (FL). The transcerebellar diameter (TCD) is said to accurately date pregnancy but it is not commonly used.

Although most women who have babies with IUGR may not have any known risk factors it is important to take note of any characteristics that puts a woman at increased risk for having a baby with IUGR, so that there would be increased surveillance of these women. These characteristics may be present at booking or may develop later during the antenatal period; the etiological factors enumerated above provide a list of most of the risk factors to be taken into consideration.

Measurement of the symphysis-fundal height (SFH), a simple procedure which is part of routine antenatal care, may be the means by which suspicion of IUGR will be aroused. The sensitivity of SFH measurement in detecting IUGR has been reported to be as low as 40% and as high as 86%, with specificity between 79% and 93%. Despite its drawbacks it is a simple, inexpensive and easy-to-learn method of assessing fetal growth. It has twice the detection rate of uterine size assessment by fundal palpation in the diagnosis of not only SGA but large for gestational age (LGA) babies as well⁽⁹⁾. A

This measure is used to measure the distance from the top of the symphysis pubis to the dome of the fetal fundus. The measurement, in centimetres, is usually within 3cm of the gestational age, in weeks, between 20 and 38 weeks gestation. A SFH that lags by more than 3cm or is increasing in disparity with gestational age may signal IUGR. If in addition there is a clinical impression of reduced fetal volume (though this may be subjective) or inadequate maternal weight gain or decreasing maternal weight this should strengthen the suspicion.

The diagnosis of SGA/IUGR has to be confirmed by ultrasound examination. Fetal weight estimation has the best sensitivity and specificity for identifying babies who will be SGA at birth. Its measurement is based on the use of equations, which relate fetal weight to two or more of the biometric parameters mentioned above (i.e. BPD, HC, AC, FL). The ultrasound machine automatically calculates the weight once the appropriate parameters are known. The estimated fetal weight should be compared with fetal monograms and not neonatal ones. This is because there is some indication that babies born preterm are smaller than those of the same age remaining in utero. Measurement of the HC/AC ratio may help differentiate symmetrical from asymmetrical IUGR. The ratio is greater than 1 until approximately 36 weeks, at which time it becomes 1 or less. It is normal in symmetrical and increased in asymmetrical IUGR. Other methods that have been used, but are uncommon or are still in the process of being evaluated include the transcerebellar diameter/ abdominal circumference (TCD/AC) ratio, cheek to cheek diameter (coronal view) of the fetal face, subcutaneous tissue thickness at the level of the mid-calf, mid-thigh or in the anterior abdominal wall, and estimation of fetal liver size or weight.

Assessment of amniotic fluid volume using the amniotic fluid index (AFI) may, in some instances, aid the diagnosis of IUGR. A reduced AFI, with no history of ruptured membranes, not only suggests IUGR but may indicate its severity and has implications for its management.

Where gestational age is uncertain and the first ultrasound scan is performed late in pregnancy, diagnosis of IUGR may be difficult. If the HC/AC ratio is normal then symmetrical IUGR may be missed unless there is significantly reduced AFI. A high HC/AC ratio may be suggestive of asymmetrical IUGR. In any case a repeat ultrasound scan 2-3 weeks later to assess fetal

growth may resolve the issue.

Management of IUGR

The management of IUGR depends on the gestational age at diagnosis, the confirmed underlying aetiology, the chances of extrauterine survival and the level of expertise and technology available.

When the diagnosis of IUGR has been made, it is necessary to look for any maternal condition that could have given rise to it (e.g. chronic hypertension, pre-eclampsia, diabetes mellitus, etc.) and treat as appropriate.

A detailed ultrasonic evaluation of the fetus should be undertaken to exclude any structural anomalies. If there is a structural anomaly, which is incompatible with extrauterine survival then, nothing more may need to be done. The situation will have to be explained to the parents and subsequent course of action discussed with them. If the anomaly is compatible with life it is necessary to exclude a chromosomal abnormality. This is also desirable in early onset (before 30-32 weeks) or severe IUGR. Chromosomal studies may be performed on amniotic fluid cells (obtained by amniocentesis), placental tissue (obtained by placental biopsy) or fetal blood (obtained by cordocentesis). Cordocentesis may be performed in highly specialised centres; its advantages are that the fetal karyotype result is available within 48-72 hours and infection screening and assessment of fetal acid-base status can be performed. However it is associated with a procedure-related fetal mortality of 1%. Repetitive cordocentesis is not recommended because of the associated risks.

It is useful to know if one is dealing with a small but normal fetus or with a growth restricted one. The general measures in management include:

- admission to hospital to ensure bed rest, in left lateral position, in the hope of improving blood flow to the uterus, and to ensure close supervision
- improving the diet if necessary
- advising cessation of smoking and of use of alcohol or illicit drugs
- maternal hyperoxygenation (though not a long term solution it can improve intrauterine environment so that corticosteroids can be administered to enhance fetal lung maturity; it may also improve the fetal condition and allow a vital one or two weeks to be gained in the extremely preterm fetus)

It is necessary to ensure close supervision of both

mother and fetus, with delivery of the fetus if there is evidence of deterioration of fetal or maternal condition. Where it is likely that delivery may occur before 34 weeks gestation corticosteroids may be administered.

Fetal well-being needs to be closely monitored and the methods that may be used to achieve this include:

- fetal movement count (FMC)
- non-stress test (NST)
- biophysical profile (BPP)
- umbilical artery Doppler blood flow studies (UAD)
- Ultrasound evaluation of fetal growth every 2-3 weeks.

FMC is performed daily and at least a count of 10 is expected in 12 hours; it is important to recognise its low specificity. NST is performed every other day, except when there is reduced FMC in which case it may be reassuring to have it performed daily. With regards to the BPP this is performed weekly unless there is ruptured membranes in which case it may be performed 2-3 times a week. If at any time the NST is non-reactive or the BPP score is 6/10 the baby should be delivered.

In situations of oligohydramnios the baby should be delivered at 36 weeks.

Doppler velocimetry is the best fetal surveillance technique for predicting hypoxia or acidaemia. Where UAD is available this is performed every fortnight. Reversal of end diastolic velocities is an indication for delivery. If there is only absence of end diastolic velocities the fetus may be delivered if it has reached 34 weeks gestation; otherwise daily NST and 2-3 times weekly BPP are continued. A combination of absent end diastolic velocities with oligohydramnios is an indication for delivery.

Another indication for delivery is absence of fetal growth in two ultrasound scan evaluations 2-3 weeks apart.

With an otherwise normal but small fetus there may not be any abnormalities in any of the tests of fetal surveillance. Fetal growth continues at a slower than normal but fixed velocity. Conservative management is continued till about 38 weeks gestation when the baby is delivered.

Labour And Delivery

It is obvious from the foregoing that management of every case of IUGR must be individualised. Labour and delivery should be conducted in a centre with neonatal intensive care facilities in view of the potential perinatal problems. The underlying etiology and severity of growth restriction,

gestational age, and presence or absence of abnormal fetal surveillance results determine the mode of delivery. A normal but small fetus may be allowed to go through labour so long as there is no complicating factor, such as abnormal NST, or malpresentation. Growth restricted fetuses with normal surveillance results may be managed in the same way. It is important to emphasise, however, that where growth restriction is marked or there is oligohydramnios Caesarean section may be the best course of action. The likelihood of severe fetal distress in labour in such situations is increased considerably because there is likely to be underlying uteroplacental insufficiency, which may be aggravated by labour; an associated lack of amniotic fluid will predispose to cord compression. Continuous electronic fetal monitoring during labour (with fetal scalp electrode where appropriate) is mandatory; where this is not available delivery by Caesarean section may be the best way of ensuring a favourable neonatal outcome in fetuses with significant degree of growth restriction.

During labour the mother should lie in the left lateral position and supplemental oxygen administration may be useful. The threshold for Caesarean section should be low: any fetal heart rate abnormalities or anything more than thin meconium staining of the liquor may be considered an indication for Caesarean section.

Fetuses with a major anomaly incompatible with life should be allowed to go through labour and vaginal delivery. Fetuses with a non-lethal anomaly should be managed according to the expertise necessary to manage the anomaly postpartum. Those amenable to surgery should be delivered as late as possible in gestation; in general, the older and bigger the fetus the easier the surgical correction.

Personnel skilled and experienced in neonatal resuscitation should be present at delivery. A depressed infant with low Apgar scores should be anticipated and should that happen resuscitative efforts, aimed to clear the airway, establish ventilation, support circulation and correct any metabolic acidosis should be promptly initiated. If there has been meconium staining of the liquor the airway should be suctioned after delivery of the head before the shoulders are delivered; neonatal intubation and removal of meconium from the respiratory tract may be necessary. Early cord clamping may be beneficial in reducing the risk of hyperviscosity. Exposure to cold temperatures must be avoided.

Problems of the SGA/IUGR infant

The perinatal morbidity and mortality rate of the SGA/IUGR infant is increased 2-6 times over that of the general population.

There is an increased risk of perinatal asphyxia. The uteroplacental function, which may already be at a precarious level, may be further compromised by uterine contractions during labour. The effect will be even worse where the fetus has been in a state of chronic hypoxia/acidaemia prior to the onset of labour. Meconium aspiration is more common and will contribute to the asphyxia.

hypoglycaemia may occur because of limited glycogen stores and the increased glucose needs associated with hypoxia.

As a result of chronic intrauterine hypoxia there is increased erythropoietin production, which leads to polycythaemia and hyperviscosity. This could give rise to multiple organ thrombosis, heart failure and hyperbilirubinaemia.

In view of the poor subcutaneous fat stores the infant is at increased risk of developing hypothermia.

There may be electrolyte disturbances such as hypocalcaemia.

It is generally agreed that SGA infants are at increased risk of being short later in life. In a large population based study in Sweden, it was observed that although the majority of children born SGA/IUGR will show excellent catch-up growth soon after birth, approximately 10% will remain short at two years of age. The majority of these will remain short in later life comprising about 22% of adults with short stature.⁽¹⁰⁾

There is no agreement on the effect of SGA/IUGR on neurological and cognitive function. Some studies have shown increased prevalence of minor neurologic dysfunction and behavioural and learning disabilities; others, however, have not demonstrated any significant difference in intelligence scores. It is possible that socioeconomic and antenatal factors may have an important influence on the observed different outcomes.

It has become increasingly obvious from recent epidemiological evidence that people whose intrauterine growth was restricted are at increased

risk of developing coronary heart disease, stroke, hypertension and insulin-dependent diabetes mellitus.⁽¹¹⁾ This has led to the "fetal origins hypothesis" which proposes that the nutrient and hormonal milieu of the fetus, in situations of inadequate nutrient supply, alters gene expression resulting in developmental adaptations that lead to permanent changes in physiology and metabolism which in turn predispose to cardiovascular, metabolic, and endocrine diseases in adult life.

Prevention

In developing countries a general improvement in the nutritional status of the population is likely to reduce the risk for IUGR. In malaria endemic areas, adequate antimalaria prophylaxis has been shown to reduce incidence of IUGR especially in the primigravid. Cessation of smoking, in developed countries, will also have an impact on the incidence of fetal growth restriction.⁽¹¹⁾ As regards medical treatment to prevent the condition, although low-dose aspirin treatment started early in pregnancy (before 17 weeks gestation) reduces the risk of IUGR, a specific group of women most likely to benefit from this treatment has not been identified. Low-dose aspirin may therefore not be used routinely in pregnant women until those most likely to benefit from it have been clearly identified.⁽¹²⁾

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Intrauterine Fetal Death

SA Obed

Introduction

Very occasionally the mother and the health care providers are jolted by the realisation that the fetus has died in utero. The demise may occur either prior to the onset of labour (antepartum) or during labour (intrapartum). Antepartum fetal demise may occur any time in the course of pregnancy. Starting from fertilization, an unknown number of embryos die in the first nine days before implantation (and before the woman can even miss a period). This type of loss has been estimated to be about 40% of all pregnancies.⁽¹⁾ After implantation, a varied proportion from 12%-31% of pregnancies dies before delivery.^(2,3)

Definitions

Early fetal deaths (i.e. abortions) are unfortunately not well documented. Late fetal deaths are classified as macerated stillbirth (death before the onset of labour or more than 24 hours before delivery) and fresh still birth (death during labour or less than 24 hours before delivery).

Incidence

The incidence of stillbirths in most developing countries is about ten-fold that of the advanced countries⁽⁴⁾. The incidence rates of stillbirth in some West African countries range from 52.1 to 69.3 per 1000 births^(4,5,6) while that of

Sweden and USA range from 5.8 to 6.6 per 1000 births.⁽⁷⁾

Predisposing Factors

The background of the mother and the community in which she lives has a large influence on fetal demise. The health of the whole community and its nutritional status are important determinants. Other factors are the efficiency of the health care service and the cooperation between the mother and the health care providers. The age, parity and socio-economic status of the mother tend to influence the pregnancy outcome.⁽⁶⁾

The age at which the risk of fetal demise is lowest is between 20 and 29 years. In those patients younger than 20 years there is a tendency of poor

antenatal attendances, being unmarried and in a low income status. In those older than 29 years there are increased incidences of hypertension, diabetes and renal diseases.^(8,9,10)

The lowest risk for fetal demise occurs at delivery of the second baby.⁽⁸⁾ The first baby may be at a greater risk since there is an increased incidence of pregnancy induced hypertension and more difficult deliveries. Beyond the fourth pregnancy there are increased risks of twinning⁽¹⁰⁾ and poor attendances at the antenatal clinics due to over-confidence of the mothers.^(6,9)

Poor maternal nutrition tends to adversely influence fetal growth⁽¹²⁾, which may eventually cause in-utero death.

Alcohol, even in low quantities can contribute to the risk of intra-uterine growth retardation⁽¹³⁾ and possibly fetal death.

The effects on the fetus of maternal smoking include deviations from normal placentation⁽¹⁴⁾, fetal growth retardation⁽¹⁵⁾ and on the immune competence. The cause of fetal death by maternal smoking may depend on the presence of other adverse factors. Exposure to occupational or environmental hazards such as radiation or lead can also contribute to fetal loss.⁽¹⁷⁾

Aetiology

The major pathological causes of fetal death are due to congenital abnormalities, asphyxia, growth retardation and infections.

Congenital Abnormalities

About 20% of fetal deaths are due to major congenital abnormalities.⁽⁸⁾ Parental genetic constitutions may result in a conception of a fetus with a lethal hereditary defect. Certain chromosomal anomalies, particularly trisomy 21 has a risk of occurrence with advanced maternal age. Other non-chromosomal abnormalities especially of the central nervous system like anencephaly and spinal bifida contribute significantly to fetal demise.

Infections

Infections contribute a great deal to fetal demise in areas of poor hygienic standards. Toxoplasmosis, rubella, cytomegalovirus, herpes simplex and syphilis with the acronym TORCHES are well known infections that cause fetal death. Pyrexia infections like malaria and urinary tract infection contribute significantly to fetal demise in the developing countries. Chorioamnionitis has also been noted in pathological studies as a major cause of fetal death.⁽¹²⁾

Asphyxia

Post-mortem evidence of asphyxia is found in about a half of in-utero fetal death.⁽⁹⁾ Acute asphyxia may occur in placental abruption, titanic uterine contractions or umbilical cord compressions. Chronic asphyxia from other diseases like hypertension may lead to poor fetal growth. In all these instances the fetus is at risk of poor placental exchange.

Hypertension

Hypertension in pregnancy, whether pregnancy induced or pre-existing can damage placental vessels with a consequent reduction in blood flow and fetal growth retardation.

Other Maternal Diseases

Sickle cell disease, anaemia and diabetes are conditions that contribute significantly to fetal deaths in the West African sub-region.

Diagnosis

Early intra-uterine death is suspected when the uterus is small for dates or when there is loss of the symptoms of pregnancy. After mid-pregnancy, loss of fetal viability is often heralded by loss of subjective fetal activity as well as fetal heart tones not heard on auscultation.

The introduction of ultrasonography has made confirmation of intra uterine fetal death easy. An early pregnancy loss may reveal disruption of the gestational sac or persistent absence of fetal echoes. Later in pregnancy, there is absence of fetal cardiac activity and collapse of the fetus if a sufficient interval has elapsed since death.

Sometimes overlapping of skull bones (Spalding sign), sharp angulation of the spine or gas in the cardiovascular system (Robert's sign) may be seen. A plain X-Ray may be used to demonstrate these signs.

Intrapartum fetal death can be detected by

absence of fetal heart tones by auscultation or cardiotocogram. Ultrasonography can also be used to confirm fetal death during labour.

Investigations

Coagulation studies are indicated in fetal demise particularly if the death is known to have occurred 3 or more weeks earlier. The general investigations usually done include full blood count, sickling if not known, urine analysis, blood grouping and detection of antibodies and VDRL.

Other specific investigations that may be indicated include blood film for malaria parasites, urine culture and sensitivities, fasting blood sugar and toxoplasmosis serology test.

Management

Coagulation studies are performed prior to treatment. If there is derangement of the coagulation factors fresh frozen plasma is given. In rare cases when the patient has hypofibrinogenemia, heparin is indicated if there is no bleeding. A one to two day course of heparin (1000 units per hour up to 48 hours) will increase the fibrinogen to acceptable levels (more than 20mg per 100ml) and restore number of platelets and factors V and VIII to normal levels. The heparin should be discontinued before induction of labour. If the patient goes into spontaneous labour while heparin is being administered, the infusion should be stopped.

It is not necessary to neutralize the heparin with protamine sulphate unless the patient is bleeding.

If the uterus is 14 weeks size or less, evacuation can be achieved by suction curettage or dilatation and sharp curettage. When the uterus is greater than 14 weeks' size, there are several options available. These include expectant therapy (observation), Prostaglandins E₂ vaginal suppositories, Misoprostol (Pg E₁) Vaginal Suppositories, Intravenous oxytocin, Hysterotomy, Caesarean Section and Destructive operation.

About 80 percent of patients with intra-uterine fetal death experience spontaneous labour within 2 to 3 weeks after the demise. Unfortunately intrauterine demise is a great emotional burden and expectant management is unacceptable to most patients. Although a great stress, women with fetal deaths may agree to expectant therapy. Another drawback to expectant therapy is the

potential for development of disseminated intravascular coagulation. The women at risk are those with a gestation greater than 16 weeks and who have retained the dead fetus for 4 weeks or more.

Misoprostol (cytotec or prostaglandin E₁) 50ug, 4 to 6 hours as vaginal suppositories or prostaglandin E₂ (Prostin) in a dose 3mg every 6-8 hours would ripen the cervix and also induces uterine contractions. Foley catheter with a large balloon (about 30ml) can also be inserted into the cervix and the balloon blown with water. This causes release of endogenous prostaglandins from the cervix and promote its ripening.

Dilapam or laminaria tents can also be used in cases when the cervix is not favourable for induction of labour. Intravenous oxytocin has had the modest use especially after 36 weeks gestation. Unfortunately, the preterm uterus is relatively insensitive to oxytocin even in high dosages. Water intoxication, from the antidiuretic effect of oxytocin, uterine rupture and cardiac arrhythmias have been reported with its use.

Hypertonic saline injected into the amniotic sack is an effective abortifacient. It is contradicted however, in patients with renal or cardiovascular disease. Its use may increase the risk of coagulation defect. A special case is the patient who has had a previous Caesarean section and whose uterus is too large for curettage.

Use of prostaglandins, hypertonic saline and oxytocin poses the threat of uterine rupture. If the previous Caesarean section was lower segment with no evidence of endometritis, labour is usually allowed with close monitoring and if available internal pressure cannula to monitor uterine contractions. If in doubt hysterotomy or repeat Caesarean section may be done.

Destructive operations have a place when the fetal demise occurs in labour with the cervical os sufficiently dilated to allow the procedure to be performed. After the delivery of the baby, the parents should have emotional support. Autopsy examination may be necessary to confirm the cause of death. The autopsy report should be discussed at length with both parents as soon as possible.

A search for the aetiology may have to be done to prevent some potential repetitions. These may include chromosomal studies, total body X-rays

and full body photographs of babies who have congenital abnormalities.

In case of advanced pregnancy lactation has to be suppressed. The use of tight cotton brassier may be very helpful. Bromocriptine, though expensive is very effective in suppression of lactation.

Controversies

Late fetal death or stillbirths comprise those born of a least 28 weeks gestation but showing no signs of life. There are, however, certain anomalous features about this definition which can lead to classification problems. Firstly, such deaths include fetuses known to have died long before 28 weeks' gestation but whose delivery was after that time, for instance papyraceous twins. Secondly in most advanced countries, where there has been tremendous improvement in neonatal care babies delivered earlier than 28 weeks of gestation have significantly higher chances of surviving and have normal development compared to those in developing countries.

Incidence

The incidence rates of stillbirths in Ghana and many West African countries are institutional based. In these countries many deliveries occur outside the health institutions. In Ghana for instance, supervised deliveries occur outside the health institutions. In Ghana for instance, supervised deliveries for the period 1994-96 was only about 40%.⁽¹⁹⁾ Thus the rates of stillbirths do not reflect on actual incidence in the population. On the other hand almost all deliveries in the advanced countries are done in health institutions.⁽²⁰⁾

Retained Dead Fetus And Living Twin

The occurrence of single fetal death in a preterm multiple pregnancy poses unique therapeutic dilemmas. Prolongation of the pregnancy could result in life-threatening maternal haemostatic failure. On the other hand, termination of the pregnancy for maternal indications would result in the birth of an immature infant.

The incidence of this problem is not widely known, but is likely to be observed more frequently with the widespread use of ultrasound scanning in obstetrics.

Prolongation of pregnancy for 8 weeks after spontaneous in-utero death of one twin at 26 weeks has been reported.⁽²¹⁾ The patient was treated with intravenous heparin and the reversal of the

consumption coagulopathy resulted in uneventual prolongation of pregnancy for the living twin to achieve lung maturity.

Destructive Operation

If labour has been prolonged and neglected and the fetus is dead, Caesarean Section or destructive operation should be performed. Destructive operation may be considered because of:

1. The risk of overwhelming infection following Caesarean section in patients who invariably already have genital infection.
2. Obsession with having a vaginal delivery and make the patient or her relations refuse consent for abdominal delivery.
3. The patient may not have access to skilled supervision in her subsequent pregnancy or may choose not to avail herself to hospital delivery. The circumstances mentioned above do not exist in developed countries because of high literacy rates, large numbers of skilled personnel and good communications. However, in developing countries an alternative method to Caesarean section for delivering the dead fetus, the result of neglected labour, even in the twenty-first century is destructive operation.

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Preterm Labour

SA Obed

Introduction

Prematurity is a leading cause of perinatal morbidity and mortality. In the developed world preterm delivery has assumed greater prominence as advances in the fields of infectious disease, genetics, neonatology and paediatric surgery have reduced morbidity from other causes. Despite the introduction of tocolytic therapy, the improvements in the diagnosis of multiple pregnancy and placenta praevia by ultrasound, the marked decrease in syphilis, apparent improved nutrition and government funded medical assistance, preterm delivery continues to be common throughout the world.

Definition

Preterm labour is defined as the occurrence of regular uterine contractions productive of cervical changes (dilatation and effacement before the end of the 37th week of gestation. Pregnancies terminating before the 28 weeks of gestation are considered abortions in most West African countries. However in the developed world earlier periods like 20 weeks in USA are used to define the lower limits of preterm labour. Since gestational age may occasionally be difficult to ascertain, some statistics refer to birth weights in defining preterm labour. Preterm labour may be considered as one occurring at birth weights between 1000g and 2500g for the countries using 28 weeks and 500g for those using 20 weeks gestation as the lower limit.

Incidence

The incidence of preterm birth is dependent on many factors and varies from one country to another and institution-to-institution. In the developed countries reported rates generally vary between 5 and 9%^(1,2) because the diagnosis of preterm labour is hard to define, rates are unavailable, although undoubtedly greater than those for preterm birth.

Risk Factors

A large number of risk factors have been associated with preterm labour. These factors include demographic, behavioral, past

reproductive history, current pregnancy complications and genital tract infections.

Demographic Risks

- * **Age** - The incidence of preterm delivery is highest for those under 15 and those above 35 years and lowest between the ages of 25 and 29 years.⁽³⁾
- * **Weight** - Pre-pregnancy weight of less than 50kg is associated with a high incidence of preterm birth.⁽³⁾
- * **Socio-economic** factors - Unmarried status and limited educational attainment are associated with high incidence of preterm birth.^(2,3,4) Education level is often used as a proxy for socio economic status because it is thought to correlate well with income level.

Behavioural Risks

- * Physical activities may increase the risk of low birth weights by prompting either intrauterine growth retardation or preterm labour. Employment and the risk of preterm delivery reports are conflicting. Probably these reports do not factor in the relative effects of unpaid domestic chores by housewives.
- * Alcohol, cigarette smoking and other substance abuse have been found to influence adversely the occurrence of preterm delivery.^(5,6,7)

Past Reproductive History

- * Previous preterm delivery - The risk of recurrence of preterm delivery after one episode is about 35% and may reach 70% after two or more.^(8,9)
- * Previous abortion - First trimester abortions do not appear to influence the risk of preterm delivery⁽¹⁾ but second trimester abortions and stillbirth are important risk factors for preterm delivery.⁽⁸⁾
- * Uterine abnormalities - Cervical incompetence is a major predisposing factor to preterm birth. Congenital anomalies like bicornuate,

didelphic or unicornuate uteri ⁽¹⁰⁾ submucosal and intra mural leiomyoma ⁽¹¹⁾ and Asherman's syndrome ⁽¹²⁾ decrease the likelihood of carrying a pregnancy to term.

Current Pregnancy Complications

- Uterine over distension - Multiple gestations is perhaps the single risk factor most predictive of preterm labour. ⁽¹³⁾ Monozygotic twins are at a higher risk than dizygotic twins primarily due to an increased rate of premature rupture of membranes and induction of labour for fetal conditions. Preterm delivery occurs in about 30-40% of pregnancies complicated by polyhydramnios. ⁽¹⁴⁾
- Congenital anomalies - The fetuses with multiple congenital anomalies are at increased risk of preterm delivery because of the associated intra uterine growth retardation from possible placental insufficiency.
- Antepartum haemorrhage is associated with increased incidence of preterm birth irrespective of the cause. ⁽¹⁵⁾
- Maternal illness - Almost any severe maternal disease may be associated with preterm delivery.
- Febrile illness including malaria and urinary tract infection are important causes of preterm labour in West Africa.

Genital Tract Infections.

A variety of pathogens including viruses and bacteria infecting the genital tract have been associated with preterm labour. Microorganisms associated with prematurity possess high levels of phospholipase A2 activities.

Phospholipase A2 is believed to cause hydrolysis of phospholipids in the membranes resulting in their rupture and also increase the synthesis of prostaglandins ⁽¹⁶⁾

Diagnosis

Early differentiation between true and false labour is difficult before there is demonstrable cervical effacement and dilatation. Uterine contractions alone can be misleading because of Braxton Hicks contractions. These contractions are irregular, non-rhythmical and painless.

Because uterine contractions alone may be

misleading, the following criteria described by Herron ⁽¹⁷⁾ are often required to document preterm labour; regular uterine contractions after 28 weeks gestation or before 37 weeks, which are 5 to 8 minutes apart or less, and accompanied by one or more of the following; progressive change in the cervix, cervical dilatation of 2cm or more, or cervical effacement of 80% or more.

Ruptured membranes impose considerable certainty of preterm labour that follows.

Other signs and symptoms that may aid in the early diagnosis of women at risk for preterm delivery include:

- (1) Passage of cervical mucus often slightly bloody.
- (2) Low backache.
- (3) Pelvic pressure due to descent of the fetus.
- (4) Menstrual - like cramps.

A determination of the gestational age must be made. When dates are uncertain ultrasound scan biometry should be done even though in the third trimester the error margin is quite high. Ultrasound scan can also be used to determine the type of growth retardation by the head - abdominal circumference ratio as in cases of asymmetrical growth.

Management

Once the diagnosis of preterm labour is suspected the patient should be admitted. A search for the aetiology should be undertaken. This includes full blood count, blood film for malaria parasites, midstream urine specimen for microscopy and culture, endocervical swab and culture and if chorioamionitis is suspected amniocentesis may be done for obtaining liquor for microscopy and culture.

The institution of care should be appraised whether it has adequate facilities and personnel in relation to the gestational age and size of the fetus. If not suitable, the patient should be referred to a tertiary centre or other centres that can give the requisite care.

The inpatient management starts with hydration if the patient is dehydrated and anti-malaria drugs and antibiotics if febrile whilst waiting for the laboratory investigations. Erythromycin has been proven to be a useful antibiotic in such circumstances. ⁽¹⁸⁾

Tocolysis

A complete history and physical examination are necessary to determine whether there are any maternal or fetal contraindications to tocolytic therapy. Although the upper limit of gestational age for preterm labour is defined as 37 weeks, labour inhibition beyond 35 weeks is usually unwarranted unless there is uncertainty regarding gestational age.

The fetus that is most likely to benefit from inhibition of preterm labour is one whose gestational age is less than 32 - 34 weeks.

Absolute contraindications to tocolysis in preterm labour include fetal death, fetal congenital anomaly incompatible with life, chorioamnionitis, fetal complications requiring immediate delivery and maternal complications requiring immediate delivery.

Relative contraindications include fetal growth retardation, fetal distress, pre-eclampsia, antepartum haemorrhage and cervical dilatation more than 4cm.

If uncertain about false labour narcotic sedation like pethidine usually abort the contractions of false labour. If it is not false labour and the baby is delivered within few hours after the pethidine administration there may be neonatal depression. Mild sedation may occasionally be indicated to alleviate maternal anxiety, which understandably, often accompanies preterm labour.

1. Beta -Adrenergic Agonists

These include such agents as ritrodine, terbutaline, hexoprenaline, salbutamol, isoxsuprine and fenoterol. The beta-adrenergic agonists exert their effect on the myometrial cell, through a membrane-mediated mechanism. Beta-adrenergic receptors are located in the outer cell membrane. The interaction of an agonist and the receptor leads to activation of adenylate cyclase, the enzyme that catalyses the conversion of ATP to cAMP. Increased intracellular cAMP activates the enzyme cAMP-dependent protein kinase. An increase in this latter enzyme reduces myometrial contractility by decreasing intracellular calcium and by reducing the effect of calcium on muscle activation.

The trophoblast also has beta 2 receptors and in this tissue cAMP increases progesterone production, which inhibits uterine contraction. Maternal side - effects of beta agonist tocolytic agents include palpitations, arrhythmias, myocardial ischaemia, pulmonary oedema, anaemia, glucose intolerance, hypokalaemia,

nausea, bloating, paralytic ileus, tremor, restlessness, rash and elevated transaminase levels. Feto-neonatal side effects include hypoglycaemia, hypocalcaemia, ileus, hypotension and death.

Administration

The patient is placed in a left lateral tilt - recumbent position and an intravenous line using 5% Dextrose if no hyperglycaemia, or half-strength normal saline is inserted.

Baseline maternal heart rate, blood pressure and respiratory rate are determined. The fetal heart rate and uterine activity are continuously monitored.

The drug infusion is introduced as a separate infusion. The initial infusion rate is the lowest recommended for the selected agent. The dosage is increased every 10-20 minutes until uterine activity is inhibited to less than one contraction every 15 minutes, or when adverse maternal effects occur. As contractions begin to space out, the infusion rate is increased less frequently. A maternal heart rate greater than 140 beats per minute or any other unacceptable side - effect requires a reduction in dose.

Once effective tocolysis has been achieved, intravenous infusion is maintained for an additional 12-24 hours depending upon the difficulty encountered in initially inhibiting contractions. The infusion rate may be tapered during this time as long as uterine contraction remains inhibited. During this time maternal intake and output are monitored, blood studies of full count, glucose levels, urea and electrolytes, and transaminases are re-assessed at 12-hour intervals.

Following this maintenance period, the infusion rate is gradually reduced at half-hour intervals until the maternal heart rate decreases to 100 beats per minute. Subcutaneous or intra muscular injections followed by oral therapy are then initiated and the intravenous infusion discontinued 30 minutes later. Alternatively, oral therapy may directly follow intravenous therapy.

2. Magnesium Sulphate

Magnesium competes with calcium for entry into the cell at depolarization. This reduces the intracellular free calcium available to participate in the actin-myosin interaction of smooth muscle contractions.

Magnesium sulphate causes periphera

vasodilatation leading to uncomfortable warmth and flushing sensation. Other side effects include nausea, headache, dizziness, chest tightness and lethargy. An initial loading dose of 4g intravenously over 20 minutes followed by a maintenance dose of 4g per hour by constant infusion is administered. If uterine contractions are inhibited the maintenance dose is tapered to 2g per hour and continued for 12-24 hours when the intravenous therapy is discontinued, the woman should receive oral beta-adrenergic agonists since there is no practical oral form of magnesium sulphate.

3. Prostaglandin Synthetase Inhibitors

Prostaglandins, especially PG F₂ and PGE₂, play a dual role in preterm labour. These compounds are probably the final pathways for smooth muscle contraction. PGE₂ induces biochemical changes in cervical collagen, which facilitate cervical dilatation. Prostaglandin synthetase converts free arachidonic acid to prostaglandin. Indomethacin is the prototype of prostaglandin synthetase inhibitors.

Indomethacin may be given as oral tablets or rectal suppositories. Maternal side effects include nausea, vomiting and gastrointestinal bleeding. Fetal side effects include preterm closure of ductus arteriosus in utero and oligohydramnios resulting from decreased fetal urine excretion.

4. Calcium Channel Blockers

Calcium channel blockers are a heterogeneous group of organic compounds that inhibit the influx of extracellular calcium across the cell membrane during the slow inward flow of calcium current of the action potential. These include Nifedipine and Verapamil.

These groups of drugs produce vasodilatation and decrease in peripheral resistance leading to lowering of diastolic blood pressure, tachycardia and transient facial flushing.

Promotion Of Fetal Pulmonary Maturation

The beneficial effect of the use of corticosteroids in maturing the fetal lungs when preterm labour occurs in women with gestation less than 34 weeks is almost conclusive when the membranes are intact.⁽¹⁹⁾ However, when the membranes are ruptured, it is believed that this acts as a stressor to initiate an outpouring of endogenous fetal steroids, which act directly on pulmonary enzyme systems.

Some other benefits of steroids apart from prevention of respiratory distress syndrome include prevention of necrotising enterocolitis, bronchopulmonary dysplasia and patent ductus arteriosus.

Steroids adversely affect glucose utilization, aggravating the diabetogenic effects of beta agonist used for tocolysis. The risk of infections may be increased. The regimen commonly used is 12mg, dexamethasone 12 hourly for two doses in 24 hours. It may be repeated after one week if the baby is not delivered and the gestation is still less than 34 weeks. Betamethasone is also used and believed to have less intense side effects.

Management Of Advanced Preterm Labour

Preterm labour may progress despite maximal therapy or the patient may present in advanced labour, precluding successful labour inhibition. The delivery should be performed at a facility equipped to care for the preterm infant since transferring the neonate post delivery reduces the chances for a successful outcome.

Once it is apparent that labour cannot be inhibited, the first step is to discontinue with the tocolysis. A decision should be made on the route of delivery depending on the prevailing circumstances. A consultation with the neonatologist should be initiated for the care of the baby soon after delivery. Antepartum administration of phenobarbitone has been found to reduce the risk intraventricular haemorrhage.⁽²⁰⁾

Intrapartum Monitoring

Both prolonged and precipitous labour are hazardous to the preterm infant. Oxytocin should be used carefully to prevent hyperstimulation. Vaginal examination should be performed sparingly because the preterm gestation is more prone to infection.

Continuous fetal heart rate monitoring if available is more accurate in predicting fetal hypoxia, Fetal distress can be prevented by avoiding the supine positioning of the patient and oxygen by face mask. Preterm labour is often an unexpected crisis for which patients are emotionally unprepared. Fear and anxiety will not only intensify pain, but also mediate release of catecholamines. Pain relief is therefore an essential element of the management of patients in preterm labour.

Virtually all the medications used for pain relief for

the mother cross the placenta and affect the fetus. The personnel in charge of neonatal resuscitation should be made aware of all medications administered to the mother during labour, especially analgesics that may temporarily depress respiration in the neonate. Continuous epidural analgesia is a safe and effective method of pain control for patients in preterm labour.

Inhalation anaesthesia for emergency operative delivery is of no danger to the fetus, provided the mother is pre-oxygenated well prior to intubation and is positioned on the operating table in a manner to avoid supine hypotension.

Delivery

In the absence of a relaxed vaginal outlet, a liberal episiotomy for delivery is advantageous once the fetal head reaches the perineum. Recent studies have not confirmed the beneficial effects of low forceps, once thought to be so important for preterm delivery.

Breech presentation is a common and important complication of preterm labour. Among other things, there is a relatively high incidence of fetal abnormalities with breech presentation, especially neuromuscular defects. Also, up to approximately 32 weeks gestation, the fetal head is relatively larger than the abdomen and thorax. Consequently, there is a substantial risk that the lower extremities, abdomen and thorax of a very low birth weight fetus may deliver through an incompletely dilated cervix, leaving the relatively larger fetal head trapped behind the cervix.

However, studies have shown that there is no difference in neonatal mortality between preterm babies delivered by Caesarean section and vaginally.^(22,23) Caesarean section, does not itself avoid trauma to the low birth weight infant. An inappropriate incision and rapid contracting of the lower segment can trap the fetal head as ominously as the incompletely dilated cervix.

Resuscitation Of The Neonate

An experienced neonatologist who has been fully oriented to the specific problems of the case should be present at delivery. In most cases of borderline neonatal viability, it is probably wiser to do tracheal intubation and begin cardio respiratory support in the delivery room and decide later in the NICU to withdraw the support if it is clearly futile than not to vigorously resuscitate the neonate in the delivery room on the grounds that the neonate is too immature to survive.

Prevention

Failure to prevent preterm labour results mainly from inability to recognise the early signs. Identification of the high-risk patient should be done early in the pregnancy or if possible pre-pregnancy. These women should be educated to modify their lifestyles, and health practices.

Modifications of lifestyle, which may help prevent labour, include cessation of smoking, and proper nutrition. Reduction in psychological stresses may also be advantageous.

Useful health practices include screening of the endocervix for *Neisseria gonorrhoea*, group B streptococcus and *chlamydia trachomatis*; and urine culture for asymptomatic bacteria. If this screening shows colonization, treatment should be offered.

Some have recommended prophylactic bed rest and avoidance of coitus during pregnancy but the efficacy of these is not proven.

Cervical cerclage is an effective treatment for incompetent cervix.

The patient should be taught to recognise uterine contraction early and to report to the hospital for evaluation promptly.

Controversies

The concept of preterm labour is bedevilled with a lot of controversies. The distinction between late abortion and preterm labour is very thin. Though in most West African countries, 28 weeks is set as the dividing line of viability, some babies have been expelled at gestations less than 28 weeks and lived for 24 hours or more. In such instances they should be certified as delivery.

Though the criteria for diagnosing labour is clear at term, the difference between false and true labour preterm may not be clear before demonstrable cervical effacement and dilatation. Braxton Hicks contraction may be confused with preterm labour. The Herron criteria may occasionally be useful but certainly not in all cases.

As a consequence of the confusion and imprecision as to the diagnosis of preterm labour, there has been corresponding uncertainty about the effectiveness of most preterm labour treatment regimens.

The use of steroids to enhance fetal lung maturation before 34 weeks gestation used to be controversial but it has been amply demonstrated that steroids are useful in such cases. However controversies still exist when the membranes are ruptured before 34 weeks. Opinion is still divided on the use of steroids under such circumstances. The benefits of steroids when the gestation is less than 30 weeks are not certain.

Phenobarbitone administration in the antepartum period to prevent intraventricular haemorrhage in preterm labour has some benefits but has no clear advantage over postnatal administration.

Often debated are the use of prophylactic forceps and episiotomy to avoid undue trauma to the head of a preterm neonate during vaginal delivery. The skull of the preterm infant is considerably more fragile and susceptible to forceps trauma than is the skull of a term infant. Consequently if forceps are applied to assist the delivery in cases of fetal distress or dystocia, they must be applied with the gentlest technique. Also there is no convincing evidence that an episiotomy is necessary in all cases. The use of an episiotomy should be determined by an assessment of the resistance of the perineum and the need to hasten delivery.

While a traumatic delivery is particularly hazardous for the preterm infant, this does not mean that Caesarean section is the preferred route of delivery. The evidence that Caesarean section of the preterm vertex infant improves perinatal outcome is insufficient to recommend its routine use^(24, 25) Caesarean section after the onset of labour does not prevent central nervous system bleeding in these infants.⁽²⁶⁾ Vaginal delivery may have some advantages, since amniotic fluid is more completely expressed from the fetal lungs as the chest is compressed at delivery, perhaps facilitating lung expansion. The placental transfusion that occurs at delivery may be greater with the vaginal route.

However, in specific situations, which might require a prolonged induction in the presence of a rigid, unyielding cervix, Caesarean section may be preferable.

The uterine incision during Caesarean section mostly preferred is the low transverse even though the lower segment is not usually well developed before term. Many authors recommend a vertical incision, thus increasing the potential long-term morbidity for the mother, as it is more likely to rupture in subsequent pregnancy than a low transverse incision.

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Vomiting In Pregnancy

Ali Samba / EY Kwawukume

Introduction

Vomiting is such a common symptom of pregnancy that many women accept it as inevitable. Approximately 50% of pregnant patients experience mild vomiting, the severe condition of hyperemesis gravidarum (intractable vomiting during pregnancy) occurring in less than 0.5 to 2% of pregnancies. Symptoms usually begin shortly after the first missed menstrual period and disappear by the fourth month of pregnancy. The vomiting is usually more likely during the first pregnancy and it usually recurs in subsequent gestations. Women with early pregnancy vomiting had better outcomes than women without vomiting.¹

Vomiting usually occurs in the morning but may occur throughout the day and patients who are in employment tend to vomit in the evening. The vomiting is usually mild and the patients do not develop fluid and electrolyte disturbances. Very infrequently vomiting may be so severe that dehydration, electrolyte and acid-base balance disturbance and starvation become a serious problem. Violent episodes of vomiting may lead to haematemesis from Mallory-Weiss syndrome. A history of fever, chills and rigors, dysuria and frequency of micturition is important as it may suggest malaria or urinary tract infection as the cause of the vomiting. Some patients may develop jaundice and have evidence of malnutrition as a complication.

Aetiology

The precise aetiology of vomiting in pregnancy remains unclear. Suggested causes include:

1. Elevated Human chorionic gonadotrophin (hCG) and/or Oestrogen levels. Vomiting is commoner in multiple pregnancy and molar pregnancy.
2. Allergic reaction to the foeto-placental unit.
3. Vitamin deficiency
4. Vomiting in previous pregnancy
5. Psychogenic factors-sufferers may have immature personality, hysterical tendencies and patients with background tendencies, which may induce fear as well as marital disharmony.

6. Endocrine disorders like diabetes mellitus, thyrotoxicosis² and Addison's disease.
7. Infectious conditions like malaria and urinary tract infection.
8. Gastrointestinal disturbances such as sub-acute obstruction, gastroenteritis and pancreatic disorders.

The entire act of vomiting comes about as a result of stimulation of the emetic centre, which is situated in the lateral reticular formation in the floor of the fourth ventricle. The vomiting centre is anatomically in close proximity with the salivation and the respiratory centre and activities mediated by these centres may all be involved in vomiting, thus the frequent occurrence of hyper-salivation and respiratory movements associated with vomiting. For most patients there is no underlying cause for the vomiting except the pregnancy itself. Precipitating factors include standing in hot places especially in the kitchen with cooking odours, spices and foods with high fat content. Hunger tends to perpetuate vomiting in a vicious cycle.

Investigations

1. Full blood count and blood film for malarial parasites.
2. Urinalysis for pus cells and ketones.
3. Urine culture and sensitivity
4. Liver function tests and Blood urea and electrolytes and creatinine.
5. Pregnancy test.
6. Ultrasound for number of foetuses and to exclude molar pregnancy.

Treatment

Seldom is the treatment of vomiting in pregnancy so successful that the expectant mother is afforded complete relief. However the unpleasantness and discomfort can be minimized. The patients require sympathetic advice and reassurance that the condition is self-limiting and moreover pregnancies in which nausea and vomiting occur are more likely to have a favourable outcome than those without nausea and vomiting.

Small meals with a high carbohydrate and low fat content is advised, she should avoid hunger and spices and eat small meals at frequent intervals

stopping short of satiation. Any food that makes her vomit should be avoided.

Anti-emetic drugs should be avoided whenever possible because of the risk to the foetus. However it is better to control vomiting with anti-emetics rather than subjecting the foetus to the adverse effect of maternal dehydration, ketosis and malnutrition. Commonly used anti-emetics are the antihistamines-Promethazine, Cyclizine, Meclozine among others. Phenothiazines have also been used. Some anti-emetics are supplied as combined preparations with Pyridoxine (Ancoloxin). There is little experience with newer anti-emetics on the market such as Metoclopramide. Both parenteral and oral preparations of these drugs are available.

Hyperemesis Gravidarum

Hyperemesis gravidarum or pernicious vomiting of pregnancy is a condition defined by intractable vomiting, leading to fluid and electrolyte disturbances and nutritional deficiencies. The onset of symptoms occurs mainly during the first month of gestation and remits by the end of the first trimester but occasionally might persist throughout pregnancy. The vomiting is usually severe to produce weight loss, dehydration, acidosis from starvation, alkalosis from loss of hydrochloric acid in the vomitus and hypokalaemia.³

The pathogenesis of hyperemesis gravidarum is poorly understood but more likely during first pregnancy in younger women and usually recurs in subsequent gestations. Intriguingly, women with early-pregnancy vomiting had better outcomes than women without vomiting.³ Possible pathogenic factors include the elevated oestrogen level of pregnancy and increased circulating chorionic gonadotrophin.⁴

Treatment of this condition requires hospitalisation for fluid, electrolyte and vitamin replacement. In some patients the mere removal to hospital without any treatment often leads to dramatic and immediate improvement. In the initial assessment of the patient, it is very important to exclude any treatable cause of the vomiting. The baseline investigations is as mentioned above.

Therapy involves correction of fluid and electrolyte deficit and acidosis or alkalosis. This requires appropriate amount of sodium, potassium, chloride, lactate or bicarbonate, which are administered parenterally until the vomiting is controlled. The patient is maintained on nil per os. Energy is provided in the form of glucose with 5% Dextrose or Dextrose Saline. Correction of electrolyte imbalance is usually done with Normal

Saline, Ringers Lactate and in some cases with Potassium Chloride supplementation.

Daily urinalysis (for ketones) and urea and electrolyte levels are done to monitor the progress of the patient. The urine output is also monitored as deteriorating cases may go into renal failure.

Parenteral anti-emetics such as Promethazine (Phenergan) are given. The intravenous route is preferred as it suppresses the vomiting better when given as a continuous infusion. In our unit, continuous low-dose infusion of phenergan is administered. To each 500ml of intravenous fluid, 25 mg promethazine is added and the patient is given at least 4 L/day. A vomiting chart is kept. The patient should be kept on NPO and continued on parenteral therapy for at least 48 hours after all vomiting has ceased or improved substantially to prevent reappearance of symptoms. Parenteral vitamin supplement, in the form of Pabrinex1&2 are given daily. Vitamin B complex injection can also be used. In most cases, following initial treatment, oral feeding can be commenced cautiously starting with dry foods in frequent small doses. Where prolonged IV therapy is envisaged, vitamin supplementation must not be forgotten. In severe cases parenteral alimentation may be required.⁵ Patients suspected of having vomiting due to a psychogenic disorder should be referred for psychiatric evaluation. Therapeutic abortion for very severe vomiting is rarely done in modern obstetrics.

Usually the vomiting tends to subside at the end of the first trimester. When vomiting persists into mid pregnancy or occurs later on in the pregnancy then a diligent search for a specific cause is essential. The later in pregnancy vomiting occurs the more likely is there a pathological cause. These include malaria, urinary tract infection, respiratory tract infection, polyhydramnios, abruptio placentae and pre-eclampsia. Others to be considered are: hiatus hernia, peptic ulceration, intestinal obstruction, hepatitis, pancreatitis, or acute gastroenteritis. Torsion of an ovarian cyst and red degeneration of fibroid may also be responsible.

Complications

Maternal complications of vomiting include:

1. Dehydration and electrolyte imbalance.
2. Ketosis.
3. Weight loss.
4. Dental caries.
5. Gastrointestinal mucosal tears (Mallory-Weiss).
6. Aspiration pneumonia.
7. Neurological damage (Wenicke-Korsakoff encephalopathy)

7. Hepatic dysfunction (Jaundice).
8. Renal damage.

There is no evidence that vomiting increases perinatal mortality rate; it is rather considered a favourable sign of a lower risk of spontaneous abortion before 20 weeks of pregnancy.⁶ There is no significant difference in birth weight or maternal weight gain in patients who experience vomiting in pregnancy. There is also less risk of foetal loss and preterm labour.

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Cord Prolapse

EY Kwawukume

INTRODUCTION

Umbilical cord prolapse is one of the many causes of fresh stillbirths, occurring once in 161-714^(1,2) deliveries. The common denominator for cord prolapse is incomplete fitting of the presenting part into the maternal pelvis at the time of rupture of the membranes^(3,4).

Cord prolapse is one of the obstetric emergencies seen in maternity units in modern obstetrics and timely delivery is the hallmark of good clinical management. Many times, cord prolapse occurs at a time in labour where the cervix is not sufficiently dilated for vaginal delivery. In such situations different management techniques are applied as temporary measures until a Caesarean section can be safely and expeditiously performed.^(5,6)

In many developing countries it is not easy to mobilise the theatre for emergency Caesarean section and patients with cord prolapse and partially dilated cervix would have to travel long distances before reaching a hospital equipped for Caesarean section. This results in fetal death. The various modalities of management of cord prolapse all aim at raising the pelvis and therefore bring the cervix to a higher level than the fundus of the uterus. Such manoeuvres will allow decompression of the cord by natural forces of gravity acting on the presenting part of the fetus.⁽⁷⁾

DEFINITION: Cord prolapse is the presence of the umbilical cord below the presenting part with ruptured membranes.

Cord presentation is the presence of the umbilical cord below the presenting part with intact membranes.

Occult cord prolapse happens when the cord lies beside the presenting part and is not detected by the examining fingers. This might lead to variable decelerations or unexplained fetal distress.

Causes and Diagnosis

Careful clinical examination is needed to identify non-longitudinal lie or an unengaged vertex presentation because the umbilical cord is at risk of prolapse. First, the obstetrician should avoid rupturing the membranes until clinical evidence justifies it. There should be ultrasound evaluation

and if possible vaginal probe to locate the position of the umbilical cord.

During digital pelvic examination, variable deceleration may be observed or the cord may be palpated. Fetal heart should be checked continuously after amniotomy to monitor the fetal status.

Cord prolapse is associated with all factors maintaining the presenting part high above the pelvis and it is primarily related to fetal presentation and secondarily to the station of the presenting part. Malpresentations as in breech, transverse lie, footling and complete breech presentations where the presenting part does not fill the maternal pelvis can result in cord prolapse. The risk of cord prolapse increases progressively for the respective breech presentations. For the frank breech the incidence is about 0.5 percent and increases to approximately 5 percent for the complete breech presentation and drastically from 15 to 18 percent for the incomplete breech presentation⁽⁸⁾.

Other conditions are polyhydramnios, preterm labour, multiple pregnancy, premature and small fetuses and contracted pelvis where the presenting part is free above the pelvic brim^(3,4).

Sudden rupture of membranes can lead to umbilical cord prolapse. This is one of the reasons why it is necessary to perform pelvic examination as soon as the membranes rupture in labour or perform sterile speculum examination in premature rupture of membranes if a decision has not yet been taken to deliver the fetus.

Management

Cord prolapse in the face of full dilatation of the cervix and a live baby poses little problem because immediate delivery by vacuum extraction or obstetric forceps could be used provided there are no contra-indications to vaginal delivery. The difficult ones are cases of cord prolapse with partially dilated cervix and a live baby. These are cases that are impossible to treat with any degree of success unless emergency caesarean section is performed without delay.

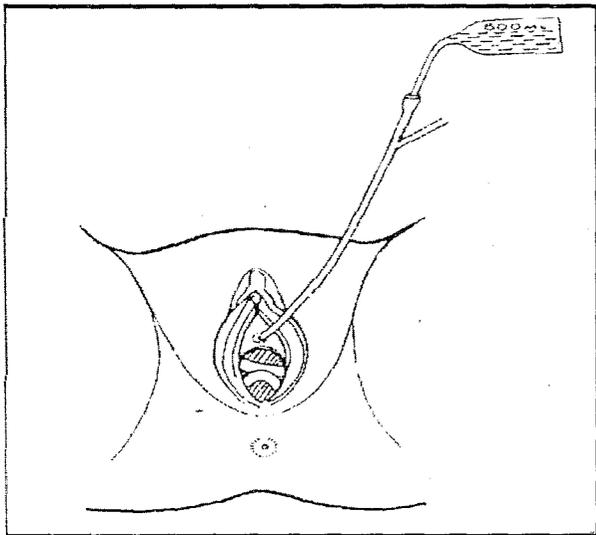
The genu pectoral (knee-chest position) and high Trendelenburg positions^(3,9) have all been used but these are tiring and irksome for the patient. Some obstetricians combine these methods with keeping

fingers in the vagina and thus preventing the presenting part from pressing the cord especially during contractions or by Funic reduction, that is, manual replacement of the prolapsed cord ⁽¹⁰⁾. These methods need experienced obstetricians.

If there is overt umbilical cord prolapse pulsation in the cord should be checked. If pulsations are not felt it might be due to cord compression and the fetus might still be alive. Fetal heart tones should be auscultated to confirm the diagnosis. Ultrasonography is necessary to confirm fetal demise if available. If the cervix is fully dilated and there is no contraindication to vaginal delivery, an assisted breech delivery, forceps or vacuum extraction is carried out. If the presenting part is not deep in the pelvis, it would be better to proceed to Caesarean section. If the cervix is not fully dilated, the cord should gently be replaced in the vagina if it is outside and a sanitary pad placed at the vulva.

Five hundred millilitres of normal saline solution is instilled rapidly into the urinary bladder with a size 16F Foley catheter and the end spigotted ⁽⁶⁾. The balloon should be inflated to retain the catheter in the bladder and the patient should lie on the left lateral position or Trendelenburg's position while preparations are made towards emergency caesarean section.

The mechanism of filling the bladder is to elevate the presenting part of the fetus and prevent it from compressing the umbilical cord and thus enhance cord blood circulation. Secondly, full bladder physiologically inhibits uterine contractions. Though, there might be contractions they would not be strong enough for the presenting part to effectively compress the umbilical cord.



Umbilical cord Prolapse: Foley's catheter in the urethra with 500 ml of normal saline placed at the end

It has been reported that out of 59 cases of cord prolapse there was one fresh still birth (FSB) which was attributed to the spigot being removed rather early before the patient was wheeled onto the operating table ⁽⁶⁾. This might have resulted in the head compressing further on the umbilical cord. Other researchers ^(7,9) in a 5-year study, combined filling the bladder and giving intravenous ritodrine in 51 patients and there were no perinatal deaths. Finally, oxygen should be administered by facemask or intranasal, oxytocin infusions should be discontinued and intravenous fluids should be given in preparation towards an emergency Caesarean section.

When there is cord prolapse and fetal death there is no hurry to effect delivery if the cervix is not fully dilated. Care must always be taken to make sure the fetus is dead. This is not always easy in a situation where facility to establish fetal death is unavailable.

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Ectopic Pregnancy

EY Kwawukume/A Idrisa

Introduction

Ectopic pregnancy still remains a disaster in Sub-Saharan Africa and has become a public health problem of epidemic proportions. It still continues to exert its toll on human reproduction contributing to increasing morbidity and mortality. At least one case of ectopic pregnancy is reported every day or every other day in Korle Bu Teaching Hospital Accra, Ghana forming about 1 in 30 to 50 deliveries. The number of ectopic pregnancies seen has certainly increased and there are multiple factors. There is more adequate management of pelvic inflammatory diseases that in the past would have rendered the patient infertile and reduce the chances of pregnancy. The increased use of potent antibiotics allow tubal patency but with luminal damage. Secondly, the use of relatively efficient intrauterine devices (IUD) has probably prevented intrauterine gestations and any pregnancy becomes extrauterine. In addition surgical treatment of diseases on the fallopian tube as seen in tubal anastomosis predisposes the patient to ectopic pregnancy. Finally, ectopic gestation that would have been mislabelled as unexplained abdominal pain or bleeding in the past is now diagnosed with improved diagnostic techniques thus increasing the incidence of ectopic gestation.

Definition: Ectopic pregnancy can be defined as any gestation occurring outside the uterine cavity. The most usual site is the fallopian tube forming ninety-seven (97) percent, while two (2) percent are uterine ectopic pregnancy (interstitial). The remaining two (2) percent include abdominal, ovarian and cervical. Majority of ectopic pregnancies occur in the distal one-third of the fallopian tubes. ^(1,2,3)

Combined intrauterine and extrauterine pregnancy is very rare and occurs in approximately 0.02 to 0.007 percent of pregnancies ⁽⁴⁾. The incidence of lateral ectopic pregnancy has not been documented but isolated cases have been reported in the literature. ^(5,6)

Fig1

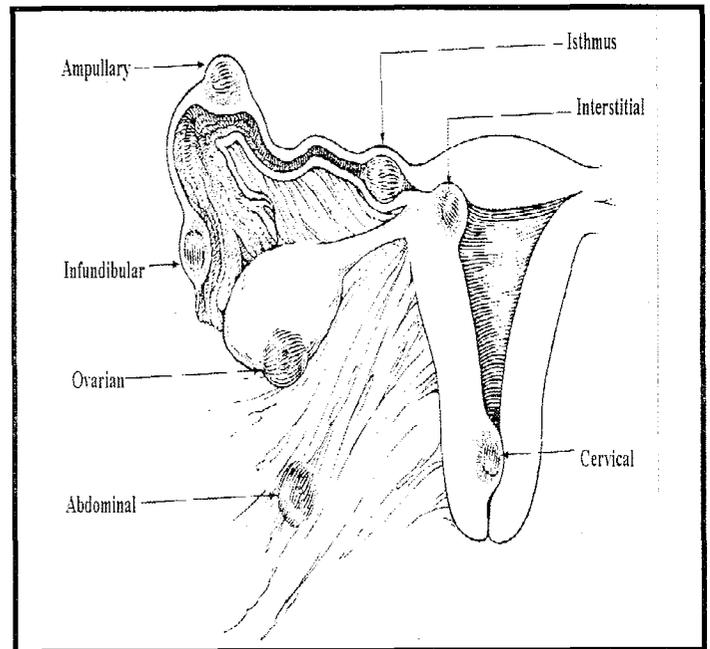


Diagram Of Various Sites Of Ectopic Pregnancy

Epidemiology

The incidence of ectopic pregnancy in developing countries is rather high as compared to developed countries. In Jamaica ⁽⁷⁾ the incidence is 1 in 28 deliveries, 1 in 21 deliveries in South Korea ⁽⁸⁾ and 1 in 24.4 deliveries in Ghana ⁽⁹⁾. In the United States of America the incidence is 1 in 241 deliveries ⁽¹⁰⁾ to 1 in 280 deliveries ⁽¹¹⁾. The increase in the incidence might be due to more liberal sexual practices and the increasing incidence of pelvic inflammatory disease ⁽¹²⁾

It is the 3rd commonest case seen at the Gynaecology Emergency Unit in Korle Bu Teaching hospital. The rate has doubled from 1 in every 44 deliveries in 1975 to 1 in 24.4 deliveries at the same centre over a decade ^(9,10). The most common form of presentation is ruptured ectopic although a few cases of unruptured are increasingly being diagnosed. The later is largely due to increased index of suspicion. The use of ultrasound and diagnostic laparoscopy in centres where available has also increased our diagnostic acumen

Pathophysiology

The fallopian tube serves as the passage for the sperm, ovum, and zygote. The ovum passes through the fimbrial end of the tube and the fimbrial mucosa and the cilia serve to pick up and transport the ovum into the fallopian tube where fertilisation takes place ^(14,15) in the ampullary site, in the inner surface of the lumen. The zygote spends approximately 72 hours before it is transported to the isthmus of the tube ⁽¹⁶⁾.

When ectopic embryo outgrows its blood supply, four different processes may occur. There is formation of a tubal blood mole, in which the blood flows around the chorionic sac, overdistend the tube and leads to intraluminal rupture of the sac into the tube. On the other hand, there may be tubal abortion of the tubal blood mole through the fimbriated end of the tube, either completely or incompletely into the abdominal cavity. Thirdly, there may be reabsorption of the conceptus as a result of either erosion of chorionic villi or mechanical overdistention of the tube. Normally the fertilised ovum eventually reaches the uterus in about 80 hours.

Majority of ruptured ectopic gestation occurs in the ampullary site, probably because the tissue between the lumen of the tube and the serosa is composed of loose adventitia and represents the path of least resistance. Expansion of the zygote in the ampullary portion results in tubal damage and destruction by the invasive trophoblast. Pregnancy continues at this site and the developing villi might erode into the blood vessels resulting in bleeding.

The isthmus portion of the tube has different anatomic features. The tissue between the epithelium of the lumen and the serosa is compact, composed mostly of muscularis. This compact structure prevents the ectopic pregnancy from eroding into the space between the serosa and the tubal epithelium. The zygote develops within the lumen of the tube itself causing destruction of the lumen.

Risk factors

1. Pelvic Inflammatory Disease (PID)

- * Tubal fibrosis
- * Scarring of intraluminal structures.
- * False passage formation
- * Altered cilia
- * Abnormal tubo-muscular action
- * Chronic pelvic infection
- * Narrowing of the tube, which may result in transport dysfunction because of tubal constriction.

2. Peritubal adhesions, e.g. Appendicitis and drainage of pelvic abscesses.
3. Congenital defects of the tube as seen in sacculations and diverticuli diseases.
4. Fibroids at the junction of the uterus and tube.
5. Benign tumours and cysts of the tube.
6. Tubal endometriosis.
7. Previous ectopic pregnancy
8. Previous tubal surgery
 - * Tubal anastomosis following previous tuba ligation.
 - * Adhesiolysis.
9. Ovarian hyperstimulation
10. IUD use. Most pregnancies conceived with IUD in-situ are likely to be ectopic. This is probably due to the relative efficiency of the device in preventing intrauterine gestations rather than from a direct relationship of the IUD to ectopic gestation.
11. Progestin-only oral contraceptives.
12. Transmigration of the fertilised ovum. This might lead to delay in transportation of the fertilised ovum and the developing blastocyst might be too big to pass through the narrowed isthmus part of the tube.
13. Exposure to diethylstilbestrol (DES).

Diagnosis of ectopic pregnancy

Any woman of reproductive age presenting with pelvic pain or bleeding should have ectopic pregnancy ruled out. Lower abdominal pain and a period of amenorrhoea are the most common symptoms of ectopic pregnancy, therefore adequate history of abnormal bleeding that may occur at the expected time of menses should be taken. Most patients might interpret abnormal bleeding as menses and the diagnosis of ectopic gestation may be missed. Major bleeding is uncommon but the amount, timing, and character of the bleeding should not obviate the possibility that ectopic pregnancy could be the diagnosis.

Few patients present with a typical picture of ectopic pregnancy and presentation might be variable, ranging from asymptomatic patient to dizziness or syncope to the patient in hypovolemic shock. Presentation also depends on the amount of trophoblastic tissue that has developed, the site of

implantation and whether or not rupture has occurred. A period of amenorrhoea of 12 to 14 weeks might be diagnostic more of cornual (interstitial) ectopic pregnancy.

Clinical signs include lower abdominal tenderness, palpable pelvic mass and positive cervical excitation tenderness. Distended abdomen might be due to haemoperitoneum with shoulder pain, which is referred pain from haemoperitoneum causing irritation of the diaphragm.

Differential diagnosis

1. Normal Intrauterine pregnancy
2. Pelvic Inflammatory Disease (PID)
3. Ruptured ovarian cyst
4. Threatened or incomplete abortion
5. Adnexal torsion
6. Degenerating uterine fibroids
7. Dysfunctional uterine bleeding
8. Bleeding corpus luteum of a normal intrauterine pregnancy
9. Appendicitis
10. Endometriosis

Pelvic Inflammatory Disease is the most common condition confused with ectopic pregnancy, especially when PID is associated with anaemia. Pelvic Inflammatory Disease is hardly ever seen in pregnancy because the decidua and membranes effectively seal off the uterine cavity preventing organisms from ascending. Therefore, if a patient of reproductive age presents with amenorrhoea and signs and symptoms of PID, think of ectopic pregnancy and investigate as such. A high level of suspicion is needed to diagnose ectopic gestation.

Labouratory Diagnostis Tests

1. Serial values of human chorionic gonadotropin (HCG, a glycoprotein produced by trophoblastic tissue) can be measured in the serum within 8-12 days after fertilisation.

- Serum *b*-subunit of HCG is positive in 100% of ectopic pregnancies.
- Urine pregnancy test for HCG is positive in only 50% of cases with proven ectopic gestation
- The serum HCG level doubles every 2 days in a normal pregnancy, which does not occur in ectopic and blighted ovum pregnancies
- At a discriminatory zone of about 6,500 mIU/ml of HCG an intrauterine gestational sac should be seen by an abdominal ultrasound at 42 days of gestation. A value of 1,300-1,400 mIU/ml at

35 days of gestation using an endovaginal ultrasound is also diagnostic. Failure to detect fetal echoes within the uterus should suggest the possibility of an extrauterine gestation. It therefore means that ectopic pregnancy can be ruled out about 7 days earlier with the vaginal ultrasound than with the abdominal ultrasound. (17)

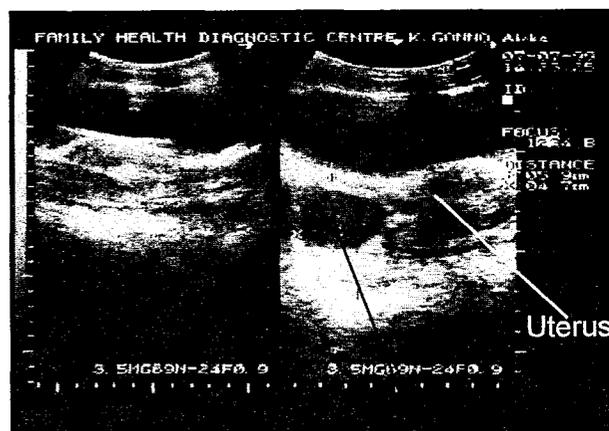


Fig 2. Scan picture of unruptured ectopic pregnancy

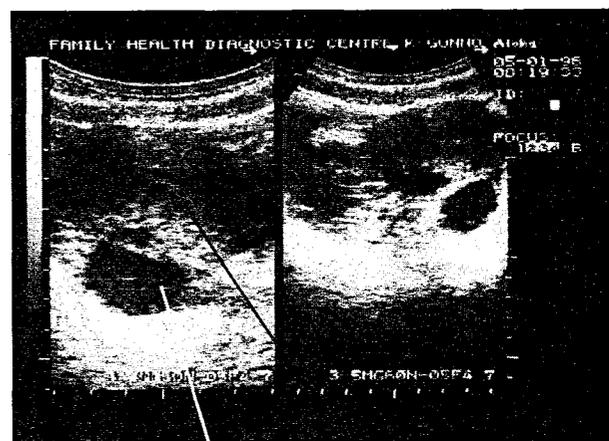
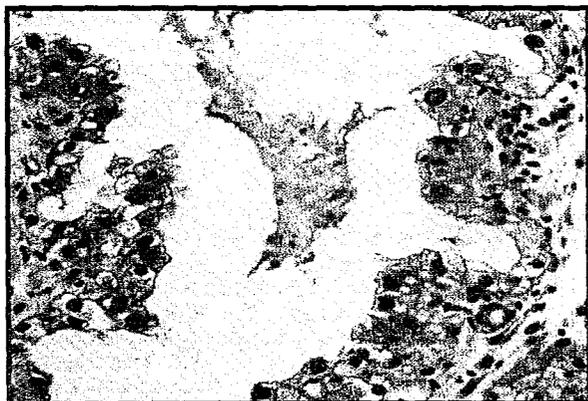


Fig 3. Ruptured ectopic gestation showing fluid in the Pouch of Douglas

2. Progesterone Assay⁽¹⁸⁾. Normal intrauterine pregnancies have values more than 20ng/ml and ectopic gestation has values less than 15ng/ml.
3. Abdominal or vaginal ultrasound. Fig 1 and 2 illustrate unruptured and ruptured ectopic pregnancies. Fig 2 shows fluid in the Pouch of Douglas and an empty uterus with a thick echogenic endometrium. The β -hCG was positive.
4. Paracentesis or culdocentesis.
5. Laparoscopy.
6. Endometrial histology for abnormal uterine bleeding.

Diagnosis might be doubtful in some circumstances of abnormal bleeding. In such cases dilatation and curettage (D&C) is performed. If there is decidual reaction in the endometrial sample without evidence of chorionic villi then ectopic pregnancy is indicated until proved otherwise.



Picture of Arias-Stella reaction

Additionally, Arias-Stella reaction in the endometrium is also suspicious of ectopic gestation. Arias-Stella reaction in the endometrium is a response to hormonal stimulation of pregnancy, producing a patchy, hyperactive and often a hypersecretory pattern.

Treatment

Management options include, conservative or radical surgical techniques, systemic methotrexate, and local injection of a variety of substances either by ultrasound guidance or at laparoscopy, and expectant management in clinically stable patients with declining hCG levels. Unfortunately, 99% of the cases seen in our sub-region have already ruptured and are treated by salpingectomy in our hospitals. We need extensive public health education for our women to report to health care centres as soon as they miss their menses. Early treatment can then be started and attempt can be made to utilise the least invasive method to manage the disease and conserve fertility if desired.

Treatment options can therefore be classified into:

- Radical surgery.
- Conservative surgery.
- Medical.

These treatment options depend on whether **the ectopic pregnancy is ruptured or not ruptured**

1. Radical surgery with ruptured ectopic pregnancy.

* Salpingectomy. This is the most common surgery in our hospitals. The whole tube is

removed

2. Conservative surgery with unruptured ectopic pregnancy.

* The pregnancy is milked from the fimbria end of the tube. No surgery is done if there is no bleeding. It must however be noted that failure rate can be high due to persistence of trophoblastic tissue ⁽¹⁹⁾

* Linear salpingostomy, either by laparotomy or laparoscopy, even if the tube is dilated as much as 4-cm. ⁽²⁰⁾ This is usually done for ampullary ectopic pregnancies. A linear incision is made on the antimesenteric side of the tube with a scalpel, electrocauter, carbon dioxide or argon laser. The products of conception are extruded, and haemostasis is achieved. This technique is employed if the tube is going to be allowed to heal by secondary intention ⁽²¹⁾.

* Salpingotomy. The tube is closed in two layers, muscularis and then serosa, with fine suture materials.

3. Medical management with unruptured ectopic pregnancy. The drug used commonly in the medical management is methotrexate, a folic acid antagonist. It inhibits synthesis of purines and pyrimidines thereby interfering with DNA synthesis and cell multiplication.

* Multidose methotrexate (MTX) with Citrovorum rescue factor (CF). Intramuscular (IM) injection with 1.0 mg/kg of MTX on days 1,3,5, and 7 (4 doses) and 0.1 mg/kg IM of CF on days 2,4,6, and 8. Methotrexate acts as a folic acid antagonist and interferes with DNA synthesis and cellular multiplication.

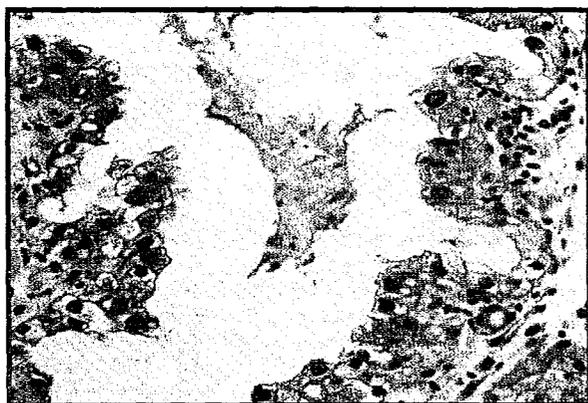
* Single dose methotrexate without citrovorum rescue factor given intramuscularly (IM) as 50 mg/m².

* Actinomycin-D. This might be used in advanced gestation (HCG > 10,000 mIU/ml) in which methotrexate has a higher failure rate.

* Potassium chloride (KCl): Can be injected into the fetal heart in advanced ectopic gestation to induce asystole. KCl may have a role in treating heterotopic pregnancy ⁽²²⁾

* Antiprogestin (RU486). This requires

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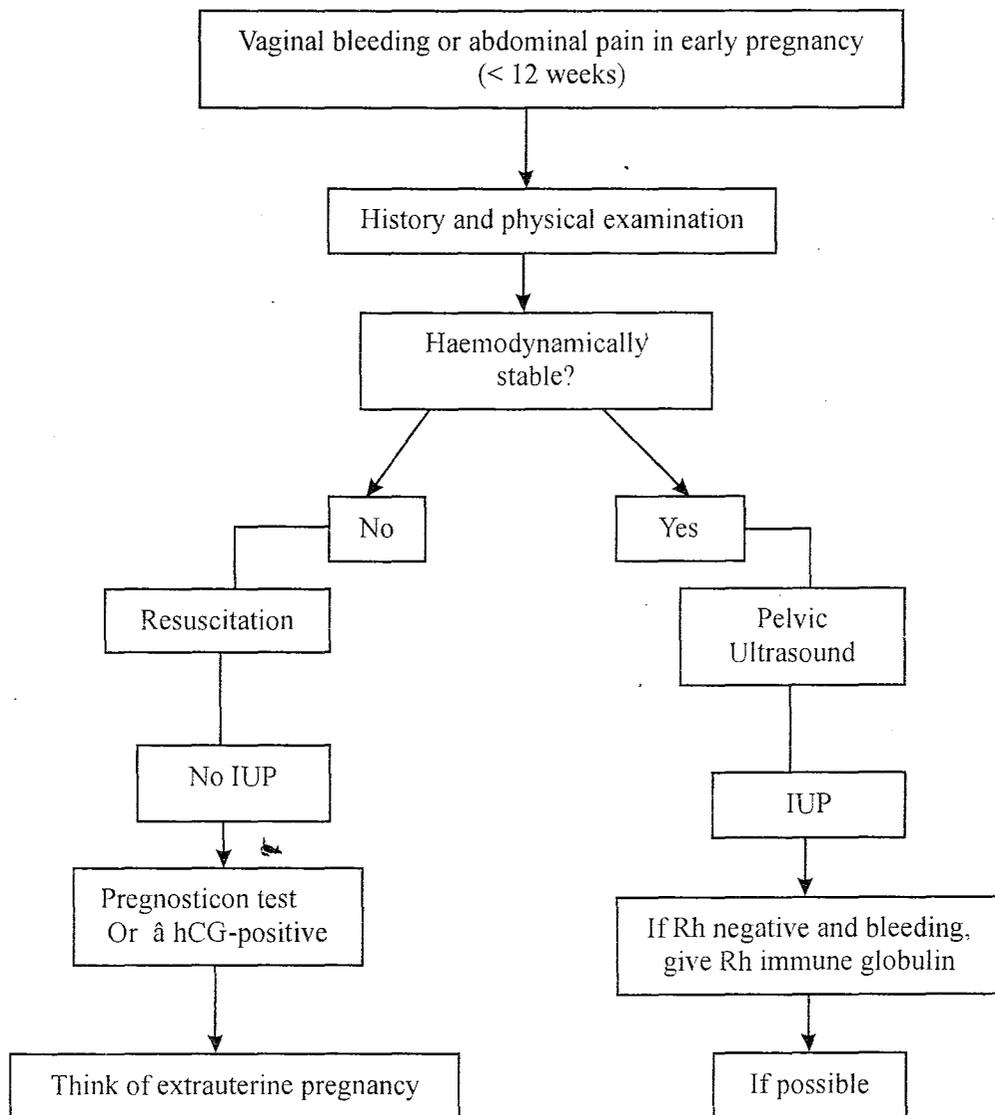
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Algorithm for possible ectopic pregnancy without invasive methods



Ovarian pregnancy. The arrow shows Villi adjacent to a corpus luteum

- substantial further testing.
- * Prostaglandins injection.
- * Hyperosmotic glucose (50%) injection directly into the gestational sac. ⁽²³⁾

Ovarian Pregnancy

Ovarian pregnancy is rare and for diagnosis one must identify Spiegelberg's criteria as:

1. The tube, including fimbria ovarica, must be intact.
2. The gestational sac must occupy normal ovarian position.
3. The sac must be connected to the uterus by the utero-ovarian ligament.
4. Ovarian tissue must be identified histologically in the wall of the gestational sac.

Abdominal Pregnancy

Abdominal gestation is also rare but probably the most serious of the extrauterine gestations. It can be classified as primary or secondary. Most abdominal pregnancies are secondary, resulting from early tubal abortion or rupture and subsequent implantation of the pregnancy into abdominal structures.

Primary abdominal pregnancy must satisfy the three criteria defined by Stufferford: ⁽²⁴⁾

1. Both tubes and ovaries must be in normal condition with no evidence of recent or remote injury.
2. No evidence of uteroperitoneal fistula should be found
3. The pregnancy must be related exclusively to the peritoneal surface and must be early enough in the gestation to eliminate the possibility of secondary implantation following primary implantation in the tube.

Diagnosis of Abdominal Pregnancy

1. History of recurrent abdominal discomfort
2. Fetal movement beneath the abdominal wall
3. Presence of fetal movements high in the upper abdomen
4. Cessation of fetal movement
5. Vomiting late in pregnancy
6. Fetal malposition
7. Closed and uneffaced cervix.
8. Hysterosalpingography if the uterus can be

- palpated differently from the fetal parts
9. Failure of oxytocic to stimulate the gestational mass.
10. Abdominal x-ray with fetal small parts in the lateral position overlying the maternal spine, ⁽²⁵⁾

11. Ultrasonography is the most reliable.

- * Sometimes fluid is seen in the Pouch of Douglas
- * Tubal mass could be seen
- * Better evaluation of pseudosac in uterus
- * Character of pelvic masses. It should be noted that ultrasound presence of an enlarged uterus and an adnexal mass is not helpful since this may simply represent an early intrauterine pregnancy and a corpus luteum.

Management of Abdominal Pregnancy

Abdominal pregnancy is not uncommon in the developing countries. Many of the cases that were managed resulted in fetal mortality, which agrees with other workers such as Martin ⁽²⁶⁾. The fetuses had a lot of congenital malformations including facial and joint deformities, torticollis and hypoplasia of the extremities.

Management of the placenta remains controversial. In our units, after delivery of the baby, the cord is clamped leaving the placenta in-situ. The abdomen is then closed. A corrugated drainage or a self-retaining catheter is left in the peritoneal cavity and the patient is given a broad-spectrum antibiotic cover.

Sometimes, an isolated vascular segment that can be clamped is attached to the placenta and the whole placenta is removed after clamping. If the placenta is attached to other structures, it is better left behind to undergo autolysis and sclerosis of its blood supply and finally for spontaneous resorption to occur. Other authorities would administer methotrexate to hasten trophoblastic degeneration. Pregnancy after the wound is completely healed is encouraging and patients can deliver vaginally.

Discussions/Controversies

1. Paracentesis (abdominal tap) or culdocentesis is the diagnosis of ectopic pregnancy.

Paracentesis or culdocentesis has a place in the diagnosis of ectopic gestation especially in areas where radiologic or ultrasound support is not available in a timely fashion. Before abdominal tap

the history and the physical examination should be reviewed again to check for cervical excitation tenderness accompanied by cul de sac fullness or bulging. If there is shifting dullness or fluid thrill then there is no place for culdocentesis. On the other hand if shifting dullness is absent but there is a strong suspicion of ectopic gestation then a tap could help in further management of ectopic gestation. In centres with accurate serum HCG values and endovaginal ultrasound probe, abdominal tap is less frequently used. Culdocentesis is negative if serous fluid is aspirated and positive if nonclotting blood is aspirated. The blood obtained is nonclotting because there is lysis of blood that has clotted previously. The haematocrit of the nonclotting blood should exceed 15% to be significant. It should also be noted that unclotted blood in the Pouch of Douglas does not mean that ectopic pregnancy has ruptured this could be bleeding from ruptured corpus luteum or any abdominal organ.

2. Laparoscopy is the Gold Standard for the diagnosis of ectopic pregnancy but should be performed after the following procedures:

- Patient has subnormal rise in HCG
- Abnormal ultrasound finding
- Negative result for dilatation and curettage
- A suspicious case of PID that does not resolve appreciably in hospital should be scoped before going home.

3. Autologous Blood Transfusion

Intraoperative blood salvage is one of the four modes of autologous blood transfusion. The others are predeposit, intraoperative haemodilution and postoperative salvage. Autologous transfusion is not new in some of our hospitals but proper documentation has not been done to ascertain its merits and demerits in modern medical practice. Autotransfusion of blood can be accomplished by using a simple system consisting of a blood-collecting device, an in-line filter system, and a container for anticoagulation, usually with citrate phosphate dextrose.

There are various equipment for collecting, washing and filtering shed blood before reinfusion. Some of these are Cobe Cell Saver, Solcotrans Plus and ID set. The last two do not incorporate washing, are relatively simple and suffice for clean flowing blood as seen in ectopic pregnancies. However, if there is cellular debris, fat and other contaminants in the operative field, washing is recommended.

The principles involved in intraoperative blood salvage are as follows:

- Shed blood is suctioned at 80-100 mm Hg into a bag containing anticoagulant, citrate or heparin in a ratio of 1:7 to 1:10 citrate to blood.
- The anticoagulated blood is filtered using a 40/60-micron blood filter
- The filtered anticoagulated blood is re-infused using a blood giving set

The main dangers about autologous transfusion are infection and possible amniotic fluid embolism. However, the necessary infection prevention technique should be practised to make transfusion safe and beneficial. In addition, old clotted blood should not be transfused and haemoperitoneum should be properly sieved before transfusion. Blood could be taken for culture and sensitivity and patient should be given broad-spectrum antibiotic as prophylactics.

If autologous transfusion is mastered and practised according to standard protocols, diseases such as HIV, hepatitis and other blood related infections could be prevented. Further technique on autologous blood transfusion is outlined in the chapter on Anaemia in Pregnancy.

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Obesity In Pregnancy

CA Klufio

Introduction

Obesity is a nutritional disorder that is characterised by excessive accumulation of fat in the subcutaneous tissues, in the omentum and viscera, and in muscles. In women, the **fat component** of body weight has a normal range of 13-24%. Obesity is present in women when more than 25% of body weight is due to fat. All mammals store fat for its heat insulation properties and as a potential source of energy in lean times. In the subcutaneous tissues and around organs, fat is a thermal insulator. Fat is an efficient source of stored energy (9kcal/g). However, excessive fat storage is a health hazard. Records of insurance policyholders consistently show that persons, who are 30 percent or more overweight, have measurably increased risks of disease and earlier death.

Obesity is common in the developed world because of gross overeating, and eating of high fat and refined carbohydrate foods so-called "junk food". Because of its high prevalence in the developed world, obesity is a major public health problem in these countries. On the other hand, in the developing world, because of poverty, shortage of food, and ignorance about good nutrition and ideal body weight, people eat excessive amounts of carbohydrates. It is even thought by some women that fatness is a sign of affluence and good living and proof that they are being well-kept by their husbands! As a result, obesity is becoming more common. Urgent and vigorous education of the public is needed to arrest this trend, before obesity becomes another disease of the developing world.

Diagnosis

How do we determine if a woman is obese or not? In other words, how do we define obesity? There are rough and ready criteria as well as more valid measures, e.g. the body mass index. But none of these actually measures the proportion of fat in the body mass of the individual; they are therefore indirect measures of obesity. The commonly used measures are:

Height and weight indices

Subscapular and triceps skin fold measurements
Empirical criteria

Height and weight indices

A woman is said to be **overweight** if her weight is 20-25% above the standard weight for women of the same age and height. She is graded as having:

Mild obesity, if her weight is 25-40% above the standard weight

Moderate obesity, if her weight is 41-100% above the standard weight

Severe (morbid) obesity, if her weight is more than 100% above the ideal weight.

a. Height and Weight Tables: These tabulate the height and frame (small, medium, or large) of a woman, against the weight range, which according to the records of insurance policyholders, is associated with the lowest mortality for that height. A woman is not overweight if her weight falls within the ideal range for her height and frame. If her weight is more than 9kg in excess of her ideal weight range, she is obese.

b. Ponderal Index: This is obtained by dividing the square of the height by the cube root of the weight
Ponderal index = $\text{height}^2 \sqrt[3]{\text{weight}}$

c. Body Mass Index (Quetelet Index): The Body Mass Index (BMI) is obtained by dividing the body weight in kilograms by the square of the height measured in centimetres
 $\text{BMI} = \text{Body weight (kg)} / \text{height (m)}^2$
BMI is the most appropriate measure for assessing weight change. The normal range is 18.5-24.9kg/m².

The chief disadvantage of height and weight indices is that they fail to take account of the variable proportions of lean body mass and fat mass in the total weight of the body. A person may be overweight because of lean mass and not necessarily because of excessive body fat, as is the case with bodybuilders.

Pre-obesity (mild Obesity)	25.0-29.9kg/m ²
Obese Class I	30.0-34.9kg/m ²
Obese Class II	35.0-39.9kg/m ²
Obese Class III	40.0kg/m ²

Subscapular and Triceps Skin fold thickness

These measure subcutaneous fat more directly than do the height weight indices. They require special callipers. Obesity is diagnosed if skin fold thickness is greater than the standard by 20% or more.

Empirical Definitions

In pregnancy, obesity is defined as weight greater than 90kg, regardless of height and pre-pregnancy weight. Morbid obesity is defined as weight greater than 115kg.

Pathogenesis

The vital processes of animals (e.g. digestion and metabolism of food, synthetic and anabolic reactions, muscular contraction, nerve impulse conduction, active transport, secretions by glands, and thermoregulation) require energy. They are endergonic. Energy is obtained for the vital processes by chemical linkage (coupling) with oxidative reactions that produce chemical energy. The oxidative reactions are exergonic. The energy produced is in the form of molecules that contain high-energy phosphate bonds, chiefly adenosine triphosphate (ATP). ATP traps energy produced in exergonic reactions and releases it for use in endergonic reactions. The rate of energy release is measured as the metabolic rate, and is controlled by the thyroid hormones. The basal metabolic rate (BMR) is 2000kcal/day. Depending on the individual's physical activity, an additional 500-3000kcal/day will be required.

The exergonic processes, themselves, depend on fuel. This is food. Food also generates heat for thermoregulation through its specific dynamic action (SDA), and by providing energy for isometric and isotonic muscular contractions. The SDA of food is the energy produced during digestion and

absorption of food. The SDA of protein is 30kcal for the weight of protein that will yield 100kcal. For carbohydrate and fat, the respective figures are 6kcal and 4kcal. This energy must come from the food itself, or from the body's energy stores. Apart from the SDA, energy output after meals is also increased by an increase in sympathetic discharge; and this increase in sympathetic discharge can be altered.

Brown adipose tissue differs from ordinary adipose tissue in many ways.

- It is present mainly around the scapulas and along the aorta.
- It has a generous blood supply.
- It has numerous mitochondria and cytochromes.
- It has many sympathetic nerve endings.
- The activity of ATP synthase is low.
- Oxidation and phosphorylation are not coupled in the mitochondria. As a result, oxidation produces heat, but not ATP.

The main function of brown adipose tissue is heat generation for thermoregulation. After eating noradrenaline is liberated at the sympathetic nerve endings. This causes lipolysis (release of free fatty acids and glycerol) and production of heat. This is termed "diet-induced thermogenesis". Brown adipose fat is absent or reduced in obese persons. The caloric value of fat is 9kcal/g; for both carbohydrate and protein it is 4kcal/g.

The first law of thermodynamics is the law of energy conservation. It states that the total energy of a system, including its surroundings, cannot be changed; it remains constant. Energy in the system can be transformed (converted) from one form into another form, (e.g. from biochemical energy into heat, from muscular energy, say, shivering, into heat, radiant energy into heat and vice versa) but energy cannot be created or destroyed. On earth, the sun is the ultimate source of energy.

According to the above law, if energy (food/caloric intake into the body (system) is less than energy expenditure, endogenous stores (glycogen, body fat and body protein) are catabolised, and the individual loses weight. On the other hand, if energy intake exceeds energy expenditure, energy is stored, and the individual gains weight. The

Excess energy is stored primarily as fat. Insulin has a leading role in the metabolism of adipose tissue.

- It promotes the uptake of glucose into adipose tissue cells.
- It inhibits the release of free fatty acids from adipose tissue.
- It inhibits the activity of hormone-dependent lipase on adipose tissue. As a result of these three properties, insulin enhances lipogenesis and inhibits lipolysis.

Therefore, in the final analysis, body weight is determined by energy balance; i.e. by the balance between caloric (food) intake and energy expenditure.

Obesity may start in childhood or in adult life. Adult-onset obesity is mainly due to adipose cell hypertrophy, and the accumulation of fat is mainly trunkal. Childhood-onset obesity is mainly due to fat cell hyperplasia, and the distribution of fat accumulation is more general.

Pathologically, there are two types of obesity. In the first type, obesity occurs because the individual overeats, and/or does not perform sufficient exercise or other physical activity to use the excess energy she takes in. In the second type, obesity develops because the individual is a poor heat loser; she loses little heat from the skin, and has a lower skin temperature. She has little or no brown adipose tissue to generate heat. She does not expend a sufficiently large proportion of the energy taken in to produce heat. She therefore has more energy to store.

Complications

Obesity is associated with increased risk of many health problems.

A. General Complications

1. Mechanical Complications
 1. Osteoarthritis, particularly of knees and hip joints
 2. Flat feet
 3. Skin disorders, especially in the intertriginous folds (intertriginous dermatitis) and fungal infections
 4. Varicose veins of the lower limbs
 5. Ventral diaphragmatic hernias
 6. Cholelithiasis and cholecystitis

7. Obesity-Hypoventilation Syndrome (Pickwickian Syndrome)

- II. Chronic (Pre-existing) Hypertension
- III. Type II Diabetes Mellitus
- IV. Atherosclerosis and Thromboembolism

1. Ischaemic heart disease
 - i. Angina pectoris
 - ii. Pre-infarction angina
 - iii. Myocardial infarction
 - iv. Sudden death

2. Cerebrovascular Disease
 - i. Transient ischaemic attacks
 - ii. Stroke
 - iii. Deep venous thrombosis and pulmonary embolism
- V. Anaesthetic Complications: Difficult/Failed Intubation
- VI. Difficult Venepuncture: Gaining venous access can be difficult
- VII. Wound Complications
 1. Wound dehiscence
 2. Wound infections

B. Obstetrical Complications

- I. Feels more uncomfortable, and suffers easy fatigability and exertional dyspnoea more than the nonobese patient does
- II. Oedema of the feet and legs is more common
- III. Hypertensive Diseases of Pregnancy
 1. Chronic hypertension
 2. Superimposed pregnancy induced hypertension
 3. Eclampsia
- IV. Diabetes Mellitus
 1. Type II diabetes
 2. Gestational diabetes
- V. Difficulty in Abdominal Palpation, and in:
 1. Diagnosing malpresentation
 2. Managing malpresentation by external cephalic version
 3. Diagnosing twins
 4. Estimating gestational age
 5. Diagnosing inappropriate fetal growth: excessive or restricted intrauterine growth
 6. Because of the above there is a greater need for imaging. Even so, imaging, (e.g. ultrasound scan for the fetus and placenta) may not give as clear a picture as it would in the non-obese.

- VI. Difficulty in auscultation of the fetal heart: If cardiotocography (CTG) is not available, fetal distress in labour may go unrecognised
- VII. Macrosomia from obesity per se, and from obesity plus diabetes. Obesity increases the likelihood of macrosomia 4 - to 12-fold. Macrosomia predisposes to shoulder dystocia, traumatic delivery, and injury to both baby and mother, and birth asphyxia
- VIII. Post-term Pregnancy
- IX. Increased use of Induction of Labour because of:
 1. Post-term
 2. Hypertensive disease
- X. Prolonged Labour from:
 1. Macrosomia
 2. Ineffective uterine contractions
- XI. Increased use of Syntocin Augmentation of Labour
- XII. Increased Perinatal Morbidity and Mortality from:
 1. Placental insufficiency from post-term and from hypertensive disorder
 2. Shoulder dystocia and traumatic delivery
 3. Diabetes mellitus
 4. Pre-term delivery as management for hypertensive disorder
 5. Failure to diagnose malpresentations
 6. Prolonged labour
 7. Birth asphyxia
- XIII. Increased Rate of Caesarean Section
- XIV. Failure of Lactation
- XV. Thromboembolism
- XVI. Urinary Tract Infection
- XVII. Postpartum Haemorrhage

Hypertension

Hypertension is a major contributor to the increased death rate in obesity. Obesity predisposes to chronic hypertension⁽¹⁻⁵⁾. The obese have: (a) a greater than normal blood volume, (b) increased stroke volume, and (c) increased cardiac output. Increased cardiac output causes hypertension. It also means increased cardiac work, and this can cause heart failure.

In the series of Kliegman and Gross, 7% of women who weighed more than 200lb (90kg) had chronic hypertension in pregnancy⁽¹⁾. Johnson et al. found an incidence of 28% in the obese, compared with 3% in the non-obese⁽²⁾. Pregnancy induced hypertension and superimposed pregnancy induced hypertension are also more common⁽²⁾.

Diabetes Mellitus

Obesity predisposes to diabetes. In the non-pregnant population, diabetes is four times more common in the obese than in the non-obese. Pregnancy is diabetogenic. Both obesity and pregnancy cause: (a) hyperinsulinaemia in subjects with intact islets of Langerhans, and (b) decreased sensitivity to insulin. High insulin blood levels cause increased endocytosis-internalisation of insulin-receptor complexes, resulting in a reduction in insulin receptors (down-regulation) in target organs, including adipose tissue. This contributes to the insulin resistance seen in obesity and pregnancy. Obesity and pregnancy together is therefore a strong predisposing risk factor for diabetes mellitus and gestational diabetes⁽²⁻⁵⁾. In the study of Johnson et al., the incidence of all types of diabetes was 10% in the obese and 2% in non-obese controls; for gestational diabetes, the respective figures were 8% and 0.7%⁽²⁾.

Macrosomia and Shoulder Dystocia

The association between obesity and macrosomia is well known. The association is independent of diabetes, although the two usually go together. Spellacy et al. found that in women who weighed more than 90kg, 33% of their infants weighed 4500-5000g at birth; and 50% of their infants weighed more than 5000g. Shoulder dystocia occurred in 7.3% of the infants weighing 4500-5000g, and in 14.6% of the infants who weighed more than 5000g⁽⁶⁾. Johnson et al.⁽²⁾ found the incidence of shoulder dystocia to be 5.1% in women weighing more than 250lb (115kg) compared with 0.6% in those weighing less than 200lb (90kg).

Difficult/Failed Intubation during Anaesthesia

Morbid obesity is a risk factor for difficult intubation. The short neck of the grossly obese is responsible for this. Movements of the chest are not easy to observe.

Syntocin Augmentation and Increased rates of Caesarean Section

Because of hypertension, diabetes, post-term pregnancy, and macrosomia, the obese have increased rates of syntocin induction and augmentation of labour and increased use of abdominal delivery⁽²⁾.

Wound Complications

Obese women are more prone to wound dehiscence and wound infection^(2,3,7) than are non-obese women.

Adipose tissue is relatively fragile, has a poor blood supply, and tends to heal slowly. Antiseptic preparation of the skin below the panniculus is difficult and usually unsatisfactory. Postoperatively, it is difficult to keep this area dry. Incisions in this area are prone to infection. Surgical operations take much longer than in the non-obese. It has been established that the risk of wound infection is positively related to the duration of the operation. Inadequate exposure in the obese may encourage excessive retraction, causing additional trauma to tissues. This will result in a higher risk of infection.

Wound dehiscence is more common. The rectus sheath in the obese is at times tenuous, and may not hold stitches well. The increased intra-abdominal pressure also predisposes to dehiscence.

Artherosclerosis, Ischaemic Heart Disease and Cerebrovascular Disease

Obesity increases the levels of serum lipids, particularly the levels of low-density lipoproteins (cholesterol) and very low-density lipoproteins (triglycerides). The hypercholesterolaemia and hypertriglyceridaemia promote atheromatous plaque formation and thrombosis.

Thromboembolism

Artherosclerosis, longer surgical operating time, restricted respiratory movements and prolonged immobilisation and venous stasis increase the risk of thromboembolism. In pregnancy and after delivery, there is an increase in clotting factors and platelet adhesiveness. This increased coagulability of the blood further raises the risk of thromboembolism.

Peripartum Heart Failure

The combination of morbid obesity and hypertension is a predisposing risk factor for peripartum heart failure⁽⁸⁾.

Obesity-Hypoventilation Syndrome (Pickwickian Syndrome)

This syndrome consists of hypoventilation, somnolence at inappropriate times, ventilation-perfusion imbalances, and carbon dioxide retention (pCO_2 48mmHg). It may be due to decreased

compliance of the chest wall and consequent increase in the work of breathing, fat in the respiratory muscles, and limited mobility of the diaphragm from increased intra-abdominal pressure.

Cholelithiasis and Cholecystitis

Pregnancy predisposes to gallstone formation. Real-time ultrasound scan investigation during the second and third trimesters has revealed increased gallbladder volume before and after eating. Incomplete emptying encourages biliary sludge and cholesterol gallstone formation. Biliary sludge was seen in 25% of postpartum women, but after a year, only 4% of the 25% still had biliary sludge⁽⁹⁾. The high progesterone levels in pregnancy may be responsible for the dilatation and incomplete emptying of the gallbladder in pregnancy. Gallbladder tissue was shown to have both nuclear and cytosolic receptors for estrogens and progesterone⁽¹⁰⁾.

Management

During Antenatal Period

1. **Weight reduction:** The patient should lose weight before embarking on pregnancy or should lose weight after delivery. Dietary manipulation to lose weight during pregnancy is not advised, because it can harm both the baby and the mother. It can result in low birth weight and can affect the health and development of the child^(11,12). However, instead of the average 10-12kg weight gain in pregnancy, the patient should be encouraged to gain not more than 6-9kg.
2. **Screening for diabetes mellitus:** Perform a fasting and a 2-hour post-cibal blood glucose measurement at booking. If normal, repeat at 28 weeks' gestation.
3. **Blood pressure measurement:** A sphygmomanometer with a cuff of the appropriate width should be used to measure the blood pressure. Standard cuffs tend to give higher readings when used in the obese⁽¹³⁾.
4. **Scan diagnosis and surveillance:** Difficulty in assessing fetal growth and determining presentation demands a more liberal use of ultrasound scan.
5. **Check for asymptomatic bacteriuria:** Use screening methods, if available, or culture of a

clean-catch midstream specimen of urine. Treatment of diagnosed cases reduces the risk of pyelonephritis and its complications⁽¹⁴⁾.

During Labour and Delivery

1. **Cardiotocography:** Because of difficulty with auscultation, CTG may be needed to detect fetal distress.
2. **Fetal weight estimation:** Estimate fetal weight at term to decide on mode of delivery.
3. **Watch for Cephalopelvic Disproportion (CPD):** Observe for CPD - rate of descent on partograph, presence of caput and moulding.
4. **Shoulder dystocia:** Be prepared and drill labour ward staff in a manoeuvre to deal with shoulder dystocia, e.g. McRobert's manoeuvre.
5. **Caesarean section:** If caesarean section is to be performed the following precautions should be taken:
 - i. **Anaesthesia:** Regional (epidural or spinal) should be preferred to general. If general anaesthesia is going to be used, the possibility of difficult or even failed intubation should be anticipated. Pre-operative assessment should warn the anaesthetist and he/she should be prepared to use alternative instruments and methods⁽¹⁵⁾. In these patients, surgery should not be started until intubation has been satisfactorily accomplished. Should intubation fail, the operation should be suspended, the patient should be ventilated by mask and cricoid pressure applied to reduce the chance of aspiration⁽¹⁶⁾. The next course of action (e.g. regional anaesthesia or local plus ketamine) should be decided while the patient is being ventilated by mask and bag.
 - ii. **Prophylactic antibiotics** should be used.
 - iii. **Heparinisation:** Low molecular weight, low dose subcutaneous heparin should be started pre-operatively and continued until the patient is fully ambulant to prevent thromboembolism.
 - iv. **Incision:** A low transverse incision is preferable to a subumbilical midline incision. The panniculus can be forcefully pulled caudad with the help of a series of Littlewood's or Lane's tissue forceps, so that the incision can be placed on the superior aspect of the panniculus, over the lower segment, instead of on the under

surface of the panniculus. The rectus sheath should be incised transversely so that, if necessary, the rectus abdominis muscles can be divided to increase exposure.

- v. **Wound closure:** Haemostasis in the subcutaneous layer should be achieved. If the wound cannot be made dry, a drain superficial to the rectus sheath should be inserted and brought out through a separate stab incision beyond one end of the wound, and a closed suction drainage instituted for 24 hours. A nylon suture, or, a polyglycolic suture material should be used. The sutures should be placed at least 1.5cm from the cut edge of the rectus sheath. Additional tension sutures should be considered. Naso-gastric suction for 24hrs to prevent abdominal distension may be useful.

During Postnatal Period

1. Watch for wound complications.
2. Watch for chest complications - atelectasis. Start physiotherapy after recovery from general anaesthesia.
3. Ambulate early and continue low dose heparin to prevent thromboembolism.
4. Check blood sugar levels of newborn for first 24 hours.
5. Encourage exclusive breastfeeding.
6. Encourage patient to lose weight.

Controversies

1. Dietary manipulation in pregnancy: Should dietary manipulation be employed to achieve weight reduction during pregnancy? The answer is no^(11,12). Dieting that stops weight gain can cause ketosis and low birth weight. The average weight gain in normal pregnancy is 10 to 12kg. The patient can be encouraged to gain no more than 6 to 9kg, but this is easier said than done!
2. Alimentary by-pass surgery: Should a morbidly obese patient who intends to become pregnant, have alimentary by-pass surgery? Stomach stapling to reduce the capacity of the stomach has not been found to lead to any complications in a subsequent pregnancy. This may be because gastric by-pass is not associated with nutritional and metabolic abnormalities⁽¹⁷⁾. Babies have been smaller than babies of untreated patients, but not to the extent of being

low birth weight. On the other hand, pregnancies after intestinal by-pass surgery have been complicated by intrauterine growth restriction⁽¹⁸⁾.

- ∩ Choice of abdominal incision for caesarean section: Should the incision be subumbilical midline, subumbilical paramedian, or low transverse? This is controversial but the low transverse as described earlier may be the best option. If longitudinal incisions are employed, a non-absorbable suture, e.g. nylon, should be used for the rectus sheath.

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Abortion

CA Klufio

Definition

Miscarriage (abortion) is termination of pregnancy before fetal **viability**. It may be spontaneous or induced.

The definition of fetal **viability** varies from country to country. WHO defines viability as 22 completed weeks from the date of onset of the last menstrual period (LMP). In Ghana, miscarriage is defined as termination of pregnancy before 28 completed weeks from the LMP, or the expulsion of a fetus weighing less than 1000g. Most countries, including the USA, define viability as 20 completed weeks, or the expulsion of a fetus weighing less than 500g.

The **leading symptom/sign** of miscarriage is a variable amount of **vaginal bleeding** (from spotting to heavy bleeding with clots) after a period of amenorrhoea of less than 28 completed weeks.

Induced Abortion

Induced abortion is defined as the termination of pregnancy by artificial means before the pregnancy has become viable. Medically (legally) induced abortion is defined as the termination of pregnancy using drugs (mifepristone *plus* misoprostol) or by surgical intervention after implantation and before the conceptus has become independently viable (22wks or 500g weight according to WHO, 28wks or 1,000g weight in Ghana).

Unsafe Abortion

WHO (1992) defines **unsafe abortion** as "a procedure for terminating an unwanted pregnancy either by persons lacking the necessary skills, or in an environment lacking the minimal medical standards, or both" (1). In other words, unsafe abortion is an induced abortion that is performed by an unskilled person and/or in an inappropriate environment and with inappropriate instruments.

Differential Diagnosis of Vaginal Bleeding in Early Pregnancy

Vaginal bleeding in early pregnancy can be due to:

- Miscarriage.

- * Ectopic pregnancy.
- * Molar pregnancy.
- * Implantation haemorrhage at the expected period.
- * Bleeding from local lesions in lower genital tract.
 - Vaginitis, particularly vaginitis caused by *Trichomonas vaginalis*.
 - Cervical polyp.
 - Cervical ectopy.
 - Cervical carcinoma.

Complications of Early Pregnancy

- * Bleeding: In variable amount (spotting to heavy bleeding occurs in 30-40% of all pregnancies before 20 weeks' gestation. About 50% of pregnancies who have a bleed will abort (2).
- * Miscarriage: 1 in 8 of all pregnancies will abort.
- * Abdominal pain.
- * Ectopic pregnancy: Occurs in 1% of pregnancies.
- * Hyperemesis gravidarum.
- * Hydatidiform mole.

Significance (complications) Health problems

Introduction: Miscarriage is the most common complication of pregnancy. Miscarriage, especially an unsafe induced abortion, is a very common cause of maternal death in the developing world, including Ghana, as shown in Tables 1 and 2^(3,4).

Table 1. 724 Maternal Deaths in KBTH 1984 1994*

Direct Obstetric Causes	No. (% of all 724 deaths)
Haemorrhage	128 (17.7)
Hypertensive disorders	127 (17.5)
Abortions	98 (13.5)
Genital infections	69 (9.5)
Obstructed labour/Ruptured uterus	40 (5.5)
Indirect Obstetric Causes	262
Total	724 (100)
Reference 3.	

Table 2. Causes of Maternal Mortality All Over The World

A. Direct Obstetric Causes	
Postpartum haemorrhage	25%
Puerperal sepsis	15%
Unsafe abortion	13%
Hypertensive disorders of pregnancy, especially preeclampsia & eclampsia	12%
Obstructed labour	8%
Other direct obstetric courses (including ectopic, mole, embolism)	8%
<i>Total</i>	81%
B. Indirect Obstetric Causes	
Examples: anaemia, sickle cell disease, malaria, et	19%
Reference 4.	

Classification of Causes of Miscarriage

Broadly, the causes are:

- * Chromosomal/genetic.
- * Maternal/environmental.
- * Idiopathic.

Miscarriage may occur during the:

- * Pre-embryo stage (6wks).
- * Embryo (organogenesis) stage (6-8wks).
- * Fetal (maturation) stage (8wks).

Clinically, miscarriage is categorised as *early* (first trimester) or *late* (second trimester).

Causes of Miscarriage

More than 80% of miscarriages occur in the first trimester; the majority occur in the embryonic period (6-8wks).

Causes of early (first trimester) miscarriage

1. Idiopathic.
2. Chromosomal:
 - * When tested by culture of amniotic fluid cells, 50% of early miscarriages have normal karyotype, i.e. they are euploid, 46XX or 46XY and 50% have abnormal karyotype; but when tested by direct cytogenetic analysis of tissue from chorionic villus biopsy, 85% of early miscarriages are abnormal.
 - 25% of all fetal loss is caused by errors of maternal gametogenesis, 5% by errors of paternal gametogenesis, 5% by errors of fertilization and 5% by errors of zygote division. Among couples with a history of two or more miscarriages, 95% are chromosomally normal.
 - * The types of chromosomal abnormalities commonly found in early abortuses are:
 - a) Aneuploidy: 95% of abnormal karyotypes are abnormalities in chromosome number. 75% occur before 8wks.
 - * Autosomal trisomies: 50% - 65% of abnormal karyotypes) are autosomal trisomies, commonly 16, followed by 22, and less commonly, 13, 18, 21. They arise from non-dysjunction during meiosis, and less commonly from balanced translocation, or chromosomal inversion. Monosomy Turner 45XO as a result of non-dysjunction is found in 7-15% of early abortuses.
 - * Triploidy from dispermy or digyny during fertilization (15%): This is often associated with

hydropic degeneration of the placenta, or incomplete (partial) mole.

- * Tetraploidy and mosaicism from errors during first division of fertilized ovum.
 - b) Structural abnormalities of individual chromosomes (5% of abnormal Karyotypes).
 - c) Parental chromosome abnormalities are rare. Most are translocations.
- 3. Antiphospholipid syndrome:
 - * Lupus anticoagulant.
 - * Anticardiolipin antibody.
- 4. Smoking and alcohol are embryotoxins: Smoking and alcohol independently increase the incidence of euploid miscarriages. The effect of each is dose-dependent. Women who smoke 15 cigarettes per day have 1.7 times the risk of non smokers. Women who drink alcohol 2 days a week have 2 times the risk of non-drinkers.
- 5. Lower genital tract infections:
 - Bacterial vaginosis.
 - * Endometrial colonisation with T-strain mycoplasmas:
 - * Ureaplasma urealyticum.
 - * Mycoplasma hominis.
- 6. Insufficiently prepared endometrium:
 - Luteal phase defect before conception.
 - * Oligomenorrhoea.
 - hCG-dependent progesterone deficiency after conception.
- 7. Pyrexial illnesses, e.g. malaria and typhoid. Any severe maternal infection, which leads to bacteraemia or viraemia can cause sporadic miscarriage because of the high temperature or because of the bacteraemia/viraemia.
- 8. LH hypersecretion during follicular phase: LH levels 10IU/L on Day 8 are associated with increased risk of miscarriage and of infertility. The mechanism may be premature aging of the oocyte or a direct effect on the endometrium that adversely affects implantation. Since LH secretion is pulsatile, and since LH has a half-life of only 30min, urinary assays may reflect LH activity more accurately than an isolated blood sample assay.
- 9. Genetic (HLA-sharing): Chromosomally (karyotypically) normal (euploid) abortuses

The **complications** of miscarriage include:

- † Maternal death.
- † Severe haemorrhage.
- † Pelvic infection.
- † Tubal infertility.
- † Ectopic pregnancy as a result of tubal damage from tubal infection.
- † Intrauterine synechiae as result of infection or secondary to curettage (Asherman syndrome = intrauterine synechiae + amenorrhoea/oligomenorrhoea + infertility).
- † Psychological/Psychiatric/Emotional problems (psychological sequelae). Miscarriage, whether spontaneous or induced can have a negative psychological impact on patients, their partners and families:
 - Anxiety.
 - Depression.
 - Grief.

- A sense of loss, failure, and insecurity about the outcome of future pregnancies.
- A sense of shame and guilt, particularly in induced abortion.

Rh-immunisation if patient is Rhesus negative and baby Rhesus positive. Always check rhesus status and give rhesus (D) immunoprophylaxis to rhesus negative women who are not already immunised to prevent sensitisation (5).

- Anti-D immunoglobulin by deep intramuscular injection within 72hrs of onset of bleeding: 250 units (50mcg) if pregnancy is 20wks, 500 units (100mcg) if pregnancy is 20wks.

- threatened miscarriage, if pregnancy continues, the outcome of the pregnancy may not be optimal (6):

- Preterm labour.
- Low birth weight.
- Perinatal deaths.
- Very slight increase in the rate of fetal abnormalities.
- Clinically and psychologically challenging for attending physician: emotional stress.

Socio-economic problems

- Admission to hospital and disruption of family life and work.
- Makes high demands on hospital resources; takes up a large number of hospital beds.

Incidence of spontaneous miscarriage

Pre-clinical embryo loss: Using -hCG measurements to detect pregnancy during the

luteal phase (chemical pregnancy), it has been found that 23% of fertilised ova are lost before the time of the next expected menstruation (7,8).

- * Clinical pregnancy loss: Of all conceptions that reach the stage when their presence can be clinically diagnosed, 10% -20% miscarry; i.e. about 15% of all pregnancies end in clinically recognised miscarriage (9).
- * Therefore, about 40% of all conceptions end in miscarriage after implantation.

Independent Risk Factors for Miscarriage

- I. Reproductive history:
 1. Increasing parity.
 2. Number of previous miscarriages.
 3. Outcome of last pregnancy: Rate is higher after a miscarriage or a stillbirth than after a live birth.
- II. Maternal age: The frequency of clinical miscarriage increases with increasing maternal age. The risk increases substantially after 35yrs. It increases from 12% at 20yrs to 26% at 40yrs. When maternal age is 35yrs, the subsequent miscarriage rate after transvaginal real time sonographic visualisation of fetal heart activity is 2 to 3 times what is found in younger women; when fetal heart activity (FHA) is visualised at 6wks the subsequent miscarriage rate is 21% in 35yr olds and 7% in 35yrs olds; when FHA is visualised at 8wks, the figures are 6% and 3%, respectively. The increase is directly related to the higher incidence of trisomies, mainly trisomies in the D and G groups of chromosomes, especially chromosome 21. Older women have a higher incidence of non-dysjunction, and less commonly, balanced translocation and chromosomal inversion than younger women. This is the reason for the higher incidence of trisomy in older women. This is why chorionic villus biopsy and/or amniocentesis for karyotyping is mandatory in women 35yrs
- III. Paternal age: The rate increases with paternal age.
- IV. Oligomenorrhoea.
- V. Live birth-pregnancy interval 3 months.

may be genetically abnormal because of gene mutations or polygenic factors. Compared with controls, significantly more recurring miscarriage couples share the same histocompatibility locus antigens (HLA) sharing at the A, B, and DR loci of chromosome 6. This segment of chromosome 6 may also carry recessive genes that are potentially lethal. Because they are on the same segment, sharing of the HLA genes would mean sharing of these lethal recessive genes. This may be the cause of the recurring miscarriage.

10. Immunological (alloimmune) factors: It has been hypothesised that maternal blocking antibody (IgG) coats foreign fetal antigens and thereby protects the fetal allograft from being rejected by the mother. Because of HLA-sharing and the similarity in genetic composition between the fetus and the mother, there is an inadequate stimulus for the mother to produce blocking antibodies to protect the fetus from rejection. It has been hypothesised that some women may miscarry because of this; they may suffer a so-called "partner-specific miscarriage". Based on this hypothesis, paternal leukocyte immunotherapy, i.e. infusion of paternal white cells into the woman, was practised. A European randomised controlled clinical trial reported no benefit from such therapy. The hypothesis, though attractive is no longer tenable.
11. Hostile endometrium: Endometrium containing a hostile leukocyte population.

Causes of late (2nd trimester) miscarriage

1. Idiopathic.
2. Antiphospholipid syndrome.
3. Lower genital tract infections.
4. Uterine anomalies: They cause miscarriage because of limited space for the developing conceptus to grow, or because of non-compliance of uterine walls so that expansion is limited, or because the blastocyst implants on a poorly vascularised area.
 - * Congenital anomalies:
 - Bicornuate: Its presence is associated with 25% incidence of miscarriage).
 - Severe septate: Its presence is associated with 25% incidence of miscarriage).
 - Severe septate: Its presence is associated with 25% incidence of miscarriage).
 - Unicornuate with rudimentary horn: This is the least common of the congenital

anomalies, but it is associated with the greatest incidence of miscarriage (50%).

- * Acquired anomalies:
 - Submucous leiomyomata.
 - Intrauterine synechiae (adhesions).
 - Adenomyosis.
- 5. Cervical incompetence:
 - * Congenital.
 - * Acquired.
- 6. Endocrine factors:
 - * Hypothyroidism.
 - * Presence of anti-thyroid antibodies:
 - Thyroglobulin antibody.
 - Thyroid peroxidase antibody.
 - * Uncontrolled diabetes mellitus: the higher the glycosylated haemoglobin (HbA_{1c}) level, the higher the miscarriage rate.
- 7. Thrombophilias: These are conditions in which deficiencies and defects in the quality of the natural inhibitors of coagulation result in procoagulant (hypercoagulability) states that predispose to thrombo-embolism, recurring early pregnancy loss, late intrauterine death and stillbirth. The natural inhibitors are Antithrombin III, Protein C, and Protein S.
 - * Protein C deficiency: The abnormality may not be a deficiency but a defect in the quality of the inhibitor.
 - * Activated protein C resistance (APCR): The vast majority (95%) of APCR are congenital, and are due to a mutation in the Factor V (Leiden) gene that results in deficiency of Factor V. Factor V Leiden is the most common familial thrombophilic condition.
 - * Protein S deficiency.
 - * Hyperhomocysteinaemia.

Clinical Categories of Miscarriage

Bleeding after a period of amenorrhoea is the cardinal sign/symptom of miscarriage. Depending on the type of miscarriage, the bleeding may vary in amount, from mere spotting/staining to life-threatening haemorrhage.

1. **Threatened:** The pregnancy is showing signs (threatening) that it may terminate. The leading sign is vaginal bleeding. The pregnancy may terminate, or the bleeding settles and the pregnancy continues, or the pregnancy dies but is not expelled (delayed miscarriage).
2. **Inevitable:** Although products of conception have not as yet been expelled, there are signs that the pregnancy will terminate. Rupture of

the membranes with loss of amniotic fluid is one of the signs that the miscarriage is inevitable.

3. **Incomplete:** Parts of the products of conception have been expelled, but some have been retained.
4. **Complete:** All of the products of conception have been expelled. The uterus is empty.
5. **Missed (delayed) miscarriage:** The pregnancy died many days (14 days) ago. No products have been passed.
6. **Blighted ovum (anembryonic gestation):** See below for the differential diagnosis of the types of abortion.

Miscarriage is usually complete if it occurs before 8 weeks or after 14 weeks. From 8 to 14 weeks gestation, part of the conceptus, usually the placenta, is retained. In other words, from 8 to 14 weeks, the miscarriage is usually incomplete.

Table 3. Clinical Categories of Miscarriage and Their Clinical Distinguishing Features

Table 3. Clinical Categories of Miscarriage and Their Clinical Distinguishing Features					
	Vaginal Bleeding	Abdominal Pain	Cervical os Dilated?	POC passed?	Uterine size versus dates
	<i>Slight, moderate,</i>	<i>Nil, mild, moderate</i>	Yes, No	Yes, No	<i>Equal, smaller, larger</i>
Threatened	Slight	Nil/mild	No	No	Equal
Inevitable	Severe	Severe	Yes	No	Equal
Incomplete	Severe	Severe	Yes	Yes	Smaller
Complete	Nil/slight	Nil/mild	No	Yes	Smaller
Delayed	Mild	Nil/mild	No	No	Smaller

= Rupture of membranes is a sign of inevitable miscarriage
 = POC=Products of conception

Investigations In Miscarriage

The sheet anchor in the investigation of uterine bleeding in pregnancy consists of the following package of tests (10): Hb, sickling: Hb electrophoresis, if sickling is positive.

- * Blood group and rhesus status.
- * Qualitative pregnancy test on urine.
- * Quantitative serum -hCG measurement.
- * Quantitative serum progesterone measurement.
- * Ultrasound scan: In the 1st trimester, the high-frequency (7.5MHz) transvaginal probe is far superior to the abdominal probe, 3.5-4.5MHz (Table 4).
- * Transvaginal endosonography in 1st trimester.
- * Abdominal ultrasound after 1st trimester.
 - * Real-time ultrasound for fetal heart activity.

Note: Ultrasonographic Scan Examination should provide Information on the following (11):

- * Gestation sac: Number, size, and quality (e.g. regularity of outline).

- Yolk sac: Presence or absence.
- Fetus:
- Number.
 - * Size (CRL).
 - * Fetal heart activity.
 - * Intra - uterine haematomata.
- Adnexal lesions.
- Peritoneal fluid.

When the mean sac diameter is 10mm, a fetal pole (the embryo) is always demonstrable. When the mean sac diameter is 20mm, a fetal pole with a CRL of 6cm and fetal heart activity are always demonstrable.

Since the LMP, by itself, cannot be relied upon, we use the serum -hCG level to determine how we should interpret the ultrasonographic findings. If the serum -hCG level in mIU/mL is:

1,000, a gestational sac with a MSD of 3mm should be visualised

1,500, a gestational sac with a MSD of 20mm, a fetal pole 5mm, and fetal heart activity (FHA) should be visualised

Table 4. Temporal Values for GA, Serum β -hCG Levels, and Transvaginal endosonography Findings in Normal Pregnancy

Days from LMP	β -hCG mIU/mL	Transvaginal ultrasonography		
		MSD	Fetal pole (CRL)	Fetal heart activity by real-time ultrasound
	<1,000	Not visualised		
33	1,000-1,500	3mm	Not visualised	Absent
34-38	>1,500	>10mm* Visualised	Not visualised	Absent
39-43	\geq 3,000	>>20mm	>>5mm	Always Demonstrable

MSD=Mean sac diameter = The mean of 3 perpendicular diameters

CRL=Crown rump length

*Yolk sac visualised

Serum β -hCG levels increase by at least 65% every 48hrs

If MSD 20mm and there is no fetal pole, i.e. an empty sac, an embryonic gestation is present.

Blighted ovum (anembryonic gestation)

This is defined as ultrasonic visualisation of a gestational sac 20mm in mean diameter without the presence of a fetal pole.

Note: A fetal pole of 5mm and fetal heart activity should be visualised when **one** of the following is present:

- * The gestational sac is 20mm in mean diameter.
- * The pregnancy is 43 days old from the LMP.
- * The serum β -hCG is 1,500mIU/mL.

Threatened Miscarriage

Some bleeding occurs in about 30% of all pregnancies during the first 20wks, and 50% of these go on to abort spontaneously.

Diagnosis

Diagnosis is based on the clinical features and investigations. The features in **Table 4** are used to clinically diagnose threatened miscarriage.

- Vaginal bleeding only slight to moderate, i.e. not severe or heavy.
- No pain or very little lower abdominal pains (uterine cramps).
- No products of conception (tissue) passed.
- No cervical dilatation.
- Uterine size agrees with dates.
- Membranes are not ruptured.

Prognosis

The patient and/or her partner may ask what the chances are that the pregnancy will go to term, or that the pregnancy will result in the birth of a live normal baby.

In two thirds of threatened miscarriage, the embryo/fetus is alive, and about 85% of these will survive, and the remaining 15% will abort. Of the third without a live embryo/fetus, 50% will have blighted ovum, 25% will be embryonic death, and 25% will be incomplete miscarriage (2).

When **fetal heart activity (FHA)** is visualised by transvaginal real time ultrasonography at 6wks, the subsequent miscarriage rate is 7%. When FHA is visualised at 8-12 weeks, the subsequent miscarriage rate is 3%. The risk of subsequent miscarriage after visualisation of FHA in the 1st trimester is 5%, i.e. $\{(7+3)/2\}$ %. If bleeding occurs when FHA is present in the 1st trimester, the risk of miscarriage increases 3-fold, from 5% without bleeding to 16% with bleeding (2).

- * If the **fetal heart rate** in the 1st trimester is less than 100 beats/min (fetal bradycardia), or higher than 120 beats/min, the risk of miscarriage rises (12-14).
- * The **duration of bleeding** also has a prognostic influence on the outcome of the pregnancy. The risk of miscarriage is 7% in those who bleed for 3days, and 24% in those who bleed for 3days (2).
- * In the presence of bleeding, an elevated **CA-125 levels** increase the risk that the pregnancy will end in miscarriage
- * In the general population, if the difference between the mean sac diameter (MSD) and the crown rump length (CRL) is 8mm, (evidence of early **polyhydramnios**), the subsequent miscarriage rate is 5%; if the difference is 5mm (early **oligohydramnios**), the subsequent abortion rate is 80% (15,16).

Bleeding in Early Pregnancy: Treatment According to Ultrasonographic Finding

The **possible diagnoses** are (10):

1. Fetal heart activity (FHA) present in uterus: Viable intra-uterine pregnancy.
2. Fetal pole, no cardiac activity: There are 2 possibilities:
 - a. Fetus viable but too early to show FHA.
 - b. Early fetal death.
3. Empty gestation sac with MSD 20mm: There are 2 possibilities:
 - a. Fetus viable but too early to show FHA.
 - b. Blighted ovum if repeat scan at 7 days shows that MSD is still 20mm.
4. Empty sac with MSD 20mm: Blighted ovum.
5. No gestation intrauterine sac: Probable retained products of conception.
6. Empty uterus, no gestation sac: There are 3 possibilities:
 - a. Very early viable pregnancy.
 - b. Complete miscarriage.
 - c. Ectopic pregnancy.

7. Suspected hydatidiform mole:
 - a. Complete mole.
 - b. Incomplete (partial) mole with a non-viable pregnancy
 - c. Incomplete (partial) mole with viable pregnancy.

Viable Intra-uterine Pregnancy

Diagnosis

This diagnosis is made when there is an **intra-uterine sac with clearly identified fetal heart activity**. At least 85% will continue. Fetal heart activity is always demonstrable by real-time ultrasound when the mean sac diameter (MSD) is 20mm, or the gestational age by the LMP is 8wks. **Note:** Because we can never be 100% certain that the LMP is correct, when the MSD is 20mm we cannot say that the fetus is not viable. We must wait and repeat the scan in **not less than 7 days**.

Treatment

- * A gentle speculum examination should be performed to ascertain that the bleeding is coming from inside the uterus and to exclude local lesions.
- * The couple should be advised to abstain from sexual intercourse until the bleeding stops. This is to reduce the risk of infection and the initiation of uterine contractions by prostaglandins in seminal fluid.
- * If the patient is rhesus negative and not already sensitised, and if the pregnancy is 12wks, anti-D immunoglobulin immunoprophylaxis against rhesus sensitisation must be given, 500IU (100mcg) (5).
- * No other intervention is necessary. Bed rest makes no difference to outcome. Synthetic progestins do more harm than good; they can cause delayed miscarriage and they can cause virilisation of the female fetus.

Fetal Pole Present but Fetal Heart Activity Absent

Diagnosis

There are two possibilities:

1. Fetus viable but too early to show FHA: This is the diagnosis if all of the following 3 findings are present.

- i. A normally situated (intra-uterine) gestation sac with a MSD of 20mm present

- ii. A fetal pole with a CRL 6mm present
- iii. FHA absent.

2. Early fetal death: This is the diagnosis if all of the following 3 findings are present.

- i. A normally situated (intra-uterine) gestation sac with a MSD of 20mm present
- ii. A fetal pole with a CRL 6mm present
- iii. FHA absent.

This is missed abortion, now preferably called "delayed miscarriage" or "silent miscarriage".

Fetus viable but too early to show FHA

Treatment

Patient can go home and return in **not less than 7 days** for a repeat scan. In normal pregnancy, in the 1st trimester, the gestation sac increases by 1.1mm per day. If the repeat scan shows no FHA and there is no increase in MSD or CRL, a diagnosis of delayed miscarriage is made and the patient treated as such.

If the patient is rhesus negative, anti-D immunoglobulin immunoprophylaxis against rhesus sensitisation is given.

Delayed (silent) miscarriage

Treatment

The uterine cavity is evacuated medically or surgically after haematological (Hb level, Blood Group and Rhesus status) and other indicated investigations have been performed. If the fetus has been dead for more than 5wks, there is a theoretical risk of disseminated intravascular coagulation and a clotting defect. However because of the small size of the conceptus, the risk is remote. Delayed miscarriage is discussed more fully below.

Empty gestation sac: MSD 20mm

If a normally situated **true** gestation sac (not a pseudosac) is present, but there is no fetal pole (fetal tissue) and the MSD is 20mm, the pregnancy may be viable or not viable. Repeat scan in **not less than 7 days**. If the MSD is still 20mm, the fetus is not viable, and the diagnosis is blighted ovum.

Treatment

Same as for delayed miscarriage.

Empty gestation sac: MSD 20mm

The diagnosis is "blighted ovum".

Treatment

Same as for delayed miscarriage.

Retained products of conception (POC)

The uterus contains tissue of mixed echogenicity, and there is no gestation sac. Usually, the tissue is retained POC. This is incomplete miscarriage. However, a retained blood clot may look the same.

Treatment

Treatment depends on the amount of tissue present, and the severity of vaginal bleeding. Medical evacuation and "expectant management" are effective in selected cases of incomplete miscarriage (17,18). In properly selected cases, expectant management can be as effective as medical treatment (19, 20). The drug used in medical evacuation is misoprostol. Misoprostol is cheap, easily stored because it is temperature-insensitive, effective PG-analogue, which is active orally and vaginally. The vaginal route is more effective⁽¹⁸⁾.

Expectant (conservative) management: When bleeding is light, the patient is stable, and the amount of POC is small, i.e. maximum diameter of tissue is 30mm.

Medical or surgical evacuation: When bleeding is slight, the patient is stable, and the amount of POC is large, i.e. 30mm, medical evacuation with misoprostol may be used (see later under treatment of delayed miscarriage).

Surgical evacuation of the uterus: When bleeding is heavy and/or amount of tissue is large. Surgical evacuation provides the quickest way of emptying the uterus and stopping the bleeding. Suction curettage for incomplete or delayed abortion is easier and safer than sharp/blunt curettage⁽²¹⁾.

Advantages of non-surgical methods of treatment

The non-surgical methods have the following advantages over surgical methods:

- The risk of clinical pelvic infection is less (17,22,23). There are no adverse effects on future fertility after expectant treatment (23).
- There is no risk of uterine perforation or cervical damage.
- General anaesthesia, regional block, or a local is not required.
- There is no risk of subsequent iatrogenic intra-uterine synechiae.

In a randomised controlled trial no difference in psychological morbidity was found between those treated expectantly and those treated surgically (24).

Empty uterus with urine pregnancy test *truly* positive

The uterus contains no tissue, or shows decidual reaction (thickened decidua) only.

The possibilities are:

- * Very early pregnancy.
- * Complete miscarriage.
- * Ectopic pregnancy.

The aim is to:

- * Avoid unwittingly evacuating an early pregnancy.
- * Avoid doing a laparoscopy and inserting sounds and manipulators into the uterine cavity when an intra-uterine pregnancy is present.
- * Avoid failure to diagnose an ectopic pregnancy.

The differential diagnosis is based upon:

- * Diligently taken history: risk factors for ectopic (previous history of PID or ectopic, infertility, assisted fertility methods) pain, syncope attacks.
- * Diligent physical examination for signs of chronic or acute intraperitoneal blood loss anaemia, abdominal tenderness, restricted abdominal breathing, abdominal distension, signs of free fluid, pelvic haematocele.
- * Serum -hCG levels:

- An intrauterine pregnancy is always seen with transvaginal ultrasonography if the level is 1,000mIU/mL. Below this level, an intrauterine pregnancy may not be seen because the pregnancy is too early to be seen. When the level is above 1,000mIU/mL and a gestation sac is not seen with transvaginal ultrasonography, it can be categorically concluded that intrauterine pregnancy is not present.

The diagnosis is either:

- Ectopic pregnancy, or,
- Complete abortion.

Laparoscopy is indicated to exclude ectopic pregnancy.

- * If the level is 1,000mIU/mL, and there is no gestation sac, the diagnosis could be ectopic pregnancy, intrauterine pregnancy, or a complete miscarriage. If the patient is stable, she can be watched and the scan and -hCG repeated after 48hrs. If by 48hrs the -

hCG has fallen to 20% of its original value, complete abortion is diagnosed.

- * If the level has fallen more slowly, or has risen, ectopic pregnancy is diagnosed, and laparoscopy performed (10).

Suspected hydatidiform mole

May be a complete mole, or, a partial mole (with gestation sac). A complete mole has classical features on ultrasound scan and cannot be missed. A partial mole can be more difficult to diagnose. Microscopy of all tissue passed or evacuated is needed to confirm either diagnosis.

Treatment

- a. Complete mole.
 - * Preoperative work-up: If uterine size 14wks perform haematological and RFT investigations, Cross-match blood, look for PIH and thyrotoxicosis; measure serum -hCG level to prognosticate risk of future malignant outcome.
 - * Evacuation with a suction curette with syntocinon drip running to minimise risk of haemorrhage. Medical induction of evacuation with misoprostol or with syntocin should not be used because they may increase the risk of deportation and malignant change.
 - * All tissue removed should be sent for histopathological examination
- b. Partial mole with non-viable pregnancy.
 - * There may be no fetal heart activity because the pregnancy is too early or because the fetus is dead, the pregnancy should be evacuated with a suction curette as described for complete mole.
- c. Partial mole with a viable fetus.
 - * The risk that the fetus has triploidy is high. Before the pregnancy is allowed to proceed, the patient and her partner should be counselled and chorionic villus biopsy or amniocentesis and other necessary investigations should be performed.

NB:

1. Any tissue obtained at the time of miscarriage, either spontaneously expelled or medically or surgically evacuated, should be examined histologically to confirm pregnancy and to exclude ectopic pregnancy or gestational trophoblastic disease.

2. When miscarriage occurs in the presence of *B. vaginosis* or endocervical infection with *C. trachomatis* or *N. gonorrhoeae*, the risk of endometritis and salpingitis is greatly increased. The routine use of antibiotic prophylaxis substantially reduces the incidence of pelvic inflammatory disease.
3. The risk of PID and its sequelae is much higher in unsafe abortion.
4. In the rhesus negative woman, with a rhesus positive pregnancy, miscarriage is a sensitising event.

Treatment of Inevitable or Incomplete Miscarriage

Miscarriage is usually complete if it occurs before 5 weeks or after 14 weeks. Between 8 and 14 weeks part of the conceptus, usually the placenta is retained. In other words, between 6 and 14 weeks the miscarriage is usually incomplete.

Management

- I. Quickly assess the patient for:
 - A. Incipient or overt shock and anaemia
 - Pallor
 - Assess vital signs: tachypnoea, tachycardia, hypotension
 - Thenar eminence blanching test

If the patient is bleeding profusely and products of conception can be seen or felt wedged in cervical canal, use ovum or sponge-holding forceps to gently remove them. This will reduce the amount of bleeding
- B. Intrauterine infection/septicaemia
 - Pyrexia
 - Tachypnoea
 - Tachycardia
 - Offensive vaginal discharge
 - Suprapubic tenderness
 - Cervical excitation (motion) pain
- C. Possible uterine perforation/generalised peritonitis
- II. Venepuncture with a large bore (18-gauge) canula or needle. Take blood for:
 - A. FBC, sickling; Hb electrophoresis if sickling is positive
 - B. BUE and creatinine
 - C. Blood Group and Rhesus type and for antibodies
 - D. Cross matching 2 units of blood if patient is anaemic or if blood volume low
- III. Set up an intravenous line
- IV. Correct hypovolaemia if present:

- Start with normal saline or Ringers lactate
- ≡ Transfuse blood when blood is ready
- Observe urinary output if hypovolaemic or if bleeding is profuse
 - In-dwelling catheter and record hourly urinary output. Should be 20ml/hr
- Assess gestational age, using
 - LMP
 - Uterine size
 - Examination of fetus if it has been expelled
 - Ultrasound scan if fetus in-utero
- Prepare for uterine evacuation by manual vacuum aspiration (MVA) under analgesia/sedation with IM 50-100mg pethidine plus diazepam 5mg and paracervical block with 10ml of 1% Xylocaine
- Give prophylactic antibiotics to cover *N. gonorrhoeae*, *C. trachomatis*, and Bacterial vaginosis
- If patient is Rhesus D negative and is not already sensitised, give anti-D immunoglobulin 50-100g IM for prophylaxis; dose according to gestational age

Septic Miscarriage

Definition

This is miscarriage complicated by uterine infection. The infection starts in the products of conception (POC) and spreads to the endometrium (endometritis), the myometrium (metritis), parametrium (parametritis) and peritoneum (peritonitis). To start with, the peritonitis is confined to the pelvis. Later, the infection spreads to involve the abdominal peritoneum as well (generalised peritonitis).

Septic abortion can complicate any type of miscarriage: threatened, inevitable, incomplete, complete, delayed (silent) miscarriage (missed abortion). Septic abortion is particularly likely to occur:

- * If pathogenic organisms were present in the lower genital tract (vagina or endocervix) at the time of the miscarriage: *Chlamydia trachomatis*, *Neisseria gonorrhoea* or Bacterial vaginosis.
- * In unsafely induced abortion.

Significance

- It is a very important cause of maternal mortality (see Tables).
- Can cause tubal damage that, in turn, can cause:

- Ectopic pregnancy.
- Tubal infertility.
- * Recurrent PID and chronic pelvic pain.

Clinical features

These are the features of intrauterine infection and peritonitis.

- * Lassitude.
- * Anorexia/nausea/vomiting.
- * Fever (37.8°C): Endotoxic shock can cause hypothermia.
- * Tachycardia.
- * Lower abdominal pains.
- * Suprapubic tenderness.
- * Maldorour bloodstained discharge from cervix.
- * Pelvic tenderness.
- * Bilateral cervical (motion) excitation pain.
- * Cervix open, if not complete miscarriage.
- * Pelvic masses may be present.
- * Signs of trauma to cervix and/or vagina may be present if the miscarriage was unsafely induced with instruments.

Additional features if generalised peritonitis is present.

- * Tachypnoea with diminished abdominal excursions during breathing
 - Abdominal distension.
 - Abdominal rigidity.
 - Generalised abdominal tenderness and rebound tenderness.
 - Diminished or absent bowel sounds.

Complications Haemolysis, especially in *Clostridia welchii* infections:

- Jaundice
- Anaemia.
- * Liver dysfunction/failure:
 - Jaundice.
 - Bleeding diathesis from a fall in clotting factors produced by liver.
- * Renal failure: oliguria.
- * Septicaemia.
- * DIC
- * Septic pelvic thrombophlebitis.
- * Septic arthritis.
- * Pelvic abscess:
 - Tubo-ovarian abscess.
 - Pouch of Douglas abscess.
- * Septic emboli resulting in abscesses in different organs.
- * Septic shock.
- * Adult respiratory distress syndrome.
- * Traumatic injury to genital tract (uterine perforation, lacerations of cervix and vagina) if miscarriage was unsafely induced.

- * Other unsafe methods of induction have their own complications. For example, insertion of chemicals into vagina and uterine cavity.

Investigations

- * HVS, endocervical, and intra-uterine swabs for:
 - Gram stain.
 - Culture and sensitivity.
 - FBC and sickling test; Hb electrophoresis if sickling positive.
 - ESR.
 - Blood Group and Rhesus type.
- * Blood culture.
- * Urine RE.
- * MSU for culture and sensitivity.
- * BUE and creatinine.
- * Coagulation studies.
- * Chest X-ray.
- * Erect abdomino-pelvic X-ray for:
 - Free gas under diaphragm in cases of uterine/intestinal perforation.
 - Gas bubbles in uterine cavity in cases of *Clostridia welchii* (Gram positive rods) infection.
 - Residual foreign body in uterus.
- * Ultrasound scan of uterus and pelvis for:
 - Retained POC.
 - Pelvic masses, including abscesses.
 - Free fluid, which might be blood or pus.

Treatment

Antibiotics

Intravenous, broad-spectrum bactericidal antibiotics to cover gram positive and gram negative organisms and aerobic and anaerobic organisms.

Ampicillin; crystalline penicillin if *Clostridia* suspected

- * Metronidazole.
- * Gentamycin.
- * Clindamycin.

A 3rd generation cephalosporin, e.g. ceftriaxone, plus clindamycin may be used.

Intravenous Fluids

Clinical indications will determine the type and volume of fluid that should be given.

Blood transfusion, if indicated

If patient is anaemic or is haemolysing rapidly, blood transfusion is given.

Evacuation of POC

Emptying the uterus of infected necrotic products

as early as possible is necessary for quick recovery and prevention of complications.

Two units of blood are cross-matched and the patient is prepared for general anaesthesia and for laparotomy.

Loads of bacteria can enter the circulation during evacuation. This can cause septicaemia and septic shock. To avoid this, it is important that the plasma levels of antibiotics should be high. When given intravenously, antibiotics reach peak levels in an hour. The patient is taken to theatre 1-2hrs after giving the first dose of intravenous antibiotics.

Examination under anaesthesia is first performed, looking for signs of trauma to the vagina and cervix and for pelvic masses. The uterine sound is used to probe for uterine perforation.

Vacuum aspiration is used to evacuate the POC. The products are sent for microbiological examination and for histopathology.

Laparotomy

is performed if:

There is evidence of free fluid (pus or blood) in the abdomen: shifting dullness and pus or blood on paracentesis.

- * Ectopic pregnancy cannot be excluded.
- * Uterine perforation/laceration diagnosed or suspected.
- * Patient not responding satisfactorily to conservative management.
- * Abdomino-pelvic masses are present and they are not resolving (getting smaller) with antibiotic treatment.

Abdominal hysterectomy

is performed if:

- * Uterine perforation/laceration cannot be repaired.
- * Clostridial infection diagnosed or suspected. e.g. if haemolysis and jaundice present
- * At laparotomy, uterus is showing signs of gangrene or necrosis.
- * Laparotomy, drainage and peritoneal toiletting did not produce an improvement in patient's condition.

Rhesus D immuno-prophylaxis

Given within 48hrs if patient is Rhesus D negative and is not already sensitised.

Monitoring Response to Conservative Treatment

Watch to assess if patient improving on current treatment. Observe carefully for incipient shock.

- Impression of general health: appearance, posture in bed, appetite, nausea/vomiting, tongue-furring/coating.
- Vital signs:
 - Respiratory rate.
 - Pulse rate.
 - Temperature.
 - Urinary output.
- Abdominal examination:
 - Abdominal excursions with breathing.
 - Distension.
 - Rigidity.
 - Tenderness and rebound tenderness.
 - Bowel sounds.
- Laboratory investigations:
 - Serial Hb and WBC.
 - Serial BUE and creatinine.
- Others.

Management of Septic Shock

- Central venous pressure monitoring.
- Intravenous fluids to maintain blood pressure and adequate kidney perfusion.
- Intranasal oxygen or oxygen by facemask.
- Vassopressor agents.
- Digitalis.
- Corticosteroids.

Follow-up

- Ultrasound scan for retained POC.

Delayed (Silent) Miscarriage (Missed Abortion)

Definition and aetiology

Delayed or silent miscarriage, (previously called "missed abortion"), is diagnosed when fetal death in utero occurs before the age of viability, (i.e. at 23wks' gestation in Ghana) and the pregnancy is retained for a prolonged period after the fetal death. In normal pregnancy, placental progesterone prevents uterine contractions and expulsion of pregnancy. In silent miscarriage, plasma progesterone levels are low, but the concentration of progesterone may be high enough at the placental site to inhibit uterine contractions. This may be why expulsion of the fetus is delayed. In threatened abortion, the administration of exogenous progestins, in the mistaken belief that

this will save the pregnancy, is a well-known cause of silent miscarriage.

Diagnosis

Diagnosis is made from the clinical features, ultrasound scan, and -hCG measurements.

Clinical features

- * Bleeding/spotting per vaginam: Bleeding may or may not occur. When it occurs, it is usually only slight. It may be continuous or intermittent.
- * Continuous brown vaginal discharge after cessation of bleeding, or, in between intermittent bleeding/spotting periods, or, may arise de novo without a preceding bleeding/spotting episode.
- * Mild lower abdominal pains, or, no pains.
- * Regression of symptoms of pregnancy, e.g. nausea, vomiting, breast tenderness.
- * Cessation of fetal movements if they had been present.
- * Failure of quickening to occur at the expected gestation (16-22wks).
- * Uterus stops getting larger and starts getting smaller and smaller than gestational age.
- * The uterus is not cystic.
- * The cervix is firm and the os closed.
- * No adnexal abnormality.

Investigations

- * Ultrasound scan (transvaginal in early pregnancy, or abdominal after first trimester) is crucial in diagnosis and management. It will provide:
 - * Diagnosis of silent miscarriage: A fetus with no cardiac activity. Serial scanning will not show growth or cardiac activity.
- * Accurate dating of the pregnancy: This will help determine the mode of treatment.
- * Pregnancy tests, which were previously positive, will become negative.
- * Serial serum or urinary hCG measurements will show progressively falling levels.

Differential diagnosis

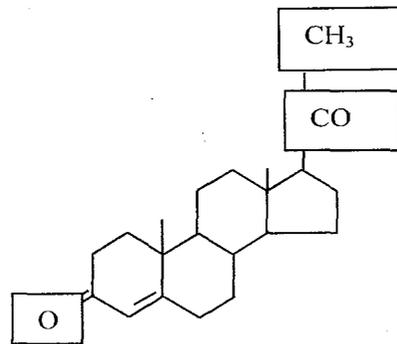
A continuing pregnancy that is younger than the given dates:

- * Chronic ectopic pregnancy.
- * A pelvic tumour without pregnancy.

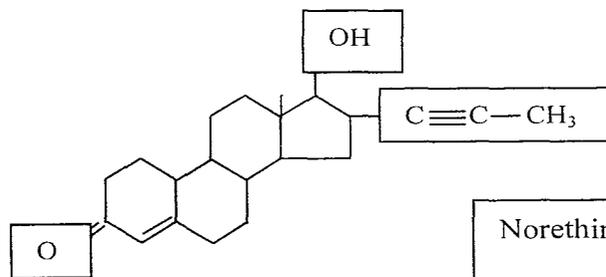
Complications

- * Infection: Risk becomes unacceptably high if membranes rupture.
- * Coagulopathy: Can occur if the gestational

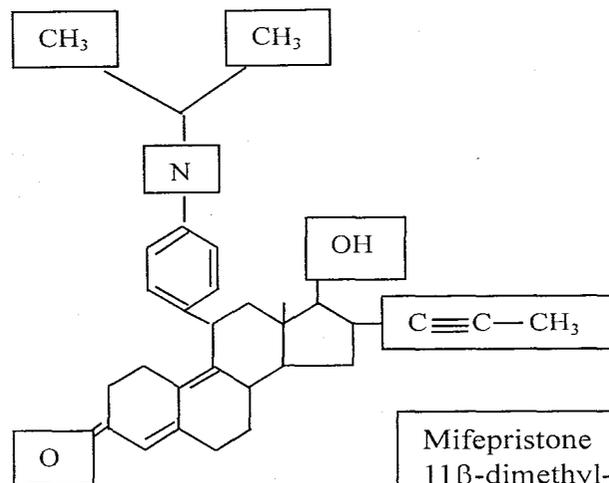
Fig 1. Structure of Mifepristone



Progesterone (21C)



Norethindrone



Mifepristone
11β-dimethyl-phenyl derivative of
norethindrone

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Maternal Mortality In The Tropics

JB Wilson / AT Lassey

Introduction

Maternal mortality remains very high in developing countries even though it has become very clear that women can be prevented from dying without high technology and a relatively low cost. The most recent figures from UNICEF and WHO quote maternal mortality as 585,000 per year (higher than the traditional figure of 500,000 when the safe motherhood initiative was initiated in 1987).⁽¹⁾

Since 1987, there has been increased awareness of the problem of maternal mortality among policy makers, health professionals and the general public.

Various studies have been undertaken that have helped clarify the nature and extent of the problem and have indicated some solutions.

The maternal mortality rates or ratios (MMR) in developing countries range from 190 per 100,000 live births in Latin America and the Caribbean, to 370 per 100,000 in Africa. Extremely high rates of over 1,000 per 100,000 live births are found in East and West Africa (Table 1).

Definition

Maternal death is defined by the World Health Organisation (WHO) as "the death of a woman whilst pregnant or within 42 days of termination of pregnancy, irrespective of the duration or site of the pregnancy, from any cause related to, or aggravated by the pregnancy or its management, but not from accidental causes"⁽²⁾. Thus death from termination of pregnancy is a maternal death since it is considered to be due to the "management" of the pregnancy. Maternal death and mortality is commonly expressed as

the number of maternal deaths per 100,000 live births or per 100,000 total deliveries. In many countries the WHO definition, which includes deaths after an abortion or ectopic pregnancy, applies but in some countries it does not.⁽³⁾

Identification and registration of a maternal death can be extremely difficult and hence the difficulty in assessing national maternal mortality ratios or rates. Doing so requires information on deaths of women of reproductive age (15-49 years), the cause of death and also whether or not the woman

was pregnant at the time of death or had recently (within six weeks) been pregnant. Globally, few countries accurately count births and deaths; even fewer register the cause of death and fewer still systematically note pregnancy status on the death form.⁽¹⁾ In many tropical countries registration systems are absent or inadequate and hence, these countries can only estimate their maternal mortality using survey techniques. Unfortunately, these surveys suffer from a number of disadvantages, including cost. In general, the high maternal mortality countries have neither adequate systems of vital registration nor the resources to rely on surveys instead.

International statistics on maternal mortality are consequently difficult to present in a comparable way as different countries have different exclusion criteria⁽³⁾. For example - some countries do not include deaths after abortion or ectopic pregnancy in the numerator whilst others like the UK until the past decade, included deaths up to one year in the numerator⁽³⁾. For the denominator some countries such as the UK use the total maternities (i.e. the total number of women delivered) whilst others use the number of live births. A larger denominator would result in a lower calculated rate or ratio.

Thus the maternal mortality ratio for the same country will be higher if the denominator is the live births instead of total births.

Although most textbooks and publications would use maternal mortality rate and maternal mortality ratio interchangeably, the rate and ratio are, strictly speaking quite different. Whereas the ratio reports the number of maternal deaths in proportion to births, the rate relates the number of deaths to the number of women at risk so that a proper death rate can be calculated. The correct denominator for the rate therefore should be the number of related pregnancies, but no record can be kept of all pregnancies since many of them do not finish with registrable births or stillbirths and some mothers can die before this stage is reached. The conventional denominator used therefore is the officially recorded number of live births or the total births where total "births/deliveries" is the total number of women delivered^(4,9) or maternities.

Maternities are the number of pregnancies that

result in a live birth at any gestation or a stillbirth occurring at 28 weeks completed gestation (prior to 1st October 1992) and at 24 weeks or more thereafter and are required to be registered by law in the UK. ⁽⁴⁾ Because of the unreliability of data on the total number of pregnancies occurring in a given period (as not all pregnancies result in a live or stillbirth), maternal mortality rates in the UK are related to the number of maternities rather than to the total number of pregnancies.

Most other countries however, use the number of live births as their denominator as these are registrable and more reliably available data.

Underlying Factors

The major causes of maternal mortality are virtually the same everywhere: Haemorrhage, Eclampsia, Infections, Obstructed Labour and complications of abortion etc. but in many developing countries there are many underlying factors that contribute to the high levels of maternal mortality. Many of these have been elucidated during the 8-year operations research conducted by the Prevention of Maternal Mortality (PMM) Network by teams in Ghana, Nigeria and Sierra Leone. The research demonstrated that saving women's lives need not cost much and that it can be achieved under the most difficult working conditions. It also revealed that even though health facilities in many developing countries are likely to be poorly staffed, equipped and maintained, the problems start well before the hospital door.

The numerous problems that characterise this tragedy in the tropics include poverty, malnutrition, ignorance and illiteracy ^(6,7). Other socio-demographic and cultural characteristics are high parity, pregnancies at the extremes of life, short intervals between pregnancies and unhealthy customs and beliefs. ^(6,7) In addition, feelings of powerlessness, fear, social taboos and other factors in the value systems of tropical countries inhibit the women's ability to voice their pain and suffering, resulting from illness and discriminatory practices. They are trapped in circumstances from which there seems little hope of escape ⁽⁸⁾. Superimposed on all these unfavourable characteristics are the poor health care delivery systems in many local communities and of particular relevant mention is the lack of effective care in pregnancy and childbirth. Sadly the high maternal mortality figures in the tropics are further plagued by under-reporting and misclassification of maternal deaths, poor case identification and recording, as well as poor data collection and storage. ⁽¹⁾

Some of the underlying factors can be grouped as:

- * Decision to seek care
- * Transportation Difficulties
- * Health Services' factors
- * Distribution of Health Facilities and Personnel

1. Decision to seek care

In many communities, the decision by a woman to seek care in obstetric complication is not taken by the patient herself. Very often it is the husband, mother in law or another family member who takes this decision. Some complications are attributed to other factors such as witchcraft, insubordination or infidelity. Thus traditional healers, spiritualists are consulted to perform rituals in the event of obstetric complication before the patient is taken to hospital. Another factor is that community midwives and TBAs often do not have the knowledge to advise or early referral.

2. Transportation Difficulties

- a. **Availability** Transport is very scarce in many rural areas of developing countries. There are areas where vehicles are available only on market day. On other days women in Obstetric emergency have to be carried long distances to a point where transport may be available.
- b. **Terrain** the 'roads' in many rural areas are rough and rugged so that a short journey takes several hours. During the rainy season, some of the roads are just impassable.

c. **Cost** Drivers tend to raise fares when they have to transport women in obstetric emergency. Apart from being an opportunity to earn more, the increased fare is geared to put the patients off. Drivers fear that women may deliver in the vehicle may soil the vehicle, or may even die with many unpleasant consequences.

3. Health Services' factors

- i. Patient's reluctance to use health facilities are based on many reasons: Negative staff attitude Health facility staff are perceived to be impatient, rude and accused of taking bribes before attending to patients.
- ii. Patients in some cases perceived the level of care at the health facilities as inadequate. Lack of drugs and equipment, inadequate number of beds with patients lying on the

floor, poor water and erratic power supply are some noted factors. In fact District and Teaching Hospitals are regarded as the Last stop due to the high case fatality rate which was the result of poor quality of care.

The cost of services at the health facilities is another deterrent factor. In many developing countries patients have to pay for services at health facilities. Often payment has to be made before treatment is started.

4. Distribution of Health Facilities and Personnel

Health facilities are often separated by long distances of poor roads and are based more on political expediency than on actual population distribution.

Many doctors and midwives do not accept to work in rural areas because of the lack of social facilities.

A new approach to dealing with maternal mortality was developed by the PMM network based on two key premises:

- a. That maternal mortality is a problem that must be directly addressed by medical treatment (unlike for example, child mortality where socio-economic programmes can make a difference). Intervention ought to be directed at the specific complications that arise during pregnancy and childbirth.
- b. That women in developing countries are often unable to get to the medical treatment that they need. Thus prevention programmes need to address obstacles to access, which requires a better understanding of social, economic, cultural as well as the medical factors involved.

Maternal mortality is a multifactorial and multisectorial problem and therefore needs different disciplines to deal with it. The different disciplines should involve Social Science, Community Medicine, Midwifery and Obstetrics. This mix of disciplines could be involved in the following:

Obstetrics to deal with the clinical aspects of pregnancy and childbirth

Midwifery this is also a clinical skill but often has more contact with the patients.

Community Health to deal with the organisation of health care and aspects of the delivery of services

Social Science to tackle the role of social and

behavioural factors

Traditionally, maternal health programs have focussed on reducing the incidence of complications among pregnant women. This is because certain groups of women are known to have much higher risks of death than do others. For instance, maternal mortality is lowest among women in their 20's yet a close look at the Matlab study shows that in sheer numbers the largest number of maternal deaths occurred in the 20-29 yr age group!

Relative risk is a useful guide for clinical practice and enables midwives and obstetricians to manage individual patients. But for public health programs the number of deaths is a more relevant indicator than the relative risk. Screening pregnant women to identify those at high risk will neglect low-risk women, when, in fact most complications and deaths will occur in this group.

The above buttresses the principle that **most obstetric complications can neither be prevented nor predicted but can be adequately treated**. Since all pregnant women are at risk of obstetric complications, they need to have access to emergency obstetric care (EMOC). Thus availability of hospitals with facilities for EMOC is central to any effort to reduce maternal deaths.

The Three Delays Model

In spite of the availability of a well functioning health facility, which can offer EMOC services, women with obstetric complication may face a number of barriers to using them. These barriers may be

- * **Economic** e.g. lack of money to pay for transport or Services,
- * **Cultural** e.g. the low value placed on women's lives; or
- * **Geographic** e.g. long distances and poor roads. Any factor that causes delay in getting treatment creates a risk to the life of the woman with obstetric complication.

The various factors that can cause delay can be grouped using a simple model called the "**Three Delays**". The Model specifies the three types of delay that contribute to the likelihood of maternal death:

1. Delay in deciding to seek care
2. Delay in reaching a treatment facility
3. Delay in receiving adequate treatment at the facility.

Delay No.1: Deciding to seek care

The delay in deciding to seek care is influenced by many factors:

- a. The knowledge of the patient and her family of serious obstetric complications
- b. Knowledge of the patient/relatives to know where to get help
- c. Cultural factors play an important role here. In many areas women are expected to bear labour pains in silence and so the more stoic the woman the more she is respected.

The lack of autonomy and low status of women also affect the decision to seek care. In some areas a woman cannot be taken to hospital without the husband's permission.

- D. The distance to the health facility, availability and efficiency of transportation, and the cost of health care and transportation influence how the people decide to seek care
- E. The quality of the health facility such as the skill of doctors and midwives, availability of drugs influence the people's decision to seek care.

Delay No. 2: Delay in Reaching a Medical Facility

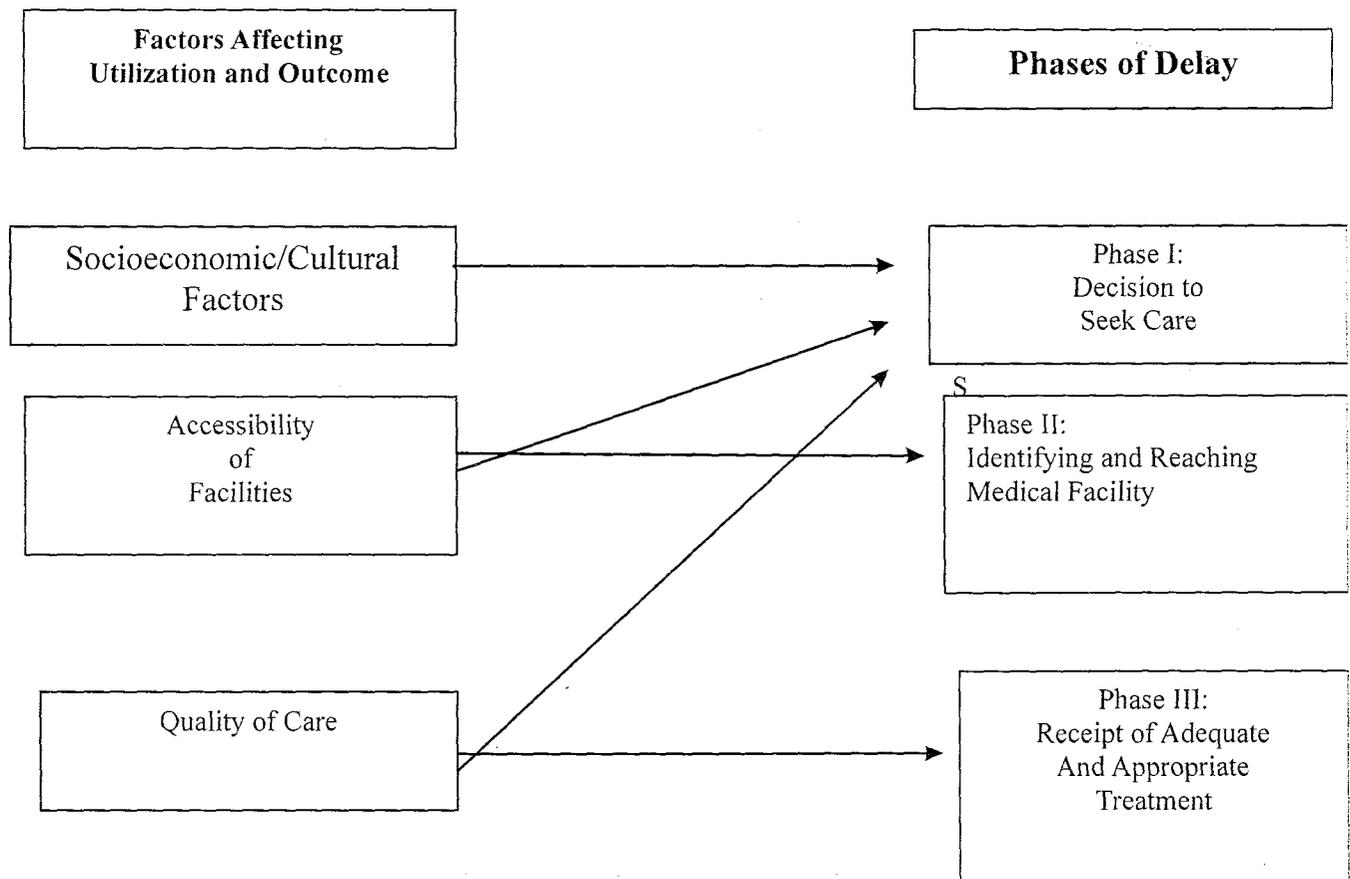
Accessibility depends on the distance and type of road from the health facility availability, cost and efficiency of transportation.

The services offered at health facility affects accessibility; for instance if a nearby health centre does not offer EMOC, the patient will have to travel a much longer distance in order to obtain care.

Delay no 3: Delay in receiving treatment

Often women overcome delays 1 and 2 only to die in hospital. This is due to a number of factors

- a. The number and skill of staff
- b. Availability of drugs and supplies
- c. General condition of the facility
- d. The attitude of staff and the promptness with which patients are attended to.



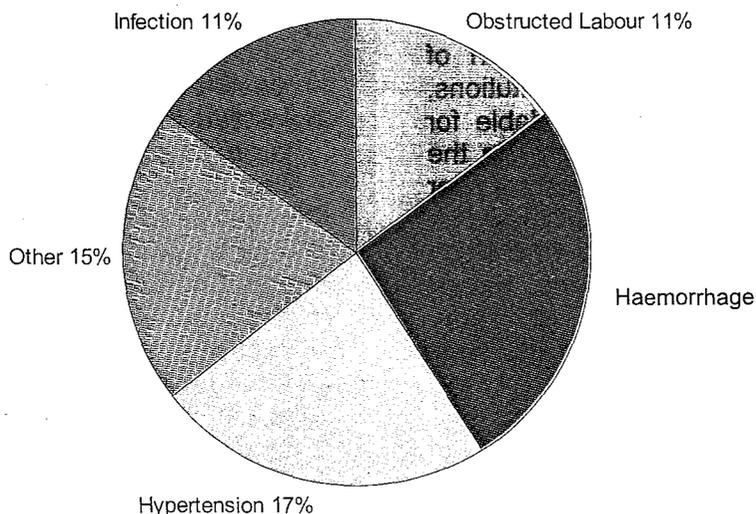
	Maternal mortality ratio (maternal deaths per 100,000 live births)	Number of maternal deaths	Lifetime risk of maternal death, 1 in:
World total	430	585 000	60
More developed regions*	27	4 000	1800
Less developed regions	480	582 000	48
Africa	870	235 000	16
Eastern Africa	1060	97 000	12
Middle Africa	950	31 000	14
Northern Africa	340	16 000	55
Southern Africa	260	3 600	75
Western Africa	1020	87 000	12
Asia	390	323 000	65
Eastern Asia	95	24 000	410
South-central Asia	560	227 000	35
South-eastern Asia	440	56 000	55
Western Asia	320	16 000	55
Caribbean	400	3 200	75
Central America	140	4 700	170
South America	200	15 000	140
Melanesia	810	1 400	21
Europe	36	3 200	1400
Eastern Europe	62	2 500	730
Northern Europe	11	140	4000
Southern Europe	14	220	4000
Western Europe	17	350	3200
Latin America & the Caribbean	190	23 000	130
North America	11	500	3700
Oceania*	680	1 400	26
Australia-New Zealand	10	40	3600

Table 1.
Revised estimates of maternal mortality by United Nations regions (1990)

* Australia, New Zealand and Japan have been excluded from the regional totals but are included in the total for developed countries.

Figures may not add up to totals due to rounding.

Direct and Indirect Obstetric Deaths in Developing Countries ⁽¹⁴⁾



In order to improve maternal mortality the following measures have to be adopted.

1. Improve availability and quality of Emergency obstetric care

- a. Improve availability of drugs and supplies.
- b. Up grade facilities and equipment.
- c. Improve staff knowledge and skills.
- d. Increase staff coverage at facilities.
- e. Improve availability of blood at facilities.

2. Improve access to health facilities.

This involves getting the patient from the community to the Health facility and also between different levels of the health service.

Various methods used dialogue with transport unions to transport women at normal charge; setting up loan or revolving funds from which families could borrow in obstetric emergency and repairing or providing transport at health facilities.

3. Socio-Economic factors

Barriers that delay can be overcome by Community health education targeting traditional birth attendants, pregnant women, husbands and in-laws. It is estimated that between 60-80% of all births in Africa, Asia and Latin America are attended by Traditional Birth Attendants (TBAs)⁽⁹⁾. "A TBA is a person who assists the mother during childbirth and initially acquired her skills by delivering babies herself or through apprenticeship to other Traditional Birth Attendants"⁽⁹⁾. TBAs do not have the scientific knowledge and life-saving skills needed to cope with obstetric emergencies and cannot therefore save lives.

In the developing countries haemorrhage is the leading cause of maternal mortality^(10,11,12). This is due significantly to the moribund condition of referred patients arriving at the health institutions, the inadequate quantities of blood available for prompt transfusion as well as inefficiency in the blood transfusion services. There is also a poor response of relatives of patients to donate blood for in the emergency situation. The rising cost of blood processing and cross matching fees further compound an already desperate situation. Maternal death inevitably results in many such cases.

Operational Research

Although a great deal of knowledge exists about the causes of maternal deaths and how to prevent them

in general, there is a need for operational research in each particular community to clarify the specific pattern of causes and to identify the potentials for improvement⁽¹³⁾.

The typical scenario of maternal mortality in the tropics as summarised in the three delays needs serious scrutiny.

Operational research should clarify the particular picture in any given community and identify substandard or avoidable factors for necessary correction. Indeed the Prevention of Maternal Mortality Network (PMM) has done some such research in West Africa⁽¹³⁾ with very useful results which need dissemination.

Time-and-motion studies of the delay in decision making in the home including how long it takes to recognise life-threatening symptoms and the time it takes for the family decision-makers to decide for the woman to seek medical help should be useful in the specific community. Similar studies down the line following the decision to seek medical help should look at how long it takes to secure transport and communication and should analyse contributory factors. Finally on arrival at the health facility, time-and-motion studies should reveal the obstacles to the early and timely delivery of appropriate medical/midwifery care for each community and district.

Controversies

Stop-gap measure of TBA training

1. which started in the Sudan in 1921⁽¹⁴⁾ and has since spread to other tropical countries has come to stay as a continuing measure instead of the training of straight midwives at the least. Indeed the cost of training a TBA is so high that some assessors have suggested that it might just be enough for a 3-year straight midwifery training course⁽¹⁵⁾. Local selection criteria and sponsorship of the trainees with bonding on return to the local community after the training should check the tendency to seek greener pastures outside the area.
2. Instead of highly equipped and sophisticated large hospitals for delivering women as in developed countries, tropical countries need to concentrate on equipping district hospitals with simple equipment and tools to be able to cope with obstetric emergencies that seek their care. It is important that the district hospital has the capacity to efficiently manage the obstetric emergencies they receive otherwise they quickly lose credibility and patronage from the community.
3. The training of non-medics such as nurses and para-medics in life-saving skills, instrumenta-

- delivery and Caesarean section is another controversial issue. Reports from Zaire (now Democratic Republic of the Congo) and Mozambique have suggested that this is feasible despite misgivings from other health workers.
- 4. Tropical countries' perception that the industrialised countries are promoting and using family planning services to control the size of their populations is a stumbling block to attempts to improve on the low prevalence of contraceptive use in the tropics.
 - 5. The provision of safe abortion services continues to evoke strong sentiments in both the developed and the developing countries. The International Conference on Women and Development (ICWD) Cairo, 1994 clearly illustrated this controversy. The compromise Communiqué in the end recommended the provision of abortion services where appropriate.

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SECTION 4
MEDICAL COMPLICATIONS
OF PREGNANCY

Malaria In Pregnancy

H.S. Opare-Addo/A.T. Odoi

Introduction

Each year approximately 300 million people are affected and over 1 million deaths occur in Africa, Asia, Oceania, Central and South America from malaria^(1,2,3). Malaria is the most important of the parasitic diseases of human beings especially for people living in Sub-Saharan Africa where about 90% of all the deaths from malaria come from^(1,2). The resurgence of the disease in many parts of the tropics accentuated by the increasing resistance of *Plasmodium falciparum* to commonly used drugs and the absence of a vaccine makes malaria a formidable adversary. Pregnant women are known generally to demonstrate an increased susceptibility to, and severity of malarial infection^(4,5,6,7). The reason for this differential response in pregnant and non-pregnant women living under endemic and hyper-endemic conditions is uncertain. The mechanism involved in the immunity of individuals in both hypo- and hyperendemic regions are well known^(3,4,6,8) however their relevance to what happens in pregnancy and the greater susceptibility of primigravidae to malaria is unclear.

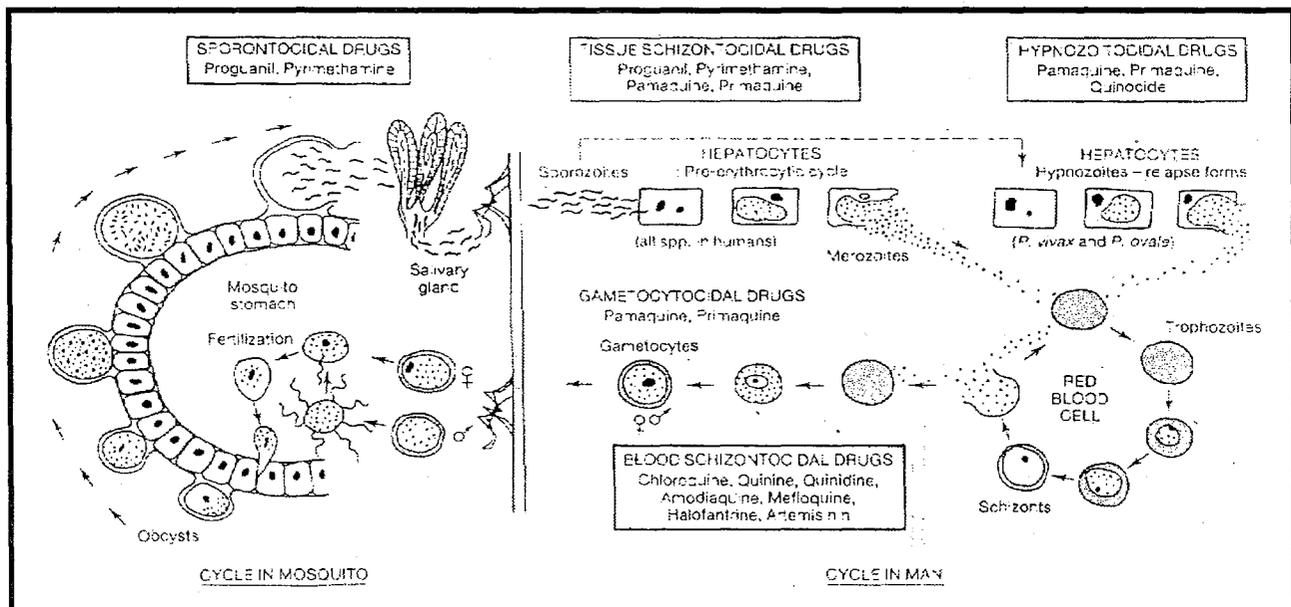
Etiology

Malaria is caused by four species of the protozoan

genus *Plasmodium*: *P. falciparum*, *P. vivax*, *P. malariae*, and *P. ovale*. Malaria is usually spread by the bite of an infected female anopheline mosquito but may follow transfusion of infected blood or use of syringe contaminated with infected blood. Parasites remain viable in stored blood for up to 2 weeks. Cases of transplacental infection have been recorded^(9, 10, 11, 12)

The life cycle of the human malaria parasite is shown in Fig. 1. During a blood meal, the female anopheline inoculates plasmodial sporozoites from its salivary gland. These small motile forms (sporozoites) circulate in the blood stream for 30 minutes before taking refuge in hepatocytes. Here single sporozoites multiply and eventually produce several thousand merozoites in a process known as intrahepatic or pre-erythrocytic schizogony. The swollen liver cell eventually bursts, discharging merozoites into the blood stream, beginning the symptomatic blood stage of the infection. In *P. vivax* and *P. ovale* infections a proportion of the intra-hepatic forms do not divide immediately, but remain dormant for months before reproduction begins. These sleeping forms or hypnozoites are the cause of the relapses that characterize infection with these two species.

Fig 1



In the blood, each merozoite enters a red blood cell and becomes a trophozoite (ringform), which enlarges, forming a schizont then a mature schizont. This then bursts releasing from 6 to 24 daughter merozoites.

Each of these is capable of invading a new red cell and repeating the cycle. Synchronous rupture of ring forms every 48 or 72 hours in the case of *P. malariae* corresponds to febrile paroxysms. After a series of such intra-erythrocytic (asexual) cycles, some trophozoites are transformed into morphologically distinct sexual forms (gametocytes), which are long-lived and relatively inert. After a female anopheles mosquito has ingested erythrocytes from infected human, the gametocytes mature into male and female gametes and go through the process of sexual reproduction within the mosquito's digestive system. This cycle produces sporozoites in about 2 weeks. The sporozoites accumulate in the mosquito's salivary gland and are infectious for humans.

Predisposing Factors, Host Response and Immunity

The severity of malaria is related very much to the species of *Plasmodium*, the level of parasitemia and the immune status of the individual specifically to malaria. *Plasmodium falciparum* is the most virulent species invading erythrocytes of any age. Therefore *P. falciparum* multiply faster and generally produce a denser parasitemia. Almost all deaths are caused by *P. falciparum* malaria⁽²⁾. *Plasmodium vivax* infects younger red blood cells (reticulocytes) and *P. malariae* older red blood cells.

Merozoites need attachment sites to be able to invade erythrocytes. Attachment is mediated via specific erythrocyte surface receptors. In *P. vivax*, this attachment is related to the Duffy blood group antigen Fya or Fyb. Most

West Africans and people with origins in this region carry the Duffy -Negative FyFy genotype and are therefore resistant to *P. vivax* malaria^(2,3,9).

The effect that malaria exerts on a population is largely governed by its epidemiological pattern. Malaria in a community may be either stable or unstable. Stable malaria occurs in regions in which there is a constantly repeated infection (endemic areas). The population has a high degree of immunity and epidemics do not occur. Unstable malaria occurs in regions in which transmission is intermittent. Communal immunity is poorly developed and dramatic epidemics can occur. Immunity to malaria is produced by a complex interplay of both cellular activity and humoral factors.

The initial response to infection in the non-immune subject is the activation of non-specific host defence mechanisms. Splenic immunologic and infiltrative clearance functions are augmented in malaria and there is accelerated removal of both parasitized and un-infected erythrocytes. The parasitized cells escaping splenic removal are destroyed when they rupture to release the schizonts. The material released induces activation of macrophages and release of mononuclear cell-derived cytokines (including tumour necrosis factor and interleukin 1), which cause fever and other pathologic effects. Temperatures of 40 degrees centigrade are schizonticidal, and thus has effect of synchronizing the parasite cycle and eventually producing the regular fever spikes and rigors that originally characterized the different malaria tertian every 2 days in *P. falciparum*, *P. vivax* and *P. ovale*; quartan, every 3 days *P. malariae*).

The global distribution of sickle cell disease, thalassaemia, and glucose-6-phosphate dehydrogenase deficiency closely resemble that of malaria before the introduction of control measures. This epidemiologic information strongly suggests that these genetic disorders affecting the red cell confer protection against death from falciparum malaria. This has been confirmed in the case of Hb AS heterozygotes (sickle cell trait). The mechanism whereby these disorders protect against severe infection has not been elucidated except in the case of Melanesian ovalocytosis. The rigid erythrocytes resist merozoite invasion. Experiment with malarial parasites in artificial culture has shown that under normal physiological conditions they grow as well in AS blood cells as in cells containing haemoglobin AA. However, under conditions of low oxygen tension they do not grow well in AS red blood cells. Perhaps malarial parasites growing in AS red blood cells can lower the oxygen tension sufficiently to cause the cells to sickle; thus ensuring their removal together with the parasites that they contain by phagocytic cells of the spleen⁽⁶⁾.

The specific immune response to malaria limits the rising parasitemia and with exposure to sufficient strains eventually confers protection from disease, but not from infection. Asymptomatic parasitemias are commonly found in adults living in holo- or hyperendemic areas. The state of premunition is specific for both the species and strain of infecting parasite. Both humoral and cellular immunity are necessary but the mechanisms are incompletely understood. Immunity to malaria is acquired gradually after repeated infection. In endemic areas this takes 5 - 10 years to develop and is partial. Immune individuals have a polyclonal increase in

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serum IgM, IgG, and IgA (although much of this antibody is unrelated to protection). Antibodies against a variety of parasite stage-specific antigens (particularly those related to the red cell surface cytoadherence protein) presumably act in concert to limit in vivo parasite replication. Infants do appear to possess mechanism to limit parasite growth and a role for maternal antibody cannot be ruled out⁽¹²⁾. Passively transferred IgG from immune adults has been shown to reduce parasitemia in children and passive transfer of maternal antibody also presumably contributes to the relative protection from severe malaria in the first months of life⁽²⁾.

Several factors retard the development of cellular immunity. These include the absence of major histocompatibility antigens on the surface of infected red cells, which precludes direct T-cell recognition; malaria antigen-specific immune unresponsiveness; and the enormous strain diversity of malaria parasites and their ability to express variant antigens on the erythrocyte surface which change during the period of infection. Immunity against all strains is never achieved. Parasites may persist in the blood for months and, in the case of *P. malariae*, for many years if treatment is not given. The complexity of the immune response to malaria, the sophistication of the parasites' evasive mechanisms and the lack of a good in vitro correlate with clinical immunity have all contributed to the slow progress towards an effective vaccine. The development of antibodies and cell-mediated immunity contribute to the pathogenesis of some of the clinical features of chronic malaria like nephrotic syndrome and tropical splenomegaly.

The Influence Of Pregnancy On The Course Of Malaria

The stress of pregnancy tends to lower the immunity acquired in the non-pregnant state. The reason has not been well elucidated. However it has been argued that when protein requirement is unusually high, as in pregnancy, metabolic channels may be altered so that, if the dietary intake is insufficient, protein is withdrawn from the immune system⁽⁶⁾. Another explanation is that cell-mediated immunity is depressed during pregnancy, though specific malarial antibodies are not decreased. Cortisol levels are increased during pregnancy, and this may contribute to decreased cell-mediated immunity.

Susceptibility to other diseases normally controlled by cell mediated immunity, such as tuberculosis, also is increased in pregnancy. Lymphocytes from pregnant women when challenged with malarial antigens show a depressed proliferative response in

comparison to lymphocytes from nonpregnant women.

As a result of this decline in immunity, pregnant women experience both increased parasitemia and clinical disease^(5,6,7,13,14). This is more common in the last trimester than in the first. In areas of unstable malaria, pernicious forms such as cerebral malaria are more common in pregnant women with resultant high mortality.

The Influence Of Malaria On Pregnancy, Labour, and The Puerperium

The breakdown of malarial immunity is most marked in first pregnancies and thus nulliparous women are more vulnerable to severe malaria and their fetuses similarly show more pronounced adverse effects^(5,6,7,13,14,15,16,17). Malaria often causes anaemia, increased uterine activity, abortion, preterm labour, fetal distress, death in utero, stillbirth and lowbirth weight^(17,18). Maternal mortalities following severe anaemia from malaria in adolescent primigravidae have been reported⁽¹²⁾.

The patients become more vulnerable to hypoglycaemia especially following treatment with certain anti-malarials. A few may also develop pulmonary oedema in the puerperium.

Different mechanisms are responsible for these changes. The anaemia following severe malaria may not only be from haemolysis but also from folate deficiency and hypersplenism. The accelerated rate of haemopoiesis needed to keep abreast with the red blood cells destruction increases the folic acid requirements, which may not be satisfied during pregnancy owing to the competing demands of the developing fetus. Severe anaemia is predominant among parasitaemic pregnant women with an enlarged spleen⁽²⁰⁾.

Hypereactive malarial splenomegaly (HMS) is common in the tropics and often associated with anaemia, which can be debilitating in patients already compromised by anaemia due to poor nutrition and pregnancy. The course of the disorder in pregnancy is commonly punctuated by episodes of haemolytic anaemia, which can be life threatening to the mother and cause increase fetal morbidity and loss.⁽²⁰⁾

Placental malaria is recognized as a common complication of malaria in pregnancy in areas of stable transmission, and is particularly frequent and severe in primigravidae^(4,5,6,21,22,23). Just as cerebral malaria results from parasite sequestration

In the brain, maternal (placenta) malaria results from parasite sequestration in the placenta. The placenta in malaria is stuffed full of parasites and in addition shows many pathological changes. A distinct sub-population of parasites, which bind chondroitin sulphate A, but not CD 36 causes maternal malaria.

Women have little or no immunological experience with this parasite⁽²⁴⁾. Parasites adhere to the surface of trophoblastic villi, eliciting the accumulation of inflammatory leucocytes in the intervillous space and the necrosis of adjacent placental tissue. Histologically, the intervillous spaces are filled with macrophages and malaria pigments. There are perivillous fibrinoid deposits and proliferation of the cytotrophoblastic cells with thickening of the trophoblastic basement membrane. There is sound epidemiological evidence that placental malaria determines low birth weight, mainly mediated by intrauterine growth restriction and increases the risk of death and disease during the first year of life⁽²¹⁾. The exact mechanisms leading to placental changes and determining the observed impairment of maternal-foetal exchange are incompletely understood. A deeper understanding of the mechanism involved in this process will be of key importance in designing effective intervention. Anti-malarial chemoprophylaxis significantly reduces placental malaria

and prevents the development of lowbirth weight babies. The placental changes coupled with the increased uterine activity associated with maternal fever and the often associated severe maternal anaemia are responsible for the deteriorating fetal well-being.

Hypoglycaemia is an important and common complication of severe malaria in pregnant women and children. Hypoglycaemia results from failure of hepatic gluconeogenesis and increased consumption of glucose of both host and fetus by parasite. The principal gluconeogenic substrates, lactate and alanine, are increased. To compound the situation, quinine, the drug of choice for severe chloroquine-resistant malaria is a powerful stimulant to pancreatic insulin secretion. This turns to aggravate the hypoglycaemia on account of the already existing B-cell hyperplasia in pregnancy. The fetus is vulnerable because it cannot switch to fat-derived energy, as the mother can.

Clinical Presentation

Patients with malaria usually present with chills and fever, often associated with headache and muscle aching. Early on, these fevers may be erratic before developing synchronous periodicity related to timing

of schizogony. Between the paroxysms of chills and fever the patient often feels well. Other presenting non-specific symptoms include malaise, dry cough, abdominal pains, nausea, anorexia or vomiting.

An attack of malaria in the last trimester may put the patient into labour. Malaria is sometimes seen in labour in endemic areas. Similarly malaria may be the cause of puerperal pyrexia and the stress of labour can precipitate acute attacks.

Physical Examination

The fever is irregular at first and in the non-immune often rises to over 40 degrees centigrade and is accompanied by tachycardia. There may be splenomegaly (more common in *P. vivax* and *P. ovale* infections), hepatomegaly, jaundice and anaemia.

Fever and related symptoms gradually subside over several weeks in untreated *P. vivax*, *P. ovale* and *P. malariae* malaria. Months or years after initial exposure, dormant forms of parasite (hypnozoites) may emerge from the liver and cause relapse. With *P. falciparum*, serious complications such as coma in cerebral malaria, severe haemolytic anaemia, disseminated intravascular coagulation, acute renal failure and acute pulmonary oedema may develop rapidly in the non-immune host; however, once eradicated from the blood, *P. falciparum* does not relapse.

Diagnosis

The diagnosis of acute malaria can often be suspected clinically. It can only be confirmed by examination of thick and thin blood films for malaria. In areas of high malarial endemicity, many people intermittently have a mild parasitemia. In these areas the presence of malaria parasites in the blood of a patient with an acute febrile illness may not necessarily be malaria. Blood films usually show a polymorphonuclear leucocytosis and some thrombocytopenia. A very rough calculation of parasite density can be made by gradation of a thick blood film on a + to ++++ basis. Accurate assessment involves determining the red-cell count and the percentage of red cells infected. Another approach is to count 200 high-power fields and record the number containing one or more parasites. Determination of parasite density is of value in assessing the response to treatment and as a prognostic guide. Malaria antibodies can be demonstrated by a number of techniques in the sera of subjects.

DRUG	ACTION	SIDE EFFECTS
4-Aminoquinoline a. Chloroquine	Acts on asexual blood parasites. Rapid blood schizonticidal. Gametocytocidal against <i>P. Vivax</i> , <i>P. ovale</i> and <i>P. Malaria</i>	Bitter taste and causes nausea. Pruritus in dark- skin patients Hypotension and cardiac arrhythmias occasional. Causes accommodation difficulties rarely and retinopathy after long-1 term prophylaxis. Relatively safe in pregnancy.
B. Amodiaquine	Rapid blood schizonticidal	Agranulocytosis rarely Hepatotoxic rarely
Quinine	Rapid schizonticidal Gametocytocidal against <i>P. Vivax</i> , <i>P. ovale</i> and <i>P. Malaria</i>	Bitter taste when taken orally. Necrosis and alogdystrophy following I.M injection. Hypoglycaemia especially following I.V treatment. Vertigo and Tinnitus
Antifolates	Dihydrofolate reductase inhibitors Inhibits pre-erythrocytic growth (causal prophylaxis) and Development in the mosquito (Sporontocidal)	
a. Proquanil	Causal prophylaxis	Well tolerated Rarely causes megaloblastic anaemia, pancytopenia and pulmonary infiltration.
b. Pyrimethamine	Speed of action depends on stage of parasite development	Well tolerated Rarely causes mouth ulcers and megaloblastic anaemia in renal failure.
Halofantrine	Rapid schizonticidal	Rarely causes diarrhoea. Not much is known about effects in pregnancy and therefore contraindicated in pregnancy.
Artesunate	Rapid schizonticidal Has potential gametocidal effect on Immature falciparum gametocytes	No abnormalities observed in patients treated with drug before pregnancy had been confirmed and also in those treated in second trimester following multi- drug resistance with other anti- malarials ^(2, 29, 30)
Mefloquine	Rapid schizonticidal	Found safe in pregnancy ^(27,28,29) . Prevalence of congenital malformation observed in those exposed to drugs estimated as 4% is similar to the general population ⁽²⁹⁾ .

Management

Antimalarial Drugs:

The main anti-malarial drugs are quinine and the amino-4-quinolines, which rapidly act against intra-erythrocytic trophozoites. The amino-8-quinolines, which are active against gametocytes, have been abandoned, as they are too toxic. The anti-folate drugs proguanil and pyrimethamine on their own are slow acting and cannot be used for treatment but for causal prophylaxis. The sulphonamides are also slow acting blood schizonticides for *P. falciparum* with weaker activity against erythrocytic stages of other species. The combination of a diaminopyridine and long acting sulphonamide was developed following the advent of resistance. The mechanism of action of the individual components proved complementary against malaria. Various newer drugs such as Halofantrine, Mefloquine and Artemisinin are now part of the armamentarium developed in the attempt to fight the emerging drug resistance.

Fig. 1 and Table 1 summarize the action of various drugs at different stages of the development of the malaria parasite. In addition, Table 1 gives important minor and major side effects including its effect in pregnancy.

Drug resistance has become a major epidemiologic concern in malaria treatment and prophylaxis. Chloroquine resistance has not been reported in infections with *P. vivax*, *P. ovale*, or *P. malariae* ^(2,25).

However, *P. falciparum* resistance to chloroquine is widespread. Three stages of resistance are defined in relation to parasitemia: Stage I (RI): Temporary disappearance. Stage II (RII): Regression. Stage III (RIII): No reduction. To be able to categorize resistance to anti-malaria one needs to count the parasites before treatment and daily for the first 7 days and again at 14, 21 and 28 days. In RI parasites disappear at day 2, but reappear and persist on any day up to day 28 (re-infection has to be excluded). RII is present if by day 2 about 25% or fewer parasites are counted. RIII exists if more than 25% are counted on day 2. If no parasite is present at day 28 then it is possible to say that they are sensitive.

Although quinine-resistant *P. falciparum* has been reported, significant resistance to this drug is limited mainly to South East Asia and may necessitate larger dosing or the addition of a second agent. Pyrimethamine/Sulfadoxine (fansidar) resistance is common among *P. falciparum* as well as *P. vivax*. Mefloquine has limited resistance, mostly confined to South East Asia

Treatment Of Acute Malaria

The treatment of an acute attack of malaria aims at reducing the pyrexia and bringing the attack to an end as quickly as possible. In endemic areas uncomplicated infection may be treated on an outpatient basis. Acetaminophen (paracetamol) lowers the fever and provides symptomatic relief. Chloroquine or any 4-aminoquinolines is given at regimen detailed in Table 2. Care is taken to ensure the patient has taken some calories before taking the drugs. Antihistamine is prescribed for patients with history of itching on taking the drug.

For the non-immune or the severely ill patient and those with the propensity to vomit and unable to tolerate oral anti-malarial drugs admission may be necessary. Injectable antipyretics, and anti-emetics such as promethazine and metoclopramide are given. Care is taken to correct/prevent hypoglycaemia and electrolyte imbalance with intravenous fluids before giving injectable anti-malarials as detailed in Table 2.

Anaemia may be acute and severe necessitating transfusion or exchange transfusion in patients with over load or pulmonary oedema. The management of chronic hyperactive malaria splenomegaly (HMS) consist of lifelong antimalarial prophylaxis supplemented by haematinics ⁽²¹⁾. Blood transfusion may be required to treat episodes of severe haemolysis. Oxygen and diuretics may be needed for pulmonary oedema.

Anticonvulsants and intensive care may be necessary for cerebral malaria. Currently there is no evidence supporting the use of osmotic or diuretic agents or dexamethasone in cerebral malaria. Infact dexamethasone prolongs the patient's return to consciousness and also causes pneumonia and gastrointestinal bleeding ⁽⁶⁾. Dialysis may be necessary for renal failure following black water fever. Severely ill malaria patients especially those with cerebral malaria or renal failure are prone to gram-negative septicaemia and would need appropriate antibiotic therapy

Fetal growth and health must be monitored. Correction of fever and hypoglycaemia often corrects transient fetal hypoxia. However, for the severely parasitized placenta, deceleration may persist and calls for delivery. The treatment of acute chloroquine-resistant malaria in pregnancy is with quinine sulphate and pyrimethamine/ sulfadoxine, Artesunate, and mefloquine alone or in combination with quinine sulphate ^(26,27,28) and many other combinations detailed in Table 3.

TABLE II

TREATMENT OF ACUTE MALARIA IN PREGNANCY WITH 4-AMINOQUINOLINE

ORAL TREATMENT

Oral chloroquine 600mg base (4 tablets) followed in 6 hours by 300mg base then 300mg base daily for 2 days.
OR

Oral amodiaquine 600mg (3 tablets) stat, then 400mg (2 tablets) daily for 2 days.

PARENTERAL TREATMENT

Intramuscular chloroquine 300mg base 6hourly until able to take drugs by mouth.
Oral chlor

TABLE III

CHLOROQUINE RESISTANT MALARIA IN PREGNANCY

ORAL TREATMENT

Oral quinine sulphate 600mg every 8 hours for 5 - 7 days.

Oral quinine sulphate and pyremethamine/sulfadoxine 3 tablets on day 3

Oral quinine sulphate + clindamycin (10mg/kg every 8 hours for 5 days)

Artesunate 100mg(2 tablets) bd on day 1 and then 50mg (1 tablet) bd for 4 days

Mefloquine 15mg/kg single dose.

INTRAVENOUS

Quinine dihydrochloride 10mg /kg every 8 hours for 5 days.

Prevention Of Malaria

WHO accepted by 1969 that, programmes of controlling Malaria were indispensable in areas where eradication was impracticable such as sub-Saharan Africa⁽¹⁾. Since 1992, WHO has encouraged national programmes which focus on early diagnosis and prompt treatment, selective and sustainable prevention, early detection, containment and prevention of epidemics and building local capacity to assess and manage the Malaria situation⁽¹⁾.

By and large vector control and drug prophylaxis are the two main approaches presently used to control Malaria. Insecticide-treated nets have been used for the last 15 years to prevent mosquito bites.⁽¹⁾ Despite the low use of insecticide-treated bed nets, national impregnated bed net programmes have had some impact on the outcome of pregnancy, particularly in reducing the percentage of premature babies in primigravidae^(1,31).

Residual house spraying using the stratified approach in high-risk areas remains an important control activity. Environmental Management such as drainage or control of water flow of rivers and other water bodies and application of larvicides in areas where breeding sites are well-defined remains important control activities⁽¹⁾.

Chemoprophylaxis or presumptive treatment during pregnancy is known to reduce the risk of Malaria infection in all pregnant women significantly and increases the birth weight of babies born to women in their first pregnancy^(1,16,23,32,33,34,35).

It also improves the pack cell volume^(16,32,33). What is not clear is whether chemoprophylaxis has advantages over early, effective treatment of clinical Malaria taking into consideration the cost of prophylaxis^(32,33,36).

Less than 20% of pregnant women in Malaria endemic areas receive regular prophylaxis although WHO recommends prophylaxis for all such pregnant women⁽¹⁾.

Chloroquine [5mg base per kilogram per week [300mg maximum] weekly] is considered relatively safe in pregnancy. The drawback is the pruritus caused in dark skinned patients and the tendency of inducing resistance. Amodiaquine is associated with a high risk of agranulocytosis and therefore not recommended for prophylaxis. Pyrimethamines have been used widely but resistant strains of both *P. falciparum* and *P. Vivax* have limited their use. Proguanil is considered the safest antimalarial to be used as prophylaxis in pregnancy. Proguanil has retained efficacy against pyrimethamine-resistant strains of *P. falciparum*. The usefulness of sulphadoxine-pyrimethamine for intermittent presumptive treatment in malarious areas and more so where HIV co-existed has been acknowledged^(32,35). The side effects of exfoliative dermatitis and other skin rashes, agranulocytosis, hepatitis and pulmonary eosinophilia however limits its use. There is the need to exclude G6PD patients and to offer supplemental folic acid to those prescribed the drug. It must be avoided also in the third trimester. Low dose of mefloquine has also been found efficacious in holoendemic areas with multidrug resistant plasmodium falciparum^(2,30).

A major break-through needed in Malaria control is the development of an effective vaccine. Vaccine development is now at a point of unprecedented opportunity, though it may take 7-15 years before an effective vaccine is ready⁽¹⁾.

TABLE IV

CHEMOPROPHYLAXIS IN PREGNANCY

Chloroquine phosphate: 300mg each week

Pyrimethamine/sulfadoxine: 3 tablets if Malaria symptoms occur.

Proguanil: 200mg per day Mefloquine 250mg each wk for 4 wks then every 2 wks.

Discussion And Controversies

Malaria is no doubt the commonest cause of febrile illness in the tropics. However conditions such as urinary tract infections and typhoid fever should be ruled out, especially in situations where initial antimalarial treatment of proven efficacy does not work. Cerebral malaria is very uncommon in immune pregnant women; however, it should be considered in a comatose febrile patient. Eclampsia may sometimes be difficult to differentiate from cerebral malaria as coma, convulsions, pyrexia and albuminuria may occur in either. Repeated generalized convulsions in a woman with high blood pressure with coma in the interval is more likely to be an eclamptic whereas if coma occurs in a hyperpyretic patient in the absence of fits cerebral malaria may be the most likely diagnosis. In an endemic area an eclamptic may have a positive blood film. The differential diagnosis may occasionally be difficult and one may have to treat for both conditions simultaneously.

HIV infection is associated with a significant increase in malaria prevalence in pregnant women of all parities with the effect apparent from early in gestation⁽³⁷⁾.

Chemoprophylaxis is never entirely reliable and Malaria should always be considered in the differential diagnosis of fever in patients who have taken prophylactic anti-Malarial drugs. Non immune subjects with Malaria should have daily parasite counts performed until thick films are negative (parasite clearance). If the parasitemia does not fall below 25 percent of the admission value in 48hrs or has not cleared by 7 days, drug resistance should be suspected and treatment should be changed.

Patients have sometimes not accepted antimalarial treatment for fear of potential toxicity to the fetus and risk of causing abortion. Chloroquine and Quinine have stimulant effect which increases as pregnancy advances. It is widely known to the general public as an abortifacient, however it is unreliable even in lethal doses. Rates of spontaneous abortion and birth defects are comparable in pregnant women taking mefloquine, chloroquine-proguanil, or pyrimethamine sulfadoxine prophylaxis in the first trimester of pregnancy⁽²⁹⁾. Standard doses of quinine do not increase the risk of abortion or preterm delivery⁽²⁹⁾.

There are controversies surrounding the management of malaria. Should people in endemic areas, with malaria parasites in their blood but without febrile illness be treated? The authors' view

is that patients with increased susceptibility including pregnant mothers must be fully treated while those with less susceptibility, with low parasitemia must be observed without immediate treatment. In the developing countries where many patients are seen in health facilities without adequate laboratory support, should patients with a febrile illness be treated for malaria empirically at such centres? We think the medical staff in such centres should use clinical features to rule out the most common causes of febrile illness before instituting antimalarial treatment.

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In the female, the urethra is approximately 3 to 4cm in length, with its external opening in close proximity to the vaginal canal. The vaginal canal in turn borders the anus and rectum.

The vagina is normally colonized with organisms from the lower gastrointestinal tract, and the distal urethra frequently is colonized with these potential uropathogens^(9,10,11). Physiologic urinary tract obstruction leading to stasis is associated with acute pyelonephritis in pregnancy. Two factors, which are believed to lead to hydronephrosis and hydro-nephrosis, are mechanical compression from the enlarging uterus and progesterone induced smooth muscle relaxation.

The causative organisms in 85 to 90 per cent of urinary infections during pregnancy are due to Enterobacteriaceae, especially *Escherichia coli*, *Klebsiella*, and *Enterobacter*. Less common urinary tract pathogens during pregnancy include *Citrobacter*, *Proteus* and *Pseudomonas* spp. *E. Coli* is not the most plentiful in faeces but its pathogenic virulence appears to derive from a number of factors. These include resistance to vaginal acidity, rapid multiplication in urine, possession of adhesions (characterised as fimbriae) allowing adherence to uroepithelial cells and production of chemicals that decrease ureteric peristalsis and inhibit phagocytosis⁽¹²⁾. Women who are susceptible to infection may differ immunologically from those who resist infection in that they are less likely to express antibody to the O antigen of *E. coli* on the vaginal epithelium and may display less effective leucocyte activity against the organism.

Clinical Presentations

Urinary tract infections present with or without symptoms:

- (1) **Asymptomatic Bacteriuria**
- (2) **Symptomatic Bacteriuria**

Lower urinary tract: Cystitis (and Urethritis)

Upper urinary tract: Pyelonephritis

1. Asymptomatic Bacteriuria

As the name implies, this refers to infection in the urinary tract without symptoms such as dysuria or frequency. The prevalence of asymptomatic bacteriuria is essentially the same in both pregnant and non-pregnant women. The incidence of bacteriuria during pregnancy is approximately 5 to 6 per cent but may be as high as 10 per cent in high-risk populations⁽¹³⁾. Conditions associated with an increased frequency of asymptomatic bacteriuria, in both pregnant and non-pregnant women, include

Screening

In non-pregnant women, asymptomatic bacteriuria carries minimal health risks thus making its routine screening unnecessary and not cost-effective. There is controversy about the value of routine screening of pregnant women for asymptomatic bacteriuria.

Early studies showed that approximately one third of pregnant women with untreated asymptomatic bacteriuria develop acute pyelonephritis.⁽¹³⁾ In a recent antenatal study in which at least 99% of women took part in at least one screening, the suggestion was that bacteriuria was highest between the 9th and 17th weeks of pregnancy. The 16th week was the optimal time for a single screen of bacteriuria calculated on the number of bacteria-free gestational weeks gained by treatment.

Some studies have questioned the value of screening. It is known that not all untreated bacteriuric women develop symptoms of acute urinary tract infection during pregnancy and those found to have sterile urine at booking may subsequently develop symptomatic urinary tract infection. This raises the point that routine screening may not be cost effective^(15,16).

Anaemia, pregnancy-induced hypertension, preterm labour and low birth weight infants are adverse effects of asymptomatic bacteriuria.⁽¹⁷⁾ Subsequent studies suggest these conclusions may have resulted from inaccuracies in matching cases and controls.⁽¹⁸⁾ The better studies were subjected to meta-analysis and this led to the conclusion that there was an association between untreated asymptomatic bacteriuria and low birth weight at term delivery and that therapy did reduce the incidence of low birth weight neonates.⁽¹⁹⁾

Treatment

The choice of drug is based on the sensitivity of the isolated organism(s) and must not be contraindicated in pregnancy. Various single drug regimens have been investigated during pregnancy and success rates ranged from 50 to 88 per cent^(22,23,24).

Nitrofurantoin and short acting sulfonamides may be used. Nitrofurantoin may cause nausea and vomiting in the last few weeks of pregnancy and may precipitate haemolytic anaemia due to glucose-6-phosphate dehydrogenase deficiency in the newborn. Short acting sulfonamides may be effective but should be avoided in late pregnancy because they competitively inhibit the binding of bilirubin to albumin.

albumin and increase the risk of neonatal hyperbilirubinaemia.

Trimethoprim, a constituent of Cotrimoxazole (Septrin) is not recommended for use in pregnancy since it is a folic acid antagonist. It is used extensively nevertheless and is probably safe, though not a drug of first choice. Ampicillin and cephalosporins can be safely used in pregnancy. A two-week course of therapy is usually adequate. Some studies have advocated the use of short, especially single-dose courses⁽²⁴⁾.

Urine cultures should be obtained one week after therapy is discontinued and then at regular intervals throughout the pregnancy. This obviates the need for continuous antibiotic therapy from the time of diagnosis until delivery.

Recurrence

Relapse and Reinfection)

Relapse is the recurrence of bacteriuria caused by the same organism, usually within six weeks of the initial infection. Reinfection is the recurrence of bacteriuria involving a different strain of bacteria, after successful eradication of the initial infection⁽²⁵⁾. Reinfections usually occur more than 6 weeks after treatment and in most patients are limited to the bladder.

Follow Up

There is no evidence that persistent asymptomatic bacteriuria in women with normal urinary tracts cause long-term renal damage or that treatment reduces the incidence of chronic renal disease⁽²⁶⁾.

In some cases, there may be some abnormality of the urinary tract and intravenous urography is then recommended. This recommendation applies when there is asymptomatic bacteriuria and any of the following⁽³⁰⁾:

- Difficulty in eradicating the bacteriuria during pregnancy.
- Episode(s) of acute symptomatic urinary tract infection during the pregnancy.
- History of acute infection(s) prior to the index pregnancy.
- Persistence or recurrence of asymptomatic bacteriuria or acute infection postpartum.

2. Cystitis And Urethritis

Cystitis frequently appears to arise de novo in most pregnant women. This was the outcome of a study in which 2/3 of women with acute cystitis initially had sterile urine cultures⁽²⁷⁾. There is no evidence that acute cystitis is associated with an increased risk of acute pyelonephritis although 40 per cent of pregnant women with acute pyelonephritis have preceding symptoms of lower urinary tract infection⁽²⁸⁾.

Symptoms And Signs:

The symptoms may be similar to those due to normal pregnancy i.e. Urgency, frequency and suprapubic discomfort.

The diagnosis of cystitis during pregnancy is based on significant dysuria, suprapubic discomfort or gross haematuria along with a positive urine culture and no systemic symptoms. If there is fever, renal angle tenderness or other systemic symptoms and signs then the upper urinary tract is involved.

A positive urine culture is diagnostic. The range of organisms found is the same as in women with asymptomatic bacteriuria. The commonest organism is *E. coli*. Others are *Proteus mirabilis* and *Klebsiella pneumoniae*. Pyuria is not diagnostic since it may be found in 10 to 15 per cent of pregnant women without infection.

Treatment And Follow Up

Treatment can be on outpatient basis with oral antibiotics best chosen on culture and sensitivity results.

Sulfonamides, nitrofurantoin, ampicillin or a cephalosporin given for 10 days are effective.

Shorter courses of therapy (3 to 5 days), especially in non-pregnant patients have been used as well.

Single dose therapy as described for asymptomatic bacteriuria has been shown effective for both non-pregnant and pregnant women. Renal infection must be excluded with confidence before single dose therapy because 40 per cent of women with early pyelonephritis initially have lower tract symptoms.

Urethritis: *Chlamydia trachomatis*, a common pathogen of the genitourinary tract may cause urethritis. This will present with frequency, urgency, dysuria and pyuria and a "sterile" urine culture. There is frequently a mucopurulent cervicitis and therapy with erythromycin is effective.

Eosinophilic Cystitis may be the cause where seemingly adequate therapy for bacterial cystitis does not eradicate symptoms⁽²⁹⁾.

3. Acute Pyelonephritis

Pyelonephritis occurs in approximately 1 to 2 per cent of all pregnancies and is the most common renal complication of pregnancy^(28,30,3).

The risk of developing acute pyelonephritis is markedly increased in pregnancy because of obstructive uropathy and stasis of urine. Untreated asymptomatic bacteriuria adds to the risk. There is a pressure gradient of approximately 15ml of water between the lower and upper ureter in late pregnancy⁽³²⁾.

Most cases of acute pyelonephritis occur during the second and third trimesters⁽³³⁾. It is unilateral and right-sided in more than half of cases and bilateral in one fourth. In most women, bacteria that ascend from the lower tract cause renal parenchyma infection.

Symptoms And Signs: These are fever, shaking chills, nausea and vomiting, and renal angle tenderness. Nearly 40 per cent of women also complain of lower urinary tract symptoms such as urgency, frequency and dysuria. The fever is generally spiking in nature, may be high and reach 40°C.

Diagnosis: Diagnosis is based on the systemic signs and symptoms together with a positive urine culture that invariably yields more than 100,000 colonies per millilitre of a single organism. In approximately 15 per cent of cases, blood cultures are positive. In cases where urine cultures are negative, the woman may have taken some antibiotics - even a single oral dose may render the urine culture negative.

Differential Diagnosis: These include febrile conditions such as malaria, and respiratory tract infections (pneumonia), which are common in the tropics.

Other urinary tract pathology such as renal calculus or acute hydronephrosis (which can be recognized on ultrasound scanning or limited excretory urography) should be considered.

The onset of labour and chorioamnionitis may mimic pyelonephritis. It is important to exclude other causes of acute abdominal pain such as acute appendicitis, biliary colic, gastroenteritis, placental,

abruption or red degeneration in a fibroid³⁴ in pregnancy, especially in the third trimester. Appendicitis is difficult to diagnose. Features that help are that pain is referred to the centre of the abdomen, vomiting is less marked, the pyrexia is as high as in acute pyelonephritis and rigors do not occur.

Rarely viraemia, listeriosis or toxoplasmosis should be considered. In the puerperium, sepsis may complicate the picture.

Treatment

Once the diagnosis is made on clinical findings the patient should be admitted and a mid-stream specimen of urine is obtained for culture and sensitivity.

Blood is taken for full blood count and sputum for film for malaria parasites. The serum electrolytes and creatinine are requested. In severely ill patients, blood cultures should be requested.

Intravenous fluids are given for rehydration and intravenous antibiotics started whilst waiting for urine culture and sensitivity results. Intravenous fluids are given until vomiting ceases and the temperature settles. Tepid sponging and antipyretics such as paracetamol may be necessary. The condition usually improves after 48 hours and the antibiotics can be given orally.

In choosing antibiotics, the aim is to give the most effective drug to eradicate a particular infection while avoiding fetal exposure to harmful drug effects. Broad spectrum antibiotics producing high blood levels and high renal parenchymal concentrations are preferred. Ampicillin and Cephalosporins are suitable. E. Coli is the most common organism isolated in urinary infections and is usually sensitive to either of them. Other antibiotics, which are used, include Cotrimoxazole (Septrin), aminoglycosides such as Kanamycin and Gentamicin. It is important to consider the potential adverse effects of Septrin and the aminoglycosides in pregnancy. The duration of treatment is usually 2 to 3 weeks although some may extend to 4 weeks. On the completion of course of treatment, urine cultures should be taken at every antenatal visit for the rest of the pregnancy.

Complications

1. Adverse effects on fetus

Acute pyelonephritis is associated with an increased incidence of premature labour and possibly also with intra-uterine growth restriction or fetal death.

2. Renal dysfunction

Failure to improve within the first 2 to 3 days suggests a complication that is frequently associated with underlying obstruction, such as urolithiasis or an anatomic anomaly.

At the time of an acute episode of acute pyelonephritis in pregnancy, the glomerular filtration rate may be reduced⁽³⁵⁾ in contrast to the usual lack of impairment of renal haemodynamics in non-pregnant patients. Acute pyelonephritis is an extremely rare cause of acute renal failure in non-pregnant subjects in the absence of complicating factors such as obstruction, stones, analgesic nephropathy and papillary necrosis. In pregnancy, however, when there are complicating factors, acute renal failure appears more likely⁽³⁶⁾.

3. Septic Shock

Gram-negative sepsis can occur in severely ill patients with acute pyelonephritis but this situation is more commonly associated with instrumentation of an infected urinary tract.

4. Haematologic changes

Endotoxin induced haemolysis may occur with resulting anaemia⁽³⁷⁾.

6. Pulmonary Injury

Up to 2 per cent of women with antepartum pyelonephritis develop varying degrees of respiratory insufficiency caused by endotoxin induced alveolar injury and pulmonary oedema⁽³⁴⁾.

5. Hypothalamic Instability

Acute pyelonephritis in pregnant women may alter the hypothalamic response so that there are wide variations in temperature. Fever as high as 42°C may be followed by hypothermia of 34°C. This reaction is mediated, probably in the anterior hypothalamus by cytokines that are elaborated by macrophages in response to endotoxin.

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Cardiac Disease In Pregnancy

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Introduction

Cardiac disease is uncommon in pregnancy. The obstetrician thus has difficulty in making an accurate diagnosis and managing heart disease in pregnancy on his own.

Although the incidence is low, symptoms such as breathlessness, or signs such as an ejection systolic murmur that are suggestive of cardiac disease, may be present in up to 90 per cent of the pregnant population as a consequence of the physiological changes induced by pregnancy itself.

In the presence of normal cardiac function the physiological adaptive changes occur to accommodate these cardiovascular alterations. However, in the presence of significant cardiac disease, decompensation occurs that may result in serious morbidity and death. Decompensation may be progressive, the clinical effects worsening as pregnancy advances. The additional stress of labour may cause further decompensation.

For women with cardiac disease, therefore pre-pregnancy counselling and early recognition of problems during pregnancy is necessary to prevent morbidity and mortality to the woman and her baby.

Although, cardiac disease is uncommon in pregnancy, it remains a significant indirect cause of maternal mortality in the western countries⁽¹⁾. The situation is masked in the developing countries by the sheer numbers of more direct causes of maternal mortality.

It is important nevertheless to make the diagnosis as early as possible since both mother and fetus may be at considerable risk.

In developing countries, one can anticipate an increase in cardiac disease in the pregnant population although rheumatic fever and rheumatic heart disease are on the decline. This is because improvements in health services and the establishment of a few cardiac centres in developing countries have ensured that more girls with congenital heart diseases are maturing into the reproductive age group. The result is that whilst consequences of rheumatic heart disease such as mitral tricuspid and aortic valve disease are on the

decline, congenital defects, Fallot's tetralogy, and patent ductus arteriosus may be seen more often in pregnancy. The relative frequency of congenital as opposed to acquired heart disease is indeed increasing^(2,3).

Certain patients are also at high risk of bacterial endocarditis. These include patients with prosthetic valves, surgical systemic pulmonary shunts, most congenital cardiac malformations and rheumatic valvular dysfunctions. A previous history of endocarditis is particularly dangerous.

The long-term health of a woman with heart disease is not jeopardized if the pregnancy is well managed provided she survives the pregnancy itself⁽⁶⁾.

Maternal Mortality Associated with Pregnancy

Group 1 - Mortality <1%

Atrial septal defect
Ventricular septal defect
Patent ductus arteriosus
Pulmonic/tricuspid disease
Tetralogy of Fallot, corrected
Bioprosthetic valve
Mitral stenosis, NYHA class I and II

Group 2 Mortality 5 - 15%

2A
Mitral stenosis, NYHA class III and IV
Aortic stenosis
Coarctation of aorta, without valvular involvement
Uncorrected tetralogy of Fallot
Previous myocardial infarction
Marfan syndrome with normal aorta

28B

Mitral stenosis with atrial fibrillation
Artificial valve

Group 3 Mortality 25-50%

Pulmonary hypertension
Coarctation of aorta, with valvular involvement
Marfan syndrome with aortic involvement.

Fetal Outcome

The outcome for the fetus with rheumatic heart disease is usually good, although the babies are likely to be lighter ⁽⁷⁾. With cyanotic congenital heart diseases, there is an increased risk of spontaneous abortion, intrauterine growth retardation and death in utero ⁽⁸⁾.

Another consideration is the 5% risk of fetal congenital cardiac anomalies, 50% of which may be concordant with the maternal lesion ⁽⁸⁾.

Functional changes in pregnancy

In normal pregnancy, maternal cardiac output increases by about 30-50 percent from a non-pregnant average of about 4.5L/min to about 6.0L/min at rest ⁽⁹⁾. The increase occurs early in pregnancy, at least two-thirds of the increase has occurred by the end of the first trimester. Peaking between 24-28 weeks the increase in cardiac output is caused partly by an increase in heart rate ⁽¹⁰⁾ and partly by an increase in stroke volume. The rise in cardiac output in pregnancy is also due to increased preload caused by increased circulatory blood volume. Blood volume rises very early in pregnancy. The total increase is about 40% and this rise is maintained until delivery ⁽¹¹⁾.

The blood pressure usually falls due to a marked fall in peripheral vascular resistance.

Cardiac output rises still further on exertion and in labour. During labour, each uterine contraction will increase cardiac output by about 20 per cent ⁽¹²⁾.

The physiological changes of increase in cardiac output and associated vasodilatation in pregnancy lead to circulatory changes mimicking heart disease:

1. Cardiac size increases and displacement of the apex beat by up to 1cm. from the mid-clavicular line should not be considered abnormal.
2. There is tachycardia and it is likely that arrhythmias are more common in pregnancy.
3. Pulse volume is increased. Jugular venous pressure waves are more prominent, though the height of the venous pressure is not increased in pregnancy.
4. The first heart sound is loud; there is often a very prominent third heart sound and an ejection systolic murmur heard over the whole praecordium in up to 90 per cent of pregnant women.

5. Venous hums, continuous murmurs usually audible in the neck, which can be modified by stethoscope pressure, may also be heard in pregnancy.
6. Peripheral oedema is very common in pregnancy.

By two weeks post-partum, the cardiac output has nearly returned to normal ⁽¹³⁾ although it may take up to 3 months for the blood pressure to return to normal.

Management of Heart Disease in Pregnancy

Pre heart disease in pregnancy is best managed with the collaboration of cardiologist. For optimum results, heart disease should be diagnosed prior to pregnancy so that appropriate counselling can be undertaken. Congestive cardiac failure, endocarditis, and other forms of decompensation should be controlled before pregnancy is embarked upon. Mortality with certain conditions like severe pulmonary hypertension is so high that pregnancy is a virtual contraindication.

Diseased valves may be replaced with prosthesis, septal defects closed and patent ductus arteriosus ligated before pregnancy is embarked upon.

Identification Of Patients At Risk

In developing countries, traditional birth attendants, churches and spiritual houses provide care to a considerable number of pregnant women. They should be taught to refer antenatal patients with breathing difficulties to a tertiary center for further assessment ⁽¹⁴⁾.

Midwives in midwifery homes and general medical practitioners should refer patients to the regional and teaching hospitals for evaluation if there are no specialists in such centres.

Evaluation In Hospital

In the regional and teaching hospitals a combined obstetric and cardiac team carries out evaluation. This arrangement minimizes the number of patient visits and maximizes the clinical experience of the health personnel.

History: The patient may already know she has heart disease and documentation is already available.

Symptoms to be evaluated are:

1. **Dyspnoea at rest or exertional dyspnoea:** this is the most frequent symptom of heart disease in pregnancy. Most normal pregnant women have a degree of breathlessness thus making its assessment difficult. ⁽¹⁵⁾ It is important to find out if the breathlessness was present before the pregnancy.
2. **Syncope:** this may be a feature of normal pregnancy. It may also occur because of dysrhythmias or other serious conditions such as hypertrophic cardiomyopathy and aortic stenosis.
3. **Chest pain:** it is uncommon in pregnancy and is usually a feature of ischaemic heart disease. Chest pain therefore merits further investigations.
4. **Palpitations:** this may indicate a dysrhythmia and should be investigated further.
5. **Haemoptysis:** This may indicate pulmonary oedema from a primary cardiac lesion

Physical signs: The changes, which occur in the cardiovascular system in normal pregnancy, have been considered earlier. Any other murmurs or heart sounds should be considered significant and investigated. Systolic murmurs are so common in pregnancy that their interpretation and significance may pose difficulties. Diastolic murmurs should however be investigated. Signs of heart failure should be looked for as well as cyanosis, finger clubbing, presence of pulse deficits and splinter haemorrhages (bacterial endocarditis)

New York Heart Association Classification (NYHA)

The criteria committee of the New York Heart Association classified cardiac disease based on clinical function. Although a more complex descriptive systems based on aetiology, anatomical defect and physiological considerations has largely replaced the NYHA functional classification, the latter is still a useful system ⁽¹²⁾.

- Class I Asymptomatic
- Class II Symptoms with greater than normal activity
- Class III Symptoms with normal activity
- Class IV Symptoms at bed rest

Class I & II cardiac disease are said to have a good prognosis although deterioration to lower classes may occur as pregnancy advances.

(See table)

Despite these general rules specific cardiac defects are associated with specific risks of maternal mortality

	Limitations Of Physical Activity	Compromised (Symptoms)
Class I	No	No
Class II	Slight	Slightly
Class III	Marked	Markedly
Class IV	Severe	Severely

This information may be useful in counselling patients planning to embark on pregnancies.

The disadvantages of the New York heart association classification are:

- I. Inability to indicate the structural severity of the cardiac condition at the time of classification
- II. The class may change with advancement of the pregnancy
- III. Not useful in prognosis.

Investigations

1. **Chest X-ray:** this is only useful when there is haemodynamically significant heart pathology.
2. **Electrocardiography:** this is not useful in demonstration of a structural abnormality. It is useful in the diagnosis of dysrhythmias. In pregnancy, T wave inversion in lead III, ST segment changes and Q waves, which would usually be considered pathological occur frequently.
3. **Echocardiography:** this is the investigation of choice in pregnancy. There is no radiation hazard and echocardiography can also give details of structural abnormalities.

Clinical Management

The first step is to assess the nature and severity of the heart lesion in the combined obstetric/cardiac clinic. The patients will fall into three groups after assessment.

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Group 1: this will comprise the majority and will show no evidence of any lesion at all. No further follow up will be required and they can attend their regular antenatal clinic.

Group 2: this will comprise patients with a mild lesion with no haemodynamic problems e.g. congenital mitral valve prolapse, which has an excellent prognosis.⁽¹⁶⁾ Such cases need no further follow up.

Group 3: this comprises those with real or potential haemodynamic implications and need careful assessment and subsequent management. If the patient has previously been well managed, her cardiac condition would have already been assessed prior to conception.

Termination of Pregnancy

The patient is first assessed as to the need for termination of pregnancy, if seen early enough in pregnancy, and secondly as to the need for surgery.

Eisenmenger's syndrome and primary pulmonary hypertension are considered as absolute indications for termination of pregnancy because of associated high maternal mortality. In all other cases, the decision whether the pregnancy should continue depends on an individual assessment of the risk of pregnancy compared to the patient's desire to have a baby. The decision, of course is made in consultation with the patient. Informed consent is essential.

Surgery: generally, the indications for surgery in pregnancy mirror those in the non-pregnant state. These include failure of medical treatment with either intractable heart failure or intolerable symptoms.

Antenatal Management

The aim is to avoid, if possible the risk factors for heart failure, and treat aggressively heart failure should it occur. The risk factors for heart failure include infections (especially respiratory tract infections), hypertension, obesity, anaemia, multiple pregnancy, polyhydramnios and arrhythmias.

The treatment of heart failure in pregnancy is the same as in the non-pregnant state.

Antibiotics

Antibiotics are required whenever there is a risk of endocarditis, as in tooth extraction, spontaneous rupture of the membranes and in labour. Protocols in labour will be determined by local conditions.

Examples include:

1. Intravenous amoxycillin 1 gram plus intravenous gentamicin 120 grams stat. Followed six hours later by 500 milligrams of intravenous amoxycillin.
2. IV Vancomycin plus gentamicin 1.5 mg/kg. To be repeated 8 hours later.

The Use of Anticoagulants

Anticoagulant, heparin, may be necessary in patients with a congenital heart disease who have pulmonary hypertension due to pulmonary vascular disease, those who have artificial valve replacements and those with atrial fibrillation.

Pulmonary Embolism and Systemic Thromboembolism

The hallmark of significant pulmonary embolism is dyspnoea and tachypnoea chest pain; apprehension, cough, trachycardia and haemoptysis may also be present. In massive embolism, hypotension, syncope or convulsions may mimic myocardial infarction.

Anticoagulant therapy is the mainstay of pulmonary embolism. Heparin is the preferred anticoagulant throughout pregnancy. Because of its large size, it does not cross the placenta or appear in breast milk. This applies even to low molecular weight heparin. If necessary, heparin is deactivated with protamine sulphate in a dose of 1mg/100u of administered heparin. Coumarin derivatives are preferred in non-pregnant patients⁽¹⁷⁾

With pulmonary embolism, the loading dose is 150u/kg/h intravenous (with a minimum of 5000 iu) or as bolus injections at a dose of 100u/kg IV. Following the loading dose, heparin can be infused at the rate of 25u/kg/h or as bolus injections at a dose of 100u/kg/h every 4 hours. An activated partial thromboplastin time should be determined 4-hourly before each dose. Once a steady state is achieved a PTT can be done daily. Intravenous heparin should be continued for 3 to 5 days or until symptoms have resolved⁽¹⁷⁾. For long-term anticoagulant therapy, this regime may be switched to subcutaneous heparin. The total daily dose is given in three divided doses.

To prevent recurrence, heparinisation should continue for 6 months.

Labour

Heart disease per se is not an indication for induction of labour. Induction should be based on obstetric indications. In some cases induction can be planned during the daytime to make use of optimum medical support.

Fluid balance should be carefully maintained to avoid pulmonary oedema and a central venous pressure line may be used to ensure this. Oxygen administration is essential throughout labour.

Adequate analgesia is essential and epidural is ideal. The second stage of labour is usually uneventful but in some, it may be necessary to shorten it using the ventouse or forceps. Excessive maternal expulsive efforts should be avoided.

Post-Partum

During the third stage of labour, oxytocin is used instead of ergometrine to avoid the risk of precipitating heart failure. In established heart failure, the patient is put in cardiac position and oxygen and intravenous frusemide are added. When post-partum haemorrhage is severe, the use of ergometrine may be justified. There is a risk of maternal post-partum haemorrhage when using heparin and therefore protamine sulphate should always be available in the labour wards. The heparin is continued for 7 days then warfarin may be recommended. The amount of warfarin secreted in breast milk is very small⁽¹⁸⁾ and so there is no contra-indication to breast-feeding.

Family Planning

1. Symptomatic valvular heart disease (rheumatic or congenital)

These patients should not use COCs (combined oral contraceptives) or CICs (combined injectable contraceptives) WHO class 4 contraceptives). The use of these poses an additional risk for blood clotting problems (oestrogen effects). Intra-uterine contraceptive devices (IUDs) may be used with caution. Prior to inserting an IUD, prophylactic antibiotics are advised if the woman is not already receiving long-acting antibiotics. The use of prophylactic antibiotics reduces the risk of infection and possible subacute bacterial endocarditis during IUD insertion.

2. Ischaemic heart disease or stroke (current or history)

These patients should not use COCs and CICs (WHO class 4).

Implants and PICs (progesterone injectable contraceptives WHO class 3) should be avoided. They theoretically pose an additional risk and are used only when other more appropriate methods are not available or acceptable.

Clients with class III-IV heart disease should consider voluntary sterilization. Even if one pregnancy has been successful, further pregnancies are extremely risky.

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Asthma In Pregnancy

VN Addo

INTRODUCTION

Bronchial asthma is a common lung disorder characterized primarily by chronic inflammation of the airways as well as obstruction and increased airway responsiveness to various noxious stimuli. There is a partially or completely reversible airway obstruction associated with inflammation and with airway hyper responsiveness to a variety of stimuli including environmental irritants, viral respiratory infections, cold air, or exercise. The substances that cause asthmatic attacks may do so by induction of specific IgE antibodies, by other non-specific immunologic mechanisms and by direct irritation (such as with gases, fumes, or chemicals)⁽¹⁾.

Asthma may improve, worsen or remain the same during a given pregnancy, but the pattern of symptom change experienced in one pregnancy is usually seen in subsequent gestations⁽²⁾.

For a patient in a stable state whose asthma is well-controlled and not steroid-dependent, the perinatal prognosis is comparable to that of a non-asthmatic gravida.

Functional changes

The subcostal angle of the diaphragm increases from approximately 69 degrees to 104 degrees during the course of pregnancy. This is associated with an elevation of the diaphragm of approximately 4 cm and a 2cm. increase in maximal transverse chest diameter as measured on chest x-ray films⁽³⁾. These changes lead to a conversion from abdominal to thoracic breathing and in part contribute to the increased maternal oxygen consumption during pregnancy.

The total lung capacity is decreased due to the elevation of the diaphragm during pregnancy while the vital capacity remains unchanged at 3200 ml. The tidal volume increases from 450 ml to 600 ml and this leads to an increase in minute ventilation during pregnancy from 19% to 50% without an increase in respiratory rate⁽⁴⁾.

These pulmonary changes lead to an alteration in blood gas chemistries. There is a decrease in CO₂ partial pressure (pCO₂) to 30 mmHg, while partial oxygen pressure (pO₂) ranges from 90 to 106 mmHg. Plasma bicarbonate levels will be reduced by secondary renal mechanisms to 18 to 22 mEq/L, with a relative alkalisation of the serum pH to 7.44⁽⁵⁾.

The effect of pregnancy on asthma

Pregnancy has no consistent effect on asthma. Some studies have found an improvement in asthma during pregnancy⁽⁶⁾, whereas others have found deterioration^(7,8). It appears that about one third of asthmatic women can expect worsening of the disease at some time during pregnancy.

Pregnancy does not cause any net change in airway resistance either in normal subjects or those with asthma. This is likely to be the net effect of several factors. Acting in opposing directions the bronchodilator influences are increased progesterone secretion, which may cause bronchodilation directly and also by increasing beta-adrenergic activity and increases in free cortisol.

The broncho-constrictor influences are the reduced residual volume, reduced paco₂ and increased prostaglandin F_{2a} secretion. Prostaglandin F_{2a} should therefore not be used in obstetric practice (therapeutic abortion, induction of labour), particularly in asthmatic patients^(9,10,11). Prostaglandin E is used more widely and is the preferred prostaglandin for use in asthmatics (II).

The effects of asthma on pregnancy

There may be a slight increased risk to the fetus of the mother with asthma but this effect is very small. When severe it can affect pregnancy outcome. These include increased incidences of abortions, pre-term labour, low birth-weight infants, and neonatal hypoxia^(12,13).

Severe uncontrolled asthma has maternal risks. Maternal deaths may be associated with status asthmaticus. Life-threatening complications include pneumothorax, pneumomediastinum, acute cor pulmonale, cardiac arrhythmias, and muscle fatigue with respiratory arrest.⁽¹⁴⁾

Pathophysiology. There is bronchial smooth muscle contraction associated with mucus hypersecretion and mucosal oedema. Biochemical effectors of these changes include **primary mediators** such as histamines, which are released from lung tissue immediately upon challenge with an allergen. **Secondary mediators** include prostaglandins, thromboxane, and leukotrienes.

Diagnosis

The diagnosis is usually made when there are recurrent episodes of wheeze and breathlessness, often associated with trigger factors such as exposure to allergens (dust, pollen), infection or psychological factors.

An arbitrary definition is a variation in peak expiratory flow rate or FEV₁ by more than 20 per cent either spontaneously or as a result of treatment.

The clinical spectrum ranges from mild wheezing to severe broncho-constriction capable of causing respiratory failure, severe hypoxaemia and death.

The functional result of acute bronchospasm is airway obstruction and decreased airflow. Patients present with chest tightness, wheezing or breathlessness making the work of breathing progressively more difficult.

Management

The management of asthma in pregnancy is based on four main components

1. Objective assessment of maternal lung function and fetal well-being
2. Patient education.
3. Avoidance or control of environmental precipitating factors.
4. Pharmacological therapy.⁽¹⁵⁾

The first component involves evaluation using laboratory and lung function tests as well as ultra sound (fetal biophysical profile). The second and third components will depend on the patient's social and environmental background. The patient should be helped to identify and avoid as much as possible those factors, which bring on an attack (Table 1).

Table 1: drugs to avoid with pregnant asthmatics

A. Asthmatic Stimuli

Non-steroid anti-inflammatory drugs (NSAIDS)
 Propranolol (beta-blockers)
 Prostaglandins (especially abortifacient types such as F_{2a})
 Known sensitizing drugs (e.g. Penicillin)
 Antihistamines
 Sulpha dioxide
 Sulphites
 Azo and non-azo food dyes
 Tobacco smoke
 Exercise
 Cold air
 Pollen, house dust, mites, animal danders, cockroach antigen

A. Adverse fetal effects

Iodides (fetal thyroid suppression)
 Tetracycline (adverse fetal bone and dentition effects)

Pharmacologic therapy

Pharmacotherapy now tends to focus on treating the underlying chronic inflammatory process with specific and preferably inhaled anti-inflammatory therapy, specifically cromolyn sodium and corticosteroids. (Table 2)

Table 2 Medications for pregnant asthmatics

1.	<u>ANTI-INFLAMMATORY</u> Cromolyn sodium Beclomethasone Prednisone Zafirlukast	(leukotriene	receptor	antagonist)
2.	<u>BRONCHODILATOR</u> Nebulized Theophylline			isoproterenol
3.	<u>ANTI-HISTAMINE</u> Chlorpheniramine			
4.	<u>DECONGESTANT</u> Pseudoephedrine			
5.	<u>COUGH</u> Gauifenesin Dextromethorphan			

Theophyllines

In the past aminophylline was used for severe acute asthma as initial therapy or as an adjunct to beta-adrenergic therapy, but currently it is being replaced by corticosteroids. Although aminophylline is no longer the mainstay of therapy for severe acute asthma, theophylline derivatives continue to be useful for oral maintenance therapy of outpatients who do not respond optimally to inhaled beta-agonists and corticosteroids.

Theophyllines have bronchodilator, immunomodulatory, anti-inflammatory and bronchoprotective properties.

Beta-Sympathomimetics

These drugs bind to specific cell-surface receptors and activate adenylyl cyclase, which increase intracellular cyclic AMP to modulate bronchial smooth muscle relaxation (14). They are given by inhalation or orally and are also used for maintenance therapy of out-patients.

Disodium Cromoglycate

Cromolyn sodium stabilizes most cell membranes, has a preventive effect on asthma mediators and is used chronically.

Steroids

Oral glucocorticoids are generally restricted to patients with severe persistent disease not relieved by inhaled agents and full doses of bronchodilator therapy.

Newer agents

Newer agents include Zafirlukast, a cysteinyl-leukotriene receptor antagonist and zileuton, a 5-lipoxygenase inhibitor. These agents interfere with the inflammatory process mediated to a significant degree by leukotrienes and cytokines.

Management of acute asthma

Treatment of acute asthma during pregnancy is similar to that of the non-pregnant asthmatic. First-line pharmacological therapy includes use of a beta-adrenergic agonist e.g. epinephrine or isoproterenol.

Intravenous hydration is given to help clear pulmonary secretions and oxygen is administered by mask.

It has recently been recognized that corticosteroids should be given early to all patients in the course of severe asthma.^(16,17) Because their onset of action is several hours it is emphasized that steroids, whether given intravenously or by aerosol, are given along with beta-agonists for treatment of acute asthma.

Management of chronic asthma depends on the severity of the disease. Beta-agonists by inhalation are generally used with corticosteroids added as necessary.

Status asthmaticus is severe asthma of any type not responding after 30 to 60 minutes of intensive therapy. Modern management should be in an intensive care unit with early intubation when the maternal respiratory status continues to decline despite aggressive treatment.

Management of labour and delivery

It is unusual for labour to be complicated by attacks of asthma. For attacks of asthma which do occur in labour conventional treatment with inhaled beta-sympathomimetics should be used first, with earlier recourse to parenteral steroid therapy if the patient does not improve rapidly.

Analgesia is preferably given using a non-histamine releasing narcotic such as fentanyl. Tracheal intubation can trigger severe bronchospasm and so some anaesthetists avoid using general anaesthesia. Lumbar epidural analgesia reduces oxygen consumption and minute ventilation. Aminophylline, ephedrine and epinephrine can cause tachycardia and potential cardiac arrhythmias. Droperidol increases airway conductance and is safe for premedication. Atropine has a bronchodilatory effect. Ketamine, as an inducing agent offers benefits because it decreases airway resistance and may prevent bronchospasm.

Shortening of the second stage by using the ventouse or forceps may be beneficial if there is any respiratory compromise.

Prostaglandin F₂ should not be used because of its bronchoconstrictor action; also oxytocin should be used rather than ergometrine because of concern that the latter may cause bronchospasm in patients with asthma.^(18,19)

Breastfeeding should be encouraged. Theophylline will be present in breast milk but the quantity is less than 10% of the maternally ingested dose. Prednisone is present in an insufficient concentration to have an effect on the newborn.

Discussions And Controversies

* Pre-pregnancy

The known asthmatic needs thorough evaluation and treatment prior to conception. This will minimize complications in the mother and ensure a favourable fetal outcome.

* Patient Education

The social and environmental background will

determine the focus of counselling and education. The patient should be helped to identify and avoid as much as possible those factors, which bring on an attack.

* Analgesia And Anaesthesia

Labour and delivery will entail the use of analgesics and sometimes anaesthetics. Labour ward staff should be conversant with the options available. General anaesthesia may be hazardous. Ketamine as an inducing agent is beneficial because it decreases airway resistance and may prevent bronchospasm.

* Oxytocics

There is concern that ergometrine may cause bronchospasm in asthmatics and should not be used. Oxytocin is preferred. Prostaglandin F₂ is bronchoconstrictor and should not be used.

* Bronchial Asthma And Acute Left Ventricular Failure

There may be difficulty in differentiating between these two conditions especially if a patient is seen for the first time.

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Diabetes

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Definition

The WHO Expert Committee on Diabetes defined diabetes as "a syndrome of chronic hyperglycaemia (i.e. the state of having excessive concentration of glucose in the blood), which may result from many environmental and genetic factors often acting jointly. The major regulator of glucose concentration in the blood is insulin; a hormone synthesised in and secreted by the β -cells of the islets of Langerhans in the pancreas. Hyperglycaemia may be due to a lack of insulin, or to an excess of factors that oppose its action. This imbalance leads to abnormalities of carbohydrate, protein, and lipid metabolism. The major effects of diabetes include characteristic symptoms, keto-acidosis, the progressive development of disease of the capillaries of the kidney and retina, damage to the peripheral nerves, and excessive arteriosclerosis".⁽¹⁾

Diabetic Groups in Pregnancy

Diabetes in pregnancy is a heterogeneous group of conditions with a common metabolic characteristic of hyperglycaemia. A patient whose pregnancy is complicated by diabetes may belong to one of the following 4 groups.

1. A patient with diagnosed, pre-existing diabetes (Type I, or Type II diabetes) may become pregnant. This is a **pre-gestational** diabetic.
2. A patient whose diabetes antedated the pregnancy but was not diagnosed until she became pregnant. The diabetes was first diagnosed in the pregnancy. The diabetes may be Type I or Type II.
3. The immunopathologic damage to the β -cells had started before the pregnancy, and was progressing, but had not yet reached the biochemical or clinical stage. This stage was reached after the patient became pregnant. The pregnancy was a fortuitous event. The patient would develop diabetes even if she did not become pregnant. The patient now has impaired glucose tolerance or very early clinical diabetes.
4. The patient developed a diabetic condition in the pregnancy, and as a direct result of the

pregnancy. Without the pregnancy, she would not have developed the condition at this time of her life. In this group, the diabetic condition would be expected to abate when the pregnancy was over, as evidenced by the return of the OGTT to normal during, or, after the postnatal period. Diagnosis of this condition carries a significantly increased probability of the later development of diabetes and impaired glucose tolerance. Some studies have found that within 20 years of the diagnosis, about 40% will have developed diabetes.^(2,3)

Patients in groups 2, 3, and 4, above, are called gestational diabetics.

Definition of Gestational Diabetes

This is carbohydrate intolerance of variable severity with onset, or first diagnosis during the index pregnancy.^(4,5)

Significance of Diabetes in Pregnancy

- The pregnancy adversely affects control of the diabetes.
- The diabetes, in turn, adversely affects the pregnancy.

Effects of Pregnancy on Diabetes

Pregnancy is a diabetogenic condition. It makes the control of pre-existing diabetes more difficult to control because of the following factors:

- I Changes in intermediate metabolism: Pregnancy is diabetogenic: increased post-receptor resistance to insulin, exaggerated post-prandial blood glucose excursions, islet cell hypertrophy and hyperplasia, increased insulin levels, low fasting blood glucose levels.
- II Changes in the renal handling of glucose: Increase in renal plasma flow (RPF) over non-pregnant levels of 30% (from 1.2 L/min in non-pregnant to 1.5 L/min); glomerular filtration rate (GFR) increases by 50% (110 ml/min in non-pregnant to 170 ml/min at 26 weeks pregnancy). As a result, filtered glucose may exceed proximal tubule absorption capacity.
- III Nausea and vomiting: Is common in pregnancy. Therefore, hypoglycaemia is more likely if subject takes insulin. On the other hand,

starvation may produce insulin resistance and keto-acidosis.

- IV. Infections: Pregnancy increases predisposition to infections. For example, furunculosis and pyelonephritis are more common. Infections may produce insulin resistance and make diabetic control more difficult.
- V. Labour: Labour is work. During labour, there is relative starvation. This may result in hypoglycaemia.
- VI. Postpartum fall in insulin requirements: Within a few hours of delivery, insulin requirements fall to pre-pregnant levels. If antepartum dose of insulin is given before or during labour, there will be hypoglycaemia postpartum.
- VII. Breastfeeding requires additional calories and may require increase in pre-pregnant insulin doses.

These are discussed in detail below.

I. Changes in Intermediate Metabolism

Intermediate metabolism in normal pregnancy is characterised by changes that allow the intermittently feeding mother to provide for the continuous and ever-increasing metabolic needs of the developing conceptus.

This adaptation to pregnancy is diabetogenic, with decrease in glucose tolerance. As a result of these changes, maternal protein catabolism does not reach the high levels it would otherwise reach when the mother is in the fasting state.

These changes increase in magnitude as the conceptus increases in size and its fuelled by a defect in the metabolic action of insulin distal to its binding with its receptor; i.e. the resistance is due to post-receptor events. The resistance is predominantly in muscle.

These post-receptor events are due to:

- The cellular effects of increasing levels of pregnancy hormones: human placental lactogen (hPL), human chorionic gonadotrophin (hCG), prolactin, free cortisol, progesterone, and estrogens.
- The changes in metabolic fuel availability that occur in late gestation.

The increased resistance to the hypoglycaemic effects of insulin markedly increases the height of the oscillations that occur in blood glucose after a meal. The exaggerated blood glucose excursions, in turn, stimulate hypertrophy and hyperplasia of the β -cells of the islets of Langerhans. Because of the increased β -cell mass, a unit of insulin-secretagogue (e.g. glucose or amino acid) causes a greater amount (2 to 3-fold) of insulin to be secreted than in the non-

pregnant state.

Thus, maternal β -cell sensitivity to glucose and amino acids is significantly enhanced to compensate for the increased peripheral resistance to insulin.

Whereas most (98%) normal women are able to counteract the increase in insulin peripheral resistance by a significant increase in their basal and nutrient-stimulated insulin secretion, a few (2%) women do not have the capacity to produce a sufficiently large increase in insulin secretion to overcome the resistance. These few are the women who develop diabetes because of the pregnancy.

Demonstrable Metabolic Changes in Pregnancy

In the Fasting State

1. **Plasma Insulin:** Fasting plasma insulin gradually rises in pregnancy and in late pregnancy it is twice as high as in the non-pregnant. This is true for normal pregnant women; it is true for non-obese gestational diabetics (GDs). It is also true for obese GDs, but in this group the rise is much higher.
2. **Blood Glucose:** In the non-pregnant, low blood glucose concentrations a switch to the metabolism of fat occur when there is caloric deprivation (fasting). Both the magnitude and tempo of these changes are exaggerated in the later half of pregnancy. Thus, after a 12hour overnight fast, normal women in the 3rd trimester manifest lower blood glucose concentrations than do non-gravid women. An extension of the fasting period to 18hrs, results in a further fall in blood glucose accompanied by stimulation of lipolysis and exaggerated ketogenesis that produce a significant increase in circulating ketones and free fatty acids (FFAs). In the non-pregnant, the levels of all these fuels, glucose, ketones, and FFAs, remain unchanged when the fast is extended to 18hrs.

In the pregnant, women, hypoglycaemia develops in spite of augmented gluconeogenic potential in the liver, apparently because the amount of amino acid substrate available for gluconeogenesis is limited.

These maternal adaptations may serve to minimise maternal protein catabolism during fasting while 'sparing' or saving some non-lipid fuels for utilisation by the conceptus. This adaptation of the maternal organism, which allows a rapid switch to the metabolism of fat during fasting, has been called "**accelerated starvation of pregnancy**".⁽⁶⁾

In the Fed State

Postprandial levels of insulin and all metabolic fuels are altered in pregnancy.

1. **Insulin:** As pregnancy progresses, the following situation develops:

- Increasing diabetogenic hormones, e.g. hPL, prolactin, increasing peripheral resistance to insulin increasing postprandial glucose levels increasing hypertrophy and hyperplasia of β -cells increasing insulin secretion response to meals
- The exaggerated insulin response to eating, starts early in pregnancy and increases in parallel with the growth of the conceptus; it disappears with the expulsion of the conceptus at parturition. By 10 weeks' gestation, the insulin response to an oral glucose challenge, i.e. glucose-induced insulin secretion, is already significantly higher than in the non-pregnant state. By mid-gestation the response is more than double the response in the non-pregnant. This is true for normal pregnant women; it is true for GD mothers, but with the following provisos:

- The peak plasma insulin concentration occurs later in GDs than in normal mothers.
 - The insulin response per unit of glycaemic stimulus (i.e. the insulinogenic index) is significantly higher in normal mothers than in GD mothers. The index is increased by about 90% in late normal pregnancy compared with 40% in GD.
- When food is ingested, the exaggerated maternal insulin response acts to dampen catabolism but is insufficient, to prevent augmented postprandial fuel excursions so that a significant portion of the ingested calories is made available for fetal anabolism. The response to feeding therefore promotes increased delivery of ingested carbohydrate to the fetus through exaggerated and prolonged hyperglycaemia, while fatty fuels in the form of triglycerides remain available for maternal oxidation, resulting in "facilitated anabolism".⁽⁶⁾

2. **Blood Glucose:** Despite starting from lower preprandial levels, glucose attains greater and more prolonged postprandial elevations in late pregnancy than in the non-pregnant. This facilitates placental transfer to the fetus.

3. **Triglycerides:** Like glucose, post-meal triglyceride levels are elevated in pregnancy.

4. **Free fatty acid (FfAs):** After eating, FfAs are suppressed to approximately the same degree in pregnant and non-pregnant women; but they rebound to the high pre-meal levels more rapidly in late pregnancy.

5. **Amino acids:** Amino acid levels are depressed during pregnancy, but they demonstrate significant postprandial rises, though not as high as in the non-pregnant.

Basis of the Changes in Intermediate Metabolism

1. Pregnancy hormones that are diabetogenic
2. Glucagon
3. Placental insulin degradation.

1. Pregnancy hormones that are diabetogenic

In pregnancy, there are hormones, which act against the effects of insulin and thereby contribute to the increase in insulin resistance. These counter-regulatory hormones are: hCG, hPL, prolactin, cortisol, oestrogen, and progesterone. With the exception of hCG, these hormones are produced in increasing amounts as pregnancy advances.

The predominant hormone is hPL. **Human placental lactogen** is a single polypeptide chain hormone. It is secreted in increasing amounts by the mature syncytiotrophoblast and is detectable by immunofluorescence techniques as early as the 3rd week after fertilisation (5 weeks' menstrual age). Its concentration rises progressively to reach a peak (5-15 g daily) at 34 weeks; after which it levels (1g secreted daily).

The rate of hPL secretion is proportional to placental mass. It is virtually confined to the maternal vascular compartment, and to a lesser extent to the amniotic space. Very little is found in the fetus, in fetal urine, or in maternal urine. HPL has much in common with hCG (96% homology in the amino-acid sequence) and to a lesser extent with prolactin (67 homology). HPL has metabolic effects, which together ensure adequate supplies of glucose, amino-acids, and FfAs to the fetus.

However, the fact that successful pregnancies have been known to occur in the absence of hPL proves that hPL is not crucial for successful pregnancy outcome. Rather, it functions as a fail-safe mechanism to ensure nutrient supply to the fetus in times of maternal starvation.⁽⁷⁾ The metabolic effects of hPL include:

- i. Lipolysis and an increase in the levels of circulating FfAs, thereby providing a source of energy for maternal metabolism and fetal nutrition.
- ii. Inhibition of both the uptake of glucose and of gluconeogenesis in the mother, thereby sparing

both glucose and protein. These actions have the following results:

- * They cause a rise in the maternal sugar level, thus facilitating transfer of glucose to the fetus.
- * They favour maternal protein synthesis over maternal protein utilisation; which in turn, ensures a source of amino acids that can be mobilised for transport to the fetus.

2. Glucagon secretion

The contribution of this to the diabetogenicity of pregnancy is insignificant in women who do not have pre-gestational diabetes. In pre-gestational diabetes, glucagon secretion increases relative to insulin secretion so that the fasting molar insulin/glucagon ratio is decreased. Although fasting plasma glucagon is increased in normal and GD pregnancy, there is a concomitant greater increase in insulin so that the insulin/glucagon ratio is elevated rather than decreased.

2. Insulin degradation by placenta

Insulin is predominantly degraded by the liver, but can also be degraded by placental insulinase. However, the human placental mass is so small relative to the weight of the mother that the amount of insulin captured and degraded by the placenta is insignificant.

II. Renal Function in Pregnancy

Very early in pregnancy, renal plasma flow (RPF) increases from the non-pregnant level of 1.2 L/min to 1.5 L/min by the end of the 2nd trimester, an increase of over 30%. Associated with the rise in the RPF, there is an even greater increase in the glomerular filtration rate (GFR) from 110 ml/min in the non-pregnant to 170 ml/min by the end of the 2nd trimester, an increase of over 50%. This increase is maintained until term. The high GFR increases the amount of glucose filtered per unit time and handed down to the proximal tubule for re-absorption, i.e. it increases the glucose load. The glucose load may exceed the re-absorption capacity of the tubules. On these occasions, the excess glucose will spill over in the urine, producing glycosuria. Probably, there is also a reduction in the inherent ability of the proximal tubule to reabsorb glucose. A third possible contributory factor in the glycosuria is the higher glucose level reached after a carbohydrate meal. Because of these mechanisms, the average renal threshold for glucose is reduced from 10.7mmol/L in the non-pregnant to 8.5mmol/L in pregnancy. Isolated light glycosuria is therefore common in normal pregnancy; 40-50% of women will show glycosuria at some time during pregnancy. Though

in most cases the glycosuria will not be because of abnormally high blood glucose levels, yet these losses will complicate diabetic control.

III. Nausea and Vomiting

Hypoglycaemia may result if the patient gives herself her insulin injection and she cannot eat because of the nausea and vomiting of pregnancy. On the other hand, if the nausea and vomiting produce severe starvation, this may cause insulin resistance and keto-acidosis.

IV. Infections

Some infections, such as furunculosis and pyelonephritis, are more common and more severe in pregnancy. They may result in insulin resistance and keto-acidosis unless promptly and adequately treated.

V. The Work of Labour

The vigorous muscular exercise of labour and the relative starvation during labour may result in hypoglycaemia if the usual dose of insulin is given before labour and intravenous glucose infusion is not set up to cover the labour.

VI. Fall in Postpartum Insulin Requirements

Within a few hours of delivery, the insulin requirements fall to pre-pregnant levels. If the insulin dose is not reduced accordingly, the patient will become hypoglycaemia. In 'true' GD, the need for insulin ceases after delivery.

VII. Breastfeeding

Breastfeeding requires additional calories, and may require an increase in insulin dose above pre-pregnant doses.

Effects of Diabetes on Pregnancy

Diabetes complicating pregnancy has implications for the fetus, neonate, and mother.

A pregnancy that is complicated by untreated diabetes, or by poorly controlled diabetes, has a high maternal mortality and an even higher perinatal mortality. Because of the adaptations in intermediate metabolism discussed above, considerable demands are placed on insulin secretory reserves to maintain normal maternal metabolism in pregnancy. When insulin is insufficient to meet these demands, as happens in the various degrees of glucose intolerance, significant derangement in maternal metabolism ensues.

in the mildest form of diabetes, i.e. in women with GD and normal fasting blood glucose (FBG) levels, these changes occur predominantly in the fed state, with exaggerated oscillations of every maternal fuel after eating.

As insulin action becomes progressively deficient, fasting hyperglycaemia supervenes in association with a further augmentation of postprandial fuel excursions.

At the extreme end of the diabetic spectrum, is the woman with insulin-dependent diabetes and absent insulin secretion, who is totally dependent on exogenous insulin for metabolic control. When insulin is withheld from such a patient, she may rapidly develop keto-acidosis owing to the heightened potential for fat metabolism that is a normal adaptation of pregnancy.

Normal metabolic patterns are more difficult to achieve with insulin administration than is the case in the non-pregnant. Thus, the entire spectrum of maternal diabetes is attended by quantitative and/or qualitative changes in the maternal fuel mixture. These changes are reflected in the metabolic environment of the conceptus.

Before the discovery of insulin by Banting and Best in 1921, it was rare for diabetic women to become pregnant. Many did not live long enough to reach the reproductive years. Of those who lived that long, many (50%) were amenorrhoeic and infertile. The maternal mortality in the few who achieved a pregnancy was about 50%. The spontaneous abortion rate was high and if the pregnancy reached viability, the perinatal mortality was 60%. This figure fell to 25% in the 1940s.⁽⁶⁾ In addition to the mortality, there was considerable maternal and neonatal morbidity.

Uncontrolled or poorly controlled hyperglycaemia, antedating the pregnancy and operating during the pregnancy, was responsible for this horrendous wastage.

Our understanding and management of the condition is now much better. As a result, there has been a dramatic improvement in outcome for both the mother and her infant. Now, hardly any mother dies because of diabetes in pregnancy; and in most centres, the perinatal mortality is now less than 3%.

Even so, this is 2 to 3 times the figure in non-diabetic mothers.

Causes of Increased Maternal Morbidity and Mortality

1. The diabetes is more difficult to control. Keto-acidosis and hypoglycaemic episodes occur more frequently than in the non-pregnant.
2. PIH: The incidence is higher.
3. Infections are more frequent and more severe and complicate control of the diabetes.
4. Macrosomia can cause dystocia that results in traumatic injury to the mother. The injury may be lower genital tract lacerations and haematomas or uterine rupture; VVF and recto-vaginal fistulae may follow. The dystocia is commonly due to shoulder dystocia, since the brain and therefore the head are not involved in the overgrowth.
5. Polyhydramnios is common, and together with macrosomia may cause maternal cardio-respiratory distress.
6. The uterine overdistension caused by the macrosomia and/or polyhydramnios, and the large placental bed commonly associated with diabetic pregnancy, predispose to primary PPH.
7. The caesarean section rate is higher. Caesarean section has higher maternal morbidity and mortality rates than vaginal delivery.
8. Retinopathy: Pregnancy is an independent risk factor for progression of retinopathy. Therefore, ideally, if retinopathy is present and the patient is considering pregnancy, the retinopathy should be successfully treated with laser photocoagulation before pregnancy.
9. Nephropathy: A dramatic increase in proteinuria in the 3rd trimester and hypertension are common.
10. Macrovascular disease: This refers to clinical disease caused by atherosclerosis of the cerebral, coronary, and peripheral arteries.

Causes of Increased Fetal and Neonatal Morbidity and Mortality.

Introduction Fetal growth determinants

Growth and development of the human conceptus occur within the metabolic medium provided by the mother, and in the final analysis, they depend on metabolic fuels and tissue-building blocks that are circulating in maternal blood. Once the allantoic placenta is established, these metabolic fuels reach the fetus in proportion to their concentrations in the maternal circulation.⁽⁶⁾ Pregnancy is therefore like a "tissue culture experience" in which maternal metabolism, as regulated to a large extent by maternal insulin levels, determines the culture medium that is provided to the conceptus.⁽⁶⁾

Alterations in this milieu, as occur in diabetes, may be expected to significantly influence both embryo and fetal development. This in fact, is the case.

Normal fetal growth

Normal growth of the fetus is controlled by the interplay of:

1. The genetic drive to growth.
2. Environmental factors.
3. Trans-placental supply of nutrients to the fetus

In early gestation, genetic drive is the dominant factor. Fetal growth up to mid-gestation is primarily a result of increasing cell numbers, i.e. hyperplasia. On the other hand, in the 3rd trimester, fetal growth is predominantly the result of increasing cell size (hypertrophy) and fat deposition. Growth during this period is primarily determined by the supply of nutrients to the fetus.

The main hormones that regulate fetal growth are insulin and insulin-like growth factors; and these are gestational age-dependent. Growth hormone and thyroxine do not have significant roles in intra-uterine growth.

Although insulin is present in the 8 to 10 week-old fetus, it has relatively little effect on growth until 20 weeks' gestation when the β -cells begins to respond to blood glucose levels. This response is related to how much glucose is transferred by facilitated diffusion from the maternal circulation to the fetus. Insulin receptor numbers become maximal at 19-25 weeks, but receptor affinity to insulin occurs only in late gestation (28 to 32wks).

Pedersen hypothesis, fetal hyperglycaemia and macrosomia

The hypothesis states that because the placenta readily transfers glucose from the maternal to the fetal circulation, maternal hyperglycaemia is transmitted to the fetus, and fetal hyperglycaemia results.

Fetal hyperglycaemia stimulates the fetal β -cells to secrete insulin, and fetal hyperinsulinaemia results. The size of insulin is too large for placental transfer. Fetal insulin cannot therefore cross to the maternal side of the placenta to help restore maternal blood glucose to normal levels. Chronic maternal hyperglycaemia, therefore, results in persistent hyperglycaemia in the fetal compartment.⁽¹¹⁾ Chronic fetal hyperglycaemia causes chronic fetal β -cell stimulation, which causes fetal β -cell hyperplasia and hypertrophy, resulting in chronic hyperinsulinaemia in the fetus. The chronic hyperglycaemia together with the chronic hyperinsulinaemia cause excessive fetal anabolism and excessive growth of insulin-

sensitive fetal tissues. The fetal organs that are particularly sensitive to the growth-promoting effect of insulin are the heart, liver, lung, spleen, adrenal pancreas, and muscle. The **brain and kidneys** are not affected.⁽²²⁾ There is thus **selective organomegaly**, resulting in **asymmetric macrosomia**, with head size remaining normal and body size increasing disproportionately. In GD and in pre-gestational diabetes without vasculopathy, a direct relationship exists between the blood glucose level and the rate of macrosomia. In non-diabetic pregnant women, the mean normal blood glucose is 5.40.9mmol/L. Strict glycaemic control reduces the incidence of macrosomia to near that of the general population, i.e. 10%.

Levels of other maternal fuels, apart from glucose are raised in poorly controlled and uncontrolled diabetes. These fuels reach the fetus in excessive quantities and contribute to the fetal hyperinsulinaemia, increase in fetal β -cell mass, and macrosomia. These fuels include FFAs, triglycerides, amino acids (particularly alanine, serine, and isoleucine).

Most of the adverse perinatal outcomes seen in the infants of diabetic mothers (IDM), have been attributed to **fetal hyperglycaemia and fetal hyperinsulinaemia**.

The incidence of all the following **complications are increased**:

1. Abortion: The incidence is much higher than in the non-diabetic population.
2. Major congenital anomalies: Hyperglycaemia is the specific teratogen; and it is dose-dependent. Establishing strict metabolic control prior to conception and maintaining it throughout the first 8 weeks of pregnancy reduces the incidence of anomalies.
 - * Sacral agenesis (caudal regression syndrome): This is the malformation most characteristic of diabetic embryopathy, but it is not the most common.
 - * Situs inversus
 - * Central nervous system malformations, particularly, holoprosencephaly, anencephaly, and spina bifida, are the most common
 - * Cardiac defects, particularly, ventricular septal defect, transposition of the great vessels, cardiomyopathy in the form of hypertrophic subaortic stenosis from hypertrophy of the interventricular septum. The incidence of congenital heart disease in the non-diabetic population is 0.6%; the incidence in insulin dependent pre-gestational diabetes is 3%; this is 5 times the non-diabetic incidence

Note: Gestational diabetes is not associated with congenital anomalies because it occurs after the first trimester

3. **Macrosomia:** Can cause a difficult and traumatic delivery. Shoulder dystocia is the cause of trauma in these babies because of an asymmetrically overgrown body with a normal-sized head.
4. **Intrauterine growth restriction:** The incidence is high when vascular or renal complications are present. IUGR may also be caused by severe PIH
5. **Prematurity:** Hyperglycaemia-related complications such as severe PIH, polyhydramnios, chronic fetal distress and macrosomia can cause spontaneous preterm delivery. Inability to control the hyperglycaemia may indicate iatrogenic preterm delivery
6. **Sudden unexplained intra-uterine fetal death:** Although it has been drastically reduced by good glycaemic control, vigilant fetal surveillance, and prompt delivery when indicated, unexplained IUDs is still 4 times the rate in non-diabetic mothers. It can occur with alarming suddenness in spite of fetal surveillance. It is therefore advisable to deliver these babies at 38 completed weeks. Clinical factors that have been found to be associated with this disaster are:
 - Poor diabetic control
 - Gestation 34 weeks
 - Vasculopathy
 - Intra-uterine growth restriction
 - PIH
 - Keto-acidosis. Note that this is more dangerous for the baby than hypoglycaemia
 - Polyhydramnios

The immediate cause of unexplained IUD is most probably **acute on chronic hypoxia secondary to changes in red cell oxygen transport and reduced placental blood flow.** The following facts could support this supposition:

- Infants of diabetic mothers, especially of poorly controlled diabetics, have polycythaemia with high Hb values, increased numbers of RBCs and nucleated RBCs in peripheral blood
- Increased erythropoietin levels
- Extra-medullary erythropoiesis, with increased fetal liver weight

The change in oxygen transport is a shift to the left of the oxygen-haemoglobin dissociation curve. The shift causes an increase in the affinity of Hb for O₂, and therefore a reduction in the O₂

released by the RBC to the tissues.

- * It is thought that hyperglycaemia and hyperinsulinaemia in the presence of minimal degrees of hypoxia can cause lactic acidosis and fetal death.
- * Reduced uterine blood flow contributes to IUGR in pregnancies complicated by diabetic vasculopathy, with or without superimposed PIH. These IUGR fetuses are more likely to suffer sudden death. Hyperglycaemia has been shown to reduce uterine blood flow.⁽²¹⁾
- * Hypovolaemia and hypotension occur in ketoacidosis and may reduce intervillous blood flow.
- * PIH is common in diabetic pregnancy. Hypovolaemia, haemoconcentration, increased blood viscosity, narrowing and vasospasm of the spiral arterioles occur in PIH, and can reduce intervillous blood flow.
- * Respiratory distress syndrome (RDS) The causes of the RDS are:
 - Hyaline membrane disease (HMD). This is the most serious cause
 - Wet lung syndrome (transient tachpnoea).
 - Aspiration syndrome.

The factors in the pathogenesis of HMD are:

- * Deficient production and/or secretion of surfactant.
- * Secretion of a surfactant that is deficient in the proportions of its constituents.
 - Structural immaturity of the lungs so that there is deficiency in elastin and in collagen metabolism

All three are more common in the infants of diabetic mothers even when gestational age is controlled. Infants delivered by caesarean section are more susceptible; male infants have a higher incidence. Infants of diabetic mothers who are delivered before term have 6 times the incidence of RDS seen in gestation-matched infants of non-diabetic mothers'.

⁽²³⁾ Hyperglycaemia, hyperinsulinaemia, or both are involved. There is a delay in the synthesis of surfactant and the connective tissue proteins, collagen and elastin. Glycogen is a substrate for surfactant phospholipid synthesis. Since insulin inhibits lung glycogen breakdown, it may act by decreasing the substrate flow from glycogen. Insulin may also antagonise the effect of glucocorticoids on lung maturation, possibly at the level of fibroblast pneumocyte factor.

Amniotic fluid L/S-ratio studies are not very helpful in diabetic pregnancy. On the other hand, phosphatidylglycerol levels are reliable since at term the levels are comparable to those of non-diabetics.

8. Polycythaemia is common and can cause hyperviscosity syndrome
9. Hyperbilirubinaemia, i.e. serum bilirubin 200µmol/L is common
10. Caesarean section: The caesarean section rate is higher. Caesarean section is associated with a higher incidence of RDS
11. Hypoglycaemia, hypocalcaemia, and hypomagnesaemia: These are common in the early neonatal period
12. Long-term sequelae in offspring: Fat, neural tissue, pancreatic islets, and other tissues, which are capable of only minimal or no post-partum differentiation, may be permanently affected by the metabolic insult of maternal diabetes in utero. That children of diabetic mothers have an increased risk of adiposity, impaired intellect, and diabetes in later life may suggest that this does occur.

Reasons for Improved Perinatal Outcome

The perinatal mortality associated with diabetes has dropped 10-fold in the past 50 years compared to a 5-fold fall in the general population. The reasons for this dramatic fall are multifactorial but 4 important advances in management which have contributed to the fall are:

1. Tight glycaemic control before and during the whole of pregnancy: Tested programmes, including and home glucose monitoring have made this possible.
2. Fetal surveillance to assess fetal welfare: This has allowed prolongation of pregnancy to near term in most cases, and prompt delivery of babies at risk of intrauterine death
3. Assessment of fetal lung maturity before elected termination of pregnancy has helped to reduce the incidence of RDS
4. Improved understanding of the needs of the neonate, together with improved technology in neonatal intensive care

Diagnosing Diabetes in Pregnancy

The pre-gestational diabetic is already diagnosed. The gestational diabetic is diagnosed during the pregnancy, using:

- Historical screening
- Physical signs and symptoms in index pregnancy
- Urine testing for glucose
- Blood glucose tests.

I. Screening Tests

Historical screening

1. Sociodemographic: ethnicity, locality noted for Type II diabetes.
2. Family history: First degree relative.
3. Past obstetric history: macrosomia, unexplained perinatal death.

Physical signs and symptoms in this pregnancy

1. Maternal weight 90kg, or, 20% above ideal weight.
2. Polyhydramnios.
3. Estimated fetal weight 4,000g
4. Severe or recurrent candidiasis or furunculosis
5. Polydipsia and polyuria.

Investigative tests in this pregnancy

1. Qualitative test for urine glucose, using glucose-specific dipsticks. Repeated or heavy glycosuria is suggestive and indicates OGTT.
2. Random blood glucose (RBG), i.e. untimec sample with respect to the last meal, but not fasting, is taken at booking visit, and if negative repeated at 28-32 weeks' gestation. Interpretation is as follows. If the RBG level is:
 - i. <7.0mmol/L, it is normal and the patient does not have diabetes.
 - ii. 7.0-10.9mmol/L, an oral glucose tolerance test (OGTT) is performed.
 - iii. >11.0mmol/L, the patient is a diabetic and no further testing is necessary.
3. The 50g, 1-hour oral glucose challenge test

ii. Diagnostic Tests

1. **Fasting blood glucose**
A fasting blood glucose level above 6.0mmol/L is diagnostic of diabetes mellitus.
2. **Random (untimed) blood glucose**
A random blood glucose value 11.0mmol/L is

Interpreting the OGTT Results	Venous Plasma Glucose in mmol/L	
	Fasting Level	2-hour Level
Normal subject	<6.0	<8.0
Impaired glucose tolerance	6.0-7.9	8.0-10.9
Diabetes mellitus	>8.0	>11.0

diagnostic of diabetes mellitus.

3. Oral glucose tolerance test (OGTT)

This is the universally traditional test for diagnosing glucose intolerance of varying severity. It is the "gold standard".

Procedure for the OGTT

- For at least 3 days before the test, the subject should be on an unrestricted diet including a minimum of 150g of carbohydrate daily.
- Prior to testing, the patient should fast overnight for at least 10hrs and for not longer than 16hrs.
- She should remain seated during the test.
- She should not smoke during the test.
- A fasting blood glucose specimen is taken.
- The patient is given 75g of glucose in water to drink within 5 minutes.
- One hour later, a second blood glucose specimen is taken.
- A 3rd blood glucose specimen is taken 2hrs after the glucose drink

Note: If the patient is symptomatic, i.e. if polydipsia and polyuria are present, the fasting blood sugar or the random blood glucose will diagnose diabetes mellitus. The OGTT will not be necessary, and is in fact contraindicated.

The Meal Challenge Test (Glucose profile)

As a determinant of maternal hyperglycaemia under normal living conditions, the meal challenge test is more valid than the glucose tolerance test. However, it has not been widely used for

diagnosis; but it makes physiological sense to use it in deciding if a woman diagnosed

by the OGTT as a gestational diabetic, needs glycaemic control.

Procedure for the Meal Challenge Test

- At 9am, a fasting (pre-breakfast) venous blood is taken for glucose pre-breakfast sample
- Patient is given breakfast, consisting of 350 calories and 40g of carbohydrate
- Two hours after breakfast, venous blood is taken for glucose post-breakfast sample
- At 1pm, just before lunch, blood is again taken for glucose pre-lunch sample
- Patient is given lunch, consisting of 350 calories and 40g of carbohydrate
- Two hours after lunch, venous blood is taken for glucose post-lunch sample

Interpreting the Meal Challenge Test Result.

Blood glucose levels 8.0mmol/L are abnormal.

Medical Management

Introduction

The aim of medical management is to reduce perinatal morbidity and mortality and maternal morbidity and mortality to the levels seen in the general population. The principle underlying successful management is satisfactory glycaemic control before conception and during pregnancy and labour. Satisfactory glycaemic control reduces the incidence of the complications that are responsible for the increased morbidity and mortality.

Preconception Clinic

- * Counsel couple.
- * Achieve euglycaemia before conception.
- * Manage complications that deteriorate in pregnancy nephropathy, retinopathy and macrovascular disease
- * Counsel on contraception
- * Establish rapport between medical team and patient and her husband

Joint Antenatal Care (Team Approach)

Antenatal control of diabetes

- Initial admission for stabilisation
- Weekly antenatal visits
- Monthly glucose profile, and restabilisation, if necessary
- Diet and weight control
- Exercise
- Insulin therapy

Effective monitoring of therapy

- Blood glucose profiles
- Glycosylated haemoglobin
- Urinary ketones and glucose

Medical management of delivery

- Spontaneous labour
- Induced labour
- Elective caesarean section

Postpartum management

- Reduction in insulin dosage
- Breastfeeding

Components of Successful Medical Management

I Pre-Conception (Pre-pregnancy) Clinic

Objectives of Pre-Conception Clinic

1. To counsel the couple

To counsel the couple on the effects of pregnancy on diabetes and vice versa and their implications for the mother, pregnancy, and child.

2. To achieve normoglycaemia before conception

To achieve normoglycaemia before conception so as to reduce the risk of congenital anomalies down to the risk in non-diabetic pregnancy.

3. To establish rapport between the couple and members of the medical team

The importance of co-operation between the couple and the medical team is stressed. This promotes compliance. Next to availability of the medical facility for proper diabetic management, compliance is the most important predictor of pregnancy outcome.⁽²⁴⁾ Issues discussed include:

- i. Fertility: Well controlled diabetes has no effect on fertility
- ii. Genetic counselling: Type I diabetes is a consequence of insulin deficiency due to an immunologically induced destruction of the β -cells of the islets of Langerhans in genetically susceptible individuals. The offending factor may be a virus or a chemical. The disease is therefore caused by the interplay of hereditary and environmental factors. In the general population, the prevalence of Type I diabetes is 0.15% (roughly 1 in 1000) all over the world and it is the same in all mammals. Siblings of Type I diabetics have a 5% to 10% risk of developing diabetes. The offspring of a parent with Type I have a 5% lifetime risk of developing Type I diabetes. The rate of concordance for Type I diabetes in genetically identical twins is 50%. This is higher than is seen in dizygotic twins and would suggest a strong genetic predisposition. On the other hand, since the concordance is not 100%, genetic factors alone cannot be responsible for the disease; environmental

factors must play a part. Only 10% to 15% of all Type I diabetes cases are familial; the rest occur sporadically in the community. A family history is more common in Type II diabetes than in Type I. For the offspring of Type II maturity-onset diabetes, the lifetime risk is 50%.

- iii. Effects of pregnancy on existing chronic diabetic complications:
 - A. Micro-vascular (retinopathy and nephropathy)
 - B. Macro-vascular disease (cerebral, cardiac, peripheral arterial disease)
 - C. Metabolic and micro-vascular (neuropathy)

4. To manage diabetes complications that deteriorate in pregnancy

Diabetic retinopathy

Diabetic retinopathy is the result of retinal arteriolar and capillary endothelial cell damage, basement membrane thickening, and pericyte damage. It is classified into three levels of increased severity:

Non-proliferative (background) retinopathy: characterised by exudates, micro-aneurysms, and small red dot haemorrhages in the retina.

Pre-proliferative retinopathy

Proliferative retinopathy: characterised by growth of new capillaries in the retina, retinal neovascularisation on the optic disc or elsewhere. These new vessels may extend into the vitreous and rupture, causing vitreous haemorrhage. In some cases of vitreous haemorrhage, organisation may be followed by scarring and traction detachment of the retina, resulting in permanent blindness.

Pregnancy is an independent risk factor for progression of retinopathy. Therefore, retinopathy should be successfully treated with laser photocoagulation before pregnancy. The presence of retinopathy without hypertension or renal disease does not adversely affect fetal survival, gestational age, or birth weight.

Nephropathy

Overt nephropathy is defined as the presence of persistent dipstick proteinuria (300mg/day), or, an albumin excretion rate 300mg/day, or, a total protein excretion rate 500mg/day, in the absence of bacteriuria. Diabetic renal disease progresses through distinct stages as follows:

- a. Increased size of the kidneys from hyperplasia of nephrons with basement membrane, elevated GFR by 30-40% (hyperfiltration). Strict glycaemic control can reverse these changes. If they do not regress, they may progress to:
- b. Glomerular mesangium thickens and contains

- deposits of albumin, IgG, fibrin, and platelet degradation products. Later there is:
- c. Mesangial expansion and glomerular scarring and shrinkage. Exercise induced proteinuria is present. This is early glomerulosclerosis. May lead to:
 - d. Incipient nephropathy and later, overt nephropathy. GFR is decreased, glomerulosclerosis causes substantial nephron loss; hypertension, retinopathy, arteriosclerosis, coronary artery disease, and neuropathy are commonly present.
 - e. End-stage renal disease: Almost total loss of glomerular function leads to azotaemia, electrolyte disturbances, and death.

Pregnancy does not worsen diabetic renal disease if the diabetes is well managed. However, a dramatic increase in proteinuria in the 3rd trimester and hypertension are common. The heavy proteinuria is associated with oedema. The proteinuria and hypertension fall to pre-pregnant levels after parturition. Nephropathy increases the incidence and severity of the following complications:

- IUGR
- Fetal distress
- PIH
- Preterm delivery: Over 50% do not reach 37 weeks; over 20% do not reach 34 weeks; all do not reach 40 weeks
- Perinatal death occurs in about 10%. A serum creatinine level of >250mol/L is incompatible with successful pregnancy

Macro-vascular disease

This refers to clinical disease caused by atherosclerosis of the cerebral, coronary, and peripheral circulation. Maternal mortality is high in pregnancy complicated by diabetic coronary artery disease.

5. To counsel on contraception

The couple must prevent pregnancy until satisfactory diabetic control has been achieved. They must space and limit their family.

1. Joint Antenatal Care

1. Obstetrician
2. Diabetic physician
3. Diabetic neonatologist
4. Dietician
5. Diabetic nurse specialist
6. The patient's family doctor

The risks of diabetic pregnancy may result from metabolic, obstetrical, or neonatal complications. Therefore, a multidisciplinary team approach to management gives the best results.

III. Antenatal Control of Diabetes

1. Initial admission
2. Weekly clinic visits
3. Readmission each month for blood glucose profile, and re-stabilisation, if necessary
4. Diet
5. Exercise
6. Insulin therapy
7. Danger of keto-acidosis

Initial admission

The patient is admitted as soon as pregnancy is diagnosed. The objectives of admission are:

- * Blood glucose profile and stabilisation
- * Assessment of vascular complications
- * General diabetic education
- * Baseline obstetric evaluation
- * Teaching the patient how to inject insulin and how to use reflectance meter to monitor capillary blood glucose if the patient has the capacity to learn

Dietary Control, Weight Gain And Exercise

Calorie intake should be 1800 to 2000 calories per day, or, 30-35cal/kg of ideal body weight per day. The higher amount, i.e. 35cal/kg, is used if the pre-pregnant weight was less than ideal body weight. If the patient was obese before onset of the pregnancy; i.e. if the pre-pregnancy weight was greater than 120% ideal body weight or, more than the 90th percentile, then 30cal/kg is used.

The diet should be individualised for the woman, and should take into account her ethnic background and the type of food she and her family normally eat. This will reduce disruption of the family's eating habits and enhance compliance.

Calories are distributed between 3 evenly spaced meals and a bedtime snack. The snack is included to make sure the duration of overnight fasting is less than 12h.

Energy distribution is as follows:

- * Carbohydrate: 200g or more of a high fibre carbohydrate provide 50% or more of the total calories. Large amounts of concentrated and refined sugars that may cause rapid swings in circulating glucose levels should be avoided.
- * Fat: 60g or less of fat provide 30% or less of the total calories
- * Protein: 100g of protein provide 20% of the total calories

Weight gain: On this calorie intake, women of normal pre-pregnancy weight should not gain more than 10kg by term; not more than 3kg in the first 20wks of pregnancy, and not more than 7kg in the second half of pregnancy. Women who were overweight before pregnancy may gain a little less than the 10kg, and those who were less than ideal body weight may gain a little more than the 10kg by term.

Exercise: Exercise in diabetic control; it reduces insulin requirements. Hospital in-patients have less exercise than they have when at home. Their insulin requirements are therefore higher. They must be encouraged to exercise in hospital, unless there is a contraindication.

	1 st Trimester	2 nd trimester	3 rd trimester	Soluble/Isophane ratio
Type I	0.86(52)	0.95(57)	1.19(72)	20:10
Type II	0.86(52)	1.18(71)	1.62(97)	20:10

Table 2. The pharmacological basis of the insulin adjustments

Insulin	Onset of action (min)	Peak action (h)	Duration of action (h)	Glucose value controlled
Soluble	30-60	2-4	8	2h post-breakfast, 2h post-dinner
Isophane	60-120	4-8	16-24	Post-lunch, fasting

Insulin therapy

Insulin is usually given as a mixture of regular (fast-acting, soluble, insulin zinc crystals, crystalline insulin) and isophane insulin suspension (NPH N for its neutral pH of 7.2, P for its protamine zinc content, and H for Hagerdon's Laboratory, which formulated it).

The two are mixed in the proportion 20 regular to 10 isophane. The insulin dosage must be adjusted to produce and maintain a normal blood glucose profile, i.e. pre-prandial concentrations 6.0mmol/L, and 2hr post-prandial values 7.0mmol/L. Two injections are given daily: one before breakfast, and the second before dinner.

The insulin requirements for Type I and Type II diabetes increase throughout pregnancy, with the increments required for Type II being significantly higher than for Type I. For both types, the increments occur in 3 phases that correspond to the trimesters of pregnancy. This triphasic pattern mimics the increases in the diabetogenic hormones of pregnancy.

The average daily insulin requirements in the 3 trimesters are shown in Table 1. In general, a woman with well-controlled diabetes can expect to almost double her pre-pregnancy dose by 30wks. After 30wks the need for increasing insulin usually steadies.⁽¹²⁾

Pharmacological basis of insulin adjustment

The pre-breakfast soluble insulin is adjusted to normalise the 2hr post-breakfast glucose value. The pre-breakfast isophane insulin is adjusted to normalise the 2hr post-lunch glucose value. The pre-dinner soluble insulin is adjusted to normalise the 2hr post-dinner glucose value. The pre-dinner isophane is adjusted to normalise the fasting glucose concentration (Table 2).

Table 1. Daily insulin requirements in units/kg body weight. Daily doses for a 60kg woman are given in brackets. Adapted from Langer et al⁽²²⁾

Satisfactory glycaemic control values

Fasting and pre-prandial levels should be between 3.0 and 6.0mmol/L and 2-hr post-prandial levels should be between 4.0 and 7.0mmol/L.

Diabetic ketoacidosis

This complication is particularly disastrous for the baby and must not be allowed to occur. Because of the tendency for "accelerated starvation" in pregnancy, pregnant diabetic patients may develop keto-acidosis very quickly and in the presence of only mild elevations of blood glucose. Keto-acidosis may occur during intercurrent illness or because of inadvertent interruption of insulin therapy. The patient must be strongly counselled to always take

her insulin even if the meal has been vomited. She should then take 10g glucose (two teaspoonfuls of granulated sugar) and report to hospital. If because of nausea or neuroglycopenic uncooperativeness she is not able to take the sugar, the husband should give her 1mg subcutaneous injection of glucagon. This will release enough hepatic glycogen to raise the blood sugar, and she should then take a small snack.

In diabetic subjects, intravenous tocolysis with -sympathomimetic drugs, e.g. salbutamol, can cause rapid metabolic deterioration to hyperglycaemia, hyperinsulinaemia, and ketoacidosis. The concurrent administration of glucocorticoids to mature the lungs has an additive effect.

These drugs should not be used in diabetics. Urinary ketones should be measured whenever blood glucose levels exceed 8.0mmol/L. Ketonuria in the presence of this level of hyperglycaemia is an indication for admission and assessment of acid-base status and serum ketone levels.

V. Adequate monitoring of diabetic control

- The meal challenge blood glucose profiles (glucose series)
 - Fasting (pre-breakfast): 3.0-6.0mmol/L
 - 1h or 2hr post-breakfast: 4.0-7.0mmol/L
 - Pre-lunch: 3.0-6.0mmol/L
 - 1h or 2hr post-lunch: 4.0-7.0mmol/L
 - Pre-supper: 3.0-6.0mmol/L
 - 2h post-supper or at 2100hr: 4.0-7.0mmol/L
- Glycosylated haemoglobin (HbA_{1c}) estimations
- Urinary glucose and urinary ketones
- Compliance

The meal challenge blood glucose profiles

In the normal non-diabetic subject, the pancreas maintains normoglycaemia by constantly monitoring the blood glucose level and constantly responding appropriately to it. Therefore, if true or nearly true normoglycaemia is to be achieved in the pregnant diabetic, the blood glucose needs to be sampled many times during the day, both before and after meals, and the insulin dose adjusted accordingly. This requires admission of the patient. Once glycaemic control has been achieved, with stability in the level of diabetic control, a less stringent number of samples, e.g. three per day, can be used. ⁽²²⁾

Glycosylated haemoglobin (HbA_{1c})

Glucose may react with N-terminal amino acid residues, e.g. valine, of the α -chains of HbA to form a Schiff base (an aldimine), which may undergo Amadoric re-arrangement to produce a stable ketoamine (glucose-CH₂-NH-B-A), i.e. HbA_{1c}. The reaction occurs continuously and slowly throughout the life-span of the RBC, i.e. 120 days. Because the product is stable, HbA_{1c} accumulates throughout the life-span of the RBC. The concentration of HbA_{1c}, i.e. the proportion of haemoglobin that has been glycosylated, is a function of the blood glucose concentrations over a long period of time. Therefore, the concentration of HbA_{1c} is correlated to the degree of diabetic control, so that a quantitative determination of HbA_{1c} reflects the patient's mean blood glucose concentration in the previous 4 to 8 weeks. Monthly measurements are therefore more than adequate in providing retrospective information on the validity of home control status. Accelerated haemolysis, as may occur in haemoglobinopathies and in malaria, may affect the results.

Capillary blood glucose (mmol/L)	Units soluble insulin added to 500ml infusion	Time (hrs) for infusion (rate in drops/min)	Supplementary subcutaneous insulin units given every 6hrs
<2.0	Nil	2h (84)	0
2.0-3.9	Nil	6h (28)	0
4.0-7.9	6	6h (28)	0
8.0-11.9	6	6h (28)	6
12.0-15.9	6	6h (28)	10
≥16.0	Call diabetic physician		

affect the results.

In summary, the applications of glycosylated haemoglobin during pregnancy are:

- * When measured in the late first or early second trimester, it may reflect average blood glucose control during the critical period of organogenesis. If an elevated value is obtained, it may thereby predict an increased risk for major congenital anomalies
- * When measured serially at any time during pregnancy, the values may provide evidence for patients, that their efforts towards better diabetic control are effective.
- * When measured at any time during pregnancy, they provide an additional 'check' on the accuracy of patients' self glucose monitoring results.

In normal, non-diabetic subjects, the glycosylated haemoglobin concentration is 6.0%; in well controlled diabetes it is below 8.0%; when the control has been less than good, the figure is between 8.0 and 11.0%; in poorly controlled diabetics it is above 11.0%, and may be as high as 18%.

Urinary Sugar And Urinary Ketones

Urinary glucose monitoring adds little to the assessment of capillary values. Because of the lowered renal threshold for glucose in pregnancy, urinary glucose levels may rather mislead. On the other hand, because of the increased propensity for fat catabolism during most of gestation and the attendant risk of ketonaemia, urinary ketone measurements are important. Urinary ketones must be measured daily, or at any time the fasting blood glucose, or pre-meal blood glucose is above 8mmol/L. Ketonuria in the presence of normal or low blood glucose suggests inadequate dietary intake; but ketonuria in the face of even mild hyperglycaemia may indicate incipient ketoacidosis.

V. Medical Management of Delivery

1. Spontaneous labour
2. Induced labour
3. Caesarean section

Capillary blood Glucose (mmol/L)	Units soluble insulin added to 500ml infusion	Time (hrs) for infusion (rate in drops/min)	Supplementary subcutaneous insulin units given every 6hrs
2.0	Nil	2h (84)	0
2.0-3.9	Nil	6h (28)	0
4.0-7.9	6	6h (28)	0
8.0-11.9	6	6h (28)	6
12.0-15.9	6	6h (28)	10
>16.0	Call diabetic physician		

Table 3. IV infusion of 500ml 5% dextrose

The neonatal outcome is best if the baby is born from a maternal glucose environment of 3.0-6.0mmol/L.

1. Medical management of spontaneous labour

- * Capillary blood glucose is measured hourly and the level kept in the range 3.0 and 6.0mmol/L.
- * The mother should not take her full regular dose of insulin while in labour.
- * An intravenous infusion of 5% dextrose is set up.
- * Insulin administration is adjusted as in Table 3 below.
- * Light food and oral fluids as desired are allowed,

provided blood glucose remains between 3.0 and 6.0mmol/L.

- * The IV dextrose drip is discontinued when the mother is able to eat light meals after delivery.

2. Medical management of induced labour

- * The usual insulin (soluble and isophane) is taken the day before.
 - * The morning insulin and breakfast are not given on the antenatal ward.
 - * Capillary blood glucose is measured hourly from 0600hrs.
- On labour ward:
- * ARM is performed and intravenous syntocin infusion set up at 0800hrs.
 - * One dose of 8 units of soluble insulin is given

subcutaneously.

Light breakfast (tea and toast) is given immediately after the insulin injection.

An IV infusion of 5% dextrose is set up, and insulin administration adjusted as described in Table 3 below.

3. Medical management of caesarean section

Usual insulin dose (soluble + isophane) is taken the day before.

The morning insulin and breakfast are not given. Capillary blood glucose is measured hourly from 0600hrs.

An IV infusion of 5% dextrose is set up, and insulin administration adjusted as described in Table 3 above.

Above infusion is continued until the mother is able to eat after the caesarean section.

Note: Plastic adsorbs insulin. Therefore, to know how much insulin is actually going into the patient, it is necessary to flush and saturate any plastic tubing for insulin infusions with 40 to 50ml of the insulin solution prior to infusion.

4. Medical Management of Postpartum Period

Insulin dosage re-adjustment

The IV 5% dextrose infusion is stopped when the patient is able to eat.

For pre-gestational diabetics, approximately half of the pre-pregnancy dose of insulin is given on the first day.

Blood glucose is measured as follows: fasting, 2h post-breakfast, pre-lunch, 2h post-lunch, and 21.00h

Insulin dosage is adjusted as indicated by the blood glucose profile.

For gestational diabetics, insulin is only given if blood sugar values demand it.

5. Breast feeding

Calorie intake should be increased as over 24 hours the patient loses

approximately 50g of carbohydrate in breast milk. The additional

carbohydrate should be spread over the day and taken prior to breast feeds, including night feeds.

The capillary blood glucose should be 6.0-8.0mmol/L, slightly higher than it was during pregnancy.

* The mother's food should always precede breastfeeding to guard against the risk of the mother becoming hypoglycaemic during feeds.

* The mother should always keep a drink and some biscuits hand during breastfeeding, in case the feeding takes longer than expected.

* The mother should ensure good hygiene and care of the breasts to avoid breast infection.

Obstetrical Management

I. Antenatal obstetrical management.

1. Confirm and date pregnancy; exclude fetal anomaly.
2. Complications associated with diabetes: prevent, diagnose early, manage.
3. Monitor fetal growth to detect excessive growth, or IUGR.
4. Fetal surveillance for fetal wellbeing.

II. Timing of delivery.

III. Mode of delivery.

IV. Management of delivery.

V. Family planning.

I. Antenatal Obstetric Management

1. Confirm and date pregnancy, exclude fetal anomaly

- i. Confirm early pregnancy by ultrasound scan, or -hCG subunit measurement.
- ii. Date age of pregnancy by early ultrasound scan.
- iii. Use early ultrasound scan and targeted scan at 18-20wks to exclude fetal anomaly, especially central nervous system and cardiac lesions.

2. Observe For Complications Associated With Diabetes

The patient must be closely observed for the complications that are known to be associated with diabetes. Appropriate management should be instituted when they are diagnosed. The bad prognostic signs are PIH, pyelonephritis, ketoacidosis, and failure of the mother to follow medical advice.⁽²⁴⁾

- ii. PIH or superimposed PIH: The incidence of PIH in diabetic pregnancy is four times what is found in non-diabetic mothers. The incidence is higher still in the presence of nephropathy and diabetic vascular disease. The perinatal mortality rate in diabetes complicated by PIH is 20 times what is found in diabetes without PIH (6% versus 0.3%).⁽²⁵⁾ Maternal and fetal surveillance using clinical and laboratory investigations are imperative in this condition. Worsening of the condition or

signs of failing fetoplacental function will demand termination of the pregnancy.

- ii. Polyhydramnios: Polyhydramnios is much more common in diabetic than non-diabetic pregnancy. The following hypotheses have been advanced to explain the association between diabetes and hydramnios:

- a. Fetal hyperglycaemia causes fetal polyuria

- b. High concentration of glucose in fetal urine oncologically draws fluid through the membranes into the amniotic fluid

The complications of hydramnios include preterm labour, premature rupture of membranes (PROM), cord prolapse and placental abruption when the membranes rupture. If it is severe and its evolution is rapid, it can cause maternal cardio-respiratory embarrassment.

In all cases of clinical hydramnios, an ultrasound scan to exclude twins and fetal anomaly is mandatory.

Treatment consists of bed rest in a propped-up position. Indomethacin administration to reduce fetal urinary output is useful. If all else fails and the mother is acutely distressed, a slow amniocentesis while suppressing uterine activity with magnesium sulphate may be performed.

- iii. Preterm delivery: Preterm labour can start spontaneously, or after PROM. Pregnancy may be interventionally terminated prematurely in the interests of the mother, fetus, or both. As discussed earlier, the -mimetic drugs and glucocorticoids are contraindicated in diabetics.

- iv. Pyelonephritis: Pyelonephritis complicating diabetic pregnancy demands aggressive management. Once diagnosed, prompt therapy with intravenous administration of the appropriate antibiotics should be started. An ultrasound scan examination of the kidneys and urinary tract should be performed to exclude obstructive nephropathy and a perinephric abscess. The patient should be followed up with repeated MSU cultures to detect recurrences as soon as possible. It is advisable to periodically screen pregnant diabetic patients for asymptomatic bacteriuria

3. Monitor Fetus For Excessive Growth Or IUGR

- I. Symphysio-fundal height: serial measurements.

- II. Ultrasound scans measurement of BPD and abdominal circumference.

- III. Ultrasound scan estimate of fetal weight.4.

Fetal surveillance for fetal well being from 32wks

In the past sudden unexplained intrauterine fetal death (IUD) occurred in 10-30% of gestations complicated by IDDM. Although death occurs most often after the 36th week, and among patients with poor control, hydramnios, PIH, fetal macrosomia, or vasculopathy, sudden IUD does occur in women with satisfactory control and with no obvious complications. In the past a policy of 'delivery before term' was advocated and practised to prevent the tragedy of sudden IUD. However, this resulted in increased neonatal deaths from RDS and was therefore abandoned. Now, antenatal fetal surveillance in the 3rd trimester is used as a component of the management of the pregnant diabetic. The objectives of antenatal fetal testing are:

- i. Early detection of fetal compromise, which allows early delivery so that intrauterine morbidity does not progress to IUD.
- ii. Assurance that the baby is in good health allows delivery to be postponed to 38 completed weeks in the confidence that IUD is most unlikely. Neonatal morbidity and mortality from RDS are thereby reduced. Postponement beyond 38wks can be risky and it is not wise.

The surveillance tests performed include:

- i. Fetal kick count.
- ii. Biophysical profile (antenatal CTG plus ultrasound scan assessment of amniotic fluid volume and fetal parameters tone, gross movement, breathing).

Doppler umbilical artery velocimetry for end-diastolic blood flow.

II. Timing of Delivery

It is well known that infants of diabetic mothers who are delivered preterm have a higher incidence of RDS than gestation-matched infants of non-diabetic mothers.⁽²³⁾ Therefore, if elective termination of pregnancy before 38wks is contemplated, lung maturity should first be ascertained before the termination is performed. Lung maturity can be determined by estimating the Lecithin/sphingomyelin ratio of amniotic fluid and the phosphatidylglycerol concentration in amniotic fluid.

III. Mode of Delivery

Vaginal delivery is preferred because caesarean section delivery has a higher incidence of RDS. However, the following may be indications for caesarean section.

1. Macrosomia with fetal weight estimated at more than 4000g.
2. Previous shoulder dystocia.
3. Where immediate termination of the pregnancy is indicated but the Bishop's score is unfavourable, i.e. less than 4.
4. In the presence of another indication for abdominal delivery.

IV. Management of Delivery

Intrapartum fetal monitoring should be employed.

V. Family Planning

Contraception is important in the management of diabetic pregnancy because it has been clearly shown that the high incidence of congenital malformations in the infants of pre-gestational diabetics can be reduced to the incidence in non-diabetic mothers by establishing and maintaining euglycaemia before conception and through 8wks gestation. It is therefore crucial that pregnancy be planned. It is therefore important that the patient should not become pregnant until normoglycaemia has been achieved. It is advisable to limit the number of babies to two or three. The best option for family completion is permanent surgical contraception, preferably vasectomy. If vasectomy is not possible, tubal ligation should be offered.

For reversible contraception, the low dose oral contraceptive pill is safe. The progestogen-only pill, the medicated intrauterine contraceptive device, and the barrier methods may also be used.

Medical Management of Non-Insulin Dependent Diabetes

The non-insulin dependent diabetic (NIDD) is admitted and monitored as is done for the insulin-dependent diabetic (IDD).

Diet, energy allowance and weight gain: Initial treatment is always dietary. The rules about weight gain and energy allowance and distribution are the same as for the IDD.

Insulin therapy: The criteria for initiating insulin therapy are the same as for IDD. There is generally considerable insulin resistance in NIDD, and a starting dose of 40units daily is often needed, rising rapidly to 100units or more daily if obesity is severe and the patient does not comply with dietary advice. Hypoglycaemia is most unusual. ⁽¹²⁾ The

blood glucose values aimed at are the same as for the IDD.

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Anaemia In Pregnancy

Ivy Ekem and SA Obed

Introduction

Anaemia is defined by the value of haemoglobin (Hb). It is present when the blood haemoglobin value is below the reference value for the age, sex and place of residence of the individual. For females of child bearing age, the normal range is 11.5-16.5g/dl. During pregnancy however, physiological changes that take place entails that the normal values are lower. In a well-conducted longitudinal study in women with adequate iron, the lowest recorded value was 10.4g/dl. Thus a pregnant woman is said to be anaemic when the haemoglobin falls below 10.4g/dl⁽¹⁾. If at the onset of pregnancy the haemoglobin is already lower than 11g/dl, the patient should be treated as anaemic.

Physiological changes and their effect on the haemoglobin level are:

1. An increase in plasma volume of approximately 50%, which occurs during the first 2 trimesters of pregnancy with a corresponding increase in red cell mass of only 20-30%. This results in a dilutional anaemia, which does not reflect a reduction in the oxygen carrying capacity of the blood. Transfer of iron to the foetus in the third trimester and parturition blood loss. These together with the decrease in maternal red cell volume imply a doubling of the daily iron requirement i.e. from 15mg/day in the non-pregnant female to 5mg/day in the 2nd and 3rd trimesters of pregnancy.

2. Folic acid requirements are increased due to increased nucleic acid synthesis in mother and fetus.

Other physiological changes of relevance are adjustments, which allow for immune tolerance of the fetus. These can have pathological consequences e.g. an increased prevalence and severity of plasmodium falciparum malaria infection, an increase in the incidence of aplastic anaemia and increased severity of autoimmune haemolytic anaemia⁽²⁾.

Classification of Anaemia

Anaemia may be classified morphologically or aetiology.

Morphologically, the normal red cell shape and

colour is described as normocytic and normochromic. In situations of anaemia, these characteristics remain or change and this signifies one or the other aetiological factor or factors. The three main morphological distinctions are:

1. Normocytic and normochromic anaemia e.g. dilutional anaemia of pregnancy, bleeding, infection.
2. Microcytic and hypochromic anaemia e.g. iron deficiency,
3. Macrocytic anaemia e.g. Folate or vitamin B12 deficiency.

When the cause is multifactorial, the picture may be dimorphic. Other morphological changes in the red cells themselves, the white cells and the platelets give clues to the cause of the anaemia.

Kinetically, anaemia may be due to:

1. Excessive loss of red cells as in acute or chronic bleeding.
2. Excessive destruction as in haemolysis. Haemolytic anaemia may be due to inherited causes as in sickle cell disease or acquired causes as in autoimmune haemolysis, and haemolysis due to infections like malaria and septicaemia.
3. Inadequate production as occurs in
 - a. Factor deficiencies like iron, Folate, vitamin B12 and protein all vital to the synthesis of haemoglobin.
 - b. Bone marrow atrophy as in aplastic anaemia
 - c. Infiltration of the marrow as in leukaemia
 - d. Miscellaneous as in endocrine disorders, chronic renal failure, chronic inflammatory disease and cirrhosis of the liver.

Anaemia in pregnancy

Maternal mortality in developing countries worldwide is 400/100 000. In Korle-Bu, Ghana, it is 734.4/100 000⁽³⁾ and in the Netherlands 5/100 000. Two to twelve percent of maternal mortality in tropical Africa is primarily due to anaemia.

When anaemia is not the primary cause, it is frequently a contributory factor, especially following haemorrhage. In Korle-Bu, the five leading causes

of mortality are haemorrhage (17.7%), hypertension (17.5%), abortion (13.5%), sickle cell disease (11.0%), and genital infections (9.5%). Anaemia contributes directly to 3 of these.

Anaemia is said to be moderate if the Hb is 7-10g/dl, severe if its <7g/dl and very severe if its <4g/dl. Published rates of prevalence for anaemia in pregnancy in Africa is 35 - 36%⁽⁴⁾ with 20% of the cases below 8g/dl and 27% below 7g/dl. Severe anaemia often occurs in the third trimester¹. Unpublished data by Acquaye et al show prevalence in Ghana of 65% with the highest prevalence in northern Ghana.

In industrialized countries, the incidence is less than 20%⁽⁴⁾.

Aetiology

Any of the various causes of anaemia in the general population can cause anaemia in pregnancy. In tropical Africa, certain causes predominate. The causes however vary from region to region. In many regions, nutrition is a major problem and with the increased nutritional demands in pregnancy, nutritional anaemia i.e. iron and folate deficiency are very common. The contributory factors to the poor nutrition are several, notable amongst them are poverty, ignorance, adherence to taboos and poor cooking habits. Malaria, hookworm infection, HIV and sickle cell disease are the other main causes.

Iron deficiency.

Iron deficiency is the most common cause of anaemia in pregnancy, and may be present together with other aetiological factors. In a study done in Northern Nigeria, the frequency of biochemical diagnosis of iron deficiency rose from 25% in the first trimester to 40% in the third, and from 18% in primigravida to 35% in grandmultiples⁽⁵⁾.

Diet - Pregnancy represents a state of negative iron balance. To remain in positive iron balance, the non-pregnant woman needs to absorb 2-2.5mg of iron per day. In the first trimester, this requirement decreases on account of the amenorrhoea, but increases to 5mg per day in the second and third trimester due to an increase in maternal red cell mass and foetal iron demands. Unless the nutritional intake is supplemented, the pregnant woman will be deficient in stores by term. It takes 3-6 months of regular daily oral supplementation to build up the Hb to normal and get enough body stores. It is literally impossible in our environment to build up stores on diet alone. Pregnancy at frequent intervals compounds the problem. So is it in teenage mothers who need the iron not only for the

There are populations in Africa like the Hadza in Tanzania, Kung Bushmen in Kalahari, Maasai in Kenya who have a naturally high intake of haem iron and thus do not have a high incidence of iron deficiency. This is the same for the South African Bantus who drink beer brewed in iron pots.

Haemorrhage May be acute, as in abortions, pre-, intra-, and postpartum haemorrhage. Abortions account for 12.9% to 13.5% of maternal mortality in Korle-Bu, Accra, Ghana and are the third leading cause^(3,6). Haemorrhage may be chronic as occurs in hookworm infestation, which happens to be the major cause of haemorrhage especially in the belt stretching from Cameroon to Zambia^(7,5). Folate deficiency. Folate deficiency is relatively common in West Africa. In a study done in Ibadan in 1968⁽⁹⁾ it was diagnosed in up to 75% of severely anaemic women. The causes were attributed to nutritional inadequacy, malaria and other infections, haemoglobinopathies and a high frequency of twinning. Folate requirements are doubled in pregnancy 800ug/day as compared to 400ug/day in the non-pregnant woman. The demand is higher towards the end of pregnancy and in multiple pregnancies. Haemolysis from any cause in sickle cell disease or malaria increases the requirement drastically because of erythroid hyperplasia (An increase in the red cell precursors in the bone marrow).

Pyrexia of infection can also lead to acute megaloblastic arrest of erythropoiesis due to inactivation of dihydrofolate reductase an enzyme essential for the synthesis of folate coenzymes.

Malaria

The incidence of malaria is increased in pregnancy. In a rural area in Uganda 62% of antenatals were parasitaemic⁽¹⁰⁾. It is seen most commonly in primigravida and peaks in mid pregnancy. The main effect is anaemia. The anaemia is due in part to splenomegaly with hypersplenism and also haemolysis. The frequency of palpable spleen like parasite density increases in pregnancies of all gravida classes with a peak in the second trimester. Maternal malaria can have disastrous consequences especially in the non-immune. It can be followed by abortion especially in the first trimester, and also low birth weight.

Sickle cell disease

In Korle-Bu, Accra, Ghana, sickle cell disease is among the four leading causes of maternal mortality^(3,6). 1-2% of children in Ghana and most of sub-Saharan Africa are born with haemoglobin SS, SC

Milder forms with improved prognosis are seen in pregnancy i.e. HbSC, Sthal, SS inherited with thal or with the Senegal globin haplotype. Due to folate deficiency as well as acute sequestration crises, bone-pain crises, pre-eclampsia during labour, operative deliveries necessitated by pelvic deformity and bacterial infections, maternal mortality in sickle cell disease in Africa remains above 10%⁽¹¹⁾, 20 times higher than in the normal pregnant patient.

HIV/AIDS

HIV 1 is epidemic in East and Central Africa. HIV 2 is prevalent in West Africa and high frequencies of both can be found in many areas in sub Saharan Africa. In Africa women are likely to have several pregnancies subsequent to infection with HIV thus running a rapid clinical course. Anaemia, eucopaemia and thrombocytopaenia are common in AIDS.

Presentation

Anaemia progresses through 3 stages i.e.

- I. Compensation with breathlessness only,
- II. Decompensation with breathlessness at rest and
- III. Cardiac failure.

Without treatment, 50% of the last group will die. Moderately anaemic women have a reduced tolerance to exercise and are unable to take good care of their children⁽¹²⁾. Malaria, folate deficiency and anaemia from any cause can lead to abortion or perinatal death. Acute haemorrhage presents with hypovolaemia and shock.

Even moderate anaemia results in reduced oxygen tension of amniotic fluid and intrauterine foetal hypoxia. There is an increase in premature delivery, foetal distress and low Apgar scores. Thus, there is a direct correlation between the Hb of anaemic mothers and birth weight. The deficiency of folate per se, leads to premature delivery.

In mothers presenting with Hb 7g/dl and below, and delivering soon afterwards, foetal loss is increased.

Diagnosis

To make a definitive diagnosis, it is important to take a thorough history including dietary, gynaecological, obstetric, and social history, do a physical examination and request appropriate investigations.

History

Enquiry into the person's diet is very important. Pica is common; it is both a cause and a symptom of iron deficiency. Direct enquiry should be made into: The

duration of symptoms of anaemia, whether acute or chronic; drug and other chemical use; burning sensation of the tongue, brittle and discoloured fingernails, diarrhoeal bouts, haemorrhoids, amount of blood lost per pregnancy, abortion and menses, changes in weight; fever, urine colour, cough, SCD, G6PD deficiency, work, and parity.

Examination

Look for presence or absence of pallor, jaundice, cardiopulmonary disease, enlarged lymph nodes, spleen and liver, size of uterus as well as per vaginam and per rectal examination. Sternal tenderness and petechiae are to be looked out for in the rare case of leukaemia in pregnancy.

Investigations

General Estimation of haemoglobin (Hb), packed cell volume (PCV), red cell indices (MCH, MCV and MCHC), total white cell count and differential, platelet count, reticulocyte count, film for comment and malaria parasites, erythrocyte sedimentation rate, and sickling and Hb electrophoresis.

The bone marrow should be examined especially in very severe anaemia. Although an invasive test, it is inexpensive, simple and gives meaningful results. Malaria pigment, iron stores and megaloblastic erythropoiesis should be looked for in the marrow.

The urine should be examined for colour, bilirubin, urobilinogen, blood, parasites, casts, protein, and pus cells.

The stool should be examined for colour, consistency, occult blood, ova and parasites.

The serum should be checked for the levels of urea and electrolytes and bilirubin.

Specific - As dictated by suspected cause of the anaemia. Serum iron and iron binding capacity in iron deficiency, B12 and folate levels in megaloblastic anaemia. The interpretation of the results is as important as the tests themselves.

Iron deficiency It causes a microcytic and hypochromic anaemia, the thalassaemias may give a similar blood picture. Serum ferritin, although one of the best non-invasive tests of iron stores, is unreliable in pregnancy because as an acute phase reactant it is raised. If due to parasitic infestation there will often be an eosinophilia.

Folate and vitamin B12 deficiency

They both cause a megaloblastic blood picture. Folate deficiency is however much commoner than B12 deficiency. Definitive diagnosis is dependent upon blood levels of these substances.

Malaria The parasites are found within the red cells using thick and thin blood films. Occasionally they may be so scanty as to be missed. Clinical judgement is therefore important. Haemolysis may or may not be prominent.

Sickle cell disease The Hb electrophoresis will in most cases be adequate.

HIV Detection of the antibody in the patient's sera using enzyme linked immunosorbent assay (ELISA) as the screening test and confirmation with a different and usually more specific test. In 60-70% of cases, there is anaemia and leucopaenia. More than 40% have thrombocytopaenia.

In haemolysis, direct antiglobulin test and Glucose-6-phosphate enzyme assay should also be done. In marrow aplasia and also suspected marrow infiltration, a marrow trephine biopsy will be required.

Treatment

Prevention

Since 1987, when WHO called to action "promotion of safe motherhood", there has been no improvement in maternal survival. The scandal of the century (Wendy Graham). Anaemia in pregnancy is avoidable in the majority of cases.

In the short term, oral ferrous sulphate and folic acid should be given daily at doses of 200mg and 5mg respectively to all pregnant women to prevent nutritional anaemia.

Malaria should be prevented; Proguanil is safe and effective at 100mg daily.

In the medium term there is the need for intensive health education of both boys and girls on safe sex, avoidance of early pregnancy, family planning, and appropriate balanced diet especially during pregnancy. Other measures that can be taken include fortification of staple foods with iron and folate, improvement of health facilities in numbers and quality as well as access to them, and premarital counselling in sickle cell disease.

In the long term Africans should aim at eradicating poverty, illiteracy and ignorance our greatest enemies.

Iron

In proven deficiency and in grandmultiples, oral ferrous sulphate should be given at 200mg 3 times daily and continued till delivery or 3 months after the Hb has reached normal whichever is longer. The rate of response once treatment starts should be a rise in Hb of 0.15 - 0.2g/dl daily or 2g/dl every 3 weeks. The reticulocyte count rises and peaks within 5-10 days.

When there is no response often the patient is not taking the medication, is bleeding or has malabsorption. It is also important to reassess the diagnosis as thalassaemia trait, sideroblastic and other anaemia might all be present with microcytosis and hypochromia. There may also be other contributory factors to the anaemia.

Parenteral treatment is usually unnecessary except in situations where compliance cannot be assured or there is proven malabsorption. Iron sorbitol (Jectofer) is given at doses of 50-100mg iron/day deep intramuscularly. Total dose to be given is calculated as follows:

$$\text{Dose} = (15 - \text{patients Hb g/dl}) \times \text{body weight (kg)} \times 3.$$

Jectofer should not be given intravenously. Iron dextran may be given slowly intravenously over 6 hours after a test dose. There may be exacerbation of malaria and urinary tract infections after the infusion and patients should be properly covered.

Folic acid

It is given at a dose of 5mg daily. There is no evidence that higher doses are more beneficial. In each case one should ensure that there is no coexisting cobalamine deficiency otherwise its neuropathy will be precipitated. Treatment is monitored as in iron deficiency.

Blood Transfusion

If anaemia is very severe and the mother is in danger of heart failure, blood transfusion is recommended to raise and maintain the Hb above 5g/dl and if delivery is eminent, above 6g/dl. With HIV/AIDS, and the difficulty in recruiting voluntary blood donors, blood transfusion should be used only to save the mother's life. In acute haemorrhage and operative blood loss >30% of blood volume or 500mls where tissue oxygenation is not maintained by plasma expanders, blood transfusion is indicated. As far as possible autologous blood transfusion is recommended i.e. intra operative blood salvage.

Intraoperative Blood Salvage

It is one of the four modes of autologous blood transfusion. The others are predeposit, intraoperative haemodilution and postoperative salvage.

Autologous transfusion reduces or eliminates the possibility of transfusion reaction and/or the transmission of infectious disease. Intraoperative blood salvage can be used in most procedures so long as there is no contamination (sepsis or penetrating bowel wounds) of the surgical site or contamination with malignant tumour cells.

It has frequently been used in orthopaedic, vascular, cardiac, obstetrics and gynaecology and neurosurgical procedures. Blood salvaged intraoperatively should not be transfused to any other patient. It can be stored at room temperature for up to 4 hours and at best should not leave the operating or recovery room. If for one reason or the other it must be kept longer, the unit (properly labelled) should be sent to the blood bank for storage at 1 - 6°C for up to 24 hours. There must be a written down protocol for identification of the unit and its release so as to prevent accidental release to the general pool or the wrong patient.

Some years ago, blood shed at ectopic pregnancies was scooped, sieved through gauze into a donor bottle and re-infused. This method is no longer acceptable. There are equipments available for collecting; washing and filtering shed blood before re-infusion e.g. Cobe Cell Saver. Others like the Solcotrans Plus and ID set (yet to be marketed) do not incorporate washing, are relatively simple and suffice for clean flowing blood as in ectopic pregnancies. In operative fields where there is the likelihood of cellular debris, fat and other contaminants, washing is recommended.

The basic principles are as follows:

1. Shed blood is suctioned at 80-100mmHg into a bag containing anticoagulant - citrate or heparin. The recommended ratio is 1:7 to 1:10 citrate to blood.
2. The anticoagulated blood is filtered using a 40/60 micron blood filter.
3. The filtered anticoagulated blood is re-infused using a giving set.

NB:Detailed instructions always come with the "Instructions for Use" accompanying each equipment.

Direction in the future

The study of aetiology in pregnant women in our region is beset with poor laboratory facilities.

Chemical measurements of iron and folate deficiencies are altered by inflammation and acute malaria. New reference values will need to be established for pregnant women in tropical Africa.

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Sickle Cell Disease In Pregnancy (SS, SC β ThAL)

EY Kwawukume

Introduction

Sickle cell disease refers to sickling disorders in which the sickling gene is present with another abnormal gene affecting the production (quantitative) or the structure (qualitative) of haemoglobin. In West Africa, HbC and HbS are both seen in pregnancy unlike the thalassaemias. Dawson documented that Haemoglobin C was much less widely distributed than HbS and its highest prevalence was in northern Ghana⁽¹⁾ while HbS was commoner in the southern sector of Ghana. Hospital statistics from Korle Bu Teaching hospital which is located in the southern part of Ghana showed that during the second half of 1999, out of 50 sickle cell disease patients enrolled in the antenatal clinic, SC was 72%, followed by SS 24%, SF and S/bThal 2% each. This might be due to intermarriages due to inter-country travelling for businesses and other reasons. Haemoglobin C patients are also prone to crisis but recurrence is less frequent than HbS disease. Most patients are symptomless except during pregnancy, probably in the 3rd trimester where dramatic changes might occur with acute sequestration, anaemia and thrombotic episodes.

The 3 common forms of haemoglobinopathies are:

- Sickle Cell anaemia. (SS)
- Haemoglobin SC disease
- Sickle/-thalassemia.

Basic Pathology (Haemoglobin molecule)

There are two pairs of polypeptide chains (alpha/beta) and each chain has 146 aminoacids. There is haem group, which is attached to each chain. Haem can combine reversibly with oxygen molecules.

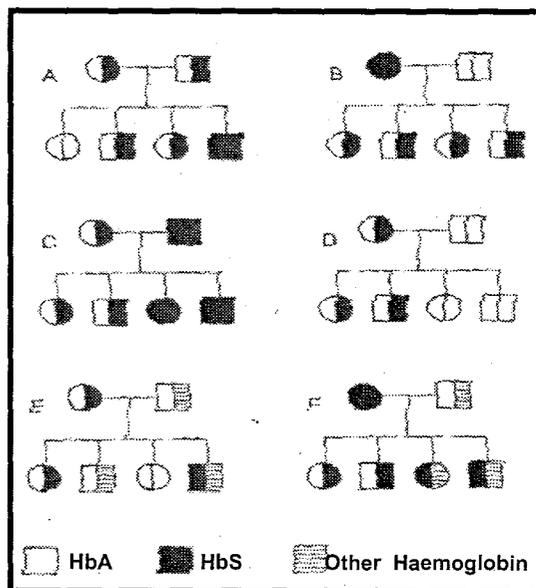
Haemoglobin A is the normal adult haemoglobin and the polypeptide chains are represented as 2A2 α 2 β . The sickle cell haemoglobin is 2A2S and haemoglobin C as 2A2C.

The difference in physical behaviour between the normal HbA and the abnormal haemoglobins HbS or HbC is in the b-globin of the haemoglobin molecule. In HbS, valine replaces glutamic acid in position 6 in the amino acid sequence. The substitution of valine for glutamic acid at the sixth locus produces all the characteristics of HbS. In HbC, the change involves

the substitution of lysine for glutamic acid in the same position.

When two sickle cell genes are inherited, the homozygous state SS genotype results. The inheritance of one-gene results in the heterozygous carrier state known as sickle cell trait (AS) and they do not normally suffer from sickling phenomena. It has also been documented that the presence of the S haemoglobin confers partial protection from *P. falciparum* malaria for AS persons.⁽¹⁾

Inheritance of common sickling disorders.



In thalassaemia the genetic materials which code for the formation of each pair of the globin chains may be absent, reduced in amount or abnormal in function. If the disorder affects the b-chain, (called b-thalassaemia) the production of b-chain is reduced but there will be no such impairment in α -chain synthesis. Consequently, during adult life HbA production is reduced whereas HbA₂ and HbF are present in increased amounts. This is because HbA₂ and HbF have two alpha chains each and two new chains known as delta and gamma chains respectively. They do not have b-chains.

Clinical features

Sickle cell disease is caused by sickling of red blood cells, which results from polymerisation of deoxyhaemoglobin S. There are many predisposing

factors to sickle cell crises such as malaria infestation, pneumonia, urinary tract infection, dehydration, acidosis and reduced oxygen. Sickling depends on the concentration of deoxyhaemoglobin S within the red cell and the type of the other haemoglobins present. SS red blood cells contain only haemoglobin S and require less deoxygenation for sickling than do SC cells. In addition, haemoglobin C fits less readily into polymers than haemoglobin S therefore haemoglobin SC is less likely to sickle than haemoglobin SS. As a consequence, haemoglobin SS disease is a severer disorder than haemoglobin SC.

Sickle cell can present in various acute forms resulting in crises. These crises present as sudden attacks of pain in bones, commonly termed "rheumatism". The pain may move from limb to limb and can be debilitating to the patient. Occasionally, there might be abdominal pain simulating surgical emergencies.

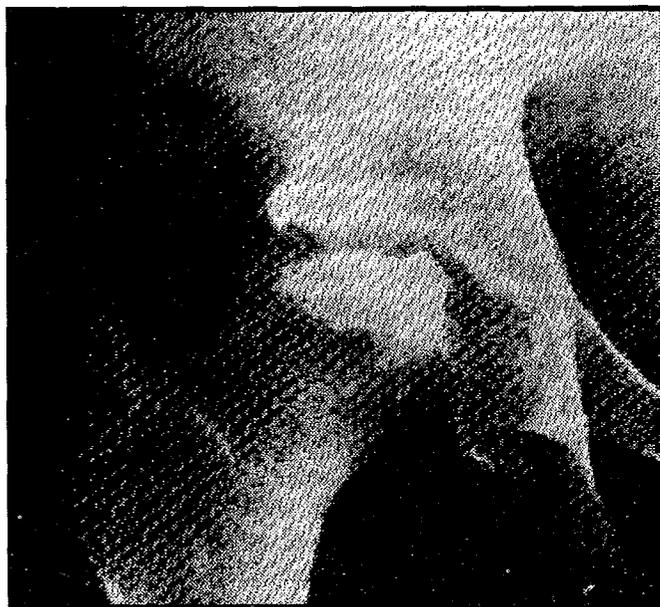
The urine might be dark from urobilinogen and a tinge of jaundice might be observed on the sclera. Another clinical presentation in sickle cell haemoglobin SS is anaemia from haemolysis, sequestration and aplastic states. The bone marrow might not keep pace with the rate of destruction of the red cells resulting in anaemia. The pathogenesis of the haemolysis is not completely understood and might be due to membrane damage but immunologic destruction or physical trauma may also be involved. Blood might be sequestered in the spleen causing splenomegaly.

The spleen might be enlarged in childhood but usually shrinks or becomes impalpable in adulthood with haemoglobin SS. It is usually enlarged in malaria endemic areas and in HbC disease. Stunted growth and skeletal deformities are also noticed.

There is forward protrusion of the upper incisor teeth with swelling of the forehead and bossing of the maxilla (gnathopathy).

Macroscopic tissue damage such as avascular, aseptic necrosis of the head of the femur and pulmonary infarction might be due to embolisation of necrotic bone marrow or tangled sickle cells occluding the microvasculature.

As the patient walks into the consulting room abnormal gait may be noticed.



Picture of avascular necrosis of the head of femur

There might be frank haematuria from renal papillary necrosis⁽²⁾ and evidence of old dactylitis from infarction of the phalanges and leg ulcers.

The detection of sickle cell anaemia and homozygous thalassaemia in-utero in the first and second trimesters of pregnancy is now possible through the complex biochemical techniques for globin chain analysis and genetic mapping.⁽³⁾

Because of these techniques, genetic counselling of patients with affected fetuses including those with haemoglobinopathies are offered therapeutic abortion in the developed countries though it is at present not practised in the vast majority of developing countries where the need is greatest.⁽³⁾

In our sub-region some patients would terminate a pregnancy to avoid the problems associated with managing sickle cell disease children. On the other hand some women would not terminate pregnancy because of religious and moral reasons.

Good counselling in sickle cell disease is important and questions asked by the couples should be answered in simple terms with explanations about the various possibilities of the medical significance of the unborn child. If they request the genotype of the fetus during the antenatal period then chorionic villus sampling^(6,7) can be done as early as the 10th week of gestation. Most women with sickle cell anaemia are aware of the disease because of the

recurrent crisis that they experience from childhood but patients with SC disease or S/thalassemia may not. In a study on prenatal diagnosis of sickle cell anaemia on a sample of Nigerian population, ⁽⁸⁾ it was found that the majority of the respondents (69.5%) appreciated the role of both parents in the transmission of the disease. However, only 18% heard of sickle cell anaemia for the first time through sickle cell counsellors, 23% through the news media, 29% through friends and relations, 21% obtained the information through health workers, while 5% had never heard of sickle cell disease before the interview. As many as 44% of the respondents were aware that sickle cell anaemia could be diagnosed in pregnancy, which is not routinely done in our subcontinent.

In a study done in Nigeria using polymerase chain reaction-based technology (PCR) combined with chorionic villi sampling, prenatal diagnosis of sickle cell disease in 50 pregnant women at risk of bearing children with sickle cell anaemia were diagnosed. ⁽⁹⁾ DNA was extracted from the villus and subjected to either PCR and restriction enzyme (Dde 1) analysis or to PCR-ARMS procedure or both procedures when the results by the first procedure were equivocal. Screening might become more readily accessible in our sub-region because many parents might want to avoid the problems associated with managing sickle cell anaemia. Good counselling is needed to achieve reasonable results

Diagnosis

- × Family history of sickle cell disease.
- × Sickling test. Add two drops of 2% sodium metabisulphate to a drop of blood and cover the slide with a small glass slide and seal the edges with vaseline to create deoxygenation. Examine the slide after 20 minutes. This test would show typical sickling of the red cells in SS, AS and SC patients
- × Haemoglobin electrophoresis. This test would show complete identification of haemoglobins A, S and C
- × Examination of stained preparation of blood film
 - × showing target cells, polychromasia, nucleated red cells, Howell-Jolly bodies and sickle forms

Complications

- × Haemolytic crisis leading to anaemia
- × Bone pain crisis
- × Acute sequestration crisis

One of the basic causes of most of the complications of SCD is that sickle cells in the deoxygenated state can cause damage to the microvasculature. Normal HbA continues to be in solution in the deoxygenated state whereas the solubility of HbS is decreased. As the reduced Hb comes out of solution it forms the typical tactoids of sickle cells. Repeated sickling damages the red cell membrane and this prevents the red cell from returning to its normal shape even when the haemoglobin becomes fully oxygenated. ⁽¹⁰⁾ Also increased stickiness of red cell membranes on blood vessel walls might cause damage and precipitate crisis ¹¹⁻¹⁵.

In addition, the diameter of some capillaries is only 2 μ m as compared with normal red cells with a diameter of 8 μ m. Sickled red cells cannot squeeze through such openings as in normal red cells and they therefore become trapped at such points in the circulation, resulting in increasing blood viscosity and reducing blood flow in the microcirculation. In the end, there is intravascular occlusion and tissue infarction.

Secondly, the survival of red cell in SCD is shortened causing chronic haemolysis, which may require repeated blood transfusion with transfusion hazards.

There is also anaemia, which might lead to heart failure. Frequent crisis predisposes the patient to preterm labour and premature delivery. In addition, there may be spontaneous abortions, the risk of getting an abnormal gene, stillbirth and increase in perinatal mortality and intrauterine restriction or retardation. ⁷⁻¹²

Maternal mortality has been very high in sicklers amounting to about 15.7 % between 1991 and 1993. This was second to haemorrhage ⁽¹⁶⁾. In an 11- year review in the same Institution (1984 to 1994) ⁽¹⁷⁾ mortality from sickle cell disease was 11% being the fourth leading cause of maternal death.

Sickle Cell mortality is usually due to severe anaemia, embolism following bone marrow

infarction and acute sequestration of red cells. This is compounded by inefficient blood supply and lack of involvement of senior persons in the initial management of crisis.

In a series of 190 HbC pregnant patients in Ibadan, the mortality was reduced from 14 % in the first 85 cases to below 5% by improvements in management. ⁽¹⁾ It was also observed that by providing information and education about SCD and improving nutritional status, malaria prevention and early detection of bacterial infections the maternal mortality was recorded as 1.8%, which was comparable to the overall maternal mortality rate for the same maternity unit in Cotonou of the Republic of Benin. ⁽¹⁸⁾

It is advocated that standard protocols should be made available in the labour wards and lying-in wards for doctors and midwives to follow regularly. Secondly, senior faculty members should be involved in the initial management of sicklers with crisis to prevent mortality

Broad-spectrum antibiotics should be given as prophylaxis any time a sickler ruptures her membranes in labour.

Heparin use in sickle cell disease patients

Bone pain crisis becomes very common during the last month of pregnancy, just before and during labour and immediately after delivery. At this time marrow embolism is likely to complicate infarction of the bone marrow. Heparinisation is indicated. Secondly, the phenomenon of systolic hypertension and albuminuria in a sickler with bone pain crisis should not be misconstrued as preeclampsia. This is rather pseudotoxaemia of pregnancy and it indicates imminent marrow embolism. Heparinisation is indicated to avoid mortality.



Marrow embolism in a SCD patient

A sickler with bone pain crisis with severe dyspnoea and chest signs of pneumonia should be critically evaluated to rule out fat or marrow embolism. Heparin is again indicated to probably stop further marrow and/or fat emboli lodging in the pulmonary arterioles.

In diagnosing pneumonia or what is commonly called "chest syndrome" in sicklers one should consider infarction first and not bacteria infection. It is a life threatening condition, which mimics adult respiratory distress syndrome and heparinization and blood transfusion should be considered.

Finally, if bone pain crisis does not resolve within 48 hrs of initial management with intravenous fluids, antibiotics and analgesics, the patient should be heparinized until the crisis resolves.

In heparinization, start with an initial dosage of 10,000-15,000 international units with maintenance of 5,000 to 15,000 IU every 4 to 6 hours. Low molecular weight heparin should be used since it is easier to manage in our circumstance. Clotting time should be more than 17 minutes. It is essential to keep protamine sulphate, 5-10 ml of one percent (1%) solution, which is a heparin antagonist in readiness of any complication.

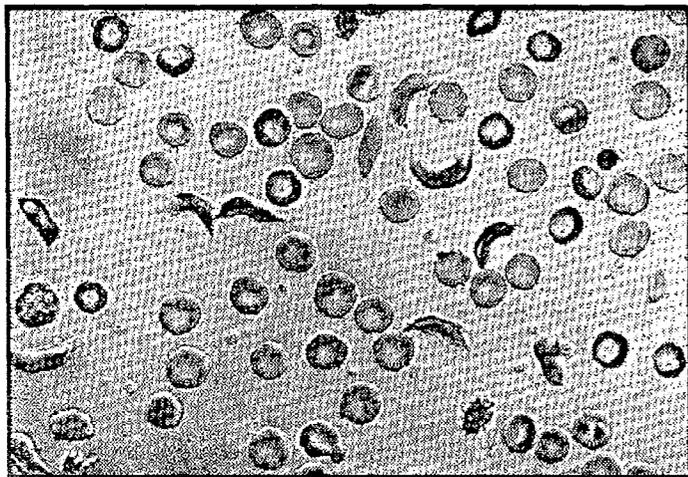
Complications

Maternal complications

- × Anaemia
- × Frequent crisis
- × Chest syndrome
- × Chronic transfusion
- × Cerebrovascular accident
- × Heart failure
- × Pyelonephritis
- × Difficult postpartum course
- × Preterm labour
- × Fat or marrow embolism
- × Pseudotoxaemia
- × Maternal mortality

Fetal complications

- ✗ Spontaneous abortions
- ✗ IUGR ⁽⁷⁻¹²⁾
- ✗ Risk of getting abnormal gene
- ✗ Stillbirth and increased perinatal mortality
- ✗ Prematurity



Picture of blood film showing sickled cells, target cells etc in sicklers

Vitamins and Iron administration in Sickle Cell Disease

Folic acid requirement is needed more in pregnant women with haemolytic anaemias, including sickle cell patients, than their normal counterparts. It is therefore necessary to supplement the diet that is normally rich in folic acid with prenatal folic acid tablets.

Iron requirements in patients who have not been transfused are not different from those of normal pregnant women. However, those patients who have been repeatedly transfused might have increased iron stores and additional parenteral therapy poses the danger of haemosiderosis. Serum ferritin should be checked before transfusion.

In a study done in Ibadan, Nigeria, ⁽¹⁹⁾ ferritin and serum iron levels were measured in adult patients with sickle cell anaemia and pregnant sickle cell patients. The serum ferritin levels were within normal range for both the adult and the pregnant sickle cell

patients. It was however noticed that in the pregnant women, significantly lower ferritin levels were recorded than in the non-pregnant women. There was, therefore, a reduction in ferritin levels in pregnancy. This could be due to the increased iron requirements and the demand by the developing fetus. Serum iron was below normal in 30% of the pregnant women and only 6% of all the adult patients had serum iron levels below the normal levels. Given the adequacy of ferritin levels in the patients studied and the very low incidence of below normal serum iron levels, it was concluded that the sickle cell anaemia patients in the area of study have adequate levels of iron and ferritin in their serum. Iron should therefore be given only in proven cases of iron deficiency anaemia.

In any case, when in doubt and facilities are not available to check blood ferritin levels then oral iron could be given instead of parenteral iron. The body might absorb the necessary amount and excrete the excess. It should be remembered that the only routes for the loss of transfused iron are pregnancy and menstruation.

Sickle Cell Disease Patients and Oral Contraceptive Pills

Some doctors are not comfortable for one reason or the other to prescribe oral contraceptive to sicklers for the fear of thrombosis. It should be remembered that vaso-occlusive crisis are not primarily due to coagulation defects but rather to impacted sickled cells in the microvascular and small vessels resulting in intravascular occlusion and tissue infarction. The risk of thrombosis in sicklers is no greater than that of other women who take low dose oestrogen containing oral contraceptives.

Again, some physicians would not give the OCP to sicklers because it is considered that they have liver disease with jaundice. The jaundice in SCD is primarily due to haemolysis and not estrogen dependent. The ideal contraceptive should be advised bearing in mind the pathophysiology of the disease state.

Any contraceptive with low dose oestrogen or progesterone only pill could be recommended. Ideally, barrier methods are preferred but sterilisation is a better choice especially if the woman has completed her family.

Contraception and Family Planning should always be discussed with sicklers because the problems of pregnancy and delivery put the sickler at increased risk in each pregnancy.

Contraception

- ✗ Combined OCP low dose oestrogen
- ✗ Norplant
- ✗ Progesterone only Pill
- ✗ Tubal ligation (BTL)
- ✗ Vasectomy for the husband

Management

Adequate management of sickle cell disease in pregnancy necessitates close observation with careful evaluation of all symptoms, physical findings and laboratory investigations. It should involve obstetricians in high-risk clinics with support from the haematologists, neonatologists, midwives and related maternity staff.

In general, the following steps should be taken to improve the life of sicklers:

- administration of prophylactic antimalarials and treatment of malaria
- prevention and adequate treatment of anaemia
- control of painful crises
- prevention and control of infection
- availability of blood transfusion services
- the use of heparin in the treatment of pseudotoxaemia
- adequate facilities for obstetric operations
- availability of good nursing care
- adequate antenatal care, labour and the puerperium⁽²⁰⁾
- education of the public about the disease

Induction of labour and caesarean sections should be carried out for obstetric indications but in the presence of repeated crises and frequent transfusions one should consider elective delivery at 37 completed weeks of gestation or at the appropriate time depending on the severity of the disease.

The results of anaesthesia are good so long as attention is paid to adequate oxygenation and ventilation, and to the maintenance of circulating volume.⁽²¹⁾

At the first antenatal visit the Hb electrophoresis should be done to establish the type of haemoglobinopathy. Other investigations should include blood group and rhesus status, full blood count, reticulocyte count, blood-film for malaria parasites, mid-stream specimen of urine for routine examination and culture and sensitivity, routine stool examination and serum iron and total iron binding capacity. The G6PD and the LFTs should also be

documented. X-ray of the chest is taken with abdominal shield if chest signs are positive.

OPD investigations - First visit

- * Hb electrophoresis
- * FBC and reticulocyte count
- * Bf for mps
- * Blood group and Rh
- * Blood cultures
- * G6PD status
- * MSSU for R/E & culture
- * Stool R/E
- * LFTS
- * Serum Fe & TIBC

During the follow-up visit the haemoglobin, blood film for malaria parasites, routine examination of urine, and other investigations as required should be performed.

Since malaria is endemic in our sub-region a curative dose of chloroquine 600 mg is given at the first attendance, followed by prophylaxis with chloroquine 300 mg or pyrimethamine 25 mg every week. In addition, 5 mg of folic acid is given daily to prevent macrocytic anaemia and iron medication should be given in proven cases of iron deficiency.

Indications for Transfusion

Sickle cell disease patients are more prone to chronic blood transfusion because of repeated crisis, notably haemolytic and sequestration crisis. Blood might be sequestered into the reticuloendothelial system such as the liver and the spleen, which presumably act passively by mopping up damaged cells. The haemoglobin levels might drop markedly and this might be accompanied by inability to carry out daily activities, marked fatigue, dyspnoea, tachycardia and headaches. Transfusion might be indicated with these features. The absolute indications for blood transfusion in our hospitals are

- (1) All cases of crisis with haemoglobin below 8gm/dl.
- (2) All cases with haemoglobin below 8gm/dl at maturity of 36 weeks or more.
- (3) All cases with haemoglobin of 7g/dl or below irrespective of the maturity and;
- (4) Where clinical judgement so indicates.
- (5) Repeated crisis to reduce concentration of sickle cell haemoglobin in blood

The blood for transfusion should contain haemoglobin AA. Haemoglobin SS or AS blood should not be transfused.

If the patient's crisis is not frequent during pregnancy or more severe than they were during her normal life there is no need to transfuse. In addition, there is no need for transfusion if the patient has no more symptoms that could be due to anaemia than do other women at her stage of gestation

Indications for admission

Sickle cell patients should be admitted if the haemoglobin is less than 7gm% irrespective of the maturity. In the presence of crisis, infection, pre-eclampsia and any complication the sickler should be admitted and managed appropriately.

Sickle Cell Patient with bone pain crisis

If the sickler is admitted with bone pain crisis the investigations should be completed if they had already not been done and the following management regime should be started:

- Chloroquine course while waiting for blood film for malaria parasites
- Broad spectrum and potent antibiotics such as Cephalosporins (Zenacef) or Augmentin.
- Analgesics such as Diclofenac suppository or injectables, Pethidine or Morphine.
- Intravenous infusions: 4 litres in 24 hrs as:

- (1) N/S 500ml
- (2) Ringers Lactate 500ml
- (3) N/S 500ml
- (4) 5% Dextrose 500ml
- (5) N/S 500ml
- (6) Ringers Lactate 500ml
- (7) N/S 500ml
- (8) Ringers lactate 500ml

* Oxygen, about 2 to 3 litres should be administered with face mask

Oral fluid can be advised if there is any doubt about the adequacy of parenteral fluid especially in patients whose peripheral veins have been thrombosed during prior treatments with intravenous fluids or those with moderate disease who are not vomiting. Oral hydration should be tried while searching for a vein. The patient should be advised to drink till the urine is "gin clear".

Management in Labour.

After completing the necessary investigations, two pints of blood should be cross-matched and blood should be given if the haemoglobin is less than 8gm/dl. Intravenous fluid should be maintained and antibiotics started if the membranes rupture. Cord blood for haemoglobin and electrophoresis should be taken after delivery.

Puerperium

The mother could stay in hospital for about three days for the various investigations to be done. The urine should be examined and the fluid balance chart should be monitored. Oral medications are also started and the parents are educated about the nature and prognosis of the child's disease. They are also counselled about future pregnancies and all the newborns are screened.

Exchange transfusion is practiced in some centres in the sub-region. The goal of exchange transfusion in sicklers is to achieve more than 50 % of normal red cells. Exchanged blood should be screened to eliminate sickle cell trait (AS). Exchange transfusion is not used routinely in our hospitals. The indications are as stated above but in addition it can be done for a patient with normal haemoglobin level to prevent increasingly severe crises

Discussion and Controversies

The Future for Sickle Cell Disease

Hydroxyurea is the experimental drug classified as an S-phase antineoplastic agent (pregnancy category D).⁽²²⁾ Large doses of the drug are teratogenic in animals^(23,24) although several case reports suggest the agent may have minimal teratogenic effects on the developing fetus. Fourteen cases of hydroxyurea therapy were reported in pregnant patients with acute or chronic myelogenous leukaemia, primary thrombocythemia and sickle cell disease. Three pregnancies were terminated by elective abortion; one woman developed eclampsia and delivered a phenotypically normal stillborn infant. All other patients delivered live, healthy infants without congenital anomalies.⁽²⁵⁾ It was also observed that two women became pregnant while taking hydroxyurea for sickle cell anaemia and delivered live infants with no congenital anomalies.^(25,26,27)

Though these isolated cases have been reported, there is the need to follow larger numbers of exposed children whose mothers received hydroxyurea during pregnancy to assess the fetotoxic effects.

There are no known controlled studies in pregnant women taking hydroxyurea therefore caution must be exercised in its use in pregnancy.

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Renal Disease

JD Seffah

Introduction

Renal functions may be reduced in pregnancy from primary renal diseases or systemic disorders. The health of the mother may be threatened and the foetal outcome may be impaired.

The main causes of maternal mortality in Ghana, i.e. infections, haemorrhage and hypertensive disorders are usually accompanied by renal disorders if not properly managed. Placental abruption, eclampsia and septic abortion may require haemodialysis to treat renal failure. ⁽¹⁾

There are physiological adaptations to renal function and volume haemostasis during pregnancy. These have to be understood in order to give the appropriate clinical and laboratory interpretations to renal disorders. Women with renal problems may want to find out if they can become pregnant.

Physiologic Adaptations

- * Dilatation of the urinary tract may lead to errors in 24 hrs creatinine or protein excretion.
- * There is urinary stasis because of increased progesterone secretion, which relaxes smooth muscles leading to asymptomatic bacteriuria.
- * The glomerular filtration rate (GFR) reaches a maximum of 50% at the end of the first trimester. ⁽²⁾
- * GFR increases less than renal plasma flow (RPF) and therefore filtration fraction (FF) decreases ⁽²⁾
- * Values considered normal in the non-pregnant woman may reflect decreased renal function during pregnancy.
- * Urate concentration is higher in pregnancies complicated by pre-eclampsia.
- * Glycosuria in pregnancy reflects a change in renal physiology and not necessarily hyperglycaemia. Urinary proteins exceeding 500mg in 24hrs is abnormal. Increasing proteins in chronic renal disease does not always imply deterioration.
- * Osmoregulatory changes are important in central diabetes insipidus and "transient diabetes insipidus of pregnancy" which remits postpartum.

Pre-pregnancy Counselling

Individuals show no symptoms until the GFR falls to less than 25%, hence the need for counselling to patients with known chronic renal diseases before they get pregnant.

Important factors for successful pregnancy are:

- * The type of chronic renal disease
 - * General health of the patient with a diastolic BP < 80mmHg
 - * Renal function plasma creatinine <250 umol/L
 - * Plasma urea < 10mmol/L
 - * Absence of proteinuria
 - * Review of pre-pregnancy drug administration.
- Prepregnancy renal status needs to be classified to guide in the prognostication of the patient.

The prevalence of hypertension and proteinuria is less in CLASS A but diseases such as SLE, focal glomerular-sclerosis must be watched carefully. Pregnancy is best avoided in CLASS B. Patients in CLASS C are usually amenorrhoeic/and or anovulatory. ⁽³⁾ Management of chronic renal disease during pregnancy depends on good Antenatal care, which involves: -

- * monitoring the BP regularly and carefully,
- * assessing renal function,
- * assessing nutritional status in the heavily proteinuric
- * assessing the size, development and well-being of the fetus and patient
- * early detection of asymptomatic bacteriuria or confirmation of UTI. ⁽⁴⁾

Pregnancy in renal transplant patient is recommended when there has been good general health for 2 years after transplantation. In addition there should be no proteinuria; no significant hypertension; no evidence of graft rejection; no evidence of pelvic-calyceal distension plasma creatinine of 180 umol/L or less.

CHRONIC RENAL DISEASE	EFFECT ON PREGNANCY
DISEASE	EFFECT
1. Chronic glomerulonephritis	No adverse effect until hypertension sets in. Coagulopathy and UTI may superimpose
2. Focal glomerulosclerosis	Hypertension and renal deterioration are common. Fetal losses are likely
3. IgA nephropathy	Hypertension may be uncontrollable
4. Pyelonephritis	Exacerbation by bacteriuria. Multiple organ involvement including adult respiratory syndrome may develop
5. Reflux nephropathy	Increasing hypertension and worsening renal function
6. Urolithiasis	Infections are common. Little data on lithotripsy in pregnancy available
7. Polycystic disease	Usually minimal reduction of function
8. Diabetes nephropathy	Oedema, Pre-eclampsia and infections increase. The lesion per se is not affected
9. S.L.E.	If disease is in remission for more than 6 months, prior to conception, prognosis may be good. Steroids should be increased after delivery.
10. Periarteritis nodosa	Fetal and maternal prognoses are poor
11. Scleroderma	Rapid deterioration of pregnancy. Reactivation may occur postpartum
12. Previous UTI	Renal function may decrease. May have associated malformation which may require C/S to avoid disruption of continence mechanism e.g. after repair of VVF
13. After nephrotomy, solitary kidney	May be associated with other malformations of the urinary tract and pelvic kidney. Pregnancy is well tolerated. Dystocia rarely occurs with a pelvic kidney.
14. Wegener's granulomatosis	Limited information. Proteinuria is common. Immunosuppressives are safe. Avoid cytotoxics.
15. Renal artery stenosis	May present as chronic hypertension or recurrent PET. Transluminal angioplasty can be undertaken if appropriate.

Modified after Davidson. Causes of chronic renal disease and their effects.

Drug therapy

Prednisolone 15mg/day or less; and azathioprine 2mg/kg/day or less are recommended.

Potential mothers should be informed about possible increased risks for the fetus and counselled against pregnancy if hypertension or renal insufficiency is present.

Pregnancy Management

Antenatal care

Hospital based antenatal care in conjunction with nephrologist is required for surveillance of renal function in cases of graft rejection, hypertension/pre-eclampsia, ⁽⁵⁾ maternal infection, IUGR and Pre-term labour. Elective radiological exam should be deferred until 16 weeks after delivery because of teratogenicity.

CLASSIFICATION CLASS	PLASMA CREATININE (Umol/L)
A. Mildly impaired renal function	<125
B. Moderate renal insufficiency	≥ 125
C. Severe renal insufficiency	≥ 250

Criteria for admission include:

- Deterioration of the renal function
- development of moderate hypertension
- signs of intrauterine growth retardation and or deterioration of placental function, and
- change in rate of weight gain,
- symptoms of impending eclampsia.

Labour and Delivery

Preterm labour is common especially when renal function is poor. Vaginal delivery should be the aim. Ensure careful monitoring of maternal fluid balance. Transplant patients may have pelvic osteodystrophy, which will give rise to disproportion requiring Caesarean section. ⁽⁶⁾ Previous urological surgery may make lower uterine segment incision difficult.

Neonatal Complications

- Neonatal problems may arise because of:
- Preterm delivery.
- Respiratory distress syndrome.
- Depressed haemopoiesis.
- Lymphoid/thymic hypoplasia.
- Adrenocortical insufficiency.
- Septicaemia.
- CMV infection.
- Hepatitis B surface antigen carrier state.
- Congenital abnormalities.
- Immunological problems.
- Reduced T lymphocytes.
- Reduced immunoglobulin levels.
- Chromosome aberrations in leucocytes. ⁽⁷⁾

Acute Renal Failure [after Davidson] ⁽³⁾

This involves sudden decrease in GFR, rising plasma urea and creatinine levels and a decreased urine output <400ml in 24 hrs.

Associated acute liver disease should be diagnosed promptly because haemolytic-uraemic syndrome (HUS) has a bad prognosis. Active treatment of acute renal failure is essential

The aim is to prevent uraemic symptomatology, acid-base and electrolyte disturbances and volume problems.

Conservative management should be used judiciously. When unsuccessful, dialysis will be necessary. Dialysis may be 'prophylactic' in cases of ARF prior to development of uraemic symptoms.⁽⁷⁾ The method of dialysis should depend on facilities and clinical circumstances.

Controlled anti-coagulation with heparin is desirable. The onset of labour usually occurs soon after the dialysis. Blood loss should be quickly replaced after delivery.

The neonate may be dehydrated because of the urea and other solutes within its circulation, which can precipitate an osmotic diuresis^(7,8).

SOME CAUSES OF OBSTETRIC ACUTE RENAL FAILURE

- Volume contraction/ hypotension	APH, PPH Abortion, Hyperemesis gravidarum Adrenocortical failure
± Volume contraction/hypotension and coagulopathy	APH (abruptio placenta) PET/Eclampsia Amniotic fluid embolism. Incompatible blood transfusion. Drug reactions Acute Fatty liver Haemolytic uraemic syndrome
± Volume Contraction/ Coagulopathy and infections	Septic abortion Chorioamnionitis Pyelonephritis Puerperal sepsis
± Urinary tract destruction	Damage to ureters during Caesarean section and repair of cervical/vaginal lacerations, pelvic haematoma, and haematoma.

Controversies In Renal Disease

1. Termination of pregnancy because of very high levels of serum urea or creatinine in renal diseases raises an ethical issue. The patient's wishes should be considered very well.
2. Radiological assessment of the urinary system in pregnancy introduces a dilemma because of teratogenicity. C.T. scan or MRI may be too costly as substitutes.
3. Experience in renal biopsy in obstetric practice is sparse because clinical circumstances rarely justify the risk of biopsy, which could be postponed till after delivery.
4. Steroids for contraception produce subtle changes in the immune system. Low dose oestrogen/progesterone can aggravate hypertension and increase thrombo embolism. Careful surveillance is needed when prescribed.

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Thyroid Diseases In Pregnancy

JD Seffah

Introduction

Derangement of thyroid function in pregnancy is not common in our practice. One reason is that hyposecretion and hypersecretion of the thyroid hormones are usually associated with infertility.

The thyroid gland enlarges in normal pregnancy. This is due to a combination of increased vascularity and cellular hyperplasia.

In areas where dietary iodine is little, there is reduced plasma iodine. The thyroid gland must hypertrophy in order to respond to the normal demands of pregnancy for increased thyroid hormone. Goitre results. However, where intake of iodine is high, no compensatory hypertrophy of the thyroid gland occurs.⁽¹⁾

The estrogen effect of pregnancy induces the liver to secrete more thyroid binding globulin (TGB) by the 10th week of gestation. The GFR also increases during pregnancy. These are some of the reasons for physiological adaptation for the pregnant woman to cope with the increased MBR (basal metabolic rate).⁽²⁾⁽³⁾

Physiologic Adaptations

Increased GRF results in a rise in renal clearance of iodine and subsequent decrease in plasma inorganic iodine concentration. The thyroid responds by increasing the uptake of iodine to maintain euthyroid levels of thyroid hormone production.

It should also be noted that:-

- Iodine deficiency follows geographic variations
- Iodine deficiency is unlikely to occur in plasma concentrations above 0.03ug/100ml
- Intake of more than 2000 ug/day can harm the mother and foetus
- Thyroxine-binding globulin (TBG) rises two-fold during pregnancy due to hepatic biosynthesis secondary to estrogenic stimulation. Since 85% of thyroid hormones are bound to TBG, increases in total T4 and T3 levels occur. Although increased globulin elevates total T3 and T4, the free T3 and T4 levels remain unchanged.
- The basal metabolic rate increases slowly due to increasing uterine demand.

Placental - Fetal Thyroid Physiology

The placenta is relatively impermeable to T4, T3 and TSH^(4,9)

The placenta contains the deiodinase enzymes that degrade T4 and T3

Thyroid stimulating immunoglobulins cross the placenta in maternal Graves' disease.

Thiouracil, propranolol and iodine cross the placenta and impair fetal thyroid function. Placenta hCG has a weak thyroid stimulating capacity and may account for minimal thyroid stimulation.

Levels of 300,000 miu/ml hCG are necessary to induce clinical hyperthyroidism. [2]

Fetal Thyroid Function

The fetal thyroid gland concentrates iodine by the 10th to 12th week of gestation when TSH in the pituitary and TRH in the hypothalamus can be detected.

After 30wks, T4 levels continue to increase despite declining TSH levels.

T3 levels are very low

Reverse T3 (rT3) is present by 28 wks.

At delivery, TSH increases, so do T3 and T4 which peak at 24 hrs after delivery [3].

Amniotic Fluid

The thyroid hormone concentration rises and peaks at 25-30 weeks. T4 rises faster than T3.

In the second half T4 decreases while T3 increases.

Reverse T3 (rT3) increases and peaks at 17-20 wks.

The source of the hormones may be fetal though the relationship is not well defined.

Fetal hypothyroidism has been treated successfully by administering the thyroxine directly into the amniotic fluid.⁽⁶⁾

Disorders Of Hyposecretion

Primary hypothyroidism results from failure of the gland itself. Hashimoto's thyroiditis, idiopathic myxoedema or radioactive iodine¹³¹ ablation of the thyroid gland may be the cause. Hashimoto's thyroiditis is common in patients with diabetes and autoimmune disorders.^(3,7)

Secondary hypothyroidism occurs as a result of loss of pituitary TSH stimulation. This may be due to

macroadenomas, craniopharyngiomas or ischaemic necrosis.

Cold intolerance, easy fatigability, constipation, dry skin and weight gain accompany hypothyroidism.

Diagnosis:

For primary hypothyroidism serum T4 is low and Serum TSH is high. As TBG is raised in pregnancy, the total T4 may remain normal. Thyroid antimicrobial and antithyroglobulin antibodies may be elevated.⁽⁴⁾

Outcome: Inadequate treatment causes spontaneous abortions, stillbirths, abnormal offsprings, and developmental retardation.

Treatment is given as:

Oral L-thyroxine 125 - 150ug/day.

Then follow-up determination of TSH every 3 weeks should reveal levels <6uU/ml and T4 concentrations in normal range.

Congenital and Neonatal hypothyroidism

Most infants appear normal at birth. There are no sequelae to neonatal hypothyroidism if treatment starts 3/12 after birth.

Aetiology

Primary hypothyroidism

Thyroid dysgenesis	1 in 4,000
In born errors of thyroid function	1 in 30,000
Drug induced	1 in 10,000
Endemic hypothyroidism	1 in 7
Secondary and tertiary	1 in 60,000 ⁽⁴⁾

Diagnosis:

There may be mental retardation, abnormal growth, deaf-mutism, spastic strabismus and abnormal sexual maturation. These are secondary to iodine deficiency occurring in utero.

At birth, an umbilical hernia, a large posterior fontanelle, dry skin, hypothermia, constipation and respiratory difficulties suggest hypothyroidism.

Confirmatory Labouratory Results

Serum T4 <4ug/100ml
Serum TSH > 80uU/ml

Treatment:

L-thyroxine: initial dose is given as 10ug/kg/day of oral thyroxine as single dose.

The requirement decreases to about 5 ug/kg at 12 months.

Disorders Of Hypersecretion

The causes are:

- Graves' disease in 95%
- Toxic multinodular goitre
- Toxic uninodular goitre
- Subacute thyroiditis
- Metastatic follicular cancer⁽⁴⁾

Symptoms and signs may manifest as heat intolerance, increased appetite, restlessness, increased cardiac output with systolic flow murmurs and pulse rate >100b/min. Graves' disease may show infiltrative eye signs (proptosis) disordered movement and pretibial myxoedema.

Labouratory confirmation of **diagnosis:**

- Elevated serum T4 and TSH <0.1uU/ml.,
- Elevated T3 index i.e. T4 x R T3U/100,
- Occasionally only T3 is elevated thus implying toxicosis in toxic nodular goitre.

Outcome:

PIH may worsen maternal prognosis. Neonatal morbidity and congenital malformations are more common with hyperthyroidism. Postpartum exacerbations may occur in Graves' disease, as there is decline in fetal T-cell suppressor function.

Treatment by either drugs or surgery

Thioamide drugs propylthiouracil (PTU) and methimazole inhibit iodination of tyrosine and hence decrease thyroid hormone biosynthesis. Also PTU decreases peripheral conversion of T4 to T3. PTU does not prevent the effect of the previously formed hormone and its effect is slow and takes 4-6 weeks.⁽⁸⁾⁽⁹⁾

Severe Hyperthyroidism:

May be treated with propranolol and thioamide. Intrauterine Growth retardation (IUGR), small placental size, neonatal bradycardia, hypoglycaemia and respiratory depression may be associated with maternal beta-blocker ingestion. Iodine treatment as temporary measure for not more than one week may be useful. Lugol's solution inhibits maternal iodine uptake and thyroid hormone secretion.⁽⁹⁾

Thyroid storm manifests as fever, dehydration, and cardiac failure. The mortality rate is 25%. Infection may also precipitate it.⁽¹⁰⁾

Treatment of severe hyperthyroidism

needs a combination of the following: -

1. IV fluids
2. Oxygen per mask or intranasal.
3. Antipyretics
4. Antithyroid drugs which are given as
 - a. Propylthiouracil 400mg orally 8 hrly or
 - b. Methimazole 30mg-40mg via rectal suppository 8 hourly or
 - c. Sodium iodine 1g in 500ml of fluid intravenously every 24 hrs
 - d. Lithium 300mg orally every 8 hrs.
 - e. Dexamethasone 2mg orally or (IM) every 6 hrs will block peripheral conversion of T4 to T3.
 - f. Plasma exchange used along with antithyroid drugs in difficult pregnant hyperthyroid cases has been described

Surgery:

Large doses of PTU may impair fetal thyroid function and surgery may instead be needed. Surgery needs preoperative medical treatment. It is best done in the 2nd trimester. Thyroid replacement hormone may be given immediately on detection of subnormal thyroid hormone levels after surgery.

Controversies in thyroid disease

1. Antithyroid drugs cross the placenta, and block thyroid hormone biosynthesis. TSH rises and goitre forms. But monitoring the dosing avoids impairment of intellectual function in children.
2. Infants can develop neonatal Graves' disease even if the mothers' hyperthyroidism is well controlled with PTU because there is transplacental transfer of thyroid-stimulating antibodies. The antibodies remain in the maternal circulation regardless of the treatment of the mother. For this reason, patients who have hypothyroidism after subtotal thyroidectomy or radiation therapy for Graves' disease are also at risk for a fetus with thyrotoxicosis. Thyroid replacement therapy may be necessary for the mother who has had a subtotal thyroidectomy but would require PTU for the sole purpose of treating fetal hyperthyroidism caused by autoantibodies.
3. The complications of transplacental transfer of thyroid-stimulation antibodies include in-utero fetal death, prematurity, IUGR, a widespread fetal autoimmune reaction with lymphatic hypertrophy and thrombocytopenia, fetal goitre, and fetal exophthalmos.
4. Aspirin should not be given in thyroid storm because it increases the percentage of free T4 and theoretically the hyperthyroid state.
5. The use of plasma exchange in life-threatening situations during pregnancy remains controversial though it is a potential therapy.
6. Radioactive iodine 131 I ablation of the thyroid in the hyperthyroid should not be done in pregnancy because it is teratogenic.
7. Surgery is hardly performed in pregnancy for the hyperthyroid except when large doses of PTU have to be given with poor compliance.
8. Breastmilk is essential for the infant and breastfeeding is necessary but with some reservations. PTU is excreted in small quantities in breast milk and can theoretically suppress the infant's thyroid function though the amount excreted is small. There is therefore a need for close monitoring of the newborn's thyroid function before permitting breastfeeding.
9. Thyroid storm can be described as a hypermetabolic state with fever and change in mental status. It usually manifests itself in a woman with unrecognised hyperthyroidism. It is a life-threatening complication and can occur during labour, caesarean section, or in conjunction with antepartum or postpartum infection. Treatment is symptomatic and also to block thyroid hormone release as outlined above.
10. Sometimes thyroid function is tested in a woman with hyperemesis gravidarum with a viable gestation because the disease has been associated with abnormal values on thyroid studies. The majority of these women have no other clinical signs of hyperthyroidism and treatment is not needed. However in a small number of cases, hyperthyroidism exists clinically, and treatment of thyrotoxicosis can help alleviate the hyperemesis gravidarum. (10,11,12,13)

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SECTION 5
SURGICAL PROCEDURES
IN OBSTETRICS

Caesarean Section

EY Kwawukume

Introduction

Caesarean section is one of the most commonly performed surgical procedures in obstetrics and it is certainly one of the oldest operations in surgery.

⁽¹⁾ The incidence is about 15 to 21 % in most West African Countries and would have been higher if there had not been acceptance of vaginal birth after caesarean section and some types of breech presentations.

The origin of caesarean section has always generated debate in most circles. It has been hypothesised that the great Julius Caesar was born in this manner and the mother lived many years after his birth. Critics believed that in 100 B.C when Julius Caesar was born this operation would have been fatal resulting in mortality for the mother or the child and do not therefore believe in this hypothesis.

Probably, this term was derived from the decree in Roman law, which made it mandatory for the operation to be performed on women dying during childbirth, a term called *lex caesariae*. Secondly, the Latin verb *caedere* means to cut; an abdominal birth is termed *partus caesareus*.

Definition of Caesarean Section

Caesarean section can be defined as delivery of a fetus through a surgical incision into the uterine wall after 28 weeks of gestation. It therefore means that non-surgical expulsion of the fetus/embryo from the uterine cavity or tubes following uterine rupture or ectopic gestation are not included.

Indications For Caesarean Section

As shown in Table 1 below, the caesarean section rate in Korle Bu Teaching Hospital from 1988 to 1999 is about 21.0 % (range 17-25 %). This is high but comparable to other tertiary health institutions in Africa. ⁽²⁻⁵⁾ About 20 years ago the rate of Caesarean section in Korle Bu Teaching hospital was 10.9 % ⁽⁶⁾. This rate has doubled in the present era in the same institution. The increase in the rate might be due to the following factors:

- High incidence of cephalopelvic disproportion being detected earlier in the developing world.
- A lower tolerance for taking risks.

- Fear of malpractice being levelled by other obstetricians and probable litigation from patients.
- Convenience of the physician.
- A couple's expectation of a perfect baby, as well as a woman's previous experience of a difficult labour, might play a part in the decision to perform a Caesarean section.
- Repeat Caesarean sections are high
- After repair of vesico-vaginal fistulae (VVF) subsequent deliveries are mostly by caesarean sections to guard against the break down of the repair.
- Sometimes retained second twins are delivered by Caesarean sections because by the time the patients reach hospitals there might be fetal distress or abnormal presentation or abnormal lie.
- Limited use of oxytocic augmentation increases resort to Caesarean section.

Vaginal Delivery After Previous Caesarean Section

Previous Caesarean section is the commonest indication for Caesarean section, as seen in Table 2. In the developed countries, trial of vaginal birth after Caesarean section (TVBACS) has become an accepted alternative to routine repeat Caesarean section especially in tertiary hospitals where electronic fetal monitoring and the capabilities to perform instant Caesarean section are available. ⁽⁷⁾ In the developing world though there are not enough electronic fetal monitors TVBACS for women with prior Caesarean sections is practiced. ⁽⁸⁻¹⁰⁾

A trial of vaginal birth after a Caesarean section achieved 50-80% vaginal delivery in some African countries. ⁽¹⁰⁾ A study in a rural area in West Africa achieved 66% of vaginal delivery after TVBACS. ¹⁰ A common risk of trial of vaginal birth after Caesarean section is uterine rupture with severe haemorrhage. It is mandatory that TVBACS should only be done in a hospital with personnel and facilities to perform repeat emergency Caesarean section, repair of ruptured uterus or hysterectomy if necessary.

Cephalopelvic Disproportion And Caesarean Section

As seen in Table 2, Cephalopelvic disproportion is the second common indication for Caesarean section in Ghana. In most reported series it accounts for about 20.8-53 % of all Caesarean sections.⁽¹¹⁾ in the tropics. The reason might be that in black African countries, women generally have narrower pelvic dimensions than their Caucasian counterparts.^(12, 13)

Unrecognised cephalopelvic disproportion might lead to obstructed labour, which is common in our rural areas, resulting in both fetal and maternal morbidity and mortality. The fetal head usually becomes impacted in the pelvis with excessive moulding and overriding skull bones resulting in fistula formation. Caesarean section for such patients is difficult and the technique of delivery of the impacted fetal head at Caesarean section as outlined below should be practised to avoid maternal morbidity and mortality.

Delivery of Impacted Fetal Head at Caesarean Section

The U-shaped incision with a broad base can be used for the delivery of the impacted fetal head⁽¹⁴⁾. The U-shaped incision is made into the lower segment of the uterus with the convexity of the incision pointing towards the pelvis. The straight low transverse incision does not create enough space when the head is deeply wedged within the pelvis, and attempts at delivery through the incision at times result in lateral extension of the wound. The uterine arteries may be involved, resulting in massive blood loss.

Additionally, before cutting into the uterus an assistant in sterile gloves should dislodge the fetal head by applying upward pressure through the vagina until the surgeon feels a sizeable part of the head abdominally. Delivery would then be easier.

Secondly, the obstetrician could get hold of the feet and perform breech extraction or use a single blade of the Wrigley forceps to dislodge the impacted head.

Indications for Caesarean Section

Fetal indications

- Fetal distress especially in the first stage of labour.
- Abnormal presentations that persist after attempt at external cephalic version, such as transverse lie and oblique lie.
- Face presentation mento-posterior.
- Multiple gestations triplets or greater

gestations in which the lie of the leading twin is abnormal.

- Macrosomia fetus weighing (>4,500gm).
- Footling breech.
- Very low birth weight (<1,500gm).
- Fetal abnormality hydrocephalus, conjoined twins and spina bifida.
- Vasa praevia.

2. Maternal indications

Severe preeclampsia with unfavourable cervix for vaginal delivery.

- Previous classical Caesarean section.
- Previous extensive uterine surgery (myomectomy) in which the uterine cavity was entered.
- Obstructive pelvic tumours (fibroids).
- Previous third-degree perineal tear and repair.

Previous reconstructive vaginal surgery.

- Large vulva condylomata
- Vulva herpes simplex virus

3. Maternal-fetal

- Cephalopelvic disproportion (relative).
- Failure to progress (arrest of dilatation in the active phase of labour or arrest of descent in the second stage of labour).
- Major placenta praevia
- Abruptio with a live fetus
- Absolute pelvic disproportion

Skin incisions

Skin incisions could be vertical or transverse. Each type has its own advantages and disadvantages.

1. Vertical incision

This incision could be sub-umbilical or could extend above the umbilicus. It is less vascular and gives good exposure of both the abdominal and pelvic organs. In emergency conditions with massive haemorrhage this incision could be indicated.

2. Pfannenstiel Incision.

Pfannenstiel introduced Pfannenstiel incision in 1900⁽¹⁵⁾. It is widely used because of its excellent cosmetic results. There is early ambulation and a low incidence of wound disruption, dehiscence and herniation. Pfannenstiel incision might take a longer time to perform than sub-umbilical midline vertical

incision because it involves dissection of the anterior rectus sheath and when extended into the external and oblique muscles, may result in injury to

the Iliioinguinal and Iliohypogastric nerves. The incision might also cause increased blood loss because of the increased dissection and also could limit the views of the upper abdomen. In general, Pfannenstiel incision is commonly used to perform Caesarean section primarily because of its Cosmetic value and also for its early ambulation.

3. Cohen's Incision

This incision was originally introduced for abdominal hysterectomy in 1954 and has since been used for Caesarean section.⁽¹⁶⁾ It is a straight transverse incision, placed slightly higher than Pfannenstiel incision. The anterior rectus sheath is incised in the midline for 3 cm but the muscles are not separated from the sheath and the peritoneum is bluntly opened in a transverse direction. The opening is widened by traction in a transverse direction with the help of the assistant.

4. Maylard Incision

This is also a transverse incision, which involves cutting the rectus muscles transversely and ligating the inferior epigastric artery. It provides better access to the pelvis than the Pfannenstiel incision

Uterine Incisions

There are many types of uterine incisions used in Caesarean sections. The most commonly used are the low transverse incision that is employed in more than 95 % of all Caesarean sections in our hospitals and the classical incision. The low vertical incision and the U-shaped incisions⁽³⁾ are occasionally used.

The advantages of transverse incision over a classical incision are:

- Less risk of entry into the upper uterine segment.
- Greater ease of entry.
- Less repair and easier reperitonealisation.
- Less likelihood of adhesion formation to bowel or omentum especially when it is not possible to apply the bladder flap above the top of the incision line.
- Less likelihood of subsequent uterine rupture during pregnancy. Lower uterine segment scars almost always rupture only during labour, but upper segment scars may rupture before labour and is certainly difficult to anticipate.
- Vaginal Birth After Caesarean section (VBAC) is possible.
- Technical ease of bladder dissection.
- Less intraoperative bleeding.

Advantages of classical incision (incision into the upper segment) are:

- Rapid entry into the uterus.
- No lateral extension into the vessels of the broad ligament.
- If the lower uterine segment is poorly developed delivery by classical incision is advantageous without lateral extension.
- Easy entry into the uterus when there is fibroid in the lower segment.

Despite these advantages the classical incision is hardly used in our hospitals because of the many disadvantages, especially subsequent uterine rupture and adhesion formation.

Sometimes there are difficult situations that might require an extension of a transverse incision to a "J"-shaped extension into the upper segment on the most accessible side. This incision is better than the inverted "T"-incision, which will form a weak scar due to poor healing. Both the J-shaped and the inverted T-incisions have been shown to be frequently associated with intraoperative complications and prolonged hospital stay.⁽¹⁷⁾ In our hospital the U-shaped incision is commonly used when difficult circumstances to deliver the fetus are encountered after performing lower uterine transverse incisions. The J or the inverted T-incisions are not normally used.

Risk factors during caesarean section in the presence of placenta praevia

- There can be severe bleeding because of placentation in the lower segment that has less contractile tissues than the upper segment.
- There is also the possibility of placenta accreta especially in the presence of multiple caesarean sections or previous uterine operations.
- Difficulty in delivery of baby in anterior or major praevia.

If there is anterior placenta praevia, first cut through the anterior muscular wall and carefully insinuate the hand between the uterine wall and the placenta to reach the membrane. Rupture the membrane and quickly deliver the baby. In certain situations it may become necessary to cut through the placenta to deliver the baby. There is a risk of severe bleeding and only experienced persons should do this. Do not cut through the placenta or the fetus could be you exsanguinated.

If it is posterior placenta praevia, deliver the placenta and bring the uterus to lie on the anterior

abdominal wall. If the skin incision were Pfannenstiel, then the anterior abdominal wall will be a splint for the uterus exposing the posterior placenta bed. Figure of eight stitches can be placed to control haemorrhage, which is common with posterior placenta praevia before closing the uterine incision.

Preoperative Procedure⁽¹⁸⁾

Caesarean section, like any surgical procedure carries major risk for both the mother and child. The indications should be explained to the patient and her consent obtained before the operation. Some of the complications that should be covered in the consent of the woman undergoing a caesarean section are: Bleeding, infection, injury to internal organs (including uterus, fallopian tubes, ureters, blood vessels and bowel), and injury to infant and possible need for blood transfusion.

General or regional anaesthesia (epidural or spinal) can be used for elective caesarean sections. In an emergency, general anaesthesia is often used because of the time needed to place an effective epidural. There is the risk of aspiration of gastric contents because of full stomach if the patient had previously eaten causing instant death or stormy post-operative complications. Aspiration is common because there is decreased cardiac sphincter tone of the stomach, increasing the likelihood of gastric reflux in pregnancy.

Blood should be taken for haemoglobin estimation, and sickling. Group and cross match two units of blood if this had not been done already. The operative area should be shaved and the bladder emptied. A self-retaining catheter (Foley's catheter) may be passed for continuous drainage.

Reassure the patient about the procedure and allay her fears about the operation and continuously communicate with her.

Procedure for Caesarean section

1. Place the patient on the table with a left lateral tilt to prevent supine hypotensive syndrome.
2. Wash hands with soap and water and dry with sterile towel.
3. Put on sterile gown and sterile gloves.
4. Under anaesthesia, clean abdomen with antiseptic solution and drape the patient.
5. Perform abdominal incision:
 - Enter abdomen through low transverse incision 2-3 cm above symphysis pubis or perform sub-umbilical midline incision.
 - Go through subcutaneous tissue and incise the rectus sheath that is composed of the aponeurosis of the external oblique muscles and the aponeurosis of the transversus abdominis.
 - Digitally separate rectus muscles.
 - The surgeon and assistant elevate the peritoneum with artery forceps at least halfway toward the umbilicus so as to avoid bladder injury, particularly in patients undergoing repeat caesarean section.
 - Incise the peritoneal fold between pairs of artery forceps and extend incision up and down exposing the uterus.
 - Pack paracolic gutters with moist abdominal packs if necessary.
 - Feel for dextro-rotation of the uterus and correct as applicable.
6. Perform uterine incision:
 - Place Doyen's retractor in the lower half of the abdominal wound.
 - Identify and lift the fold of uterovesical peritoneum.
 - Incise the fold of peritoneum transversely.
 - Push the lower leaf of the peritoneum downwards to displace the bladder downward, thus exposing the lower uterine segment.
 - Reposition the Doyen's retractor behind the bladder.
 - Incise the exposed muscle of the lower segment transversely, so that membranes bulge through the wound
 - Rupture the membranes.
7. Perform delivery of the fetus:
 - In cephalic presentation, deliver the head using hand, head lifter or Wrigley's forceps, as appropriate. A high head or floating head might give rise to difficulty in delivery. Always deliver the fetus in a flexed position similar to the fetal attitude in utero.
 - In breech presentation, conduct breech extraction.
 - If the baby is lying transversely, grip one or both legs and deliver by breech extraction.
8. Clean baby's face and suck nostrils and throat.
9. Clamp the umbilical cord in two places and divide with scissors and hand over the baby to the paediatrician or assistant.
10. Maintain haemostasis:
 - Make sure oxytocic has been given.
 - Apply Green Armitage forceps on the bleeding venous sinuses in the uterine incision.

11. Deliver the placenta by controlled cord traction.
12. Close uterine incision in two layers:
 - Close 1st layer of the uterine muscle using 1/0 chromic catgut or polyglycolic (Vicryl) suture in a continuous manner.
 - Suture the second layer of the muscle to invert the 1st layer.
13. Suture the uterovesical peritoneal flaps with 2/0 chromic catgut.
14. Suck blood and liquor from peritoneal cavity.
15. Remove the abdominal packs and check for haemostasis.
16. Inspect the ovaries, tubes and uterus.
17. Close the abdominal incision in layers:
 - Suture the abdominal incision in layers.
 - Suture the rectus sheath with Vicryl (No. 0) or nylon.
 - Suture the subcutaneous tissues with few interrupted or continuous catgut sutures, if necessary.
 - Close the skin with interrupted silk mattress sutures or continuous subcuticular nylon sutures as applicable.

Complications of Caesarean Section

1. Endometritis. Common in patients with prolonged rupture of membranes, repeated vaginal examinations and prolonged labour.
2. Bleeding leading to anaemia
3. Injury to internal organs (blood vessels, uterus, ovaries, fallopian tubes and bowel.)
4. Injury to neonate
5. Wound infection
6. Wound dehiscence
7. Urinary tract infection
8. Nausea and transient abdominal distension in the first 24 hours
9. Deep venous thrombophlebitis (DVT).
10. Maternal mortality might be due in part to the complications that required the Caesarean delivery or in part to the risk associated with the surgical procedure.

There are various opinions on how to reduce Caesarean rate in the developing world. In all reported cases in Africa, previous Caesarean delivery is the leading indication for Caesarean

section. Therefore, any measure aimed at reducing the incidence of repeat Caesarean sections would certainly reduce the overall Caesarean section rate.

In Korle Bu Teaching Hospital, Accra, previous Caesarean sections over a 12-year period (1988-1999) averaged 21.1% of the total number of Caesarean sections performed. This rate is rather high and good patient selection with supervised TVBACS, despite the lack of electronic monitoring devices might reduce this section rate.

In our present era, if there is one previous lower uterine segment Caesarean section then there should always be hospital delivery. In the presence of two or more previous Caesarean sections, a repeat Caesarean section is performed. Research is still in progress to deliver two previous Caesarean sections vaginally but until the outcome of such trials, it is advisable to deliver patients abdominally.

Myomectomy And Caesarean Section.

Myomectomy during Caesarean section is relatively unknown in the obstetrics literature. As a general rule in the 20th century, elective myomectomy was not encouraged during a Caesarean section because of the vascular uterus that might bleed profusely.

Often in Africa obstetricians encounter huge fibroids during Caesarean section. The question is whether to remove, incise through them when they are located in the lower segment and deliver the fetus, or, leave them behind for interval myomectomy.

Technique Of Bloodless Myomectomy At Caesarean Section:

- The fetus and placenta are first delivered by the usual Lower Uterine Segment Caesarean Section (LUSCS)
 - The uterine muscles are sutured and the pelvic peritoneum closed.
 - A tourniquet is tied round to encompass and compress both uterine arteries at the base of the broad ligament and the vessels in the infundibulo-pelvic ligament. This achieves a relatively bloodless field.
 - Routine myomectomy is performed.
 - The layers of the uterus are sutured up to the serosa
 - The rubber tourniquet is removed.
- The incision areas are inspected for bleeding and individual stitches are placed at bleeding points if necessary to maintain haemostasis.

Fig 1 shows intramural fibroids being removed during a caesarean section.

Probably, primary myomectomy at caesarean section might be the way forward in obstetric practice in this Millennium.



Uterus

Fibroids

TABLE 1: NUMBER OF CAESAREAN SECTIONS AT KORLE-BU TEACHING HOSPITAL (KBTH) 1988 1999

YEAR	TOTAL DEL	NO. OF C/S	%
1988	5,280	1,332	25.23
1989	6,718	1,791	26.66
1990	8,330	1,498	17.98
1991	10,301	1,744	16.93
1992	9,701	1,718	17.71
1993	11,786	2,149	18.23
1994	5,772	1,409	24.41
1995	11,754	2,086	17.75
1996	11,593	2,038	17.58
1997	12,273	2,370	19.31
1998	11,611	2,453	21.13
1999	10,851	2,583	23.80
TOTAL	115,970	23,171	19.98

TABLE 2: INDICATIONS FOR CAESAREANS AT KORLE-BU TEACHING HOSPITAL 1988-1999

YEAR	Previous Caesarean	CPD	Hyperten-sive Disease In Pregnancy	Fetal Distress	Malpresenta-tion And Malposition	APH	Cord Prolapse	Others
	(%)	(%)	(%)	(%)	(%)	(%)	(%)	(%)
1988	272 20.42	258 19.37	91 6.83	150 11	138	10 157	12 30 2.25	209 16
1989	378 21.11	383 21.38	114 6.37	100 6	160	9 174	10 42 2.35	179 10
1990	389 25.97	386 25.77	123 8.21	119 8	149	10 177	12 29 1.94	129 9
1991	495 28.38	317 18.18	167 9.58	136 8	138	8 169	10 34 1.95	288 17
1992	498 28.99	261 15.19	188 10.94	169 10	103	6 219	13 37 2.15	243 14
1993	668 31.08	196 9.12	324 15.08	173 8	169	8 236	11 38 1.77	345 16
1994	417 29.60	88 6.25	202 14.34	118 8	74	5 100	7 22 1.56	88 6
1995	284 13.61	293 14.0	511 45.47	248 12	165	8 691	33 35 1.68	930 45
1996	288 14.13	240 11.78	110 5.40	222 11	157	8 301	15 36 1.77	959 47
1997	337 14.22	273 11.52	169 7.13	287 12	177	7 291	12 43 1.81	1,055 45
1998	333 13.58	378 15.41	174 7.09	322 13	152	6 301	12 30 1.22	1,034 42
1999	539 20.87	438 16.96	174 6.74	430 17	213	8 411	16 38 1.47	660 26
	4,898 21.14	3,511 15.15	1,950 8.42	2,474 11	1,795	8 3,227	14 414 1.79	6,119 26

Psychological aspects of post-caesarean section

The main aim of obstetric care can be summarized as a physically healthy mother and infant but the psychosocial aspects are also important. There is a growing awareness of the psychological aspects of obstetrics and any woman about to undergo caesarean section should be properly counselled to withstand the stress of the operation. Some of these psychosocial outcomes are maternal happiness with childbirth, postpartum depression, post-surgery stress disorder, the relationship of the mother and the infant, breastfeeding practices and the sexual relationship with the husband or partner. The stress of coping with the baby and other siblings in the house and feelings about future births can be depressing to the mother.

Post-caesarean distress might be experienced in various ways. Some women might feel that there is the loss of an idealized birth they had hoped for during pregnancy. They therefore have a natural disinclination towards the operation

mainly because their peer groups insult them openly because they have not been able to deliver vaginally, which they consider the natural way. Some are afraid because they think operations are associated with mortality especially emergency operations. Others might have interrupted relationship with the baby especially when general anaesthesia is used and they feel the loss of the experience of birth and might think their baby is not really their own. Sometimes, women feel their body contours are deformed when they think of possible caesarean section scars and if these feelings are expressed as negative feelings then they think their body has been mutilated.

Another feeling about post-caesarean distress is anger at hospital workers because they feel the operation was unnecessary and probably there was inadequate support from hospital staff. Some patients make statements such as "doctor I can deliver by myself." There is therefore a feeling of lack of involvement of the patient in decisions to operate on her.

To prevent post-caesarean psychological distress the women should be provided with clear and

realistic information to reduce fear before surgery and to enhance emotional well-being after surgery.

⁽¹⁹⁾ Secondly, negative feelings that may follow Caesarean section under general anaesthesia should be addressed and unnecessary separation between the mother and child be minimised. Probably regional anaesthesia for Caesarean section might have the advantage of allowing the mother to be awake and witness the birth experience. Finally, there should be postnatal counselling and debriefing so that someone present at the caesarean section is allowed to talk through the birth with the mother soon after delivery. In one study, 58% of Caesarean mothers said that they wanted to talk with hospital staff about their feelings regarding the Caesarean, and yet only 14% were actually able to do so.⁽²⁰⁾

Women appear to value the opportunity to talk through their birth experiences with a health professional after a Caesarean and it is suggested that formal counselling for mothers who have a Caesarean delivery be introduced to minimise the emotional problems of the mothers.

Discussion And Controversies

Caesarean section by maternal request

There are various reports about women requesting delivery by caesarean section.⁽²¹⁻²³⁾ In the Changing Childbirth Report⁽²⁴⁾ published in London, women are encouraged to decide for themselves the type of care they would wish, the place of delivery and the degree of intervention. Other reports, internationally, have encouraged patients to become more involved in their treatment. In our sub-region there is little or no demand or probably some obstetricians do not acknowledge maternal request as an indication for Caesarean section and therefore do not agree to carry out the operation, or if they did, record another indication for it. Women certainly have reasons for requesting elective and repeat Caesarean sections and although they might not be medically indicated, many of the reasons would be understandable. Performing a Caesarean section when it is not clinically indicated has traditionally been considered unacceptable, but current views are changing. To probably arrive at a consensus opinion regarding the justification of Caesarean section for maternal request, maternal indications should be recognized and the reasons recorded for a logical approach to be taken on the issue. Maternal indication for Caesarean section should be carefully considered and documented because with the changing trends in medical

practice, Caesarean section that is thought of as a maternal request today may become an acceptable medical indication in the near future. Although vaginal delivery is associated with pelvic floor trauma and later problems of prolapse and incontinence, the patient should be counselled sufficiently about prophylactic Caesarean section and the current standard of care.

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Cervical Incompetence

K Nkyekyer

Introduction

The desire and effort to reproduce sometimes end in disappointment when pregnancy terminates in spontaneous abortion. Where this is repetitive, especially in the second trimester, there may be a feeling of frustration and almost of hopelessness. In societies in which childbearing is a cardinal expectation, repetitive pregnancy losses may produce a sense of inadequacy and failure, especially on the part of the woman. Mid-trimester pregnancy losses may be particularly distressing in view of the fact that fetal movements may have been felt but the joy and hope of carrying a baby are abruptly ended by previsible termination of the pregnancy.

Definition

Cervical incompetence may be defined as previsible or preterm termination of pregnancy due to cervical dilatation in the absence of any increase in baseline uterine activity or in the presence of mild uterine activity. The observed cervical dilatation is out of proportion to the degree of uterine activity. It usually occurs in the mid-trimester although in some situations it may present late as preterm labour. Typically, there is gradual painless dilatation of the cervix with bulging membranes which later rupture followed by expulsion of the fetus.

History of Cervical Cerclage

The first recorded reference to the cervix as a cause of pregnancy loss was an observation in 1658 that there may be situations in which the orifice of the womb is so slack that it cannot rightly contract itself to keep in the seed" (1). More than two hundred years later, in 1865, Gream suggested that dilatation or division of the cervix might result in the inability of the uterus to retain the conceptus till term (2). In 1874 Emmet described the procedure of trachelorrhaphy (3); this procedure was used by Sherman who in 1902 reported his experience of using it on three women with recurrent pregnancy losses, two of whom subsequently had successful pregnancies (4). In 1948 Palmer and Lacomme described the "gaping internal os" as a cause of recurrent abortion and also described a surgical

procedure for the treatment of the condition (5). Two years later, in 1950, Lash and Lash described the "incompetent internal os of the cervix" together with a surgical repair procedure (6). All the above-mentioned repair procedures were performed in the non-pregnant state. It was in 1955 that Shirodkar reported the first successful surgical procedure performed during pregnancy for the management of incompetence of the cervix. The procedure involved the placement of a suture around the cervix at the level of the internal os, and the general term cervical cerclage was used to describe it (7). McDonald, in 1957, described his much simpler purse-string cerclage technique (8). Many modifications of these basic techniques have subsequently been described. For example in 1961, Hefner et al described the Wurm procedure, naming it after Dr. Roger S. Wurm, an Australian doctor who first described it to them. This involved placing two mattress sutures at the level of the internal os: one suture run between 12 and 6 o'clock and the other between 3 and 9 o'clock (9).

Incidence

The incidence of cervical incompetence has been reported to be 2.7-18.4 per 1000 live births (10). This wide range is a reflection of the lack of uniformity of definition and diagnosis. Every reported study may have greater or lesser numbers of patients who do not have cervical incompetence. The number of patients in any study depends to a large extent on local selection factors.

Causes Of Cervical Incompetence

The causes of cervical incompetence may be congenital or acquired.

Congenital

Uterine anomalies, particularly such Mullerian defects as bicornuate uterus, uterus didelphys and septate uterus may be associated with incompetence of the cervix. It must be remembered, however, that the majority of women with such defects have uneventful pregnancies. Women who have had in-utero exposure to diethylstilboestriol (DES) may have defects predisposing them to the occurrence of cervical

incompetence. These include short cervix, which is flush with the vagina, non-fibrous soft cervical tissue, short cervical canal, and abnormal lower uterine segment. In congenital causes cervical incompetence may be part of a more complex Mullerian malformation.

There is evidence to suggest that in some women with cervical incompetence the defect may be physiological rather than anatomical. Studies on cervical biopsies from women with cervical incompetence, in both the pregnant and non-pregnant states, have demonstrated reduced strength when compared to controls (11,12). This reduction in strength may be due to abnormal composition of the cervix. In one study it was found that the mean percentage of cervical collagen was significantly smaller in women with cervical incompetence compared to women without incompetence (67% vrs 83%), while the mean percentage of muscle was higher in the incompetence group (22% vrs 9%) (13). Another study found no difference in collagen content but rather reported higher collagen extractability and collagenolytic activities in the incompetent cervix. The authors suggest a higher collagen turnover in the incompetent cervix and that the newly synthesised collagen is not yet fully cross-linked and therefore has a low biomechanical strength (11). The elastin content of the cervix has also been reported to be less in women with clinically diagnosed cervical incompetence than in controls (14).

Acquired

These are mostly traumatic and are the commonest causes of cervical incompetence. They may follow vaginal delivery or gynaecological procedures. Spontaneous vaginal delivery, especially where this has been precipitated, and operative vaginal delivery may result in cervical lacerations, which may not be recognised because there may not have been any associated significant bleeding. These lacerations may later be palpable to the fornix or even higher. Excessive and forceful dilatation of the cervix for procedures like termination of pregnancy may cause mechanical disruption of connective tissue fibres and thus lead to incompetence. Surgical procedures like cervical amputation and deep cone biopsy predispose to cervical incompetence.

It is also postulated that in some situations of cervical incompetence in which the cervix is visually and palpably normal the incompetence may be due to bacteria-mediated activity. Intra-amniotic or decidual invasion by bacteria may release cytokines some of which can modify the structure of collagen and other connective tissues and thereby lead to

incompetence. This mechanism may also lead premature rupture of membranes caused by the same collagenolytic activity or to preterm labour by the production of prostaglandins (15).

Diagnosis Of Cervical Incompetence

In the diagnosis of cervical incompetence the history in a previous pregnancy is of critical importance. Classically, there is recurrent spontaneous midtrimester abortion characterised by relatively painless dilatation of the cervix, rupture of the membranes and expulsion of the fetus. In some situations termination of the pregnancy may occur later in pregnancy, presenting as preterm labour, with subsequent pregnancies ending at progressively earlier gestational ages. Diagnosis, based on the history as it is, is thus a retrospective one. It is not necessary for one to wait till recurrent abortions occur before the diagnosis is made; if there has been only one abortion with the classic features the diagnosis may confidently be made, especially in the presence of other risk factors.

Investigations

There are some tests, which may be performed in the non-pregnant state to determine if there is cervical incompetence. These include the easy passage of number 8 Hegar dilator through the internal os, the Foley traction test (in which the force required to pull a size 16 Foley balloon filled with 1 ml of water through the internal os is measured), and hysterosalpingography showing dilated internal os and a widened isthmus. The usefulness of these tests is doubtful, since cervical anatomy and function in the non-pregnant state are not the same as in the pregnant. Where there is evidence of a torn cervix, especially laterally and reaching almost into the fornix a possible incompetence of the cervix may be suspected.

The clinician has the responsibility to decide, on an individual basis, which patient needs treatment for cervical incompetence. In the patient with a typical history most clinicians would place a cerclage suture in the cervix. The problem arises where the patient has had a previous midtrimester or early third trimester pregnancy termination but without a classic history. Such patients may be followed up by digital cervical examinations performed at weekly or fortnightly intervals, with timely placement of cerclage when cervical softening and effacement and/or dilatation occur. These patients must be advised to report promptly such symptoms as vaginal discharge, lower abdominal discomfort, or the sensation of a lump in the vagina (8). It must

be recognised that with this approach there is always the possibility that cervical changes may occur and result in labour and delivery during the interval between cervical examinations. It must also be pointed out that changes in the cervix do not necessarily indicate imminent abortion or preterm delivery. For example, in a study involving one hundred women whose pregnancies went to at least thirty-six weeks 15% of nulliparous and 72% of parous women had a cervical dilatation of 1 cm by the sixth month. Thirty six percent (36%) of parous women had a dilatation of at least 2 cm (16). In another study, 16% of nulliparous and 17% of parous women had an open cervix at 21-28 weeks gestation. The preterm delivery rate in these women was not significantly different from that of those with closed cervixes (17).

On the other hand, and even better still, patients without a classic history may be followed up with serial ultrasound assessment of the lower uterine segment and the cervix. The length of the cervix, the dilatation of the internal os and of the endocervical canal, the width and length of the isthmus, the prolapse of membranes through the internal os and the response of these parameters to stresses such as transfundal pressure, coughing or standing may be useful in confirming the diagnosis.

Ultrasound assessment of cervical length is more accurate than that performed through digital examination (18), and usually by the time cervical changes are detected by digital examination the changes may have become advanced. With regards to cervical ultrasonography transvaginal ultrasound has two main advantages over the transabdominal. The bladder is empty in transvaginal ultrasound while in the abdominal the bladder has to be filled and this may falsely elongate the cervix. In addition the anatomical landmarks of the internal and external os are more precisely seen with transvaginal ultrasound (19).

Guzman et al defined ultrasound diagnosis of cervical incompetence as progressive shortening of the endocervical canal length to less than 2 cm or a single measurement of less than 2cm. They found a significant correlation between the endocervical canal length measurements between 15 and 24 weeks gestation in the studied pregnancies and the earliest gestational age at delivery in previous pregnancies (20). In another study Guzman et al found that between 15 and 24 weeks gestation competent cervixes had a non-significant rate of endocervical canal shortening of 0.03 cm per week while incompetent ones had a significantly greater rate of endocervical canal

shortening of 0.41 cm per week (21). Wong et al studied the effect of an upright maternal position on the cervix as a functional test for cervical incompetence. They found that a greater than 33% decrease in cervical length in the upright position compared to the supine was associated with a significantly increased risk of preterm delivery when compared to those with less than 33% decrease in cervical length (87.5% versus 4%, $p < 0.0005$). They also found that when a cervical length of less than 2cm was combined with the postural change, the sensitivity for prediction of preterm delivery was 100% (22). It has also been reported that in women at risk for cervical incompetence, shortening of the endocervical canal length in response to transfundal pressure requires treatment with cervical cerclage because it is associated with progressive cervical changes over one to three weeks (23). Indeed transfundal pressure was found to be a more effective technique than coughing or standing position in eliciting cervical changes during pregnancy, and more sensitive in detecting the cervix that had progressive second trimester shortening (24). Mahran suggested that an internal os diameter of 15 mm or more in the first trimester or 20mm or more in the second trimester was diagnostic of incompetent cervix (25). Varma et al considered an endocervical canal width of greater than 7 mm with herniation of amniotic membrane an ominous sign (26). It must be mentioned, however, that just like in the case of digital assessment of the cervix, changes in the cervix may occur in between ultrasound examinations, and sometimes rapidly too, leading to pregnancy loss. A case has been reported in which an ultrasound examination showing a short but closed cervix was followed by digital vaginal examination twenty minutes later which revealed 4-5 cm dilatation (27).

It is possible that in future magnetic resonance imaging (MRI) may play a role in the diagnosis of cervical incompetence (28). In a case report cervical incompetence was diagnosed in a pregnant woman by MRI; in that patient ultrasonography had failed to provide conclusive evidence of extrauterine herniation of the amniotic sac (29).

In some situations it may be necessary to conduct tests to exclude other causes of recurrent pregnancy losses. These include tests for diabetes mellitus, thyroid dysfunction, lupus anticoagulant, chromosomal anomalies and cervical infections with organisms such as mycoplasma and ureaplasma.

Treatment

In women with classic history of cervical incompetence, cervical cerclage procedures during pregnancy are the standard treatment procedures for the condition. The usefulness of the surgical procedures has been questioned because there is little scientific evidence to establish their superiority over non-surgical modalities of treatment. Randomised controlled trials, which will answer these concerns, may be difficult to carry out since it will be considered unethical to withhold surgical treatment from women with classical history or signs of cervical incompetence. Most reported studies are case series in which pregnancy outcomes in women undergoing cerclage are compared with their previous pregnancy outcomes. Success rates of up to 90% have been reported for cerclage procedures. Such results must be interpreted in the light of the results of other studies in which subsequent pregnancies among women with repeated midtrimester losses were found to have a 70% success rate without any intervention (30,31). A fetal salvage ratio, defined as the ratio of fetal survival rate after cerclage to that before cerclage, has been suggested as a means of standardising the results of the procedure (32). However, results from various centres are difficult to compare because of differences in diagnostic methods, case selection, operative techniques and standards of neonatal care. In a study of cervical cerclage for the treatment of cervical incompetence in 207 Zambian women, post-cerclage survival rate of 87.4% and a fetal salvage ratio of 1.68 were reported (33). Others have reported fetal salvage ratios greater than two (34).

In women in whom the history is not classical, transvaginal ultrasonographic serial evaluations of the cervix, with secondary intervention as indicated, has been found to be a safe alternative to traditional primary prophylactic cerclage. It can save a number of women from unnecessary interventions (35,36). The Medical Research Council and the Royal College of Obstetricians and Gynaecologists of the UK conducted a multicentre randomised trial of cervical cerclage involving one thousand two hundred and ninety-two pregnant women whose obstetricians were uncertain whether to recommend cervical cerclage. Most of these women had a history of early delivery or cervical surgery. Cervical cerclage was compared with a policy of withholding the operation unless it was clearly indicated.

Although there were significantly fewer deliveries before 33 weeks and fewer very low birth weight

infants in the cerclage group, there was no statistically significant difference in the overall rate of miscarriage, stillbirth or neonatal death. The use of cervical cerclage, however, was associated with increased medical intervention and a doubling of the risk of puerperal pyrexia (37).

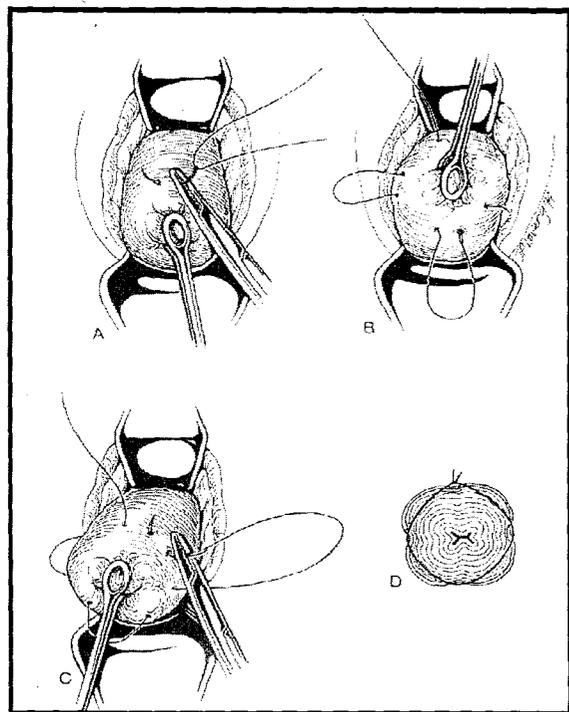
Cerclage Techniques

The cerclage techniques in widespread use are the Shirodkar's and McDonald's procedures, which are transcervical and are more commonly performed as elective prophylactic procedures. Because of it being simpler the McDonald procedure is the preferred method in West Africa. The two procedures appear to have similar outcomes (38).

The operation is usually carried out as an elective procedure in early second trimester (14-16 weeks). This is so that pregnancies, which may have terminated spontaneously in the first trimester presumably because of fetal chromosomal anomalies, are not maintained by the cerclage. It is important, before the cerclage, to perform ultrasound to rule out fetal structural anomalies.

Contraindications to cerclage include uterine bleeding, ruptured membranes, uterine contractions, major fetal anomalies and vaginal or cervical infections. Infections should be treated before surgery is attempted. In current practice a 4-mm Mersilene tape is what is commonly used; however where this is not available silk or nylon suture material may be used. In some hospitals in the sub-region what is commonly used is No.2 silk suture doubled on itself and threaded onto a large round-bodied needle.

General or regional anaesthesia may be used. Some prefer the latter because patients recovering from general anaesthesia may cough and retch, which may put undue strain on the cerclage.



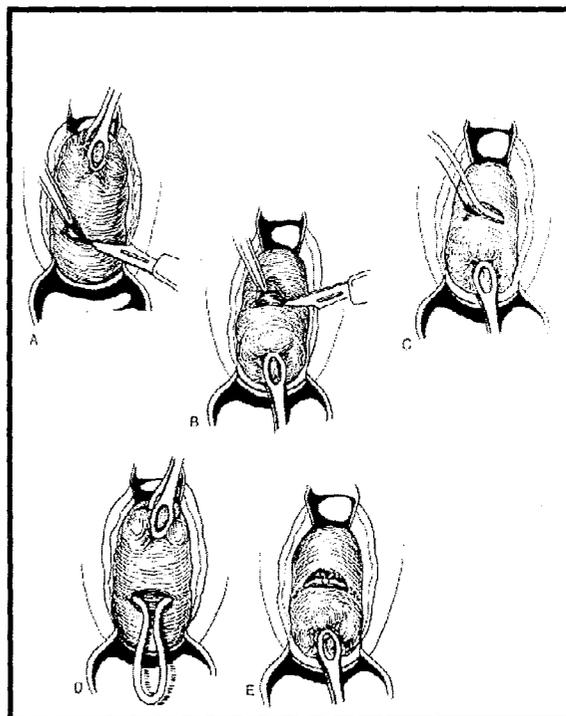
McDonald procedure

The patient is placed in a lithotomy position and after cleaning and draping the bladder is emptied. A speculum is applied to retract the posterior vaginal wall and enable the cervix to be visualised. A sponge-holding forceps each is applied to the anterior and posterior cervical lips and the cervix drawn down.

The junction of the rugose anterior vaginal mucosa with the smooth cervical mucosa is identified, which corresponds approximately to the level of the internal os. Placement of the cerclage suture is started just below the above-mentioned junction (in order to avoid going through the bladder) and four to six bites are taken circumferentially to complete a purse-string.

The first bite is taken starting from just before 12 o'clock and with the last bite the needle comes out just after 12 o'clock. The suture is placed deep into the cervical tissue, but not through the endocervical canal.

The needle is then removed and the suture pulled and knotted tight enough anteriorly to almost close the internal os; about four throws are used for the knot and the suture ends left long (2-3 cm) to facilitate identification and manipulation when it comes to removal.



Shirodkar procedure

The initial steps in this procedure up to the identification of the junction of the rugose anterior vaginal mucosa with the smooth cervical mucosa are the same. A transverse incision about 2 cm long is made just below the junction and the bladder dissected away by blunt dissection using the gloved finger until the uterovesical peritoneal fold is reached. The cervix is then pulled forward toward the symphysis pubis, the junction of the rugose posterior vaginal mucosa with the smooth cervical mucosa is identified and another transverse incision about 2 cm long made just below the identified junction. In the original procedure, an aneurysm needle was used to pass the suture submucosally round the cervix, from the posterior incision into the anterior. In current practice a large atraumatic needle may be used to achieve the same results. The knot is tied anteriorly, anchored to the cervix with a couple of 3-0 silk sutures and the vaginal mucosa repaired with the knot buried. The suture is anchored to the cervix posteriorly with a single 3-0 silk suture and the posterior vaginal mucosa repaired.

Post-cerclage management

Perioperative antibiotic therapy is advisable, although it may be mentioned that some have observed no difference in outcome between those patients given antibiotics and those not so treated (39). The use of tocolytics in the perioperative period is controversial and is perhaps best reserved for those with uterine irritability. When transcervical cerclage has been performed as an

elective prophylactic procedure, bed rest may be advised during the first twenty-four hours followed by mobilisation and increasing activity. The patient may be discharged home after a couple of days although cerclage as a day-case procedure has been reported to be appropriate (40)

Studies do not show any benefit in staying in hospital for more than a week (41, 42). However, in some cases the obstetric history is such that both the doctor and the patient feel more comfortable if the patient remained in hospital. This is especially so when there have been previous cerclage failures. When discharged, patients must be advised to avoid coitus or the insertion of any object in the vagina. They may gradually resume normal activity but must avoid strenuous physical activity. They must be advised to report any increased vaginal discharge, vaginal or backpressure or pelvic cramps. They may follow the routine antenatal clinic attendance schedule but may need to be examined every fortnight or so to determine the integrity of the cerclage.

The cerclage suture is usually removed at 37-38 weeks of gestation. However it is removed earlier if there is

- excessive vaginal bleeding
- intrauterine fetal death
- persistent uterine contractions
- rupture of the membranes

In a review of the management of cervical cerclage after preterm premature rupture of membranes it was found that latency seemed to be increased if the cerclage was kept in place, but maternal and neonatal infectious morbidity was also increased. It was suggested that in women in early gestational ages, keeping the cerclage in place may be warranted until labour ensues, while in more advanced gestations, it seems preferable to immediately remove the cerclage upon diagnosis (43).

With McDonald's suture removal can be performed without anaesthesia; however, with Shirodkar's because there may be the need to incise the vaginal mucosa and dissect in order to access the suture, general anaesthesia may be required.

Emergency Cerclage

Transcervical cerclage may be performed as an emergency procedure. This may happen in patients who do not have the classic history that necessitates prophylactic cerclage in early second trimester. Such patients are managed expectantly, with cerclage reserved for those who manifest

cervical change detected on clinical or ultrasound examination. On the other hand there may be situations in which women with no history suggestive of cervical incompetence may be found in an index pregnancy to have cervical effacement and dilatation without any uterine activity. Women in such situations may be considered for emergency cerclage and in one series a neonatal survival rate of almost 50% was reported (44). The fetal salvage rates of emergency cerclage are considerably less than those of the elective procedure. The incidence of complications, often due to infection, is high. Many patients require prolonged hospitalisation or bed rest and few reach full term (45). In spite of these, most reports recommend the emergency procedure as being beneficial as it may be the only way of prolonging pregnancy in the situations in which they are required (45-48).

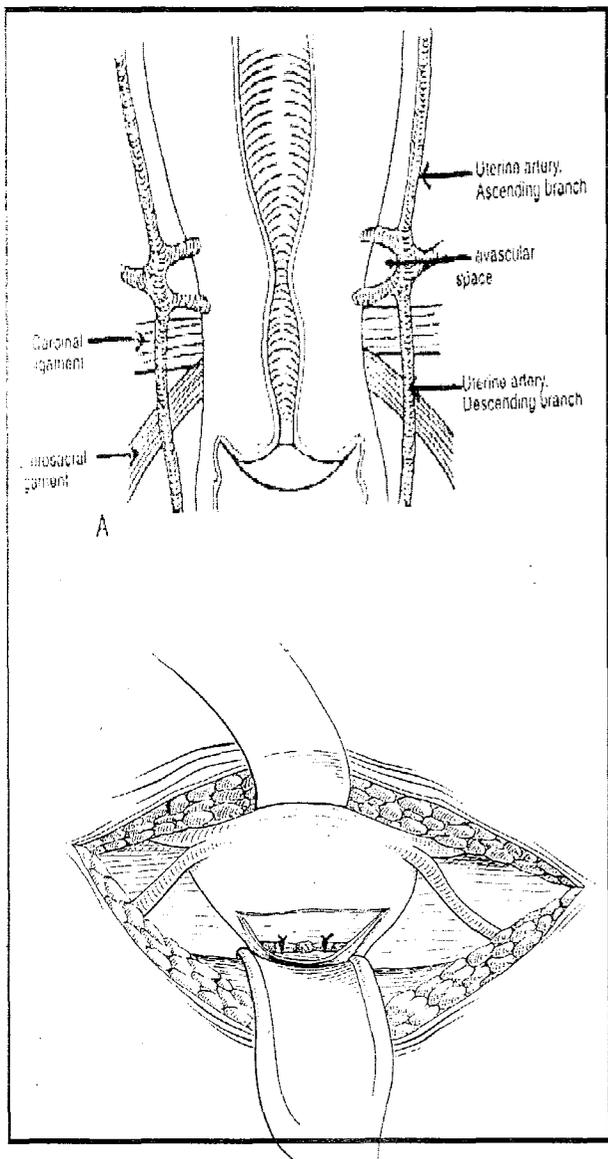
Pre-operative Procedure

When a diagnosis is established, the patient is placed on bed rest in the Trendelenburg position and uterine activity closely monitored. An ultrasound evaluation of the fetus is necessary and tocolytics may be administered if necessary. There should not be any contraindication to the insertion of the cerclage suture. Cervical cultures should be obtained to rule out infection with specific organisms such as group B streptococci. Prophylactic antibiotics are administered, and a 24-hour delay may be prudent, especially if uterine activity is marked, so that cerclage is not inserted in a woman who is in the process of aborting spontaneously.

Bulging Membranes And Cerclage

One major problem that may be encountered in emergency cerclage is bulging membranes and how to deal with them. Various techniques have been described in the literature to reduce the membranes. A Foley catheter with a 20-ml balloon in which the distal end has been cut off flush with the bulb may be used. The inflated bulb holds the membranes away from the internal os while the cerclage suture is placed, after which the bulb is deflated and the catheter removed (49). Another method involves the use of 6-10 stay sutures placed at the edges of the cervix (with the patient in deep Trendelenburg position), traction on which causes the membranes to move back into the uterine cavity (50). Bladder distension with up to 1000ml of normal saline may lead to a retraction of the membranes into the uterine cavity and allow cerclage placement (51). An inflatable bag has also been used to reduce bulging membranes (52)

Transabdominal amniocentesis has been used as a measure to temporarily reduce amniotic fluid volume and tension and assist in spontaneous reduction of the membranes (53). A combination of some of these methods may be necessary when dealing with advanced cervical dilatation and membrane prolapse.



Transabdominal cervical cerclage (TACC)

In a highly selected group of patients transabdominal cervicoisthmic cerclage may be the only means of achieving a high rate of fetal salvage. It is beneficial in patients with cervixes which are either extremely short, congenitally deformed, deeply lacerated (in all of which situations the cervix is in such a state that it is impossible to insert a vaginal suture), or previously failed transvaginal cerclage procedures (54,55). Post-TACC fetal salvage rates of 69.2% to 93% have been reported, while in the same studies pre-TACC salvage rates ranged between 5.2% and

18% (54-55). It has been performed both as post-conceptual and pre-conceptual procedures (54); postconceptional procedures are usually performed at an earlier gestational age than in transvaginal cerclage, between 10 and 14 weeks, after ultrasound confirmation of fetal viability. During the surgical procedure the abdomen is entered through a midline subumbilical or a Pfannenstiel incision. The uterovesical peritoneal fold is incised transversely at its reflection onto the uterus and the bladder flap carefully dissected downwards by blunt dissection, taking care to avoid injury to the venous plexuses present laterally. The uterus is brought up into the abdominal incision. The uterine artery on one side of the cervix is visualised splitting into ascending and descending branches; the relatively avascular space medial to the branches of the uterine artery but lateral to the cervix is identified and enlarged. A 5 mm Mersilene tape swedged onto a needle is placed through the avascular space from anterior to posterior. The same process is repeated on the other side, this time passing the suture from posterior to anterior. The band is tied snugly anteriorly in the region of the internal os with a single knot and the free ends of the knot secured to the tape by No.3-0 silk sutures placed about 1-2 cm from the knot. The bladder flap and the abdomen are closed routinely.

Invariably Caesarean section is required for delivery, the cerclage being left in place for future pregnancies. Where a preterm fetus needs delivery, laparotomy may be required to divide the band.

It may be mentioned that laparoscopic approach to the placement and removal of transabdominal cervical cerclage has been reported (59).

Complications

Haemorrhage is more likely with Shirodkar's procedure and with transabdominal cerclage. Cervical trauma, uterine contractions and rupture of membranes may occur intraoperatively. Rupture of membranes is more likely to occur during emergency cerclage. Postoperative complications include infection (including chorioamnionitis) and suture displacement. Some of the late complications are fistula formation and cervical stenosis. Scarring may cause cervical dystocia in labour, or result in deep cervical lacerations, which may extend into the broad ligament. Puerperal pyrexia was reported to be more common in a cerclage group than in a control group of women (MRC/RCOG). Potential fetal sequelae include prematurity, sepsis and intrauterine death.

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Operative Vaginal Delivery, Forceps Delivery And Vacuum Extraction

A.T. Odoi and H.S. Opare-Addo

Introduction:

To achieve a safe vaginal delivery the attending doctor will, in certain situations, have to perform operative vaginal delivery using the obstetric forceps or the vacuum extractor. While forceps delivery is widely used in Western Europe including the United Kingdom, the United States of America, and is still used in many developing countries, vacuum extraction has largely replaced forceps delivery in most developing countries, and in many countries in Northern Europe⁽¹⁾.

Whether the operator chooses the forceps or the vacuum extractor, the cardinal rules for success and safety for both the mother and her baby are:

- * Adequate skill and experience of the operator.
- * The choice of appropriate equipment/instrument.
- * Good case selection and judgement
- * Strict adherence to the prerequisites for the operation.
- * To aim at performing only simple procedures.

In the developing world where most deliveries occur and maternal morbidity, mortality, and perinatal mortality are unacceptably high, non-specialist doctors man most of the health facilities. Hence for safe delivery outcome, doctors at all levels must have the requisite knowledge, guidance, skill, and experience to perform operative vaginal delivery.

Forceps Delivery

Background

Ever since their invention in the seventeenth century by the Chamberlains, the obstetric forceps has revolutionised obstetric practice.

To improve its efficiency, this instrument has undergone several variations and modifications by other workers. Hence to date over 600 different obstetric forceps have been described⁽¹⁾. It is therefore important that the operator knows the type that will be suitable for a particular situation.

Forceps delivery is less commonly practised in the developing world compared to vacuum extraction as stated earlier for various reasons. Hence fewer and fewer doctors are acquiring the expertise in the use of the instrument with the passage of time. Senior colleagues with experience in forceps delivery should therefore endeavour to impart this knowledge and experience to their younger colleagues.

The Instrument

The forceps are a paired instrument made up of left and right parts or halves which articulate by crossing of their shanks. The parts of the forceps are:

a. The blades.

These are the parts of the instrument that are used to hold the fetal head. The blades of virtually all the obstetric forceps (with notable exception of the Kielland's) have two curves: the cephalic curve that fits the lateral aspects of the fetal head, and the pelvic curve, that corresponds to the curvature of the maternal pelvis. Because the Kielland's forceps is used for rotation of the foetal head it has virtually no pelvic curve.

b. The Shanks

This is the portion where the two halves cross each other and are articulated by the locks. The shanks may be generally parallel or overlapping. They may be short (e.g. Wrigley's) or long (e.g. Kielland's)

c. The lock.

This is the point of articulation of the 2 halves of the forceps. The lock may be sliding or non-sliding. The Kielland's forceps has sliding lock that allows it to be used to correct asynclitism of the fetal head.

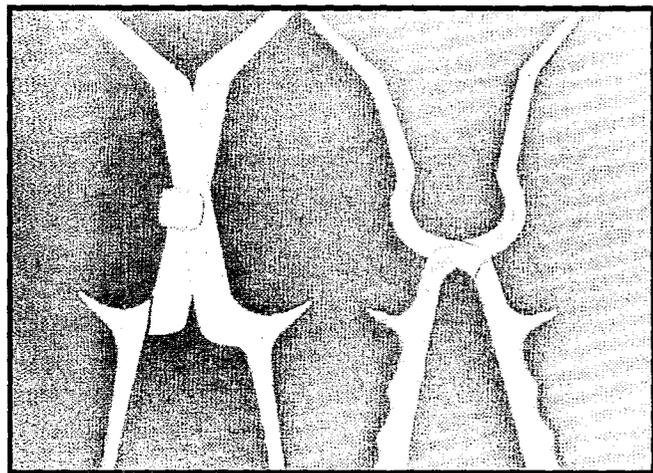
d. The handles.

These are where the doctor holds the instrument. They often have finger grips (or finger guides), which enhance traction.

The two most commonly used obstetric forceps in

the authors' centre are the Kielland's and the Wrigley's, as shown below

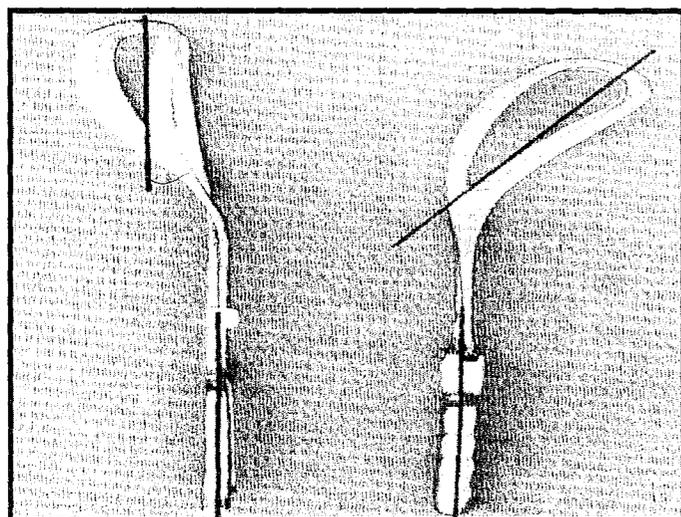
Fig. 1



Sliding lock
Kielland's

Non-sliding lock
Wrigley's

Fig. 2



A
A-Kielland's

B
B Wrigley's

There are some special features of the Kiellands forceps that are worth noting. This forceps, which was first described by Kielland in 1956, was designed purposely for delivery of the incompletely rotated fetal head. The shape is such that the axis of the blade lies parallel to the axis of the handle hence traction follows direction of the handles, so-called axis traction. The blades hardly have any pelvic curve; hence it is very satisfactory for the rotation of the fetal head. It also has a sliding lock. These features make it possible to apply the blades in cases of asynclitism. Finally, there is a knob on each finger grip (finger guide) that must point to the

occiput when the forceps are applied.

Operative Classification

Forceps delivery is classified according to the station of the presenting part when the instrument is being applied. These are (i) mid forceps delivery, (ii) low forceps (iii) outlet forceps. High forceps (when the foetal head is not engaged) is no more practised due to significant complications to both the mother and the baby.

i. Mid-forceps

This is when the forceps is applied on an engaged head above station plus 2 (+2). The Kielland's forceps is usually used here.

ii. Low Forceps

The fetal head is at station plus 2 or below but the fetal scalp is usually not visible at the vulva. Sub classified into Type A: rotation of the fetal head of equal or less than 45° and Type B: more than 45° rotation.

iii. Outlet Forceps

The fetal head is on the perineum (station plus three) with the foetal scalp visible without separating the labia. The sagittal suture is in the anteroposterior diameter of the pelvis or the foetal head is in right or left occipito-anterior, or occipito posterior position with head rotation less than 45°. Outlet forceps is the simplest and this is what is recommended for non-specialist doctors.

Caution: it is easy to confuse low and outlet forceps, and use these interchangeably. The key is the position of the sagittal suture and the visibility of the fetal scalp without significant caput succedaneum.

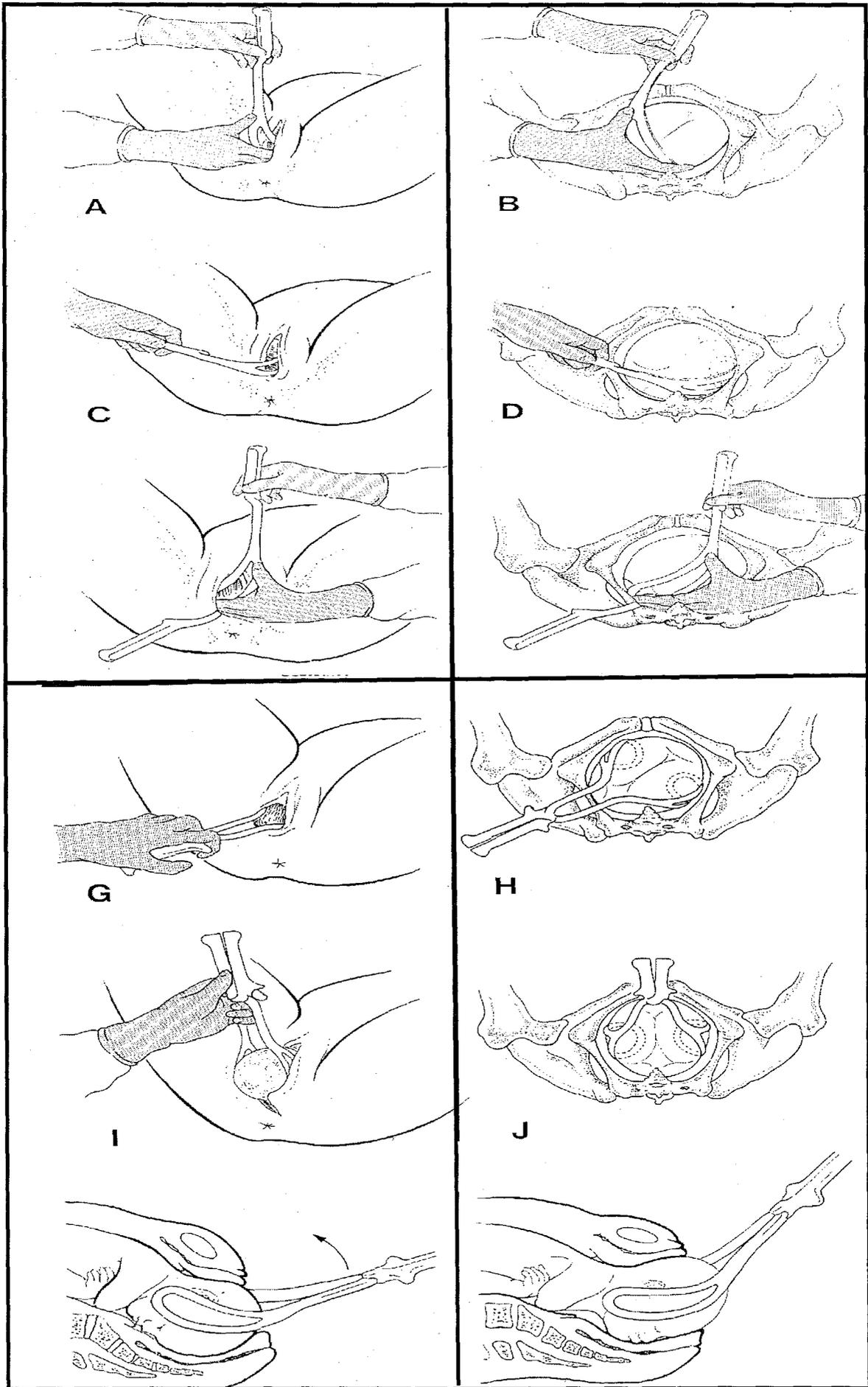
Indications for Forceps Delivery

Forceps delivery may be used electively in situations where excessive maternal expulsive efforts are not desirable, or an emergency for varied maternal and fetal indications.

Maternal Indications:

1. Elective:

- In situation where shortening of the second stage is required to minimise stress on the mother's cardio-pulmonary system
- Cardiac disease
- Severe hypertension/Pre-eclampsia
- Severe anaemia
- Pulmonary diseases including tuberculosis
- Thyroid disease



2. Emergency:

- * Maternal distress or exhaustion
- * Intra-partum haemorrhage in the second stage e.g. abruptio placentae.
- * Eclampsia
- * Sickle Cell disease patient in imminent crisis
- * Delayed second stage of labour:
 - Lack of or delayed progress due to
 - Poor maternal expulsive effort
 - Non-co-operation of the mother
 - Rigid perineum. With rigid perineum episiotomy alone may not be enough and operative delivery may be required.

Fetal Indications:

Fetal indications are almost always emergencies, to prevent fetal jeopardy.

1. Fetal distress in the second stage of labour
2. Cord prolapse at full cervical dilatation
3. Abruptio placentae in the second stage
4. The after-coming head in breech presentation.
5. It may be used electively to deliver a premature baby.

Conditions that must be fulfilled before embarking on Forceps delivery:

To avoid or minimise complications to both the mother and the fetus, certain pre-requisites must be met before one attempts a forceps delivery.

1. The cervix must be fully dilated:
 - The temptation to apply forceps with **nearly-dilated** cervix must be avoided at all costs.
2. The membranes must be ruptured
3. Suitable presenting part; mostly the vertex, infrequently the after coming head of breech in breech deliveries; and occasionally with face mento-anterior presentation.
4. The position of the vertex must be known with certainty
5. Fetal head must be well engaged
6. There should be no cephalo-pelvic disproportion
7. The estimated foetal weight must be less than 4kg
8. The bladder must be empty
9. Adequate analgesia (general anaesthesia, regional, pudendal block or local infiltration of the perineum)
10. Episiotomy must be performed
11. There must be adequate facilities for neonatal resuscitation
12. Above all the experience and skills of the operator is most crucial.

Contraindications to Forceps delivery

Forceps delivery is contraindicated if any of the above conditions cannot be met. In particular forceps must be avoided in situations of significant moulding of the fetal head with significant caput formation, deceiving the doctor to believe that the head is deeply engaged. Any likelihood of cephalopelvic disproportion is a contraindication because the forceps occupies space in the maternal pelvis, reducing further the available space for the fetus and increasing the risk of trauma.

Trial forceps where the operator attempts forceps delivery knowing that there is a probability of failure must be avoided. If such a trial fails (failed forceps) the complications are increased.

The Technique of Forceps delivery

It is not the intention of the authors to give detailed description of all the types of forceps delivery here. The emphasis is on simpler procedures that can be undertaken even by non-specialist doctors with requisite experience. Hence only low and outlet forceps will be described here. The reader is referred to the appropriate textbook⁽¹⁾ for the description of the other types of forceps delivery. However for every type of forceps delivery, the following must be noted:

1. The halves of the forceps are designated left or right in relation to the maternal pelvis.
2. Either half is held in the corresponding hand of the accoucher (the left hand holds the left half of the forceps).
3. To ensure that the blades are applied correctly it is usually helpful to assemble them as it would fit the fetal head before starting the actual application process.
4. The delivery comprises three processes:
 - a. Application: Applying the blades of the forceps to the fetal head.
 - b. Adjustment and Articulation: Adjusting the two halves of the forceps to fit properly together and lock.
 - c. Traction: Applying traction in the direction of the axis of the maternal pelvis.

The Technique

The technique for low forceps delivery with the fetal head in the left occipito-anterior position is described. The Wrigley's forceps is used here, but others prefer the Neville-Barnes or Simpson's. After ensuring that all the pre-requisites for forceps delivery are met the patient is put in lithotomy

position with the legs in stirrups.

Adequate anaesthesia by pudendal block, spinal/epidural, or general anaesthesia is given to enhance the patient's co-operation and prevent maternal injury.

The vulva and perineal area are surgically prepared and draped to expose the introitus. The bladder is catheterised to ensure that it is completely empty.

Full cervical dilation, ruptured membranes, head position and station are all re-checked.

- The two halves of forceps are assembled to ensure that they lock properly.
- The left blade is applied first:
The index and middle fingers of the right hand are inserted between the fetal head and the left vaginal sidewall. The left hand then holds the handle of the forceps. The blade is inserted between the vaginal fingers and the fetal head, with the vaginal fingers guiding it (the blade) along the fetal head, while the thumb helps to gently push the blade in place. At this point the handle should be almost perpendicular to the floor (Figure 3 A&B). The handle is next swept gently downwards along the mother's right thigh (C&D). Examination is then performed and necessary adjustments made to ensure that the blade is well applied.
- The right blade is then applied in the same way using the opposite hands (E-F)
- The two halves are then adjusted and articulated by locking of the shanks. The sagittal suture should be equidistant between the two blades.
- Soft tissue entrapment is ruled out.
- Traction is then applied along the axis of the pelvis, first downwards and backwards (G-H) until the occiput passes under the pubic arch, then horizontally, and finally upwards and forwards fig. (J-L) to complete the delivery of the head.
- Episiotomy is performed with the crowning of the head or as the perineum is stretched.
- After delivery the fetal head, the cervix, vaginal walls, and perineum are inspected for any laceration. The episiotomy is then repaired.

Complications of Forceps delivery

It is difficult to accurately assess the impact of forceps delivery on morbidity to both the mother and the baby. This is because the factors that lead to the intervention in the first place (e.g. prolonged labour, fetal distress) may have impact on the outcome. However there is no doubt that the operator's inexperience and poor judgement lead to increased complications.

Maternal Complications

Maternal complications include:

- complications of anaesthesia (especially if general anaesthesia is used)
- extension of episiotomy and perineal tears leading later to dyspareunia
- vaginal lacerations occurring during the application of the blades and during traction
- cervical laceration with upwards extension leading to profuse haemorrhage
- bladder damage that may result in vesico-vaginal fistula. These lacerations require prompt examination in theatre under anaesthesia and proper repair
- sepsis predisposed by prolonged labour and genital tract trauma during the delivery.
- spinal sprain and nerve root damage due to excessive traction force

Fetal Complications.

Common complications to the foetus include

- asphyxia
- skull lacerations
- supratentorial haemorrhage
- subdural and subarachnoid haemorrhage following skull fractures and compression
- cephalhaematoma
- Facial abrasion from undue compression or slipping of the forceps
- facial nerve damage leading to facial palsy.

On the whole the risks of these complications are very low with low forceps, but increases to about 10%-20% with mid-forceps, especially with rotational forceps delivery.

With adequate operator's experience and skill, good case selection and judgement, appropriate choice of instrument and strict adherence to the pre-requisites for forceps delivery these complications could be prevented or markedly reduced.

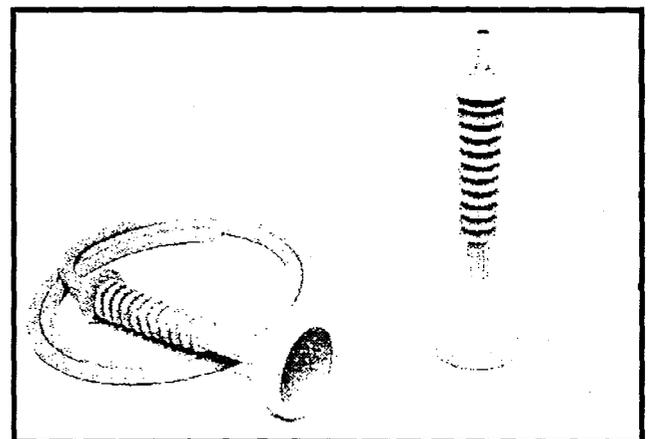


Fig .4 Silastic caps for Vacuum Extraction

Background and the principles of Vacuum extraction

The concept of vacuum suction seems to have originated from the application of vacuum to reduce depressed skull fractures in the mid early 1600's. In 1705 James Young, a surgeon in the Naval Hospital in Plymouth, tried putting the principle of vacuum extraction in obstetrics into practice without success. Arnot (1829), Simpson (1848), Souby Saleh (1857) all designed vacuum instruments. It was however not until Malmstrom of Sweden introduced his metal cap in the 1950's that the use of vacuum extraction became generally accepted in obstetric practice.

The principle of vacuum extraction entails the use of a cap (or cup) attached by a tubing to a pump that creates negative pressure (vacuum) of 0.8kg/cm^2 so that when the cap is applied to the fetal scalp this negative pressure is transferred to it. This enables enough traction force to be applied on the fetal scalp so that the fetal head is pulled along the axis of the maternal pelvis. By gradually increasing the negative pressure the fetal scalp is sucked into the cap forming a caput succedaneum (Chignon) to which the traction is applied. This chignon leads to airtight seal within the cap, tubing and the pump, and a firm grip of the cap on the scalp. This in turn enables adequate traction force to be exerted on the fetal scalp during uterine contractions as the mother bears down so called push-pull effect, to effect delivery of the baby's head.

The cap must be properly positioned on the sagittal suture very close to or just on the posterior fontanelle so that the rim facing the anterior fontanelle is about 3cm from this (anterior) fontanelle. When traction is applied on fetal scalp at this position it leads to flexion of the fetal head, hence this point is called the flexion point of the fetal head. If the cap is not properly positioned head deflexion or asynclitism may result.

The Equipment

The Malmstrom's equipment is made up of a rigid metal cap with curved-in rounded rim (like a ring) and a circumferential bulge, the outside diameter of which is 6 cm. This cap is attached to a traction chain that is covered by rubber tubing (which transmits the vacuum), at the centre of the back of its dome. This apparatus is in turn connected to a vacuum pump, which originally was a hand pump. There is a vacuum bottle through which air from the cap and tubing passes before going on to the vacuum pump. This bottle is partially filled with water and collects blood and amniotic contents from the tubing, preventing them from entering the

pump. The main variation of the Malmstrom's design is the Bird's modification (1969), which separates the vacuum unit (the tubing) from the traction unit (Chain). Bird also designed the 'posterior cap' for which the rubber tube is attached to the lateral wall of the cap. This was specifically designed for occipito-lateral and occipito-posterior positions of the vertex.

To date there are several changes in the equipment:

- * The metal cap now has smaller sizes (diameters) 5cm, 4cm and 3cm together with the 6cm cap.
- * There are now soft caps- silastic and plastic, which enable quick creation of vacuum with airtight seal without the formation of significant chignon, and are associated with less scalp trauma. However these are associated with higher failure rate compared to the rigid metal cap, especially with occipito-posterior position of the vertex. Unlike the metal cap, this soft cap has only one size of 6 cm in diameter and is dome-shaped. (Fig. 4)
- * There are now electrical vacuum pumps, which enable quick creation of negative pressure with safety. This is very useful in situations where every second wasted matters, for example in cases of severe fetal distress or cord prolapse.

In the authors' centre the electric pump is what is commonly used in conjunction with either the metal or the soft cap.

Indications for Vacuum Extraction

The indications for vacuum extraction are mostly the same as those for forceps delivery. In addition the vacuum extractor can be used in situations where the forceps should not be used, such as in cases of cord prolapse or severe foetal distress when the cervix is not fully dilated but is about 8 cm dilated. It can also be used to deliver the second twin in vertex presentation when the head is not engaged.

Maternal Indications

1. Elective:

In situations where excessive maternal expulsive effort could be detrimental to her cardio-pulmonary system and shortening of the second stage of labour is required.

- * Cardiac disease
- * Severe hypertension/pre-eclampsia
- * Severe anaemia, including **sickle cell** anaemia in imminent crisis
- * Pulmonary disease including tuberculosis
- * Thyroid disease

2. Emergency

- * Maternal distress or exhaustion
- * Intrapartum haemorrhage in the second stage, for example abruptio placentae.
- * Delayed second stage of labour
Lack of, or delayed progress due to
 - poor maternal expulsive effort
 - conduction anaesthesia
- * Eclampsia
- * Sickle cell disease patient in imminent crisis

Fetal Indications:

Fetal indications which are almost always emergency include

10. Fetal distress in the second stage of labour
10. Cord prolapse when the cervix is 8 cm or more dilated
10. Abruptio placenta in second stage
Second twin with vertex presentation.

Conditions that must be fulfilled before embarking on Vacuum Extraction

Again these are mostly the same as the prerequisites for forceps delivery.

1. The cervix must be at least 8cm dilated.
2. The membranes must be ruptured
3. The vertex must be presenting
4. The position of the vertex must be known with certainty
5. The fetal head must be engaged (except in cases of second twin with vertex presentation)
7. There should be no significant cephalo pelvic disproportion
7. The bladder must be empty
8. The operator must have the requisite experience or if he/she is now acquiring the skill, an experienced operator must be present.
9. The operator must have the willingness to abandon the procedure when it fails (see failed vacuum)
10. Anaesthesia and/or episiotomy are not always required for vacuum extraction.

Contraindications for vacuum extractions

Ventouse delivery is contraindicated, if any of the above-listed prerequisites is not met. It is contraindicated if the gestational age is less than 32 weeks, due to increased risk of cephalhaematoma. It is also to be avoided if fetal scalp blood sampling has been done, due to increased likelihood of bleeding from the site.

The Technique of Vacuum Extraction

Before describing the procedure of ventouse delivery the following must be noted.

1. The mother must be counselled, including the possibility of caesarean section should the vacuum extraction fail
2. Both the metal and soft caps must, ideally, be available.
3. If the head is flexed and synclitic the soft cap must be used. If the head is deflexed, or asynclitic, or malpositioned the Malmstrong's or Bird's cap must be used.
4. Before applying the cap to the fetal scalp the operator must assemble the apparatus and test for sustained negative pressure on the palm of the gloved hand.

The procedure:

This involves

1. Application of the vacuum cap
2. Creation of the negative pressure
3. Traction.

With appropriate indication and all the prerequisites met, the patient is put in lithotomy position, and draped. The bladder is emptied with a sterile rubber catheter. The cervical dilation, fetal head position and station are rechecked. If indicated, anaesthesia is administered by local perineal infiltration or pudendal block

1. Cap Application

The metal cap

The largest suitable cap is selected to minimise fetal scalp trauma. The cap is dipped into an antiseptic solution such as savlon to lubricate it (lubricating creams are best avoided, else the cap will slip off the foetal scalp). The index and middle fingers of the left hand are inserted into the introitus to depress the perineum. The cap is gently introduced into the vagina and placed at the flexion point of the fetal head described earlier. Soft tissue entrapment is excluded by running a finger around the entire rim of the cap.

The Soft cap

The cap is dipped into antiseptic solution such as savlon to lubricate it. The rim of the cap is then folded in to reduce its diameter. The introitus is then exposed with the fingers of the left hand and the cap is gently and carefully introduced into the vagina. It is applied to the flexion point of the fetal scalp and soft tissue entrapment ruled out as described for the metal cap.

2. Creation of the Vacuum

Once any entrapped maternal tissue is released from the cap, negative pressure is created manually with the hand pump or electrically to 0.2kg/cm². Soft tissue entrapment is again checked for and any maternal tissue that has been sucked into the cap is released. The negative pressure build-up is continued at the rate of 0.2kg/cm² every 2 minutes if the Manual Pump is used, or steadily without pulsing, if the electrical pump is used, till it reaches 0.8kg/cm². This leads to the formation of Chignon in the cap.

3. Traction

The index finger of the left hand is placed on the cap near its periphery to stabilise it while the middle finger is placed on the foetal scalp to check for slipping of the cap as well as descent of the head with traction. With uterine contraction and the mother bearing down, traction is applied along the axis of the maternal pelvis. This traction must always be at right angles to the cap. The operator should not attempt to rotate the foetal head. There will be autorotation as traction proceeds. If the cap is felt to be dislodging or air is being sucked into the apparatus, traction should be suspended. The cap position should be rechecked and soft tissue entrapment must be excluded and the possibility of successful vaginal delivery reassessed. If delivery is likely to succeed the vacuum is recreated and the traction process resumed. Traction can be continued till the head is completely delivered, or can be stopped when the head crowns. The negative pressure is then released, the cap is detached from the head and delivery completed as in normal vaginal delivery. When indicated episiotomy is performed when the head crowns or the perineum is stretched.

If obvious descent is not noticed after 3-4 pulls, or the cap pulls off twice, the procedure should be deemed to have failed and another method, preferably Caesarean section resorted to.

After delivery the baby is examined for scalp lacerations, and the mother, genital tract lacerations and haematoma. The cause and significance of the chignon is explained to the mother and reassurance is given about its rapid disappearance.

Failed Vacuum Extraction

Failed ventouse delivery as defined above can occur if the case is not well selected, the appropriate cap is not selected, the cap is not well applied, traction force is not exerted properly, or the apparatus is faulty. The commonest cause of

failure is improper traction where the traction force is applied non-perpendicularly to the cap. Unrecognised cephalo-pelvic disproportion and malposition of the vertex are also causes of failed vacuum extraction. The beginner is therefore advised to start with simpler cases of outlet deliveries before attempting those at higher stations. After failed vacuum the complications on the baby are much less, if any, compared to those following failed forceps delivery.

Complications of Vacuum extraction

Maternal Complications

- * Periurethral and perineal lacerations: these occur during insertion of the cap if improper technique and/or force are used.
- * Vaginal laceration and/or haematoma. The vaginal wall may be trapped or sucked into the vacuum cap leading to laceration with haemorrhage, or haematoma formation.
- * Cervical laceration: During first stage extraction part of the cervix may be partially or completely avulsed. The cervix may also tear, with upward extensions into the lower uterine segment with profound haemorrhage. This may happen during first stage extraction if slow traction is not applied initially to enable the cervix to be fully dilated.

Fetal/Neonatal Complications

- * Scalp trauma: scalp abrasions are fairly common with the metal cap.
- * Cephalhaematoma: In difficult deliveries cephalhaematoma may occur. Rarely there may be subgaleal haematoma, tentorial tears and even intracranial haemorrhage.
- * Neonatal asphyxia has been noted with prolonged extractions, but the indication for the intervention may compound the problem.
- * Neonatal jaundice: There is increased incidence of neonatal jaundice associated with vacuum extraction compared to spontaneous delivery and forceps delivery.

Over all the complications of vacuum extraction are less serious compared to those of forceps delivery. With operator's experience, good case selection, and adherence to the prerequisites for the procedure, these complications could be avoided or markedly reduced. So far no increase in long-term neurological deficits has been noticed compared to spontaneous deliveries.

Discussion

Operative vaginal delivery by forceps or vacuum extraction plays a very important role in modern day obstetrics. This role is even more crucial in the African setting where there is much aversion for abdominal delivery, and Caesarean section is regarded as a reproductive failure⁽²⁾. Secondly, after Caesarean section the patient may not report again to hospital for subsequent deliveries for various reasons⁽³⁾, including the fear that she may undergo another caesarean section. Thus she stands at a high risk of uterine rupture with its poor outcome in our setting⁽⁴⁾. Any method that enhances a safe vaginal delivery should therefore be fully embraced.

There is regional variation in the preference for particular equipment. While the obstetric forceps remains popular in most of the English-speaking countries including the United Kingdom and the United States of America, as well as Eastern Europe and South America, the ventouse is most popular in Asia, Northern Europe and particularly Africa⁽⁵⁾. For example while forceps delivery is very frequently performed at the authors' centre, the incidence of vacuum extraction at this centre is about one in fifteen deliveries.

A similar incidence has been recorded in Nigeria⁽²⁾. The popularity of the vacuum extractor in the developing world may be partly due to the advantages this equipment has over the forceps.

Advantages of the ventouse over forceps:

- The technique of vacuum extraction is easily learnt and the skill more easily acquired compared to the forceps. Thus in the developing world where specialists are lacking in most health facilities, it is far easier to train the non-specialist doctors to use the vacuum extractor.
- The vacuum cap does not occupy space in the maternal pelvis. Cephalo-pelvic disproportion is very common in the developing world, and is the commonest indication of Caesarean section in Ghana⁽⁶⁾. Many cases of delayed second stage of labour are due to mild disproportion or malposition. Most of these cases can be successfully and safely delivered with the vacuum extractor⁽²⁾. Because the blades of the obstetric forceps occupy space in the maternal pelvis, its use in this situation is associated with increased complications to both the mother and the baby, including neonatal **Erb's** and **facial palsy**⁽⁷⁾.

- Anaesthesia is not a pre-requisite⁽⁸⁾. Since there are inadequate anaesthetists in our health facilities, and the ventouse can be used with just perineal-infiltration analgesia when necessary, this equipment is preferred.
- Natural forces come into play: The mother pushes to assist delivery as occurs in spontaneous delivery. Thus she feels satisfied that she participated in the delivery process. Secondly, traction leads to autorotation of the fetal head, as occurs in spontaneous vaginal delivery. Thus the ventouse can be used in occipito-posterior and occipito-transverse positions with little complications compared to the forceps⁽⁷⁾.
- In-built safety mechanism: The cap pulls off from the fetal scalp when excessive traction force is applied. Thus it is less likely to exert too much traction force on the fetal head. Hence if a less experienced person has misjudged the case to be suitable for vacuum extraction, after two pull-offs he changes his mind for Caesarean section with less complications to the fetus. On the other hand with the forceps the operator can apply excessive traction force, which may lead to severe maternal and neonatal complications⁽⁹⁾.
- Failed vacuum extraction is associated with fewer complications to the mother and the baby, partly because of its in-built safety mechanism described above.
- The ventouse can be used in the first stage of labour. This is a very significant advantage over the forceps in the African setting. Many women who would have undergone Caesarean section for fetal distress or cord prolapse in late first stage (cervical dilatation of 8 cm to 9 cm) are successfully and safely delivered by vacuum extraction. Thus they are spared abdominal delivery and have the confidence to return to the hospital in subsequent pregnancies.
- Over all there are lower maternal and fetal/neonatal complications compared to the forceps^(1,10).

With these advantages the vacuum extractor is gaining wider acceptance even in the United States as recently trained Fellows are using this equipment more often^(11,12).

Advantages of the Forceps over the Ventouse

The forceps nevertheless has some advantages over the vacuum extractor:

- In experienced and skilled hands it effects faster

delivery and hence very useful in cases of severe fetal distress or cord prolapse if the cervix is fully dilated and other pre-requisites are met.

2. It can be safely used to deliver the premature infant with birth weight less than 1.5 kg where the ventouse is contraindicated.
3. It can be used to deliver the after-coming head in breech delivery.
4. It is very handy and does not easily develop faults.

It is therefore useful to learn the use of both instruments and have both available in every obstetrics-practising health facility.

Complications:

In spite of their extensive use over the years both the forceps and the ventouse continue to be associated with complications. Low et al found that the most frequent infant morbidity associated with use of the Soft silicone vacuum cap was neonatal jaundice⁽⁸⁾ while Kuit et al noted more scalp injury with the metal cap⁽¹³⁾. Subgaleal haemorrhage^(14,15), subdural haematoma and cerebral infarction⁽¹⁶⁾ and even skull fracture⁽¹⁷⁾ have been reported in association with vacuum extraction. These latter complications are, however, very rare and are associated with difficult deliveries with long application of the cap. With proper case selection, proper technique, and adequate experience these serious morbidities should not occur with the use of the ventouse.

The forceps is more commonly associated with morbidity than vacuum extraction^(7,10,18). This emphasises the need for adherence to the prerequisites for the procedure and limiting oneself to simpler procedures such as 'lift out' outlet forceps.

Controversies:

There are some controversies associated with the use of both instruments.

1. Should non-specialist doctors be taught and encouraged to practise forceps delivery?

The Division of Family Health of the World Health Organisation (W.H.O.) encourages the use of both the obstetric forceps and vacuum extractor by non-specialist doctors in district hospitals with limited access to specialist services⁽¹⁹⁾. The case for encouraging operative vaginal delivery in the developing world has already been made earlier in

the discussion. The authors strongly agree that to reduce maternal and perinatal morbidity and mortality, especially in the developing world, non-specialist doctors, who see more of our pregnant women in the district hospitals, must be taught and encouraged to practice operative vaginal delivery. They must follow the prerequisites for the procedure strictly, and limit themselves to simple cases. Because the forceps is associated with more complications they must use it only when the vacuum extractor is not available or is faulty, and even then they must limit its use to the gentle lifting out of the fetal head at the pelvic outlet.

2. Should nurse-midwives be taught and encouraged to practise operative vaginal delivery?

A case can be made for nurse-midwives in the developing countries to practice operative vaginal delivery. Midwives in both the public and private sectors conduct the overwhelming majority of deliveries in these countries. Hence no matter how few operative vaginal deliveries they perform, the overall positive impact will be enormous. The authors recommend that because forceps delivery requires more skill, expertise and anaesthesia, and is associated with more complications, with failed forceps leading to more complications compared to the ventouse, nurse-midwives should not practice forceps delivery. They should be taught the principles of vacuum extraction as they apply to the practice of nurse-midwifery and be given strict guidelines to its use.⁽²⁰⁾ They must select the simplest of cases, that is where the fetal head (not caput succedaneum) is at the pelvic outlet. The Ministry of Health of Ghana now trains and encourages nurse-midwives to perform simple vacuum extractions.

3. Does rotational forceps delivery have a place in contemporary obstetric practice?

Evidence continues to accumulate against midforceps delivery, especially rotational forceps. Apart from the increased likelihood of maternal and neonatal trauma^(7,21) there is deterioration of fetal acid-base balance associated with rotational delivery with the Kiellands' forceps⁽²²⁾.

We recommend that if intervention is very imperative, for example for fetal distress when the fetal head has not descended to station +2 or below, vacuum extraction or caesarean section must be done. This is far better than adding the trauma of difficult mid-pelvic forceps delivery to an already distressed baby.

4. Creation of negative pressure in vacuum extraction:

Does continuous (one-step) creation of the vacuum compromise efficiency and safety compared to the conventional intermittent vacuum creation?

In some situations (e.g. cord prolapse) every second counts for the baby. Randomised controlled studies have shown that rapid creation of the vacuum significantly reduces the duration of a vacuum extraction procedure without compromising to efficiency and safety⁽²³⁾.

5. Do we have to reduce the vacuum in-between contractions and does maintenance of vacuum and traction force in-between contractions (to prevent loss of fetal head station) lead to faster delivery and better outcome?

No differences in speed of delivery, maternal or neonatal outcome could be demonstrated if the level of the vacuum was reduced or maintained in-between contractions, or traction was maintained or reduced in between contractions⁽²⁴⁾. The authors recommend that the vacuum should be maintained but the traction can be stopped in-between contractions. This will allow both the operator and the mother some rest and also enable the operator to recheck for any soft tissue entrapment, and descent of the fetal head with the previous traction.

Conclusion

The proper use of the obstetric forceps or the vacuum extractor remains a safe and effective modality in achieving the universal goal of obstetric care - a healthy mother and baby. Such safe vaginal delivery is most crucial in the African setting. Because of its advantages over the forceps the vacuum extractor is gaining worldwide acceptance and is now the preferred instrument in most parts of the world. But the easier application of the ventouse may encourage misuse. Given the apparent association between difficult operative deliveries and increased morbidity, it is incumbent on the operator to attempt delivery only when vaginal delivery seems to be a safe option. Furthermore the operator must be willing to reassess the attempt if initial efforts are not met with signs of successful vaginal delivery.

The ultimate choice of the route of delivery and the mode of operative vaginal delivery should reflect a consideration of the maternal and/or fetal condition, fetal presentation, position, and station

of the fetal head, and the operator's experience. The safe use of both instruments requires continued experience, attention to guidelines and prerequisites, attention to subtleties of design of the instrument, and limitation of oneself to simpler procedures. These have been stressed throughout this chapter.

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Episiotomy And Third Degree Tear

EY Kwawukume/ Ali Samba

Episiotomy is the surgical enlargement of the vaginal orifice by an incision of the perineum during the last part of the second stage of delivery. Except for cutting the umbilical cord episiotomy is the most common operation in obstetrics. Carl Braun¹ coined the term episiotomy and a report as far back as 1741 suggested the first surgical opening of the perineum to prevent severe perineal tears.²

The place of episiotomy in modern obstetric practice has been questioned and the practice of routine episiotomy no longer exists. There are wide variations in rates of episiotomy around the world being 62.5% in the USA³, 30% in Europe^{4,5} and with higher estimates in Latin America. In Argentina episiotomy is a routine intervention in nearly all nulliparous and primiparous births.⁶ The rate at the Korle-Bu Teaching Hospital in Ghana is 15-17%.⁷ Diverse rates suggest that the practice of routine episiotomy is not justified. Too frequent recourse to episiotomy suggests bad management of the second stage. An episiotomy must be performed at the correct time and place and repaired properly within as short a time after delivery as possible.

Indications

- When the perineum threatens to tear as in:
 - persistent occipito-posterior position.
 - extensive scarring of the perineum from previous repair of tears or female genital mutilation or very tight perineum.
- Delay in second stage of labour because of soft tissue resistance
- Breech delivery.
- Fetal distress and cord prolapse to facilitate quick delivery
- Instrumental vaginal delivery; all cases of forceps delivery require episiotomy but not all ventouse cases require it.
- Preterm labour to minimize the stress on the foetal head
- Macrosomic babies to increase manoeuvrability in the event of shoulder dystocia.

Types Of Episiotomy

There are three types:

- Mediolateral episiotomy, which is the commonest, is cut at 45 degrees angle from the

midline towards the ischial tuberosity avoiding the anal sphincter. It can be made on the right or the left.

- Midline (central or median) episiotomy is cut in centre of the perineum separating the bulbocavernosus and superficial transverse perineal muscles.
- The J-shaped incision. This type of incision has the advantage of medial incision and provides better access than the mediolateral. The incision is made tangential to the top of the anus.

Whichever type is used, the incision must start from the midline. The first two types have their advantages and disadvantages.

Midline Advantages And Disadvantages

- Easy to repair
- Faulty healing is rare
- Less painful in the puerperium
- Dyspareunia rarely follows
- Anatomical result almost always excellent.
- Blood loss smaller
- Extension through anal sphincter and rectum common.

Mediolateral Advantages And Disadvantages

- More difficult to repair
- Faulty healing more common
- More painful in the puerperium
- Dyspareunia occasionally follows
- Blood loss greater
- Anatomical result not as satisfactory in 10% of cases (depending on the operator)
- Extension into anal sphincter less common.

Technique Of Episiotomy

The incision is made when the presenting part is distending the perineum with contraction (crowning) but before sufficient tissue bruising and devitalisation occurs and tearing of the perineum is imminent. If it is performed too early, blood loss from insidious oozing will be unnecessarily

excessive. The site of the incision may be influenced by the presence of previous scar or imminent rupture. If the perineum is already tearing it is better to continue the line of tear than to create another competing wound.

Prior to performing the episiotomy, adequate analgesia is obtained by local infiltration with 10ml of 1% plain xylocaine (lidocaine). Where regional or general anaesthesia has been instituted already, there is no need for the local anaesthetic.

The operation varies from an incision 2cm long to one extending the length of the perineum. A mediolateral episiotomy is performed with an 8in straight scissors with blunt points. The fingers of one hand are inserted behind the fouchette and a straight cut is made between them. It is important to make sure that the incision starts from the midpoint of the fouchette. Any oozing between contractions can be controlled by pressure with a perineal pad or artery forceps.

Repair Of Episiotomy

There are many ways of closing the episiotomy incision but haemostasis and anatomical reconstruction without excessive suturing are essential for success with any method. It is important to repair an episiotomy as soon as possible after delivery for the following reasons

1. Delay results in unnecessary blood loss.
2. Discomfort to the patient.
3. Increased risk of infection and
4. Oedema of the tissues making repair more difficult

Usually the local anaesthetic given before the incision is adequate for repair but further infiltration should not be withheld if required. However excessive volumes cause tissue oedema and devitalisation.

The suture material used for the repair is thought to influence the outcome of the repair. Typically chromic catgut 0 or 2/0 on a round-bodied needle is used. The use of absorbable synthetic material polyglycolic acid for the repair of episiotomy is associated with less perineal pain, analgesic use, dehiscence and re-suturing compared to catgut suture material.⁸

A pack with a tail is inserted in the upper vagina to facilitate identification of the apex of the incision and to prevent blood from the uterine cavity flooding the operating field. The suturing is started from the apex as a continuous suture to close the vaginal mucosa and submucosa. As the tissues are quite friable large bites should be taken but the epithelium of the vagina must not be inverted. Good alignment of the hymenal ring is important and the

suture is terminated at the ring. The muscles layers (pubococcygeus, superficial and deep transverse perineal muscles) are interposed with 3 or 4 interrupted sutures to eliminate any dead space and to ensure effective restoration of the perineal body. The bulbocavernosus muscles if avulsed and retracted are re-approximated. The perineal skin is closed with subcuticular catgut with no tension. Alternatively, vertical mattress suture may be used to include the superficial layer. The use of a continuous subcuticular technique for perineal skin closure is associated with less short-term pain than techniques employing interrupted sutures.

It is especially important to ensure that a backing of muscle tissue supports the skin of the fouchette. Omission of this precaution increases the risk of haematoma formation and breakdown in the short term and uncomfortable scar tissue and dyspareunia in the long term.

At the end of the operation the vaginal pack is removed and the vagina inspected for any lacerations and to ensure proper repair of the episiotomy.

After Care

Adequate pain relief with analgesics like Paracetamol or Brufen is helpful. Perineal toileting is required after maturation or defecation. Patients are advised not to sit in warm or hot water, as is the practice in the community. They could use tap water with a little salt or antiseptic solution added. Antibiotics are not routinely given to patients with episiotomy. Where pain is persistent or severe, it is essential to examine the perineum as this may signify a large vulval, paravaginal, or ischiorectal haematoma or abscess.

Complications Of Episiotomy

Maternal

A. Early complications include:

1. Haematoma. This can occur if haemostasis is not adequately achieved before closure. The haematoma may become infected. It is best to evacuate any large, growing or infected haematoma.
2. Dehiscence. Superficial dehiscence is usually due to infection or trauma such as early coitus. It can be managed by re-suturing once there is healthy granulation tissue or by allowing healing by secondary intention.
3. Infection: The reported incidence of infection in episiotomy is 0.05 to 3.0%. It may be simple infection limited to the skin and superficial fascia along the incision line. This usually resolves with broad-spectrum antibiotics but if infection does not respond within 24 to 48

hours of antibiotic therapy, then the episiotomy needs to be opened and debrided. Other more serious but uncommon infections include necrotising fasciitis and myonecrosis.

B. Late complications include:

1. Dyspareunia
2. Psychosexual problems and morbid fear of subsequent delivery
3. Rarely endometriosis at episiotomy site, squamous cell tumour of the vulva at the episiotomy site, and the scar becoming a metastatic site particularly for cancer of the cervix.

Foetal risks include

- a. Laceration of the lid
- b. Castration (in breech birth)
- c. Increased risk of vertical transmission of HIV infection
- d. As in the mother the risk of hypersensitivity to the local anaesthetic (xylocaine).

Discussion/Controversies

The risks and benefits of episiotomy have been analysed by various groups and there is no consensus as to its role though the benefits are thought to outweigh the risks.

The suggested maternal benefits of episiotomy are the following:

1. Reduction in the likelihood of third degree tears.^{2,3,9}
2. Preservation of the muscle relaxation of the pelvic floor and the perineum leading to improved sexual function and a reduced risk of faecal and urinary incontinence.^{10,11}
3. Being a straight, clean incision, an episiotomy is easier to repair and heals better than a laceration.
4. For the neonate, it is suggested that the prolonged second stage of labour could cause foetal asphyxia, cranial trauma, cerebral haemorrhage and mental retardation. During delivery episiotomy may also reduce the possibility of shoulder dystocia.

On the other hand hypothesised adverse effects of routine episiotomy include:

1. Extension of episiotomy by cutting the anal sphincter or rectum or by unavoidable extension of the incision.
2. Unsatisfactory anatomic results such as skin tags, asymmetry or excessive narrowing of the introitus, vaginal prolapse, recto-vaginal fistula and fistula in ano.¹²

3. Increased blood loss and haematoma
4. Pain and oedema in the episiotomy region
5. Infection and dehiscence.¹²
6. Sexual dysfunction.

Thus the restrictive use of episiotomy appears to have a number of benefits compared to routine use or no episiotomy at all. There is less posterior perineal trauma, less suturing and fewer complications, no difference for most pain measures and severe vaginal trauma, but there was an increased risk of anterior perineal trauma with restrictive episiotomy.

Third Degree Tear.

This is a tear that extends to involve the anal sphincter. When the rectal mucosa is involved it becomes a fourth degree tear. The external anal sphincter is commonly torn and the ends retract under the skin. Failure to recognize the extent of the tear and to effect a complete and accurate repair can lead to faecal incontinence or a recto-vaginal fistula. Episiotomy is thought to prevent the occurrence of third degree tears.^{2,3,9} Tearing is likely with a narrow sub-pubic arch, poorly flexed head, precipitous labour, big baby, shoulder dystocia and assisted vaginal delivery.

Third degree tears need to be repaired in theatre by an experienced surgeon under general or regional anaesthesia. The rectal mucosa and muscle are repaired in two layers with stitches about 0.5cm apart and then covering this layer with a layer of fascia. The sphincter ends are carefully identified and drawn up with tissue forceps to facilitate firm suturing with 2 or 3 interrupted or figure of eight sutures with the knots tied towards the gut lumen. The remainder of repair is same as for episiotomy. The anus should accommodate a finger after sphincter muscles are approximated and to ensure that the vaginal introitus accepts two fingers at the end of the repair. Good after care is important. Regular toileting and inspection are essential and undue strain on wound by faeces must be avoided. Stool softeners should be used for a week. Liquid paraffin should be avoided. No attempt should be made to restrict bowel action.

The role of prophylactic antibiotics has not been established but Metronidazole reduces the risk of autoinfection with bowel organisms especially *Bacteroides* species. It is important to advise elective episiotomy during subsequent births.

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SECTION 6
POSPARTUM CARE

Puerperium

EY Kwawukume/Rebecca Acquaaah-Arhin

Introduction

The word **puerperium** originates from the Latin words **puer** (child) and **parere** (to bear)⁽¹⁾

Definition: A period of about six (6) weeks, that begins from delivery of the placenta until the reproductive tract returns to the normal non-pregnant state. This period can be divided into:

1. Immediate Puerperium, which is twenty four (24) hours after delivery.
2. Early Puerperium, which is between the second to seventh day after delivery.
3. Late Puerperium, which is from the second week through to the sixth after delivery.⁽²⁾

Puerperium is the period during which the mother's altered anatomy, physiology and biochemistry return to normal. However, some organ systems resume their non-pregnant functions much later than others, especially in lactating mothers⁽²⁾. Puerperium is easily overlooked by obstetricians due to the sense of achievement felt after the delivery of a live infant by a healthy mother. There is however the need to be vigilant during this period because death occurring during this period also qualifies as maternal death. The World Health Organisation (WHO) defines maternal death as "the death of a woman whilst pregnant or within 42 days of termination of pregnancy, irrespective of the duration or site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental causes".⁽³⁾

After an uncomplicated spontaneous vaginal delivery, the patient may be discharged 24 hours post partum. Rising hospital cost and unavailability of beds have led to reduced duration in the lying in period, even patients delivered by Caesarean section are discharged after four to five days if there are no complications.

In all cases a patient should be allowed to ambulate and use the bathroom after delivery as soon as she can comfortably do so.

Management of the Normal Puerperium:

Immediate Labour Ward Management

First Hour

To prevent and control post-partum haemorrhage 0.5 mg of Ergometrine or in cases of hypertension or cardiac disease, 5 iu of syntocinon is given intramuscularly with the delivery of the anterior shoulder of the fetus in singleton pregnancy or delivery of the after coming head in breech. This is followed by active massage of the uterus through the abdominal wall after delivery of the placenta to stimulate uterine contraction and prevent postpartum haemorrhage. Immediate repair of genital tract traumas and episiotomies are done. Bonding of mother and baby is initiated. The mother is made comfortable and suckling of the baby is started.

Patients are kept on the labour ward until the blood pressure and pulse are stable and there is no abnormality, which requires close observation. Free choice of fluids is allowed, unless the patient is on intravenous fluids for a reason. After two hours mother and baby are transferred to the post-natal ward.

Next twenty-three hours: These events take place in the Post Partum Ward

- * Observation for haemorrhage
- * Analgesia
- * Rest
- * Bladder emptying
- * Maternal nutrition
- * Breastfeeding

Analgesia

Operative deliveries require adequate analgesia regularly. Medio-lateral episiotomies cause a 60% more pain post partum than median episiotomies. The pain is made worse by urination and improves within 2-3 days if it is not complicated by

haematoma. To reduce the pain and infection, patients are encouraged to wash the perineum with water regularly or sitz bath after urinating to avoid direct contact of acidic urine with the episiotomy.

After-pain from uterine contractions, particularly during breastfeeding requires reassurance or mild analgesia.

Persistent pain with breastfeeding should be investigated. It may be due to cracked nipples, which can be treated by applying expressed milk to the nipples and allowed to dry, and better positioning of the baby during breastfeeding.

Bladder Care

Many patients are unable to void after delivery because during labour the bladder wall and urethra become oedematous and bruised, or there is loss of reflex detrusor activity. They may not be able to relax due to perineal or abdominal pain or paralyzing effect of epidural anaesthesia. The patients should be encouraged to void every 6-8 hours after delivery. The bladder may become over distended or incompletely emptied causing stasis of urine and predisposition to urinary tract infection. In such cases, the patient should be catheterised under aseptic conditions. If residual volume is more than 100mls then an indwelling catheter should be left in-situ for 24-48 hours. But if symptoms suggest urinary tract infection then a clean catch mid stream specimen or catheter specimen should be taken for culture and sensitivity.

Blood pressure, pulse, temperature are recorded every four hours for twenty-four hours, then twice a day provided all readings are in the normal range. Each day the patient's breasts are checked; the uterine fundus is palpated to ensure that it is involuting appropriately. The perineum is checked for the colour of the lochia and the calves palpated to check for signs of DVT. The mother's psychological state should be observed, looking for signs of depression and instability. The onset of successful breastfeeding must be ensured.

Next six days:

- Detection and prevention of sepsis
- Establishment of breastfeeding
- Prevention of DVT
- Rest
- Psychological support
- Physiotherapy, especially after operative delivery.

Contraceptive advice.⁽⁴⁾

Discharge:

If general condition is satisfactory after spontaneous vaginal delivery, mothers are discharged after twenty-four hours.

Prior to discharge, mothers are counselled about breastfeeding and care of the perineum. They are encouraged to practice exclusive breastfeeding for 6 months. Babies receive the first dose of oral polio vaccine and the BCG vaccine before discharge. The mother is discharged on haematinics.

The first post-natal visit after discharge is at two weeks. At this visit the baby is weighed and fully examined. Enquiries about breastfeeding are made and appropriate counselling is given. A general examination of the mother is done with particular emphasis on genital tract. A vaginal examination is not necessarily done unless indicated by the patient's complaints. Counselling about contraception is emphasised.

The next visit is at six weeks. If general examination of the mother and baby shows no problem they are discharged from the clinic. The baby receives the second dose of oral polio and DPT vaccines. Subsequent immunisations are done at the child welfare clinics.

Patients with problems are seen more frequently at the postnatal clinic.

Where required, maternity leave forms should be filled before discharge.

Anatomical and Physiological Changes: Uterine Involution

This starts after the delivery of the placenta and marked reduction in uterine size and the effect of the "living ligature", that is, myometrial activity contributing to haemostasis by compressing the intramural perforating blood vessels shutting off the large blood flow to the placenta.

Intensity of uterine contraction increases significantly immediately after delivery. Then an hour or two after delivery, it decreases smoothly and progressively and then stabilises.

Release of proteolytic enzymes and migration of macrophages follow withdrawal of oestrogen and progesterone post delivery into the endometrium and myometrium. There is decrease in the amount of cytoplasm and the size of individual myometrial cells. However the increase in connective tissues and elastin in the myometrium, blood vessels and the increase in the total uterine cell numbers are permanent. Hence uterine size increases slightly after each pregnancy.

After completion of third stage, the uterus is found in the midline, below the umbilicus, approximately sixteen weeks of gestation, weighs 1000 grams and discoid when relaxed, and globular when contracted.

One week after delivery, its 500 grams and in the true pelvis region. At six weeks, it's 50-60 grams.⁽⁵⁾

Lochia

This is the discharge from the genital tract during the puerperium. It is red for the first three days when it consists of blood and the decidual debris. The colour then becomes pink when it contains mainly white blood cells, decidual debris and some red cells. By the end of the first week, it is yellowish-white in colour consisting mainly of serous fluid and white blood cells.

Ovarian Function

Breastfeeding induces a reduction in gonadotrophin releasing hormone, luteinizing hormone and follicle stimulating hormone, resulting in amenorrhoea. Reduced suckling precipitates the return of ovulation. During lactation, menses before six months are mostly unovulatory, and fertility remains low⁽⁵⁾.

The mean ovulation time in non-lactating women is 70-75 days and 190 days in breastfeeding mothers. A study of lactating women⁽⁶⁾ recorded a mean time of 36 months before the return of menses. The menses return in 7-9 weeks in non-lactating women.

On average β -HCG becomes negative 2 weeks after delivery. During the first trimester, serum β -HCG is much higher than at term. After a first trimester loss it takes about 5 weeks to become negative.⁽¹⁾

Cardiovascular and haematological changes

Following delivery there is a transient rise in both diastolic and systolic blood pressures for the first four days⁽⁸⁾. There is then a progressive fall in blood pressure with pre pregnancy pressures being reached for majority of women by six weeks after delivery.

The blood volume falls progressively after delivery. This brings about an initial rise in haemoglobin levels on the first day after delivery. This is followed by a sharp fall to a minimum level on the fourth and fifth days. It then rises to the level on the first day by the ninth day.⁽⁷⁾

Complications of the puerperium:

Postpartum fever puerperal pyrexia

Most post-partum chills are common but not necessarily indicative of infection. A slight temperature elevation is common after a difficult labour and delivery or instrumental or abdominal

delivery, but it usually falls to normal within 24 hours. A sustained rise in temperature suggests further examination. It could be due to severe breast engorgement, mastitis, endometritis, urinal tract infection, wound infection, intra-abdominal or retroperitoneal abscess or thrombophlebitis.

Puerperal pyrexia

Puerperal pyrexia is defined as rise in temperature to 38°C or more on two or more occasions 24 hours after delivery.⁽⁸⁾ Commonest causes are

1. Genital tract infections
2. Urinal Tract infections
3. Respiratory tract infections
4. Breast infections
5. Thrombophlebitis
6. Other non-obstetric causes.

Investigations

Full clinical investigation (including breast examination)

Mid stream urine

Endocervical and high vaginal swabs

Blood culture

Sputum culture (if possible)

Infections are mainly polymicrobial. Examples. Streptococci, staphylococcus, coliforms. bacteroides, mixed anaerobes, pseudomonas. chlamydia, mycoplasm and rarely clostridium. Anaerobic organisms account for 80% of all cases. Complications of puerperal infections include disseminated intra-vascular coagulation, septic shock, and subsequent infertility of death⁽⁸⁾.

With shorter labours, better labour ward techniques and administration of potent antibiotics, these infections can be minimised. Mastitis mostly occurs in the second to the third week post-partum. Severe breast engorgement does not require antibiotics as in the case of mastitis, but both conditions require firm support of the breast with a good brassieres, warm bathing of breast, adequate analgesia and bromocriptine tablets if the mother is not breastfeeding.

Secondary Post-partum Haemorrhage

Secondary postpartum haemorrhage is bleeding from the genital tract that occurs after the first 24 hours following delivery up to 6 weeks after delivery.

There is usually a sudden but transient increase in uterine bleeding between 7 and 14 days

postpartum. This is due to the slough of the eschar over the placental site. Though this bleeding can be profuse, it is self-limiting and subsides within 1 or 2 hours. Persistence beyond this period requires further evaluation.

Puerperal Sepsis

Puerperal sepsis is defined as infection of the genital tract after delivery. Puerperal morbidity includes all cases of significant pyrexia in the puerperium.

Aetiology

Puerperal sepsis occurs as a result of ascending infection. Organisms from the vagina gain access to the endometrium at delivery. These organisms then colonize the endometrium and spread to involve the myometrium, tubes and ovaries. The infection can also spread through the blood stream to give rise to a septicaemia.

Prolonged labour, prolonged rupture of membranes, numerous vaginal examinations during labour, prenatal bacterial colonization of the genital tract and retained products of conception are all factors that predispose to the development of puerperal sepsis⁽⁹⁾.

Presentation

The patient is usually very ill with complaints of fever, lower abdominal pain and offensive lochia. In extreme cases the patient may have clouded sensorium and signs of meningeal irritation.

On examination, the patient is febrile and in severe cases may be pale and jaundiced. The lower abdomen is tender and the uterus may be larger than expected. There may be an offensive vaginal discharge. If there are infected retained products the cervical os will be open. In cases of severe pelvic infection, there is clinical evidence of fluid in the pelvis.

It is necessary to do a thorough examination of patients presenting with postpartum fever because it might not be a case of puerperal sepsis but rather one of malaria or primary infection in any of the systems.

Management

The following investigations must be performed on all patients presenting with postpartum fever: Full blood count, Blood film for malaria parasites, Blood culture, and Urine culture.

Chest X-ray, CSF examination and ultrasound examination are done as directed by clinical findings.

Patients must be started on broad-spectrum

intravenous antibiotics. A combination of Metronidazole and Cefuroxime is usually effective. In situations where there is difficulty in obtaining Cefuroxime, a combination of Ampicillin, Gentamicin and Metronidazole yields fair results. The antibiotics are modified when culture results are obtained unless there is already very good response to the drug regimen.

The absence of response to the appropriate antibiotics after 48 hours is suggestive of pelvic fluid collection or immunosuppression. A laparotomy might be required in such a case.

When the fever subsides and the temperature stays normal for 48 hours the antibiotics can be changed to their oral equivalents.

Where ultrasound suggests the presence of retained products, evacuation of the uterus should be carried after 24 hours of intravenous antibiotic therapy.

Prevention

To prevent puerperal sepsis, it is necessary to prevent prolonged labour. This can be done by appropriate use of the WHO partograph during labour. Patients with prolonged rupture of membranes must be covered with antibiotics in the period around their delivery. During labour vaginal examinations must be done at four hourly intervals. Doing them more frequently at the latter stage of the labour must be to determine the need for surgical intervention and such decisions must be taken quickly.

The use of prophylactic antibiotics at elective and emergency caesarean sections is also helpful at preventing puerperal sepsis⁽⁹⁾.

Obstetric Neuropathy

Prolonged labour tends to result in difficulty in walking after delivery. In extreme cases there might be paralysis of the lower limbs. Foot-drop is the commonest presentation in such cases.

Obstetric neuropathy is due to intra-pelvic pressure on the sacral and lumbar nerve roots by the fetal head⁽¹⁰⁾. The condition is usually self-limiting. Physiotherapy and the use of NSAIDs for pain are the way to manage the condition. It is however important to perform appropriate X-rays to rule out orthopaedic problems.

Postnatal care after stillbirth

In the developing world the emphasis in obstetric care is still on the reduction of maternal mortality. As a result of this mothers who lose their babies are rarely given the special comprehensive care that they need⁽¹¹⁾.

After a stillbirth, labour ward staff must make it a point to show the baby to both parents after the baby has been cleaned up. Before discharge from the post-natal ward, a doctor must meet with both parents in order to answer their questions and offer appropriate explanations.

The first visit at two weeks should be arranged to take place away from the regular post-natal clinic where mothers are all gorgeously dressed with their newborn babies. This visit could be on the antenatal ward or at a gynaecology clinic. The same doctor who met with them on the ward before discharge should see the parents. At this visit the mother must be examined and necessary investigations as determined by the circumstances surrounding the foetal death could be carried out. Any further questions of the parents must be answered. A second visit at 6 weeks is needed to ensure that the changes of the puerperium have taken place. The couple must be counselled about contraception and advised to delay pregnancy for about a year so that there is enough time for grieving. Arrangements must be made for the patient to see a particular doctor in the unit during the next pregnancy.

Deep Vein Thrombosis

Early mobilisation is encouraged even after operative delivery to reduce the incidence of venous and pulmonary embolism and thrombophlebitis.

Deep breathing and leg exercises help to prevent venous thrombosis and chest complications. Febrile patients who do not respond to prolonged potent antibiotics but appear non-toxic and have poorly localised discomfort, severe lower abdominal or flank pain should be investigated for thrombophlebitis. A Doppler scan or computed tomography may show obstructed veins. They normally respond to intravenous heparin. Subcutaneous heparin prophylactic in high-risk patients is important. Fatal pulmonary embolism is the second major cause of unexplained maternal death. Two thirds occur after delivery and one third during antenatal period. Predisposing factors are infections, venous stasis and altered coagulation during pregnancy and the puerperium. Other factors are operative delivery, trauma to leg veins from stirrup pressure in lithotomy position, older patients, obese, anaemic and cardiac patients.

Lactation

Hormones influence lactation. During pregnancy, Prolactin, Chorionic Somatotropin and Human Chorionic Gonadotropin cause increase in ductular sprouting and branching. Oestradiol and

Progesterone stimulate both ductal and alveolar growth. Serum Growth Factor and Insulin cause ductal end cell proliferation. Prolactin and Corticosteroids cause differentiation of ductal end cells into alveoli. Human Placental Lactogen and Prolactin stimulate the secretion of colostrum. Delivery of placenta and withdrawal of oestrogen initiates lactation, which is sustained by the suckling reflex, which also causes the increase of prolactin for the synthesis of milk and oxytocin for milk release. That is why a delay in suckling may affect the long-term outcome of breast-feeding.

The pre-milk (colostrum) is alkaline, yellowish, and produced in small quantities in the last few months of pregnancy and for two to three days after delivery. It has a high specific gravity, high protein, fat-soluble vitamins, sodium, chloride, beta-carotene and minerals, but fewer calories. It also contains antibodies, which play a part in the immune mechanism of the baby.⁽¹⁾

It is easier for the immature intestines to absorb and facilitate the colonisation of the newborn gut by normal intestinal flora, and protects it from pathological bacterial invasion.

Transitional milk follows the colostrum. It increases the milk volume and engorges the breast. It has less immunoglobulin but more lactose, fats and calories. The mature milk follows this, one to two weeks after delivery.

The milk that comes at the end of a feed is more concentrated in fats and has a high calorie than at the beginning.

The concentration of the Major Milk Constituents in Colostrum and Mature Milk⁵.

Constituent (per dl)	Colostrum	Mature Milk
Total energy (kcal)	54	70
Milk sugar, lactose (g)	5.7	7.1
Fats (g)	2.9	4.5
Proteins (g)	2.3	0.86
Nonprotein nitrogen (g)	-	0.32
Minerals (ash) (mg)	30.8	20.2
Cells (macrophages, neutrophils, lymphocytes)	7.8×10^6	$1-2 \times 10^9$

Cow Milk Vs Human Milk

They differ in quantity and in quality. The energy in human milk is mainly from fats and carbohydrates (99.0%), which are easily digested, compared to proteins in cow's milk, which is less easily digested and may also cause metabolic and amino acid

imbalance.

25-34% of nitrogen in human milk is in nonprotein form, which gives all the essential amino acids, including taurine, for the development of the brain. 75% of protein in human milk is easily digestible whey whilst in cow's milk it is casein (80%). Beta lactoglobulin, which is the main whey protein in cow's milk, is a potent allergen. The high casein leads to formation of insoluble curds in the infant's gut, which binds to calcium and inhibit its absorption.

The high content of polyunsaturated fatty acids and cholesterol in human milk also helps in brain development.

Although the iron content in cow's milk is far higher than in human milk, the rate of accumulation during the first six months is the same. The high fat content in human milk gives a high sense of satiety and breastfeeding alone can meet the nutritional needs of the infant up to 15 months.

Human milk contains epidermal growth factor throughout lactation whereas only the colostrum of cow's milk contains this factor. Human milk also contains some vitamins, trace elements, enzymes, sleep-inducing peptide and melatonin, which are absent in cow's milk.

Maternal nutrition has little effect on protein, fat or carbohydrate composition of the milk but maternal malnutrition decreases the quantity, water-soluble vitamins, thiamine and vitamin B12. Good and adequate nutrition and rest increases the quantity of milk produced.

Diabetic mother's have reduced insulin requirements when breastfeeding because glucose taken by the breast is considerable. By six weeks post partum fasting blood sugar of breast-feeding insulin dependent diabetic mothers are lower than those who do not breast-feed or have stopped breast-feeding or those using supplementary feeds.

Pain, fear, discomfort, anxiety, lack of privacy and other psychological influences may inhibit the milk ejection reflex at the hypothalamic level. Pre-term or ill infants should be fed with expressed breast milk.

Most drug dose in maternal milk is 1-2 % of total maternal dose. Drugs that are easily metabolised may not achieve a significant concentration in the baby. Drugs with low plasma protein binding and

increased solubility may concentrate in the baby.

Composition of Mature Human Milk and Cow's Milk⁵

Constituent (per dl)	Mature Milk (30 days)	Cow's Milk
Energy(kcal)	70	69
Total solids(g)	12.0	12.7
Lactose	7.3	4.8
Total nitrogen(mg)	171	550
Protein nitrogen(mg)	129	512
Nonprotein nitrogen(mg)	42	32
Total protein(g)	0.9	3.3
Casein(g)	0.4	2.8
Whey protein(g)	0.5	0.19
Lactalbumin(mg)	161	0.6
Lactoferrin(mg)	167	
IgA(mg)	142	
Total fat(g)	4.2	3.7
Unsaturated long-Chain Fatty acids(g)	2.9	1.0
Calcium(mg)	28	125
Phosphorus(mg)	15	96
Iron(ng)	40	100

Although the first normal menses and ovulation usually occurs between 4th 6th weeks post-partum, the patient may ovulate as early as second third day post-partum. Lactation may delay or suppress menstruation but does not provide sufficient contraception if it is not exclusive and on demand. Exclusive breast-feeding provides 98% contraceptive protection for up to six months. But if menses return within this period, the risk of pregnancy increases and therefore artificial contraception should be used.

Before discharge from hospital, patient is examined and advised on normal activities, contraception and personal hygiene. There is the need for adequate support at home and advice on when to report that is, onset of fever, bright vaginal bleeding, and severe lower abdominal pain, in order to rule out secondary post-partum haemorrhage.

For breast-feeding mothers, the mini-pill or Depo-Provera is the best amongst the hormonal contraception. This is because it does not contain

oestrogen which causes reduced milk contents of vitamins, proteins, fats and also the quantity. HIV virus has been detected in breast milk and therefore HIV positive mothers should be advised not to breast-feed.

Puerperal psychological problems

The experience of labour and childbirth may be followed by psychological reactions. Most common among them is "Maternity blues" or postpartum blues which is relatively mild and transient. The incidence ⁽¹¹⁾ is about 50-70%.⁽⁵⁾ The second common psychological problem is postpartum depression, which is more prolonged and thirdly frank puerperal psychosis.

Maternity Blues

The symptoms appear on any day within the first week after delivery and usually resolve by the tenth day.

The patient may report with weeping, restlessness, headaches, confusion, irritability, depression, mood changes, insomnia and negative feelings towards their babies.

To deal with it, the family members and all those caring for the patient need to be sympathetic and understanding because there is no therapy apart from explanation and support, and also it is transient and of short duration.

Post Partum Depression

Post partum depression has an incidence of 10-15%. Predisposing factors include patient under 20 years of age, unmarried, comes from a large family, or has low self-esteem. Other predisposing factors are patients with emotional instability, poor relationship with husband, boyfriend, her family, social and financial problems, dissatisfied with educational achievement, were separated from one or both parents in childhood with lack of support and attention.

In addition to the symptoms of maternity blues, they show inability to love their family and apathy towards their babies.

They do well on tricyclic antidepressants. In addition, the husband and the family should be educated to understand the woman's illness and to help with the house chores by caring for the other children if any. This would help to prevent her sense of entrapment and isolation. There is 50-100% recurrence in subsequent pregnancies.

Puerperal Psychosis

Puerperal psychosis has an incidence of 0.14-0.26%. Most of these patients have manic-

depressive reactions. Some are schizophrenic. They are confused and disoriented. They may also have suicidal thoughts and tendencies or are frankly delusional but the outcome is more favourable than non-puerperal psychosis. It usually lasts for about 2 to 3 months.

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Problems Of The Newborn

Jennifer Welbeck

Introduction

The neonatal period is defined as the first 28 days (4 weeks) of life.

When the newborn is delivered he has to make a transition from the fetal life to the neonatal life. Changes take place in the body and organ functions as the fetus adapts to extrauterine life and begins to function independently.

At birth, there is a rise in the systemic vascular resistance as a result of the cessation of blood flow

through the placenta.

With the first few breaths, pulmonary vascular resistance falls, the foramen ovale closes and the ductus arteriosus begins to constrict. These processes allow all deoxygenated blood returning to the right ventricle to go on to the lung and become oxygenated. The oxygenated blood returns to the left ventricle and then is pumped throughout the body.

Risk Factors for high Risk Deliveries

Table 1

Maternal Condition Conditions	Fetal Condition	Labour/Delivery
<ul style="list-style-type: none"> * Toxaemia * Acute febrile illness e.g. Malaria, UTI, Chorioamnionitis * Chronic Medical Conditions <ul style="list-style-type: none"> - Hypertension - Diabetes Mellitus - Renal Disease - Cardiac Disease - Pulmonary Disease 	<ul style="list-style-type: none"> * Prematurity * Postmaturity * Meconium stained amniotic fluid * Intrauterine growth retardation * Multiple gestation * Blood Group iso- immunisation ± Hydrops * Prenatally diagnosed congenital abnormality 	<ul style="list-style-type: none"> * Abnormal pattern/length of labour * Bleeding * Cord Prolapse/Knotting * Abnormal presentations

A. NEONATAL RESUSCITATION

Shortly after delivery, the well newborn baby gasps or cries, establishes normal breathing and becomes pink. The baby can then be handed over to the mother if she is also well, to have skin to skin contact covering both of them together with a warm cloth and to initiate breastfeeding.

Neonates who do not establish normal respiration quickly will need resuscitation. Most babies do not require resuscitation. The need for resuscitation can usually be anticipated from the history when high-risk deliveries are identified.

A person skilled in resuscitation of the newborn should therefore be present at all high-risk deliveries and with skilled assistance.

The obstetrician should therefore notify the paediatrician in advance of the impending birth of the infant. This allows the paediatrician to prepare for specific problems that may be encountered.

Goals of Resuscitation

Resuscitative efforts are directed at the following:

1. Expansion of the lungs by clearing the upper

airway and ensuring a patent route to the trachea.

2. Increasing the arterial PO₂ by providing adequate alveolar ventilation, with added oxygen if necessary.
3. Supporting adequate cardiac output.
4. Reducing heat losses to minimise oxygen consumption.

Equipment Required

1. Radiant warmer with procedure table/drying towels.
2. Suction apparatus.
3. Oxygen source with adjustable flow meter.
4. Ambu Bag capable of delivering 100% Oxygen
5. Face masks of differing sizes.
6. Stethoscope.
7. Laryngoscope with No. 0 & No. 1 blades.
8. Endotracheal tubes.
9. Drugs.
10. Syringes, Needles, Umbilical Catheters.

Once the baby is born, a continual process of evaluation and resuscitation begins

Place the baby on the warming table, dry the infant and gently suction the mouth first, followed by the nose to remove any fluid or blood. Vigorous suctioning of the back of the throat may provoke bradycardia from vagal stimulation and should be avoided.

Assess the heart rate, respiration, tone, colour and response to stimulation. See Apgar score Table 2.

On the basis of this immediate assessment (Apgar Score at 1min.) continue further resuscitation as required. Assess the baby again after 5 minutes (Apgar score at 5 min).

Table 2:

The Apgar Score

		SCORE		
		0	1	2
A	Appearance -colour	Blue/White	Blue	Pink all over
P	Pulse Heart Rate	Absent	extreme	
G	Grimace/ Reflex irritability	No response	<100/min	
A	Activity - Tone	Flaccid	grimace	>100/min
R	Respiratory effort	Absent	Some Flexion Irregular	cough/sneeze well flexed Normal

The one-minute Apgar score is an index of intrapartum asphyxia ⁽¹⁾ while the five-minute Apgar score correlates best with the eventual neurologic outcome.

The Principles of Newborn Resuscitation is the same as for adults

- A. Airway Management
- B. Breathing
- C. Circulation (adequate cardiac output to maintain cerebral oxygenation)
- D. Drug

Ventilation:

In most cases, once airway potency is ensured, bag and mask ventilation is adequate while assessing for chest wall movement. The rate should be 30 - 40 breaths/minute using the minimum pressure that will move the chest and produce audible breath sounds on auscultation.

If despite good air entry, the heart rate fails to increase and colour remains poor, then intubation is required. A skilled person must do intubation. The heart rate should increase to >100 beats/min and colour should rapidly improve with adequate ventilation.

Circulation:

If after intubation and several breaths of 100% oxygen, the heart rate remains < 60-beats/min cardiac massage should be instituted ⁽²⁾ at a rate of 120 beats/minute. Determine the effectiveness of massage by palpating the umbilical cord, femoral or brachial pulse.

If by 3 - 5 minutes after delivery, a heart rate of more than 100 beats/minute has not been achieved despite adequate ventilation with 100% oxygen, then drug therapy is given (Table 3). The most accessible route for drug administration in the delivery room is by cannulation of the umbilical vein.

Table 3:
Drugs used in Advanced Resuscitation

Drug	Dose	Indication
Adrenaline iv or Down ETT	1ml/kg of	Bradycardia
Dextrose 10%	1:10,000up 1ml 2ml/kg	To correct hypoglycaemia
NaHCO ₃ (8.4% Solution diluted to 4.2%)	1-2mmol/kg	To correct acidosis
Naloxone	(.0mg/kg iv 1ml preterm 2ml term)	Antidote for narcotic depression
Plasma, Normal Saline Ringer's Lactate or whole blood	20ml/kg	shock
Atropine	.01 - .03mg/kg iv or ETT	Bradycardia

Reasons for failure of resuscitation are numerous - varying from poor technique to congenital abnormalities. See Table 4.

Failure of Resuscitation - Table 4

- * Inadequate mask ventilation due to poor technique
- * Wrong placement of ETT
- * ETT blocked with secretions
- * Severe birth asphyxia or trauma
- * Lung disorders
 - pneumothorax
 - diaphragmatic hernia
 - lung hypoplasia
 - pleural effusion
- * Shock from blood loss
- * Upper airway obstruction eg. choanal atresia.

Comment:

The decision to stop resuscitation is always a difficult one and must be made by a senior paediatrician. After **effective** resuscitation for 20 - 30 minutes if there is no respiratory effort or effective cardiac output, continuing to resuscitate is unlikely to be productive. This decision is best made in consultation with parents if they are around.

In our environment, parents are most of the time unavailable to help make this crucial decision. Intubation of patients is usually a temporary measure to improve the condition of the infant. Since facilities for ventilator support are uncommon in our subregion (a great limitation), it is even more difficult to continue with advanced resuscitation for longer than 30 minutes. As soon as the infant shows signs of spontaneous respirations he is extubated and supported by oxygen via facemask with minimal handling. When ventilation help is required due to apnoea episodes, intermittent ambu bagging with oxygen is offered.

B. Birth Asphyxia

Birth asphyxia continues to be a major problem in the developing countries and an important cause of brain damage in children. Therefore preventing birth asphyxia is one of the main aims of modern obstetric care.

Definition

Asphyxia, meaning suffocation, is the acute deprivation of oxygen accompanied by reduced oxygen delivery to the tissues.

The biochemical definition of asphyxia is **acidosis + hypoxia + hypercapnia = asphyxia**. An Apgar score at 5 minutes of < 6 is referred to as asphyxia. The high-risk infant. (See page 1) is at risk of developing asphyxia. Any thing that impairs maternal oxygenation, decreases blood flow to the placenta, impairs gaseous exchange across the placenta, increase fetal oxygen requirement or impairs gaseous exchange at the fetal tissue will increase the incidence of birth asphyxia.

The target organs are the brain, the heart, lung, kidney, liver, bowel and bone marrow, (see table 5). In the initial stages of asphyxia, blood is shunted to the vital organs - the brain and heart, from the other systems. As asphyxia progresses, the heart and eventually the brain get compromised resulting in hypoxic ischaemic brain injury, which can be mild, moderate or severe.

Table 5:

Effects of Asphyxia	
System	Effect
Cardiac	Transient myocardia ischaemia
Renal	Acute Tubular Necrosis (ATN)/SIADH*
GI	Bowel ischaemia, NEC (Necrotising enterocolitis)
Haematological	DIC
Liver	Shock Liver
Lung	Pulmonary Haemorrhage I

* Syndrome of inappropriate ADH secretion

Management

This starts in the delivery room with neonatal resuscitation followed by stabilising the sick infant who may need:

- * respiratory support,
- * treatment for seizures,
- * prevention or reduction of cerebral oedema with fluid restriction (SIADH),
- * treatment of hypertension with colloid infusion,
- * renal support with dopamine, and
- * monitoring and treatment of hypoglycaemia or hyponatremia.

Prognosis

When hypoxic ischaemic encephalopathy (HIE) is mild, complete recovery can be expected. The prognosis is good if condition is moderate but more variable if severe with varying degrees of cerebral palsy. Several weeks after the insult, cystic lesions or ventricular dilatation from cerebral atrophy may be identified on cranial ultrasound, CT or MR scans.

C. The Preterm Infant

Introduction

The appearance, clinical problems, chances of survival and long-term prognosis of a newborn depends on the infant's gestational age.

A preterm newborn is any neonate whose calculated gestational age from the first day of the

last menstrual period is less than 37 completed weeks.⁽³⁾

Prematurity continues to be a major problem and a major cause of perinatal mortality in our subregion.

Aetiology

Most premature deliveries occur for unknown reasons. However it may occur in association with the following conditions.

1. Poverty
2. Black race
3. Malnutrition
4. Extremes of Maternal Age
5. Multiple gestation
6. Polyhydramnios
7. Acute or Chronic Maternal Illness e.g. preeclampsia
8. Previous preterm pregnancies
9. Incompetent cervix
10. Uterine malformation e.g. double uterus
11. Uterine trauma
12. Vaginal bleeding - placenta praevia, abruptio placenta
13. Closely spaced pregnancies.
14. Premature, rupture of membranes (PROM) + amnionitis.
15. Fetal conditions e.g. erythroblastosis.
16. Incorrect estimate of gestational age.

Problems Of Prematurity

These are problems related to difficulty in extrauterine adaptation due to immaturity of organ systems.

Respiratory Distress Syndrome

In respiratory distress syndrome (RDS), there is deficiency of surfactant, a mixture of lipoproteins (lecithin, sphingomyelin, and phosphatidylcholin) secreted by the alveolar epithelium, which lowers surface tension. There is alveolar collapse and inadequate gas exchange. A proteinaceous exudate develops which forms a hyaline membrane. The more preterm the baby the higher the incidence of RDS. Glucocorticoids given antenatally to the mother at least 48hrs before birth stimulates fetal surfactant production.

Within 4 hours of delivery babies with RDS develop clinical signs of

- * Tachypnoea
- * Grunting
- * Chest indrawing
- * Cyanosis

With a characteristic chest Xray - a reticulogranular appearance with air bronchograms.

Treatment with raised ambient oxygen is required which may be supplemented with continuous positive airway pressure (delivered by face mask or nasal cannula) or artificial ventilation via an endotracheal tube.

Exogenous surfactant therapy, a recent development does reduce the morbidity and mortality of preterm infants with RDS.

COMMENT:

Absence of ventilatory support makes the prognosis for preterm infants < 30 weeks poor. Respiratory support is limited to oxygen by face mask/nasal canula and intermittent ambu bagging. The complications of ventilatory support therefore are also not commonly seen.

Apnoea + Bradycardia

Episodes of apnea and bradycardia are common in very low birth weight infants less than 32 weeks gestational age. An underlying cause for the episode (hypoxia, infection, anaemia, metabolic disorder, convulsion, or aspiration) needs to be ruled out. Treatment with a respiratory stimulant such as theophylline in mild cases often helps.

Patent Ductus Arteriosus

The ductus arteriosus remains patent in many preterm infants, especially in the presence of RDS when there is shunting of blood across the ductus from the left to the right. It may be symptomless or it may cause apnea and bradycardia, increased oxygen requirement and heart failure. More accurate assessment can be obtained on echocardiography. Treatment if necessary is with fluid restriction and indomethacin, a prostaglandin synthetase inhibitor (medical treatment) or by surgical ligation.

COMMENT:

In view of the other effects of indomethacin (reduced renal function, bleeding tendencies) and the poor monitoring and support facilities indomethacin is not commonly used for medical closure

Temperature Control

Newborn infants have a larger body surface area relative to their weight than older children. Also the skin of the preterm infant is thin and poorly

keratinized. This is an important source of water and heat loss in the first week of life and adds to their difficulty in maintaining body temperature.

Incubators or overhead radiant heaters help to maintain the temperature of these babies. In the absence of incubators, babies can be wrapped up in blankets but will need to be monitored more closely.

Fluid Balance and Nutrition

The preterm's fluid requirements vary depending on the gestational age, clinical condition and whether nursed in an incubator or not.

On the first day of life, 60ml/kg of 10% Dextrose is usually required, increasing by 30ml/kg/day to 150 - 180ml/kg/day by the 5th day. This is adjusted according to the infant's clinical condition, plasma electrolytes and creatinine, urine output and weight change. A sodium containing solution is used from the second day. Because of rapid growth they have a high nutritional requirement.

Infants of 35 - 36 weeks gestational age are mature enough to suck and swallow breast milk. Less mature babies need to be fed via the nasogastric tube. As soon as possible breast milk, the milk of choice must be introduced (may be in the form of expressed breast milk -EBM). In the very sick infant total parenteral nutrition (TPN) would be the choice but since this is not available, IV fluids continue to be used with the aim of introducing EBM as soon as it is feasible.

Preterms may require supplementation with vitamins and iron when feeding is well established.

Infection

Preterm babies are at increased risk of infection either at or shortly after birth from organisms acquired from the maternal birth canal. E Coli and other Gram-negative organisms continue to be a big concern in developing countries, thus the use of gentamicin and ampicillin - as 1st line of antibiotics.

Intracranial Lesions

Periventricular haemorrhages occur in 25% of preterm babies and is readily recognised by cranial ultrasound.

Necrotising Enterocolitis (NEC)

NEC is a serious illness affecting preterm infants in the first few weeks of life (asphyxia, hypothermia are also known predisposing factors). It is thought to be due to a combination of ischaemia of bowel wall and infection from organisms colonising the gut. The infant stops tolerating milk feeds; with milk aspirated from stomach or vomiting. The abdomen

becomes distended and stool contains fresh blood. Baby may rapidly develop shock and require ventilatory support. Xray of abdomen shows distended bowel loops with thickened walls.

D. RESPIRATORY DISTRESS IN TERM INFANTS

Newborn infants with respiratory distress develop the following signs.

- Tachypnea
- Laboured breathing with chest wall recessions and nasal flaring
- Expiratory grunting
- Cyanosis

Causes of Respiratory Distress - Table 6

Pulmonary

- | | |
|----------------|---|
| Common | - Transient Tachypnoea of Newborn (TTN) |
| | - Meconium aspiration |
| | - Pneumonia |
| | - Pneumothorax |
| Less Common | - Diaphragmatic Hernia |
| | - Tracheo-Oesophageal |
| | - Fistula Pulmonary |
| | - Hypoplastic Airway |
| | - Obstruction |
| | - Choanal atresia |
| | - Pulmonary |
| | - haemorrhage |
| Non Pulmonary- | - Congenital Heart |
| | - Disease |
| | - Intracranial birth |
| | - trauma/asphyxia |
| | - Severe anaemia |
| | - Metabolic acidosis |

Affected neonates must be admitted to the neonatal unit for monitoring of heart and respiratory rates, oxygenation and circulation. A chest Xray is indicated to help find cause for treatment, which involves oxygen, ventilatory and circulatory support or surgery.

Transient Tachypnea of the Newborn

This is caused by delay of resorption of lung liquid. It is more common after birth by caesarean section. Chest Xray shows fluid in the horizontal fissure. Additional oxygen may be required while condition usually settles within 24 - 48 hours of life.

Meconium Aspiration

Meconium is passed before birth in 8 - 20% of babies in the presence of fetal distress. At birth, babies inhale thick meconium into their airways. Meconium is a lung irritant and results in both mechanical obstruction and a chemical pneumonitis. It predisposes to infection. The lungs are over inflated, accompanied by patches of collapse and consolidation. There is a high incidence of air leaks leading to pneumothorax and pneumomediastinum. Patients require oxygen therapy as well as artificial ventilation and antibiotics.

COMMENT:

Since we are limited in providing artificial ventilation the need to prevent this condition is even more apparent. Obstetricians must endeavour to monitor labour closely and intervene early as much as possible. Thick meconium at delivery should be aspirated from the airway if possible before the delivery of the chest to prevent aspiration in the lungs.

Pneumonia

Prolonged rupture of membranes (PROM), chorioamnionitis and low birth weight predispose to pneumonia. Broad-spectrum antibiotics are indicated.

Pneumothorax

This may occur spontaneously or more commonly as a complication of excessive bagging during resuscitation. This is readily demonstrated by transillumination applied to chest wall. If uncertainty insertion of a chest drain is indicated.

E. NEONATAL INFECTION

Infants are exposed to a wide range of pathogens from the birth canal. The gram-negative organisms continue to be the culprit organisms (compared to Group B streptococci in the western world). The risk of infection is increased if there has been prolonged rupture of membranes (PROM).

Presentation is usually non-specific See Table 7.

**Table 7:
Clinical Features of Neonatal Sepsis**

Temperature Instability	Apnoea + Bradycardia
Poor Feeding	Respiratory distress
Vomiting	Abdominal distension
Jaundice	Hypo/hyperglycaemia
Irritability	Shock
Seizures	Lethargy

If systemic infection is suspected investigation and treatment must be started promptly. A septic screen is performed and appropriate antibiotics started. Antibiotics of choice in our region are Ampicillin (to cover Gram positive organisms including *Listeria monocytogenes*) and an aminoglycoside (for gram negatives). If cultures come back negative, and infant has recovered clinically, antibiotics can be stopped after 48 hours. In Neonatal meningitis, additional signs of tense or bulging fontanelle and head retraction may be present. Ceftriaxone and Ampicillin are a better choice of antibiotics for meningitis. Complications include cerebral abscess, ventriculitis and hydrocephalus.

F. Neonatal Seizures

Many babies startle or are slightly jittery. This can be difficult to differentiate from seizures, which may be tonic, clonic or more subtle. About 90% of cases have a detectable cause.

**Table 8:
Causes of Neonatal Seizures**

* Hypoxic ischaemic encephalopathy/birt trauma
* Septicaemia/meningitis
* Metabolic
Hypoglycaemia
Hypo/hypernatraemia
Hypocalcaemia
Hypomagnesaemia
Inborn errors of metabolism
Pyridoxine dependency
* Intracranial haemorrhage
* Cerebral malformation
* Drug withdrawal e.g. maternal opiates
* Congenital infections e.g. TORCH'S

Whenever seizures are suspected a low blood sugar, calcium and sodium need to be excluded. An intracranial ultrasound should be performed to

identify haemorrhage or cerebral malformations. A septic screen including a lumbar puncture will be required to exclude meningitis. Treatment is directed at the cause wherever possible. Ongoing or repeated seizures are treated with an anticonvulsant. The prognosis depends on the underlying cause.

G. Neonatal Jaundice

Over 60% of all newborn infants become visibly jaundiced. This is because: -

- a. The high haemoglobin concentration falls rapidly in the first few days after birth from haemolysis.
- b. The red cell life span of newborn infants is markedly shorter (70 days) than that of adults (120 days).
- c. Hepatic bilirubin metabolism is less efficient in the first few days of life.

Jaundice is important as

- * It may be the sign of another disease.
- * Unconjugated bilirubin can be deposited in the brain especially the basal ganglia causing kernicterus.

A lower bilirubin level than in term infants may damage preterm babies. Also infants who experience severe hypoxia, hypothermia or any serious illness may be more susceptible to damage from severe jaundice. In addition, the infant's age from birth is important, as higher bilirubin levels are tolerated with increasing age.

Treatment is by phototherapy or exchange transfusion.

H. Birth Trauma

Infants may be injured at birth if they are malpositioned or too large for the pelvic outlet. Injuries may also occur during manual manoeuvres or from instrumental deliveries.

Soft Tissue Injuries include:

Caput succedaneum: - bruising and oedema of the presenting part.

Cephalhaematoma: Subperiosteal bleed confined by suture lines, usually in the parietal region.

Subaponeurotic haematoma: Bleeding below the scalp may be accompanied by serious blood loss.

Babies become clinically jaundiced when the bilirubin level reaches 83-120umole.

This can be observed most easily by observing the

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Bleeding below the scalp may be accompanied by serious blood loss.

Babies become clinically jaundiced when the bilirubin level reaches 80-120umole.

This can be observed most easily by blanching the

**Table 9:
Causes of Neonatal Jaundice**

Jaundice Starting < 24hrs of Age	-	Haemolytic disorders
	-	G6PD deficiency
	-	Spherocytosis
	-	Congenital Infection
Jaundice at 24hrs to 2 weeks of age	-	Physiologic jaundice
	-	Infection
	-	Haemolysis
	-	Bruising
	-	Polycythaemia
Jaundice >2 weeks of age	-	Unconjugated
	-	Physiologic Jaundice
	-	Infection
	-	Hypothyroidism
	-	Haemolysis
	-	Conjugated
	-	Bile duct obstruction
	-	Neonatal hepatitis

skin with the finger. Management varies according to the infant's age at onset, bilirubin level and rate of increase, the infant's gestational age and clinical condition.

Nerve Palsies

May result from traction of the cervical nerve roots at breech deliveries or with shoulder dystocia.

Erb's Palsy → Upper nerve root injury

Klumpke's Palsy → Lower nerve

Facial Nerve Palsy → Uncommon Mild cases resolve completely over a few weeks. More severe cases may have residual effects.

Fractures

Clavicle -Usually from shoulder dystocia

Humerus/Femur- Usually mid-shaft occurring at difficult breech deliveries

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Family Planning Counselling

Gladys Kankam

Counselling permeates through all interactions with clients in the health sector and it's a very vital aspect of health care but often neglected.

It increases the rapport and trust between client and provider.

Counselling becomes more imperative in family planning where the client is a healthy individual. It is a very important task of the family planning provider and effective counselling helps clients understand contraceptive choices available and it provides guidance in deciding which method is best for his or her situation.

Why Counselling

- It ensures appropriate method choice
- It increases acceptance and effective use
- It helps clients decide whether or not they need or want to use a contraceptive
- They learn about their methods of choices
- It helps them to overcome anxieties and make adequate decisions of problems that might occur
- It ensures longer usage and continuation of a method.

Client's Rights

All individuals have a right to decide whether or not they want to practise family planning and they should have the freedom to choose what they want provided it is appropriate.

These rights are:

Right to Information

All individuals have a right to information on benefits of family planning for themselves and families. They have a right to receive services regardless of their social status economic situation, religion, political belief, ethnic origin, mental status, geographical location or anything, which may place an individual in a certain group.

Right to Choice

- They have a right to decide whether or not to practice family planning
- They should be given the freedom to

- choose which method to use
- They have a right to discontinue or switch to another method

Right to Safety

- They should have safety in the practice of family planning

Right to Privacy

- Client should be able to discuss her needs and concerns in an environment in which she feels confident
- Client should be examined in an environment in which his/her consent.

Right to Dignity

Clients should be treated with courtesy, consideration, attentiveness and full respect of their dignity regardless of their social status or level of education.

Right to Comfort

Clients should feel comfortable when receiving services

Right to Continuity

Clients have a right to receive services and supply of contraceptives for as long as they need them.

Right to Opinion

They have a right to express their views on services they receive.

Qualities of a Good Family Planning Counsellor

A good counsellor understands and makes clients feel at ease by exhibiting the following characteristics

- Desire to work with help and respect people for their right to make decisions for themselves
- Tolerance for the values that differ from ones own and belief in the value of family planning, as well as self awareness of one values and limitation
- Supportive attitude towards clients and

- comfort with human sexuality
- Empathy for clients and trustworthiness
- Ability to maintain confidentiality
- Problem solver who helps clients analyse options.
- Allows the participation of clients and help clients to come out with their needs
- Has thorough knowledge about all family planning methods
- Unbiased attitude towards various family planning methods
- Exhibits professionalism

Skills need by a Family Planning Counsellor

A counsellor needs good interpersonal communication skills in order to:

- Present information clearly and in unbiased and client sensitive manner
- Create a comfortable atmosphere
- Encourage client to ask questions and ask questions effectively to encourage client to share information and feeling
- Listen and observe attentively and respond to client's concerns
- Speak the clients language and responds to clients non verbal communication (body language)
- Use appropriate on verbal communication
- Guide counsellor client interaction
- Assesses client's comprehension of the information given before the concluding the interaction.

In giving information the counsellor is brief and uses simple words that the client can understand. The counsellor repeats the most important information and instructions and is specific and concrete so that the client understands.

Clients Consent

This is a very sensitive area and counsellors need to be diplomatic as this can act as a barrier to the provision of family planning. It is ideal for a husband and wife to agree and decide to use family planning services before it is provided. However there are instances where this is not possible and a woman can take the decision alone about her health.

The Ghana National Reproductive Health Policy states: - "For married couples, spousal consent for contraceptive use is not required to receive family planning services".

Motivation (Promotion)

This is not counselling but activities that encourage the use of family planning methods. These activities may be conducted in person or through the media. While they can convey useful information these activities are usually biased. They often attempt to influence an individual or group.

Information of Education

Activities that provide facts about methods and can be done in person or through print materials or other media. The information presented may be complete or limited and may be accurate or incorrect.

Process of Counselling

Counselling may be defined as one person helping another by providing information that the other person needs and giving it in a way that the other person can understand and apply it to his/her own situation.

Counselling is an on-going process integrated into all aspects of family planning services. The activities focus on helping individuals make choices about fertility. Counselling goes beyond giving facts, it enables clients to apply information about family planning to their particular circumstances and to make voluntary informed choices.

It includes a discussion of the client's feelings and concerns. Counselling always involves a two-way communication between the client and the counsellor in which each spends time talking listening and asking questions. Counselling techniques are applied throughout the services delivery point in the family planning clinic, and integrated into each interaction with the client.

Types of Counselling

Initial Counselling: -

This is at the first contact with the client during which all the methods are described in order to help the client select appropriate method of contraception. This usually takes place at the reception. During this period in addition to telling clients about contraceptive methods they are also asked their choice of contraceptives and what they know about the methods as well as their mode of action, advantages and disadvantages. This type of counselling is done individually or in groups.

Method Specific Counselling

This is on one to one basis, prior to, during, or immediately following service provision. It is during this period that the client is given opportunity to ask questions about her chosen method or helped to choose a suitable method. The client is further informed of how that particular method works and its side effects and benefits. It is also explained to her how that method is used and clarifications are obtained from the client.

Follow-up Counselling

This occurs during the return visit for resupply or check up.

This affords the provider the opportunity to discuss any problems, answers questions, find out if client is satisfied and manages minor problems as they occur.

Steps in Counselling

These steps are in the Acronym GATHER.

G

GREET

- Pay full attention
- Be polite
- Greet the client
- Ask client's name
- Introduce yourself
- Offer a seat to the client
- Ask how you can help
- Assure on confidentiality
- Explain routine procedures (e.g. blood pressure)
- Provide privacy

A

ASK about:

- Client's needs
- Knowledge of conditions
- Concerns, doubts etc.
- Reproductive history and goals
- Basic knowledge of relevant

component using appropriate language

- Any change since last visit (old client)

T

TELL client about:

- Basic reproductive anatomy using appropriate language and model
- All family planning methods and reproductive tract infections including STD/HIV/AIDS

H

HELP according to clients:

- Plans and reproductive health status
- Sexual partner's contraceptive method preference
- Unanswered questions
- Final and informed decision

E

EXPLAIN:

- Possible waiting time
- Consent form where required
- Client's instructions and make sure that the client understands them
- Unscheduled visits

R

RETURN for follow-up:

- Enquire about any problems and side effects
- Reassure client and refer if necessary

Family Planning is an important subject in the developing world as a whole and Sub-Saharan

- Ask about any other questions
- Help choose/change services if necessary

If these are followed and integrated in all interaction with clients, there will be more client Satisfaction.

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Family Planning

BDRT Annan/RM Adanu

Family Planning is an important subject in the developing world as a whole and Sub-Saharan Africa in particular. This is because Sub-Saharan Africa has one of the highest fertility rates in the world. Ghana has a population of 19.7 million with a doubling time of 29 years. Nigeria has a population of about 90 million with a doubling time of 24 years.

(1)

This doubling time for Sub-Saharan African countries is much shorter than the doubling time of 42 years for the total world population. These figures show that developing countries, which have a marked scarcity of economic resources, have populations growing at a much faster rate than developed countries. The concerns raised by Malthus in 1798 that population growth will eventually exceed the ability of the earth to provide food, are therefore still relevant in this part of the world even though they were proved unfounded by advances in agriculture and land management.

Contraceptive Use

The knowledge of contraceptive methods in Ghana was reported in 1998 Demographic and Health Survey⁽²⁾ to be as follows: 94% of currently married women and 96% of currently married men know at least one modern method of family planning. The male condom is the most widely recognized method followed by injectables and the pill.

Eighty percent (80%) of currently married women know where to obtain a modern method of family planning.

Despite the high level of knowledge of contraceptive methods, the actual use of contraception is very low in the sub-region. Only 22% of currently married women in Ghana use some method of contraception and only 13% use a modern method⁽²⁾. In Nigeria, it is only estimated that only 6% of women use a modern method⁽³⁾.

Table 1

Trends in current use of contraception Percentage of currently married women who are using a contraceptive method. Ghana 1988 1998.			
Contraceptive Method	1988	1993	1998
Any method	13	20	22
Any modern method	5	10	13
Pill	2	3	4
IUD	1	1	1
Injectables	0	2	3
Diaphragm/Foam/Jelly	0	0	1
Condom	0	2	3
Female sterilisation	1	1	1
Any traditional method	8	10	9
Number of women	3,156	3,204	3,131

In Ghana the most widely used modern method is the pill, 4%, followed by injectables and condoms (3%). The single most widely used method continues to be periodic abstinence

In Nigeria, 20% of married urban contraceptive users use the pill and 16% of them use intrauterine contraceptive devices whilst 33% use traditional methods. Hospital data from Lagos, Nigeria show that the intrauterine contraceptive device (IUCD) is the most commonly chosen method of contraception. It is chosen by 68% of clients with 15% choosing to use injectables⁽³⁾. These figures are inverted in Ghana, where the most common method chosen at the hospital is Depo-Provera followed by intrauterine contraceptive devices.

In Ghana there has been only an increase of 2% in contraceptive use over the past 5 years. Contraceptive use has been shown to increase with age from 19% among women aged 15-19 to 26% among women aged 35-39. It then declines after this⁽²⁾.

Current estimates show that 12% of all pregnancies occurring over the past ten years did not end in a live birth. Lost pregnancies are highest among urban women aged 15-19, with about 2 in 5 women having experienced an early pregnancy loss⁽²⁾. This highlights the need for contraception.

Analysis of the Ghana Demographic and Health survey⁽²⁾ shows that 23% of currently married women have an unmet need for family planning - 11% have an unmet need for spacing and 12% have an unmet need for limiting. Combining this with the current contraceptive use level leads to the conclusion that only about half of the demand for family planning in the sub-region is currently being met.

Non-traditional method of Reversible Contraception

Reversible contraception is the temporary prevention of fertility and includes all the currently available contraceptive methods except sterilization.

The available modern methods of contraception in the sub-region are:

- * Male condom
- * Female condom
- * Vaginal Diaphragms
- * Spermicides
- * Intrauterine Devices (IUD)
- * Progestin-Only Pills (POPs)
- * Combined Oral Contraceptives (COCs)
- * Depo-Provera Injections (DMPA)

- * Norethisterone Oenanthate (NET-EN)
- * Norplant

Male Condom Description

The male condom is a sheath made of vulcanised latex, rubber polyurethane or natural membranes such as lamb caecum. Only the latex condom is widely available in Ghana.

Use

The male condom is placed over the penis prior to genital contact and maintained until after ejaculation.

Mechanism of action

It acts as a barrier to sperm thereby preventing contact between the male and female gametes. It also prevents contact between the ejaculate and the vagina.

Effectiveness

The perfect use failure rate is 3% in the first year of use whereas the typical use failure rate in the first year is 14% (4). Failure could be due to inappropriate placement, late placement, condom slippage or breakage.

Before the condom is put on, the user must check the direction in which it unrolls. All the air must then be squeezed out of the tip of the condom before unrolling it onto the penis.

After intercourse the rim of the condom must be held and the condom carefully unrolled off the penis before the loss of erection. A new condom must be used for each repeated act of intercourse.

Advantages

Bacteria and viruses including HPV cannot pass through the latex condom. The latex condom therefore, offers protection against sexually transmitted infections. The additional use of the condom is recommended for people with multiple sexual partners even if another non-barrier method of contraception is being employed.

The male condom is easily available and almost every couple can use it. It must not be used by people who are known to be allergic to latex.

Disadvantages

- * Effectiveness as contraceptive depends on willingness to follow instruction
- * Resupply must be available.

Female Condom

Description

This is a disposable, single-use polyurethane sheath. It has a flexible, movable inner ring at the closed end, which is used for insertion. There is a larger, fixed outer ring, which remains outside the vagina to cover part of the introitus.

Use

The female condom must be inserted into the vagina prior to genital contact. At the start of intercourse, the penis should be placed manually into the sheath for intercourse. During intercourse one of the couple must manually stabilize the outer ring against the perineum. This prevents the condom being lost into the vagina. The man should monitor for any friction between the penis and the device. Excessive friction can cause breakage. A new condom must be used for each act of intercourse.

Mechanism of action

It acts as a barrier to sperm thereby preventing contact between the male and female gametes. It also prevents contact between the ejaculate and the vagina.

Effectiveness

The perfect use failure rate in the first year is 5% and the typical use failure rate in the first year is 21%⁽⁴⁾.

Advantages

The female condom provides adequate protection against sexually transmitted infections.

It is the only female method that is currently able to achieve this. It is easily available over the counter and almost every couple can use it.

Disadvantages

The main disadvantage of the female condom is the detailed education and experience, which are required for successful use.

Diaphragm

Description

This is a rubber dome-shaped device that is filled with spermicide and placed to cover the cervix. There are four types of diaphragms:

- Arcing spring
- Coil spring
- Flat spring
- Wide seal

Use

The diaphragm needs to be fitted by a trained health care provider. The prescribed size for the client is the largest size that does not cause discomfort or undue pressure on the vaginal mucosa. After this the client must be taught to insert the device herself. The client must be examined after self-insertion to make sure that she has been able to get the device to completely cover the cervix.

The diaphragm must be filled with a spermicide and inserted prior to intercourse. It must be removed 8 hours after the last coital act. If repeated intercourse occurs or intercourse is more than eight hours after insertion, additional spermicide must be used.

Mechanism of action

It serves as a mechanical barrier preventing contact between male and female gametes.

Effectiveness

The perfect use failure rate in the first year is 6%. The typical use failure rate in the first year is however 20%⁽⁴⁾.

Advantages

It reduces the risk for cervical sexually transmitted diseases such as gonorrhoea, chlamydia, cervical dysplasia and PID.

It may be used during lactation after the vagina and cervix have returned to the non-pregnant shape.

Disadvantages

It does not offer protection against HIV and it may increase the risk of urinary tract infections and toxic shock syndrome.

Spermicides

Description

These are vaginal foams, creams and suppositories that contain a surfactant that is able to kill or immobilize sperm, thereby preventing fertilization. The most common surfactant that is present in these spermicides is nonoxynol 9.

Use

The creams, foams or gels are to be applied intravaginally within an hour before the start of intercourse. The suppositories or tablets must be placed in the vagina 15 minutes before the onset of intercourse.

All spermicides must be placed deep within the vagina. Sexual intercourse must be completed within 60 minutes of application.

Mechanism of action

Spermicides serve as both barriers and detergents. As barriers, they prevent entry of sperms into the cervical os. Their detergent activity enables them to attack the sperm flagella and body. This results in reduced mobility and disruption of the fructolytic activity of the sperm.

Effectiveness

The perfect use failure rate in the first year is 6%.
The typical use failure rate in the first year is 26%⁽⁴⁾.

Advantages

Spermicides are available over the counter and can be easily applied.

There is a possible decrease in HIV transmission and they may reduce the risk of cervical cancer. They may also reduce the risk of some vaginal and cervical sexually transmitted diseases.

Disadvantages

Allergic reactions and dermatitis in both men and women have been reported.

Intrauterine Device

Description

IUDs are contraceptive devices that are placed within the uterine cavity at a time that is unrelated to intercourse and serve to prevent conception.

There are two IUDs that are currently widely available in the sub-region.

These are the Copper T 380A and the Lippes loop.

The Copper T380A is a T-shaped IUD made of radiopaque polyethylene, with two flexible arms that bend for insertion but open in situ to hold solid sleeves of copper against the uterine fundus.

The Lippes loop is made of polyethylene impregnated with barium sulphate to make it radiopaque. It is coiled in the form of a spiral but can be straightened out to facilitate insertion.

Both devices have two strings attached to their lower ends. These help in locating the IUDs as well as removal of IUDs.

Use

IUDs can be placed in the uterine cavity at any time during menstrual cycle as long as the client is not pregnant. A well-trained health provider must insert them. Insertion is done using special IUD inserters.

The Copper T IUD and the Lippes loop are both approved for use for 10 years.

Removal is by grasping the strings with ring forceps or uterine dressing forceps and applying firm traction.

The recommended client for IUD use must be:
Parous

In a mutually monogamous relationship

Seeking longer-term (2 years or more) contraceptive

Nulliparous women at low risk for sexually

transmitted infections may be candidates. Client

with a previous history of PID may have IUDs if they

are currently in stable, mutually monogamous relationships.

The IUD is a good option for the following:

Women with a personal risk of thrombosis

Breast-feeding women

Smokers over 35

Women who fear hormonal side effects

Mechanism of Action

* The Lippes loop works as a contraceptive by producing a sterile inflammatory response in the uterus. This inflammatory response produces tissue injury of a minor degree which is sufficient to be spermicidal.

* The Copper IUD serves as a functional spermicide.

It releases copper ions, which inhibit sperm motility and acrosomal enzyme activation.

The IUD also creates an inflammatory reaction that results in phagocytosis of the sperm.

Effectiveness

The perfect use failure rate for the Copper IUD in the first year is 0.6%. The typical use failure rate over the same period is 0.8%. The cumulative 10 year failure rate is 2.1-2.8%.

These rates are slightly higher in nulliparous women and for users of the Lippes loop⁽⁴⁾.

Advantages

The IUD is a very effective contraceptive whose use is not intercourse dependent.

Discontinuation of the method requires a visit to the health provider and this further helps its effectiveness.

Disadvantages

Average menstrual blood loss is increased by about 35%.

There may be an increase in dysmenorrhoea. It requires a visit to the clinic before it can be used. It offers no protection against sexually transmitted infections and increases the risk of PID if a woman is exposed to sexually transmitted infections. Copper IUDs cannot be used in clients with a known allergy to copper.

Indications for Removal

- * If the client so desires
- * At the end of effective life of the IUD Tcu 380A = 10 years
- * If change in sexual practices (high risk behaviour), consider adding barrier method (condoms) or removal.
- * If treated for RTI, other STD or documented pelvic infection
- * Menopause

Post Partum IUD

This is the insertion of IUD within 48 hours of delivery.

- * Post placental insertion is performed within 10 minutes of placental expulsion
- * Immediate postpartum insertion is performed between 10 minutes and 48 hours after delivery

Insertion Techniques

- * Manual post placental
- * Use of Ring Forceps

Advantages

- * There is no return visit
- * Counselling is done at prenatal care visits
- * Pregnancy is alimated
- * Fewer bleeding, cramping and uterine perforation
- * Less expensive (no special clinic)

Disadvantages

- * Higher expulsion rate
- * Higher rate of "missing strings"

Contraindications

- * Prolong rupture of membranes
- * Intra/postpartum infection and haemorrhage
- * History of recurrent pelvic inflammatory disease
- * Bleeding disorder (e.g. P. I. H.)

Practical Points

* IUD Insertion At Caesarean Section

A counselled client may have IUD placed at the fundus during a Caesarean Section before the closure of the uterus, unless there are signs of infection. Immediate insertions during Caesarean Section have a lower expulsion rate than for vaginal insertion immediately, within 10 minutes, after delivery. Such clients also have longer continuation rate.⁽⁵⁾

* Prophylactic Antibiotics For IUD Insertion

Most authorities do not routinely recommend prophylactic antibiotics for IUD insertion, because there is no clear evidence that this definitely prevent pelvic inflammatory disease (PID) in IUD users and studies so far have found only a trivial impact on PID rates due to prophylactic antibiotics. Opinion however differs, and there are arguments to support both sides. Good infection prevention procedure and proper assessment of client, keep the infection rate low in IUD users.^(6,7)

* Warning Signs Of IUD

P	-	Period late
A	-	Abdominal pain
I	-	Infection exposure, abnormal Discharge
N	-	Not feeling well, fever chills
S	-	String missing, shorter or Longer

Progestin-only Pills (pops) Description

These are tablets that contain only a progestin. They are taken daily without any hormone-free interval. The POP is also referred to as the mini pill. The mini-pill contains lower doses of progestin than the combined oral contraceptive pills. Common types available are Micronor, which contains 0.35mg of norethindrone and Ovrette, which contains 75 micrograms of norgestrel

Use

- * The POPs must be taken on a daily basis at the same time every day in order to achieve the maximal contraceptive effect.
- * Virtually any woman who can take pills on a daily basis can use the POPs.
- * POPs are particularly suited for women with contraindications to or side-effects from oestrogen or higher dose progestin.

Mechanism of action

The main site of action of the POPs is on the cervical mucus. The pills cause the cervical mucus to become thick and scanty. This type of mucus, which is characteristic of the luteal phase of the menstrual cycle, prevents sperm penetration into the uterine cavity. In 50% of cases the mini-pill suppresses ovulation by preventing the mid-cycle LH surge. The mini-pill also causes the endometrial lining to become thin and atrophic thereby preventing implantation.

Effectiveness

The perfect use failure rate in the first year is 0.5%. The typical use failure rate in the first year of use is 5.0%⁽⁴⁾.

Advantages

- * There is decreased menstrual blood loss, with amenorrhoea in 10% of users⁽⁸⁾.
- * There is a decrease in menstrual cramps and pain.
- * The mini-pill offers possible protection against endometrial cancer.

Disadvantages

- * The POPs cause irregular menses with increased days of spotting.
- * There is also a possible increase in depression, anxiety, irritability, fatigue and mood changes⁽⁸⁾.
- * Their effect on cervical mucus decreases after 22 hours and is gone after 27 hours. Therefore, if a pill is missed the client is advised to take the pill as soon as possible and then continue with normal pill schedule. Additionally the client is to use a back-up method (condom) for 48 hours (based on mucus effect). The more pills missed, the more likely it is that pregnancy will occur. If unprotected intercourse occurs, the client

should consider the use of emergency contraception.

- * There is no protection against sexually transmitted infections.

Combined Oral Contraceptives**Description**

There are tablets that contain a combination of an oestrogen and a progestin. Each pack of pills contains 28 tablets of which 21 are active and 7 are inactive. The inactive tablets contain either sugar or iron.

COCs could be monophasic or multiphasic. Monophasic formulations have active pills that each contains the same amount of hormones. The advantage of these formulations is that they can be used to manipulate the menstrual cycle. For the multiphasic formulations, the active pills have varying amounts of progestins and/or estrogens. These formulations usually have fewer hormonal side effects.

Ethinyl Estradiol is the most commonly used oestrogen in COCs. Currently used COCs contain 35 microgram of oestrogen. COCs contain 0.25 1mg of progestin. The following are commonly used progestins with the amount used.

Norethindrone 0.5mg
Norgestimate 0.25mg
Ethinodiol diacetate 1mg

Use

The COCs must be taken on a daily basis. Unlike the mini-pill the COCs do not necessarily have to be taken at the same time every day. It is however helpful for a woman to do this or to tie in pill taking with a regular daily schedule so that the problem of missed pills is reduced.

Most healthy reproductive aged women are candidates for COCs. COCs must however be avoided in the following:

Women with a personal history of thrombosis or a strong family history of thrombosis
Recently postpartum women
Women who are exclusively breastfeeding
Smokers over aged 35
Women with severe migraine or who get migraine when on hormones

Cerebrovascular disease or coronary artery disease
 Known or suspected breast cancer
 Active liver disease
 Endometrial carcinoma
 Complicated or prolonged diabetes, uncontrolled hypertension, systemic lupus erythematosus
 Undiagnosed vaginal bleeding.

Mechanism of action

- * COCs suppress ovulation in 90-95% of cases. This is achieved by maintenance of a constant negative feedback on the anterior pituitary and hypothalamus, which results in prevention of the mid-cycle LH surge.
- * The progestins also act on the cervical mucus making it impervious to spermatozoa.
- * It also changes endometrium, making implantation less likely.

Effectiveness

The perfect use failure rate in the first year of use is 0.1%. The typical use failure rate is 5%⁽⁴⁾.

Advantages

Use of the COCs results in decreased menstrual loss, absent ovulatory pain and decreased menstrual cramps. COCs decrease the risk of epithelial ovarian cancer as well as endometrial cancer. There is also a 25% reduction in all benign breast disease.

Disadvantages

- * This method is not available over the counter.
- * Some formulations increase spotting.
- * COCs may cause mood changes and daily pill taking may be stressful
- * The risk of cervical adenocarcinoma is increased 60% particularly in long-term users. COCs may affect later stages of carcinogenesis or alter susceptibility to infection with HPV, a known risk factor. A confounding variable is that COC users tend to have more sexual partners. COC may be used by women with cervical intraepithelial neoplasm⁽⁹⁾.
- * COCs do not protect against the development of sexually transmitted diseases.
- * The risk of hepatocellular adenoma, through lower with low dose COC, is also increased. This tumour is benign but can lead to rupture of the capsule of the liver

Other Issues

* COC and breast cancer

The literature to date is copious, confusing and contradictory. This is complicated by problems related to; Latency, changes in formulation, time of exposure, and high-risk groups. Re-analysis of over fifty studies has shown an increase risk in the development of breast cancer, the resulting tumour spread less aggressively than usual.

* COC and trophoblastic disease

There is no evidence that the incidence of any form of this tumour of pregnancy is increased by past pill-use. Charring Cross Hospital workers have shown that usage of COC before human chorionic gonadotropin has reached undetectable blood levels, doubles the chance that individual requiring chemotherapy for incipient choriocarcinoma. Other researches have not shown this association.

* Management of breakthrough bleeding

The differential diagnosis includes:

- * Inadequate estrogenic or progestogenic stimulation of endometrium
- * Missing pill or altering the time of taken them.
- * Threatened / spontaneous abortion / ectopic pregnancy.
- * Genital tract infection.

Treatment includes:

- * Manage any gynaecological problem identified
- * Reassurance breakthrough common during the first 3 months
- * Increase progestin potency of the pill
- * Increase the estrogenic potency.

* Missing Pill

The greatest risk is when a client misses hormonal pills at the beginning or at the end of cycle.

- * If one pill is missed. Client is to take that pill as soon as she remembers. The next pill should be taken at the usual time.
- * If two or more consecutive hormonal pills are missed.
- * Client takes 1 pill as soon as possible then
- (i) If 7 or more hormonal pills are left, the client

should take the rest of the pill as usual.

(ii) If less than 7 hormonal pill, client is to continue taking the rest of the hormonal tablets. Omitting the placebo pill, the client starts a new package of pills on the next day after finishing the hormonal pill.

* Additionally client is to abstain from sexual intercourse or use a barrier method for a minimum of one week.

Depot Medroxyprogesterone Acetate (Depot-Provera) (DMPA)

Description

DMPA is an injection, which is given as 1ml of a crystalline suspension of 150mg of DMPA every 11-13 weeks. The injection is given intramuscularly into the deltoid or the gluteus maximus.

Use

* A trained health care provider must give the DMPA injection. The point of injection must not be rubbed after the injection since this affects the release of the drug. Rubbing the point of injection causes a larger than usual immediate release of the drug thereby affecting the duration of the contraceptive effect.

* Virtually every woman can use DMPA if she wants immediate to long-term contraception. It is especially useful for women who need to avoid oestrogen.

* DMPA must not be used in the following:

Pregnancy

Undiagnosed abnormal vaginal bleeding

Unable to tolerate injections

History of breast cancer Myocardial infarction MI or stroke

Current venous thromboembolism (unless anticoagulated)

Liver dysfunction

Known hypersensitivity to Depo-Provera

Aminoglutethimide reduces DMPA efficacy.

Mechanism of action

* DMPA suppresses ovulation by inhibiting the mid-cycle LH surge.

* It also thickens the cervical mucus and slows both tubal and endometrial motility.

Effectiveness

The perfect use failure rate and typical use failure

rate in the first year of use is 0.3%⁽⁴⁾.

Advantages

- * DMPA is one of the most effective means of contraception.
- * It is also a very forgiving method since its contraceptive effect often persists beyond the 12-week period.
- * DMPA reduces menstrual blood loss. After 1 year of use 50% of women achieve amenorrhoea and 80% achieve amenorrhoea in 5 years⁽⁸⁾.
- * There is significant reduction in the risk of endometrial cancer and a possible reduction in risk of ovarian cancer.
- * DMPA reduces acute sickle cell crisis by 70% and also decreases the risk of PID⁽⁸⁾.

Disadvantages

- * Menses could be irregular during the first several months with increased spotting.
- * Some patients are disturbed by the amenorrhoea resulting from DMPA use.
- * The method does not offer any protection against sexually transmitted infection.

DMPA Practical Issues

* Management Of Non-acceptable Prolonged Bleeding

- * Ensure client has not recently commenced taking an enzyme-induced drug.
- * Examine client to exclude an unrelated cause notable fibroid, polyp, cervicitis-retained product of conception and carcinoma.
- * Possible treatment

- Reassurance

- Oestrogen (if not contraindicated)

- One or more pack of 30 microgram COC.
- Conjugated oestrogen (Premarin 1.25mg daily for 21 days) repeated if necessary, if unacceptable irregularity follows the withdrawal bleeding.

- Other Options

- NSAID
- Giving the next dose early (but not earlier than 4 weeks since the last
- Haemostatics (tranxamic / ethamsy late has been tried but they rarely work well.
- * Correct Anaemia Iron treatment

Management Of Amenorrhoea

- * Pregnancy needs to be excluded
- * Reassurance
- * One or two cycle of COC, even though not always successful in causing a withdrawal period, can sometimes be helpful as an adjunct to reassurance.

Return Of Fertility

The return of fertility is slow, but complete. The meantime to conception is months and 95% of DMPA users conceive by 2 years. (10) Secondary amenorrhoea, however, does persist in a minority and needs to be evaluated if it last for more than 9 months after last injection. This can be treated by standard ovulation induction methods.

Diagnosing The Menopause

It can be difficult to distinguish between amenorrhoea secondary to DMPA, and the arrival of the menopause. Symptoms, especially hot flushes can be a useful guide. Raised level of follicular stimulating hormone (FSH) may also help.

*** Norethisterone Oenanthate (NET-EN)**

This is progestin steroid given on a regular basis by intramuscular injection. It is available in vehicle of benzyl benzoate and castor oil, the dose being 200mg every 8 weeks. NET-EN is less widely used.

Comparison of DMPA and NET-EN

	DMPA	NET-EN
Effectiveness	No significant difference	
Bleeding	More amenorrhoea	More irregular
Needle/pain	Smaller/less	Larger/more
Re-injection window	2-4 weeks early or late	12 weeks early or late
Duration	3 months	2 months
Cost	Less expensive	More expensive

Non-contraceptive Benefits Of Injectables

Reduction of menstrual cycle disorder

- Less heavy bleeding
- Less dysmenorrhoea
- Less symptom of premenstrual tension
- No ovulation pain
- * Reduction in the growth of fibroid
- * Less pelvic inflammatory disease. (WHO study 1985).
- * Less extrauterine pregnancies since ovulation is inhibited.
- * Protection against endometrial cancer
- * Studies in West Indies show less painful sickle cell crises.

(H) Norplant

Description

This method comprises 6 silastic implants each measuring 34mm long and 2.4mm in diameter. Each implant contains 36ng of levonorgestrel powder, which is slowly released through micropores in the implant to achieve an average plasma concentration of 0.3ng/ml over 5 years.

Use

The implants are inserted into the subcutaneous tissue beneath the skin of the woman's non-dominant upper arm. A trained health care provider does insertion.

Norplant is suitable for virtually any woman who desires effective long-term contraception. It is especially suited for women with contraindications to oestrogen use. It must be avoided in the same groups of women as for DMPA.

Mechanism of action

- * The constant release of levonorgestrel results in the cervical mucus being thickened consistently throughout the cycle.
- * Ovulation is blocked in most women in the first two years due to higher levels of levonorgestrel.
- * Endometrial thinning

Effectiveness

The typical and perfect use failure rate in the first

year of use is 0.5%. The cumulative 5-year failure rate is 1.1%⁽⁴⁾.

Advantages

- * This is a very effective means of contraception.
- * It also results in decreased menstrual blood loss and decreased menstrual cramping and pain.
- * Norplant reduces the risk of PID and it may reduce the risk of endometrial cancer.
- * Long duration of action
- * Immediate return of fertility
- * Women of all ages can use it
- * No effect on lactation

Disadvantages

- * It requires a visit to a health facility for initiation of use and for discontinuation (removal).
- * It causes menstrual irregularities and increased spotting specially in the first year of use.
- * It offers no protection against sexually transmitted diseases.

Norplant Practical Issues

* Keloids Formation

Keloid formation resulting from Norplant implant insertion is a rare event and as such history of keloid is not a reason to restrict Norplant implant insertion. However a woman with previous history should be informed of this small increased risk⁽¹¹⁾.

* Key Elements For Client Satisfaction

- * Careful gentle insertion technique
- * Proper local anaesthetic technique
- * Good counselling/client education
- * Compassionate dialogue and communication with client
- * Assured access to on-going support, follow up and removal
- * Community support and endorsement
- * Home visiting programme (where appropriate)

Other Modern Methods

The following are other modern methods of contraception that are not yet widely available in the sub-region:

- * Combined Injectable
- * Other Implants
- * Hormone-containing IUCD

- * Gynefix Copper IUCD

Combined Injectable Contraceptives (CICS)

This name Combined Injectable Contraceptives is given to a group of hormonal contraceptives, which contain both a progestin and a natural oestrogen, and it is injected intramuscularly into the deltoid or gluteus maximus monthly.

- * Progestin Components
- * Depo-medroxyprogesterone acetate (DMPA) 25mg
- * Norethisterone enanthate (NET-EN) 50mg
- * Dihydroprogesterone acetophenide 150mg.
- * Natural oestrogen components
- * Oestradiol cypionate 5mg
- * Oestradiol valerate 5mg
- * Oestradiol enanthate 10mg

Presently Norigynon (NET-EN and Estradiol valerate) and Lunelle (DMPA and Estradiol cypionate) are available in Ghana.

- * This contraceptive has the same mechanism of action as the combined oral contraceptives.
- * The perfect and typical failure rates in the first year have been reported by WHO and US trials to be 0.1-0.4%⁽⁴⁾.
- * The Oestrogen was incorporated to DMPA and NET-EN. Mostly to improve the regularity of the menstrual cycle. This Natural Oestrogen in CICS (versus "synthetic" in COC) has favourable effect on lipid metabolism and cardiovascular function. The addition of progestin to the Oestradiol (in CICS) has not been shown to lessen these beneficial effects.
- * Based on the above, CICS might be considered safer than COCs. However, they have not been used long enough.

Other Implants

Other hormonal implants that exist are Implanon, Norplant 2, Uniplant, Nestrone and Annuelle.

- * Implanon is a 4cm long single implant, which contains 68mg of etonogestrel (3-ketodesogestrel), a progestin. The implant is placed under the skin of the upper arm and can be retained for 2-3 years. The mechanism of action and effectiveness are as for Norplant.
- * Norplant 2 is a levonorgestrel implant, which comprises two rods placed under the skin

of the upper arm. It can be used for 3 years and it works like Norplant. Because there are only two rods to be placed and removed, the placement and removal procedures take less time than that for Norplant.

- * Uniplant is a single norgestrel implant, which also works like Norplant.
- * The Nestorone implant is also a single implant that is similar in action to Norplant. It can be used for 2 years.
- * Annuelle is a norethindrone implant that comprises several pellets.

Hormone-containing IUCD

The two hormone containing IUCDs in use are the Progesterone IUD (Progestasert) and the Levonorgestrel IUD (Mirena)

The Progesterone IUD is a T-Shaped IUD, which is placed in the uterine cavity. It releases 65 micrograms of progesterone a day from the vertical limb. This IUD can be retained for 12-18 months.

- * The perfect use failure rate in the first year is 1.5% and the typical use failure rate is 2.0%.
- * The progesterone IUD works as the other progestin implants.
- * It may not be used in women with diabetes.
- * High risk of ectopic pregnancy and is therefore rarely used

The levonorgestrel IUD is also T-Shaped and releases 20 micrograms of levonorgestrel from its vertical reservoir. It is active for at least 5 years and has a failure rate of 0.1%.

- * Menopausal women using HRT, with intact uteri that are unable to tolerate other progestins may use the levonorgestrel IUD as their source of progestins.

Non-contraceptive Benefits

- * Decrease menstrual cramps (progestin Releasing)
- * Decrease menstrual bleeding (progestin Releasing)
- * Decrease ectopic pregnancy (except progestasert)

Gynefix Copper IUD

This is made up of 6 sleeves of copper on a string that has one end embedded in the fundus and the other end protruding through the cervix. It has a low expulsion rate and a cumulative 3-year failure rate of 0.5%⁽⁸⁾.

Fertility After Contraceptive Use

One issue that is raised by many women who have not yet completed their family size is the return of fertility after the use of a particular contraceptive method. It is important to have this information so that women who are planning to get pregnant can be able to stop using the method an appropriate number of months before the time that pregnancy is intended.

The barrier methods do not have any long-term effect on fertility. A woman who uses barrier methods runs a risk of getting pregnant anytime the method is not used or there is a method failure.

The non-hormonal IUCDs also do not have a long-term effect on fertility.

Fertility returns promptly to baseline levels after discontinuation of Progestin only pills and Norplant.

The average delay in return of ovulation in a woman who discontinues combined oral contraceptives is 1-2 weeks. Rarely, there may be post-pill amenorrhoea of up to 6 months. With Depo-Provera, return to fertility is delayed but excellent. Ovulation is re-started after an average delay of 10 months and more than 90% of women become pregnant within two years of discontinuing DMPA.

Sterilization

For couples that have completed their family and wish to stop childbearing, the recommended family planning method is sterilization.

Sterilization is also appropriate for a woman with any health problem that is a contraindication to future pregnancy or to use of other family planning methods.

Advantages of sterilization

- * More effective than any other method of contraception.
- * It carries one-time risk of complication
- * It is safe
- * It is easy to perform depending on the operation.

Disadvantages

- * Practically irreversible
- * Not accepted by all culture or religion
- * It may be associated with a few risks.

Importance of counselling

Counselling is important for clients interested in any kind of contraception but is particularly critical for those considering sterilization. These individuals are faced with making a decision that they probably will not be able to change. Because sterilization involves surgery and is intended to be permanent, counselling is especially important. Most clients who choose sterilization remain happy with their decision. However, some clients who freely choose sterilization are later dissatisfied with their choice. Effective counselling helps minimize the possibility of postoperative dissatisfaction and regret.

Risk factors for regret include:

- * Young age
- * Fewer children
- * Uncertain about decision
- * Making the decision when under unusual stress.
- * Pressure from someone else
- * Making the decision without the partners support.
- * Marital instability
- * Unresolved conflict or doubt
- * Unrealistic expectations
- * Economic inducement

Clients with any of these characteristics should not necessarily be refused sterilization; they do, however require more thorough counselling.

The benefits of counselling are:

- * Helps to ensure informed, voluntary and well-considered decisions.
- * Increases client satisfaction and brings about marital harmony
- * Increase the quality of the family planning programme
- * Enhances the reputation of the programme and its staff
- * Contributes to high rate of contraceptive continuation.

Informed Consent

This is the client's voluntary decision to undergo a procedure with full understanding of the relevant facts. In order to make an informed choice regarding permanent contraception, the individual must be told and must understand the elements of informed choice. viz

- * Temporary methods of contraception are available to client or the client's partner
- * Sterilization is a surgical procedure

- * There are risks and benefits associated with the procedure
- * The client will no longer be able to have children.
- * The effect of this procedure is meant to be permanent.
- * There is a small risk of failure.
- * The client has the option to decide against the procedure without sacrificing the right to other services. The client may change his or her mind at any time before the procedure

Pre-operative screening is therefore important to ensure that, through counselling and the informed choice process, the client makes a voluntary and informed choice and any non-medical factors likely to cause regret are identified.

Female Sterilization

This is defined as surgery to interrupt the fallopian tube to prevent pregnancy.

Cumulative 10 year failure rates⁽¹²⁾

Method	Failure Rate
Post partial Salpingectomy	0.8%
Silastic bands	1.8%
Interval partial Salpingectomy	2.0%
Bipolar cautery	2.5%
Spring clip	3.7%

Timing of Female Sterilization

This may be safely performed at the following times:

- * Interval sterilization: any time after 6 weeks post-partum or after 1 week post first trimester abortion
- * Post partum sterilization: within 1 week (preferably 48 hours) of delivery or concurrently with caesarean section.
- * Post abortion sterilization: within 1 week after abortion.

Technically it is more difficult to perform post-

partum or post-second-trimester abortion procedure any later than the end of 7th day following delivery or abortion.

Type Of Surgical Approach

* Minilaparotomy

This is simplified laparotomy approach using an incision 5cm or less. A transverse or longitudinal incision is made under the umbilicus for post partum procedure and transverse suprapubic incision is used for the interval or post abortion procedure.

This approach may be difficult in obese women, if uterus is immobile or the tubes are adherent to surrounding structures.

* Laparoscopy

The operator uses endoscopic equipment inserted through a 1.0-1.5cm incision under the umbilicus. This is to be avoided immediate post partum and second-trimester abortion.

* Other surgical Approaches

Laparotomy may be used when sterilization is performed in conjunction with caesarean section or any gynaecological operation.

- * Non-Surgical Transcervical approaches are experimental
- * Intrauterine approaches (colpotomy, culdoscopy) are not recommended for routine use.

Methods of tubal occlusion

A. Ligation

Oldest and most common method use. It is easy to perform, safe, very effective and inexpensive.

* Pomeroy's

Most widely used method because of its simplicity and effectiveness. It involves ligation of the base of a loop of isthmic portion of tube with plain catgut followed by excision of the loop. 3-4 centimetre of the tube is destroyed, and plain catgut is used because of its rapid absorption, minimising inflammation and fistula formation.

* Parkland

Involves excision of 1-2cm of the isthmic

portion of the tube after separation from the mesosalpinx and separate ligation of the cut ends.

Other methods

In the **Irving method**, the tube is first doubly ligated and severed. The proximal stump is buried into the uterus and the distal stump into the mesosalpinx.

For the **Uchida method**, the muscular part of the tube is dissected out and 3-5cm of it is excised. The proximal end is buried and the distal tube is exteriorised.

In the **Kroener method**, a fimbriectomy is performed.

The Uchida and Irving technique have the lowest chance of failure but are technically difficult to perform adequately via a mini-laparotomy incision. The Pomeroy method is therefore the most commonly employed method.

B. Mechanical

* Falope-ring

This is applied 2-3cm from the cornu. The advantage is that 1-3cm of the tube is destroyed. However it could lead to transection of the tube.

* Clips Filshie / Hulka

The clip is applied 2-3cm from the cornu. Less than 1cm of the tube is destroyed.

Contraindication to Female Sterilization

Absolute

- * Pregnancy
- * Systemic or local infection

Relative include:

- * Severe obesity
- * Severe anaemia
- * Bleeding disorder
- * Pelvic mass.

Causes of failure of sterilization

- * Pregnancy predating the procedure
- * Failure to occlude one or both tubes
- * Spontaneous recanalization of a tube.
- * Use of non-absorbable ligature on the tube
- * Formation of a fistula in the tubal wall.
- * Failure to identify the correct structure to occlude.

- * Failure of devices (clip/ring) used to occlude the tube.
- * Application of devices to the wrong portion of the tube.

Male sterilization

Male sterilization or vasectomy is the interruption of the male reproductive capacity for the purpose of permanently ending fertility. This is accomplished by a simple, safe, inexpensive and well-accepted operation, which can be performed as an outpatient procedure. Each vas deferens is occluded or cut so that the sperm are not released into the ejaculate.

* Contraindication

Absolute: -Systemic or local infection

- Caution:**
- Inguinal hernia
 - Filariasis (elephantiasis)
 - Large hydrocoele or varicocele
 - Undescended testicle
 - Intrasrotal mass
 - Severe anaemia
 - Bleeding disorder

Operative Procedure

In addition to the traditional vasectomy technique, the no-scalpel vasectomy is now being used. The no scalpel technique involves puncturing the scrotum, delivering the vas and then ligating or cauterising it. Aseptic technique and careful haemostasis are very essential for successful procedure.

- * Vasectomy has a failure rate of 0.10%
- * Vasectomy does not affect erectile potency. Sexual ability remains the same. The body absorbs the sperm and no proven long lasting effects of vasectomy have been found.
- * Postoperative follow-up care
 - Examination of scrotal area for proper healing.
- * Advise the need to use condom for total of 20 ejaculations
- * Semen analysis
- * This is performed after 20 ejaculation or 12 weeks after the procedure.
- * If sperms are still present after above, re-evaluate the case (repeat semen analysis or second vasectomy).
- * Complication of vasectomy
 - Intra-operative bleeding
 - Post-operative scrotal swelling bruising and pain
 - Haematoma formation and infection
 - Congestive epididymitis and granuloma formation.
 - Development of sperm antibodies.

Contraception For A Woman With Medical Condition

Women with chronic medical problems may need contraception. Providing a method in such a situation can be complicated in view of the fact that by interaction, the medical condition may limit the scope of methods that are appropriate for use. It is important to offer special counselling to guide them in choosing a method. Postpartum women with serious medical problem are to be encouraged to fully breastfeed their infants.

* World Health Organisation (WHO) Eligibility Classification System

WHO has come out with classification system for initiation and continuation of commonly available methods of contraception. The suitability of method is determined by weighing the health risks and benefits relative to specific condition. WHO is committed to enabling people to select what is appropriate for them without "medical barriers"

- * **POCs:** Women with **active** (symptomatic) hepatitis should avoid using POCs unless other more appropriate methods are not available or acceptable (WHO class 3)
- * COCs, CICs and POCs may be used by women who are asymptomatic (i.e., liver function has been Normal for 3 months) or are carriers.

High blood pressure

- * COCs and CICs: Women with blood pressure (BP):
 - * $> 180/110$ should not use COCs or CICs. (W H O c l a s s 4)
 - * $= 160/100$ but < 180.110 should avoid using COCs or CICs unless other more appropriate methods are not available or acceptable. (WHO class 3)
 - * $< 160/100$ can use COCs or CICs. (WHO class 2) but with close monitory during the first few months
 - COCs and CICs: Women with arterial vascular disease as well as high BP should not use either COCs or CICs (WHO class 4)
- * Low-dose COCs and CICs cause little or no BP change in healthy clients. It is reasonable to

consider their use in hypertensive patient (close monitoring) unless there is an underlying arterial disease. (Clotting problem)

- * PICs: Women with BP > 180/110 should avoid using PICs unless other more appropriate methods are not available or acceptable. (WHO class 3)

HIV/AIDS

- * IUDs: Women with HIV/AIDS should avoid using IUDs unless other more appropriate methods are not available or acceptable. (WHO class 3) Because of risk of infection in an immunosuppressed individual
- * Individuals seropositive for HIV, or who have AIDS, always should use a condom (male or female) to reduce the risk of spreading the disease.

Seizure disorders

- * COCs, CICs, Implants and POPs: Women using anticonvulsants should avoid using these methods unless other more appropriate methods are not available or acceptable. (WHO class 3)
- * This is because the anticonvulsants induce enzymes and cause rapid metabolism of Oestrogen and Progesterin, thus decreasing the effectiveness of all hormonal method except CICs because of the higher blood level.
- * Development of inter-menstrual bleeding or spotting May indicate a decreased level of sex steroid hormones (oestrogen and progesterin) due to interactions with antiseizure drugs. If this occurs in a client using a COC containing 30-35ug EE, consider using a COC with higher oestrogen level (50 ug EE) or help her choose another method. If using a CIC or POP, help client choose another method.

Sickle cell disease

And trait

- * All contraceptive methods can be used. POCs are recommended. Implants and PICs are preferred over POPs, especially if the woman frequently is ill and not eating or drinking regularly. (Use of PICs and possibly implants may decrease the frequency of attacks)

Tuberculosis

- * COCs, CICs, Implants and POPs: Women using rifampin for tuberculosis should avoid

using these methods unless other more appropriate methods are not available or acceptable. (WHO class 3). Anti-tuberculous drugs cause more rapid metabolism of oestrogen and progesterin thus decreasing their effectiveness

- * IUDs: Women with known pelvic TB should not use an IUD because of increase risk of secondary and uterine bleeding

Uterine fibroids

- * IUDs: Women with uterine fibroids or scar tissues in the endometrium (uterine synechiae) that distort the uterine cavity should not use IUDs. (WHO class 4). Large fibroids especially if submucous can distort the uterine cavity making insertion difficult, resulting in increase risk of expulsion.
- * Although estrogens can stimulate growth of uterine fibroids, low-dose COCs (30-35 ug EE) do not appear to cause them to grow.

Emergency Contraception

Emergency Contraception refers to type of Contraception given as an emergency procedure after unprotected sexual contact to prevent pregnancy. In current usage, it is any female method, which is administered after sexual intercourse and has its effect prior to the stage of implantation of the blastocyst.

Wide use of emergency contraception could prevent a substantial proportion of millions of unplanned pregnancy that occurs every year and would lead to increase in new client recruitment.

This principle of contraception has been recognised for centuries and included that of douching with wine, ground garbage and Coca-Cola. More effective regimen like Yuzpe method however has been available since the 1960s.

Type Of Emergency Contraceptives

- * Hormonal - Emergency Contraception Pill (ECP)
- * Combined Pill. Yuzpe method.
- * Progesterin only emergency contraception pill (POEC)
- * Postcoital copper releasing intrauterine device (IUI) insertion

Mechanism of action

- * Hormonal
- * Before ovulation Prevents or postpone Ovulation by disruption of normal follicular

development and maturation. It is also possible ECPs may alter the transport of sperm or ova.

- * After ovulation alters the endometrium to impair or block implantation probably by desynchronization of the endometrium histology and blockage of oestrogen and progesterone receptors.
- * Copper IUD
- * Mainly by blocking implantation but if inserted earlier in the cycle it can also block fertilization.

Effectiveness

- * Emergency contraception is used once or infrequently; therefore traditional measures of effectiveness are not applicable.
- * Failure affected by, fertility of the individual, day of the cycle treatment started and other acts of intercourse before and after treatment.
- * It is also influenced by sexual act intervention interval. WHO trial 1998 stated that each 12 hours delay raised the pregnancy risk by almost 50% (10).
- * Typical failure rate
 - * IUD 1%
 - * Combined Pill.....2%-5%
 - * POEC 1%

A treatment failure rate of one percent (1%) does not mean however that the method is 99% effective, because most women would not have become pregnant without treatment.

Indications

Emergency contraception is meant to be used after unprotected sexual intercourse or after contraceptive "accident". The following condition may necessitate the use of this method.

- * No contraceptive has been used.
- * Contraceptive accident or misuse.
- * Condom rupture, slippage or misuse (removal incorrectly)
- * Diaphragm/Cervical cap early removal or dislodgement.
- * Erring in practice of coitus interruptus and periodic abstinence
- * IUD partially or totally expelled.
- * Women has been victim of sexual assault
- * Rape
- * Incest
- * Exposure to possible teratogen such as live vaccine or cytotoxic drug.

Currently Accepted Regimen

- * Immediate insertion of copper IUD
- Not more than five (5) days after the most probable calculated date of ovulation irrespective of number of unprotected intercourse or.
- Not more that five (5) days after any single exposure
- Can be kept for continuous contraception

Combined Pill

- Commence within 72 hours of the earliest act. Efficacy is greatest in the first 24 hours and decline thereafter (though not to nil after 72 hours)
- The method uses some of the available contraceptive containing oestradiol, norgestrel and levonorgestrel in the following dosage
 - * Ethinyl oestradiol 200 microgram
 - * Levonorgestrel 1000 microgram
 - * Norgestrel 2000 microgram
 - * Example - Microgynon 8 tablets

- In two divided doses 12 hours apart.
 - Yuzpe method.
- Ovran/PC4, 4 tablets in two divided doses 12 hours apart.
- * Levonorgestrel (LNG) alone (POEC)
 - Starts within 72 hours of the earliest exposure and efficacy greatest within 24 hours.
 - Dosage: 750 microgram LNG start and repeat after 12 hours
 - Example - Prostinor
 - Levenelle 2
 - Where above not available dosage has to be constructed with patient being reassured that a total of 50 tablets e.g. microval/norgeston, or 30 of ovrette is not an overdose.
 - This method is more effective, with fewer side effects and contraindications as compared to the combined pill (WHO trial 1998)

Disadvantages /Side Effects

- * Hormonal
- * Nausea and vomiting
 - Antiemetic medication, an hour before first dose may help reduce these symptoms.
- * Menstrual changes
 - Timing, duration and amount of next menstrual period changes in 10 15% of women treated
- * Copper IUD

- similar to those seen after routine insertion
- Pain, bleeding (spotting), risk of infection

Cautions

- * **Hormonal**
- * Pregnancy
- * Current focal migraine
- * Jaundice (liver disease)
- * Active acute porphyria
- * Several allergy to constituent
- * Serious past thrombosis

IUD

- * Pregnancy and as for IUD's generally
- * Concern with pelvic inflammatory disease especially when this is requested after sexual assault.

Follow Up

- * If no menses within 3 weeks, client is to consult clinic or service provider to check for possible pregnancy.
- * If pregnancy is not prevented, client is counselled regarding options.

Future Regimen

- * Anti-progestin mifepristone (RU 486)
- Effective when given within 72 hours
- Failure rate < 1%
- Mechanism of action
- * May delay ovulation
- * May block progesterone effect on endometrium
 - Single 10mg dose given up to 5 days after a single coital exposure (WHO Lancet 1999)
 - Vomiting occurs in 2% and delay in menstruation (71 days) in 18% of can Single dose POEC is currently being tested against 10mg mifepristone.

Future Methods

A number of contraceptives leads are being developed. These include new barriers methods, hormonal delivery system and systemic method for men.

- * **Mechanical Barrier Method**
- * Leas Shield

This is one size-fit all diaphragm-like device with a

- one-way valve to allow
 - air to escape during placement
 - uterine and cervical fluid to escape.

* Femcap

A cervical-cap like device, which comes in 3 sizes. A users size is based on her parity.

* Polyurethane Male Condom

The use of Polyurethane, instead of Latex rubber, is to improve on the feel and durability of the male condom. Some couples complain of decrease pleasure and spontaneity with the latex condom.

* Chemical Barrier

New system are being developed to

- Improve on ease of use
- Decrease vaginal tissue reaction
- Increase spreadability and adhesiveness on the vaginal wall (to protect against HIV)

* IUD

- Frame IUD
- This eliminates pressure on the uterus, thereby minimising the abdominal cramps associated with IUD.
- Two versions
- Polypropylene thread which anchors copper-releasing sleeves to the fundal myometrium
- Biodegradable anchoring cone.

Male Hormonal

* The most promising approaches are a long-action testosterone ester given by three monthly injections, coupled with an oral or injectable or implanted progestin or a prolactin analogue.

* The concept is one of suppression of spermatogenesis while maintaining muscularity and libido through the androgen content.

* Immuno Contraceptives

The immunologic approach has been explored over the last two/three decades

* For women The most advanced is the use of HCG-based approach.

* For men

The potential ones are LHRH & FSH vaccine.

- FSH: eliminates sperm production
- LHRH: affects both testosterone production and sperm production

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SECTION 7

STATISTICS

Common Statistics In Obstetrics And Gynaecology

RB Biritwum

Introduction

Observation and measurement are important activities in the practice of obstetrics and gynaecology. These activities, when scientifically undertaken could yield data, which could form the basis of improved service to the patient and the society. The day-to-day activities of the health personnel involve documentation of events in registers and patient's notes. In order to use the data generated from the practice of the health workers, the data must be collected, organized, analyzed and interpreted. These steps form the foundation of the science of statistics.

The processed data from a unit or a service are called statistics, for example hospital statistics or health statistics. The main thrust of this chapter is to promote understanding of statistical methods used in data collection and analysis for the health care environment. The skills in calculation can be obtained from other sources.

Definition and types of data.

Data are a set of numeric or non-numeric observations on characteristics of a target population. The target population could be patients, specimens or health facilities. Each target population may have many characteristics on which data can be obtained. For example, age of the patients, the sex of the patient, the weight, height of the patients and diseases of the patients can form different data sets.

Types of data

There are two types of data, numeric (quantitative) and non-numeric (qualitative).

Numeric data can be described as continuous or discrete.

Continuous data are **measurable**; they can assume an uninterrupted range of values. For example:

- age of patients in years
- weight of a new-born in grams and
- volume of urine in (mls) produced by a patient.

Discrete data are **countable**, possible values are distinct or separated. For example:

- the size of a family measured as *number* of children in a family
- the frequency of use of a health facility by a patient measured by *number* of times
- the *number* of days since onset of illness.

Non-numeric are qualitative description of categories within a characteristic. For example:

- the blood group type of a patient such as group A, group B and group O
- temperature reading recorded as high or low or normal
- a list of diagnosis made at a clinic will generate a non numeric data-set on a characteristic, the diseases seen at OPD
- if the sex of a patient is recorded as "male" or "female", the data set obtained by recording such data for many patients will be a non-numeric data set.

The levels by which data are measured in relationship to their use

Many kinds of data are collected at different levels in the health care system. For example, the temperature of a patient can be recorded in degrees centigrade or be recorded as high or normal. Recording the temperature in degrees is on a higher level of measurement than recording temperature as normal or high.

Other examples:

- the temperature of children examined at a health facility level (interval scale)
- the diseases diagnosed (nominal scale)
- the age of women who use contraceptives (ratio)
- the educational level of patients (ordinal scale).

The manner in which the data are analysed and the amount of information obtained depend in part on the 'scale' or 'level' (nominal, ordinal or interval or ratio) on which the data have been measured.

There are four kinds (or levels) of measurement scales that are used to describe the various data, which are obtained when a characteristic is observed in a target population. They are:

1. Nominal scale: the data may simply represent categories. In which case they

are referred to as being nominal either, dichotomous, or also as qualitative. For example:

- a) the sex of patient: males and females
- b) blood group: A, B, AB, and O.

2. Ordered (ordinal): if the recorded data represent an ordering, then the data are said to be ordinal. It is similar to the nominal scale but has inherent or predetermined order in the categories. For example: results of hospital management of a case could be;

- a) considerable improvement
- b) moderate improvement
- c) no change
- d) slight deterioration
- e) considerable deterioration and
- f) death.

3. Interval scale: on this scale, distances between any two points on the scale are of known sizes, and the zero point is arbitrary. Example:

- a) the temperature scale, the actual numerical value of the temperature scale is a comparison with an arbitrary point called 'zero degree'
- b) the Apgar score for grading the health status of new-born.

4. Ratio scale: for quantitative data, on this scale, the zero point is definite i.e. the zero on the scale of measurement indicates absence of the attribute:

- a) the age of patients
- b) the blood pressure reading
- c) the volume of urine produced by the patient
- d) the volume of syrup of drug.

The units of measurement of data are important in determining whether the measurement is qualitative or quantitative. When age is recorded as adult or child, we have qualitative data and measured as ordinal. Again, when temperature is recorded as normal or high instead of in degrees, the scale is qualitative and ordinal. The type of measurement will determine whether in summarizing the data one would use a proportion or calculate a numerical summary index such as a mean.

Sources of data

There are two main sources of data to the health

personnel. The routinely generated data from the practice of the health personnel, which can be found in registers or patient's notes and the data, obtained from periodic or special research studies. Data collected for special studies should follow good scientific procedures regarding the selection of the part of the population (sample) to study, the size of the sample, the representativeness of the sample as well as the instrument required for the collection of the data.

Population and Samples

In order to be able to make generalizations from sample to the target population, the sample must be

- (i) Representative
- (ii) Selected at random to minimize bias, (iii) Large enough to increase precision (iv) Covered adequately and
- (v) should be used with a good research design. A sample that is badly selected or inadequately covered remains a biased one; however big the sample size may be. The accuracy with which the observation from the sample could be used to describe the entire population will depend on the above points

Sampling Procedures:

Sample Frame:

The entire list of all the sampling members or units in the population from which the sample is to be taken.

Sampling Unit:

The individual members of the population i.e. the units which constitute the sampling frame.

Probability sampling.

Simple random sampling (SRS). The method ensures that each member of the population has equal or known chance of being selected into the sample. It is the most basic form of all sampling. It is selected by a process that not only gives each element in the population an equal chance of being included in the sample but also makes the selection of every possible combination of the desired number of cases, equally likely. There are two ways of obtaining an SRS

- (i) BALLOTING and
- (ii) use of RANDOM DIGIT TABLE

Systematic Sampling

It uses predetermined system, using sampling interval 'k' and selecting every k^{th} item in the list starting with an item between 1 and k selected at random. The method is useful when there is a list of the population or if elements or items are arranged

in rows like houses along a street, or patients arriving at a health service.

Cluster Sampling

The sample is selected in groups instead of individual sampling units. Heads of households in villages or localities. The villages are selected at random and within each village, all the sampling units are used. The advantage of cluster sampling is that it reduces costs and leads to saving of time, little administrative effort is required. However, it leads to an increase in sampling variation. In this connection, it is desirable that elements within a cluster to be dissimilar in respect of the study variable.

Stratification

Stratified sampling is used to separate heterogeneous population into homogeneous strata for sampling. Within each stratum, any of the above sampling procedure can be used. The sub sample size can be selected according to the proportion i.e. proportional allocation, or equal sample size, or the use of optimum allocation.

Multistage Sampling

The final sample is obtained by random sampling of bigger units and then further sub sampling of smaller units within the selected bigger units of the previous sampling. i.e. Regions, Provinces, Districts, and Villages. Modifications and combination of the above methods are used in practice. A very common application of cluster sampling is seen in the estimation of vaccination coverage and the determination of prevalence of diarrhoea in children under five. List of all the villages and their population sizes are compiled, cumulative frequencies are calculated, sampling interval obtained by dividing total population by number of clusters (30). The clusters to be selected for the investigation are obtained as in systematic sampling.

Non Probability Sampling

Quota Sampling

This is to make sure that the proportion in the sample should agree with the corresponding proportion in the population. The investigator has considerable control over the choice of elements that come into the sample. The population is divided into sub populations or strata. The most common variables for the division are social class, geographic area, age, sex, race and some measure of economic level. The sample size required in each stratum is computed in advance so that stratification is proportional to size. The

enumerator is instructed to continue sampling until the pre-determined "quota" has been obtained in each stratum. Selections subjective. No sampling frame has to be provided. Unfortunately, selection bias could be significant and no statement can be given on the precision of the estimates.

Sample Size Determination

How Large?

Too large a sample implies a waste of resources. Too small a sample diminishes the utility of the results. The sample size is decided on (i) the objective of the study, (ii) the level of confidence with which one will like to estimate the population parameter (iii) the level of acceptable error and (iv) in consideration with the available resources.

Sample size: This depends on (i) the objective, (ii) the design of the study, (iii) the plan for statistical analysis, (iv) the accuracy of the measurements to be made, (v) the degree of precision required for generalization, (vi) the degree of confidence with which to conclude, and (vii) the resources. A balance may have to be struck between the cost and the usefulness of the sample.

Comparison of two proportions.

Example. In a clinical trial to compare two analgesics A and B, the null hypothesis specifies that the two drugs are equally preferable. One therefore proposes to conduct a two-sided test on the null hypothesis. If on the other hand the null hypothesis is false i.e. the proportion preferring A is as high as 0.7 then the investigator wishes to risk only 5% chance of failing to conclude that A is significantly preferable to B (type II error) The question is how many patients with chronic headache are needed.

Study Design and Research Instrument

After deciding on the sample, there is a need to use a good research design and appropriate research instrument.

- A. DESCRIPTIVE STUDY
(data on both cause and effect in an individual are not known)
- B. ANALYTICAL
 - I. Non-experimental (nature determines exposure)
 - (a) Cross sectional (time sequence between cause and effect not known)
 - (b) Longitudinal (time sequence is known)

- Case-control - selection on disease
- Cohort - selection on exposure
- II Experimental - The investigator determines exposure.
- Randomized Controlled Clinical Trial.

Research Instrument

Data collection instruments or tools

Data collection tools include:

- * Data recording forms
- * Intermediate forms, tally sheets and other forms
- * Reporting forms
- * questionnaires which could be self-administered or administered by face-to-face or long-distance interviewer
- * check lists for interviewers
- * interviewing guides for focus group discussions

The same instrument can be used as a checklist or A questionnaire depending on the mode of application. Sometimes instead of designing one's own questions on every topic, one can use standardized questions that have been developed over the years, this also allows for

comparability of surveys.

Aggregation of data

Before any use can be made of the raw set of data, which consist of individual observations, (as found in a register) they have to be aggregated and summarized. Grouping extracted data is the first step in data organization. Tallying is the simplest method of extracting information from a register. The manual organization of data consists of initially counting the number of times certain values of a characteristic (variable) appear in the data set by making strokes or other mechanism for counting or tallying (refer below).

The 'picket fence' method

The frequencies should be obtained by going through the whole set of individual observations systematically putting a mark in the appropriate group. To facilitate counting, frequencies in groups of five are usually formed by putting a line through four tally marks (refer to Table 1).

Table 1 - Example of the 'picket fence' tally method

Disease	Tally	Frequency
Malaria	### ###	15
Diarrhoea	###	6
Skin infection	### ###	10
Accidents	///	3
Others	////	4
Total		38

Summarising data

There are three main methods of summarizing data (tabular, graphical and numerical)

Tabular

- * frequency distributions
- * distribution of attributes by a factor and
- * relational tables or cross tabulation.

Graphical

- * bar charts

- * Histograms
- * line diagrams
- * scatter diagrams and
- * spot maps.

Numerical Epidemiological measures for relations

- * ratios, proportions, rates.
- * Incidence, prevalence, case fatality rate

Measures of central tendency

- * mean or average, median and mode.

Measure of variation

* range, standard deviation, variance standard error

What should be calculated and analysed and how this should be done depends on the objectives for which the data were collected as well as the type of data at hand (qualitative or quantitative).

Construction of tables

A table can be constructed from a data set to show the number of observations at different values of the characteristics of the target population in the data set. The purpose is to find out whether any meaningful pattern exists in the data set. It can be used for all types of data (qualitative or quantitative). For qualitative data, the number of observations in each category is counted. The counts are referred to as frequencies.

In the health system, it is sometime necessary to use codes for the diseases diagnosed at a clinic before summaries are made. For example, reasons for admission to a female ward in a hospital. The International Classification of Diseases (ICD 10) can be used to summarize the reasons of admission.

Relational tables

A table can be constructed to show the relationship between two or more characteristics.

The concept of rows, columns and grand totals.

Table 1 Reasons for admissions to hospital females (15 years and above)

(ICD-10) Code	Disease	Frequency Cases	Dead Cases
O80.9	Single spontaneous delivery, unspecified	8,251	0
O03.9	Spontaneous abortion, complete without complication	1,504	0
O03.0	Spontaneous abortion	850	2
O46.9	Antepartum haemorrhage, unspecified	462	1
B54.0	Unspecified malaria	452	8
D25.9	Leiomyoma of uterus, unspecified	409	1
O00.9	Ectopic pregnancy, unspecified	383	5
O13.0	Gestational [pregnancy-induced] hypertension	382	3
O63.9	Long labour, unspecified	378	0
O72.0	Third-stage haemorrhage	361	4
D64.9	Anaemia, unspecified	346	4
N73.2	Unspecified parametritis and pelvic cellulitis	231	0
N83.2	Other and unspecified ovarian cysts	89	0
E14.0	Unspecified diabetes mellitus	88	24
O20.0	Threatened abortion	87	0
C50.9	Malignant neoplasm of breast, unspecified	79	7
H26.9	Cataract, unspecified	71	0
N88.3	Incompetence of cervix uteri	71	0
O16.0	Unspecified maternal hypertension	69	0
O30.0	Twin pregnancy	68	0

Table 2 Cross-tabulation of the quality of antenatal clinic attendance and the outcome of labour

	Complications during delivery		Total
	Yes	No	
Antenatal visits Before delivery			
Yes	45	15	60
No	30	70	100
Total	75	85	160

Table 2 consists of two rows and two columns forming the body of the table. The intersection of a row and a column in a table is called a cell. The column headings (YES and NO) indicate the classification of the variable (Complications during delivery). The row variable (Antenatal visits before delivery) also has two row headings (Yes and No). There are rows and columns for marginal totals and where the two marginal totals intersect gives the grand total cell.

Data presentation (graphical/diagrammatic)

Tabular data are sometimes difficult to visualize, in order to use them fully; one can turn them to diagrams or graphs. The type of graph required depends on the type of the data, (qualitative/quantitative) or discrete or continuous. Diagrams bring out the outstanding features of the data for a quick appraisal. Graphs are useful to display patterns or trends, which are not immediately obvious from the casual inspection of tabulated data.

1. For continuous data set (data that are measurable)
 - a. Histogram
 - b. Line graph
 - c. Frequency polygon
2. For discrete data set (data that are countable)
 - a. Pie chart
 - b. Bar diagram
3. Scatter diagram for displaying relationship between two characteristics of a target population such as weight and age.

Each table and figure or diagram should be self-explanatory with suitable headings giving a clear

precise description of the contents. They should also be kept as simple as possible. Large tables are difficult to read and interpret.

Common graphical presentations in obstetrics and gynaecology practice

Numerical methods for summarizing data

Sometimes, one wants to use a single figure (summary figure) as an indication of the general level of a series of measurement, i.e. to be able to describe the raw data with one or two summary figures. Such a figure may be called a measure of location, a measure of central tendency, a mean or an average. There are three commonly used summary measures for quantitative data set: the mean, the median and the mode.

A ratio is called a proportion whenever the elements in the numerator are also part of the denominator. In practice, the term "ratio" is often applied to measures where the numerator is not part of the denominator. Example is maternal mortality ratio (the number of maternal deaths due to abortion, delivery and six weeks after delivery divided by total number of live births).

The rate

The rate is a measure of how fast a particular phenomenon occurs over time or space, and therefore refers to a process, which is dynamic or changing. In public health, the incidence rate of a disease measures how fast people are developing that particular disease over a period of time. The crude birth rate measures how fast babies are being born in a population over a given time period.

Measures of disease frequency: prevalence and incidence

The major difference between the incidence and prevalence of a disease is the type of cases that we

Figure 4 The number of cases disease/conditions diagnosed at a clinic

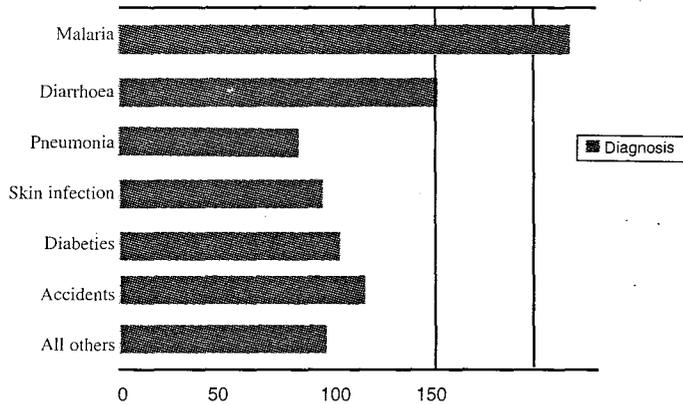


Figure 2. Histogram on the birth weight of the babies

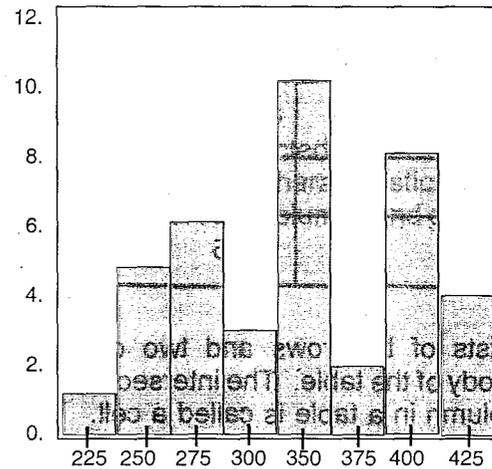
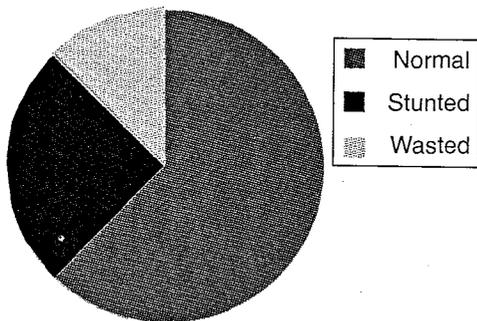
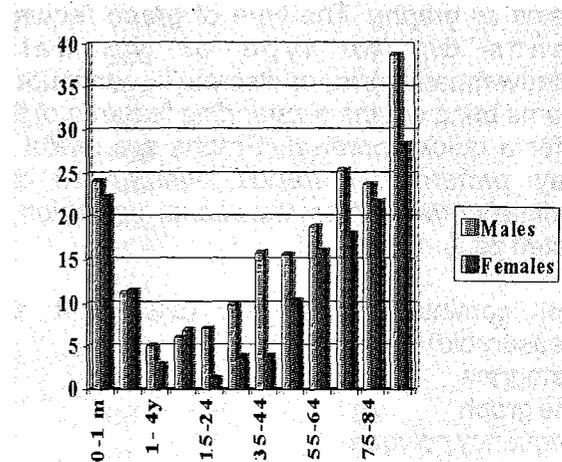


Figure 3. Pie Chart of nutritional status of children



Age and Sex-specific mortality



Bar chart, histogram, bar chart for two variables and pie diagram (clockwise)

include in the numerator.

Incidence: the number of new cases of the disease over an interval of time, usually a year. It is a measure of the number of people developing the disease.

Estimation of a population characteristics (mean blood sugar level)

Confidence Interval

It provides an interval estimation of a population parameter like the mean. It is to use the sample to pin pointing or estimating the unknown population quantity. The confidence limits can be obtained by using the standard normal distribution, the probability of the normal deviate, the z score lying between -1.96 and +1.96 is 0.95. The population mean can be written as a function of the sample mean, the standard deviation and the z score. The limits thus obtained are called 95% confidence limits on the population mean.

Tests Of Significance: Hypothesis Testing

Statistical inference is a process by which one draws a conclusion regarding a population from the results observed in a sample.

An observed difference in parameters, between two groups such as 'treated' and 'control' may be as a result of

- (i) sampling variation or chance
- (ii) Inherent differences between the treatment and control groups
- (iii) Differences in the handling and evaluation of the treatment and control groups during the course of the investigation
- (iv) The true effects of the new procedure.

Test of significance is the method to rule out sampling variation as an explanation of the observed difference.

A Statistical Hypothesis:

An assumption about the frequency function on a random variable Test of significance always refers to a null hypothesis, i.e. it answers the question, is sampling variation a likely explanation of the discrepancy between a sample result and the corresponding null hypothesis population value.

The test statistic, which is calculated, leads to the

determination of the p-value i.e. what is the chance that random sampling will produce a mean as deviant or more deviant than the observed value.

The process involves

- I. Making a null hypothesis, which gives a statement that claims a particular value for a population mean or proportion.
- ii Specifying the probability level that is sufficiently small to provide reasonable evidence against the null hypothesis value.
- iii Whether concern is with deviation on one direction or both directions.
- iv Calculation of a critical ratio or the test statistic which will lead to the calculation of the chance that random sampling from the population would produce a sample mean as deviant or more deviant than the mean observed.

Test Of Significance On A Mean.

Test of significance always refers to a null hypothesis. It answers the question: Is chance or sampling variation a likely explanation of the discrepancy between a sample result and the corresponding null hypothesis population value.

In this case, the null hypothesis will be a statement that claims a particular value for a population mean. The next step will be to calculate the chance that random sampling from such a population would produce a sample mean as deviant or more deviant than the mean observed. i.e. (p value).

The p-value corresponding to the (critical ratio, T-value, z value, Chi square value can be read from the appropriate statistical tables. The formulae and the steps are all in most statistics books and these statistics can be given at the touch of a bottom.

If the null hypothesis were true, (i.e. not significant, the following interpretation could be given)

- i. The observed result could well arise purely by chance
- ii No reason to doubt the validity of the null hypothesis. The data failed to provide sufficient evidence to doubt the validity of the null hypothesis. Not enough evidence to contradict the null hypothesis.
- iii We have to live with the null hypothesis until further evidence is obtained.

- iv The results do not provide sufficient evidence to doubt the null hypothesis.

A statistically significant result merely rules out chance alone as explaining the difference between the observed and the null hypothesis value. A result may be highly statistically significant, yet *medically unimportant*.

chi-square Test

For the comparison of two proportions, the chi-

square statistic provides an alternative method in both paired and independent samples. The hypothesis can be tested by comparing the observed frequencies to what would be expected if the hypothesis were to be true. When the data are displayed in a contingency table, the expected frequency for each cell is obtained by the expression (Expected = Row Total x Column Total/Grand Total). The chi-square is given by the sum of $(O-E)^2/E$ for all the cells, where 'O' is the observed frequency and 'E' is the expected

Table 3

Common Test Statistics	Situation	Example
Student's T-test	For group data	Mean haemoglobin level in mothers who attended antenatal clinic versus non attendant
Student's T-test	For paired data	Change in haemoglobin levels after iron supplementation
Z-score	For proportions	Proportion of
Chi square Test	Categorical variables	Association of antenatal attendance and outcome of labour
F- test for analysis of variance ANOVA	Comparison of means in more than two groups	Mean haemoglobin levels in urban, peri-urban and rural mothers
Correlation coefficient	Strength of association	Blood sugar levels and systolic blood pressure level
Regression analysis		To determine mathematical relationship between two variables

Common non-Parametric methods

Non parametric methods	Parametric methods
Sign test	Paired t-test
Wilcoxon signed rank test	Paired t-test
Wilcoxon rank sum test	T- test for group data
Mann Whitney U test	
Kendal's sign test	
Speaman's rank correlation	Pearson's correlation

frequency. The chi-square is an approximate method, and for validity, the expected frequency in each cell must be at least 5. If not one has to consider Fisher's Exact test methodology

Investigating Relationships Between Variables

Regression and Correlation

These are statistical techniques used to examine the association i.e. for investigating and quantifying the relationship between two quantitative variables. Regression in e.g. dose-response relationship studies, investigates the change in the mean value of the dependent variable as the independent variable changes. What happens to Y as X changes? Many medical and social investigation centre on the establishment of a relationship between two variables. e.g. Birth weight and parity, dental caries and sugar intake.

Correlation: Both variables are termed dependent. It is the study of the strength of the association. Quantification of the degree to which the two variables tend to relate. Do blood pressure and age tend to be related?

Regression: Mathematical model of relationship between two quantitative variables. It concerns with fitting a straight line to a scatter of points of (x,y).

Before the analysis, it is important to plot scatter diagram, which helps to visualize the relationship. It is customary to denote the abscissa as X and the Y-axis as the ordinate. The eyeball fit lacks objectivity. Different observers will fit different line and even, the same observer when confronted with the same scatter, may fit different lines. It is also not possible to calculate the sampling variation.

The best method of estimating the parameters of the line is the use of method of LEAST SQUARES. This helps to provide a mathematically derived best-fitting line, which is unique and therefore possible to develop methods of statistical inferences with respect to the parameters. It makes sure that the sum of squares of deviation of the observed points about the line is minimum.

International Classification Of Diseases (icd)

Disease classification involves the arrangement of disease entities into categories, which share similar features. It is a prerequisite of epidemiological

study as well as one of its goals.

It aims at comparability in the methods of presentation of mortality and morbidity data from different sources. The International Classification of Disease is produced to be of interest to the medical statistician and also be an instrument for collecting information into a common pool of knowledge. The main purpose of the present International classification of diseases is to provide a list of disabilities for compiling statistics and not a nomenclature of diseases.

Death Certificates

There is the need to have a uniform selection of the cause of death. Frequently, in both sickness and death, more than one morbid process is involved. More than 50% of medical certificates of deaths and hospital records of illness and disease contain more than one cause. It is agreed that the cause to be tabulated must be the underlying cause of death as stated by the certifying physician.

Health Indicators

Health evaluation is an important component of the managerial process for National Health Development. Evaluation is a very important managerial tool that allows for feedback and reprogramming.

Evaluation will involve measurement of impact and effectiveness of programmes as well as efficiency of programmes. Indicators measure the extent to which targets are being reached.

Targets are expressions of desired service performance, for example, desired output or coverage to be achieved at some time in the future.

Indicators are variables that help to measure changes directly or indirectly to determine the extent to which targets are being reached and to assess impact and effectiveness of health programmes and to provide information for programming and re-programming of health activities.

Examples of hospital health indicators

Percentage of births attended by skilled health personnel

Definition: Percentage of births attended by skilled health personnel (excluding trained or untrained traditional birth attendants).

Skilled health personnel refer to doctor (specialist or non-specialist) and/or persons with midwifery skills who can manage normal deliveries and diagnose or refer obstetric complications. Both trained and untrained TBAs are excluded.

Bed turnover:

Definition: The average number of patients managed per bed in a given period, i.e. discharges and deaths divided by available beds.

Turnover interval:

Definition: The average number of days beds lie vacant between successive patients, the number of vacant bed-days divided by number of discharges and deaths.

Average length (duration) of stay:

Definition: Average number of days patients occupied a bed during admission, total occupied bed-days divided by number of discharges.

These indicators are useful in evaluating the performance of hospitals.

The use of computers in statistics.

Statistical packages are available to produce statistical analysis on data generated in obstetric practice as well as ad hoc research. Most common statistical packages include EPIINFO, SPSS, and EXCEL. The packages can be used to produce all the statistics discussed in the above chapters and for the development of indicators.

A

Abdomen(abdominal)..... 1, 16, 22, 77, 79, 87, 88, 108, 117, 136, 163, 211, 222, 226, 231, 238, 353, 356, 357, 359

Abortion 11, 28, 32, 226, 227, 243, 289, 324
 Acidosis(acid)..... 77, 105, 108, 221, 365
 AIDS..... 11, 38, 40, 41, 42, 373, 374
 Allergens (allergic)..... 117, 205, 361
 Alpha-feto protein..... 3, 4, 12, 17, 146, 188
 Amenorrhoea..... 35, 83, 106, 380, 299, 363
 Amino acids..... 61, 186, 361
 Amniocentesis..... 1, 2, 3, 30, 146, 178
 Amniotic..... 3, 20, 30, 61, 86, 143, 179, 195, 345

Anaemia..... 3, 4, 10, 11, 12, 17, 55, 87, 89, 115, 163, 226, 382, 255, 253, 261, 262, 265, 297, 298, 303, 304, 307, 345

Anaesthesia..... 5, 68, 79, 80, 88, 93, 97, 99, 159, 222, 308, 324, 344, 348, 353, 354, 357

Analgesia..... 64, 65, 81, 93, 126, 306, 308, 343, 353,
 Anatomical(anatomy)..... 32, 86, 179, 261, 332, 352, 353, 354

Antenatal..... 1, 3, 4, 7, 13, 15, 18, 19, 20, 28, 29, 42, 77, 83, 96, 138, 303, 304

Antepartum..... 15, 18, 23, 116, 137, 140, 142
 Antibiotic..... 68, 88, 89, 123, 154, 238, 264, 353, 354, 358, 360

Antibodies..... 11, 12, 84, 360
 Apgar..... 96, 394
 APH..... 96, 394
 Arteries 18, 28, 82, 117
 Asphyxia..... 5, 84, 101, 107, 108, 123, 144, 160, 194, 222, 344, 365

B

Bladder..... 79, 80, 84, 87, 89, 123, 144
 Blastocyst..... 162
 Bradycardia..... 106, 147, 367
 Bronchial..... 17, 22

C

Caesarean Section 19, 40, 54, 70, 80, 87, 98, 100, 101, 101, 108, 113, 122, 123, 142, 159, 195, 222, 225, 249, 321, 325, 324, 327, 336, 356

Calories..... 54, 56, 289, 360
 Cardiac 4, 10, 17, 22, 33, 54, 93, 98, 194, 268, 270, 289, 345, 356, 363

Cardiotocograph..... 20, 106, 138, 159, 222
 Cardiovascular..... 20, 36, 174, 185, 194, 225, 268
 Catheter..... 78, 79, 80, 209, 357
 Cephalo-pelvic..... 68, 77, 111, 133, 224, 322, 343
 Cephalosporin..... 54, 88, 264

Cerclage..... 98, 334, 335, 336
 Cerebral..... 21, 22, 29, 102, 252, 354
 Cerebrovascular..... 221, 381
 Cervical..... 12, 59, 62, 63, 87, 98, 140, 198, 211, 226, 330, 332, 348, 359, 382

Chignon..... 347, 345
 Chloroquine..... 253, 254, 255, 264
 Chorioamionitis..... 152, 194, 363
 Chorionic (Chorion)..... 2, 3, 4, 30, 36, 205
 Chromosomal..... 1, 2, 3, 6, 12, 187, 195, 228, 229

Chronic..... 17, 55, 83, 174, 194, 302
 Coagulation..... 97, 146, 174, 195, 253, 358

Contraceptives..... 44, 307, 371, 372, 375, 376, 380, 385, 387, 388, 391

Cord..... 84, 108, 158, 208, 343, 350, 352
 Corticosteroids..... 4, 6, 34, 143, 147, 154, 239,
 Cotrimoxazole..... 80, 264
 Cyanotic..... 17, 22, 174
 Cystic..... 80, 263
 Cytokines..... 61, 151, 250

D

Dehydration..... 68, 77, 206
 Diabetes mellitus..... 10, 11, 12, 30, 110, 113, 221, 289
 Dyspoea..... 33, 269
 Dystocia 1, 4, 54, 66, 110, 112, 222, 313

E

ECG..... 99
 Eclampsia 54, 68, 89, 93, 99, 173, 175, 178
 Ectopic..... 7, 28, 86, 127, 211, 226, 243, 261
 Electrolyte..... 78, 88, 94, 119, 181
 Embryo..... 32, 42, 162
 Endocrine..... 6, 34, 61, 83, 106, 119, 126, 173, 187, 205, 230

Endometrium..... 28, 86, 103, 228, 381, 391
 Endotracheal..... 99, 126, 127, 364
 Enzyme..... 9, 12, 33, 60, 357, 361
 Ephedrine..... 97, 101
 Epidural..... 65, 98, 100, 118, 119, 125, 121, 127, 344
 Episiotomy..... 40, 65, 66, 79, 80, 99, 101, 102, 115, 140, 343, 353,

Erythroblastosis 4, 20, 30, 355
 Estrogen (see oestrogen)

F

Fallopian..... 211, 257, 392, 454
 Family(see planning)..... 215, 307, 371, 372, 373
 Febrile..... 307, 371, 373

Fetal (fetus)..... 1,5,13,17,18,20,
23,24,25,26,27,28,32,36,56,59,61,62,65,66,78,80,87,1
08,112,123,136,178,208,289,322,343,350

Fistula..... 81,82,83,354
Forceps..... 78,86,90,343,344,345,35
0
Fundus..... 149,151,385

G

Gestation..... 2,8,12,15,18,19,20,27,28,99,100,110,1
36,137,134,162,173,195,198,304,357

Glycosuria..... 17,32,36,261,378,378
Grand multipara..... 77,79,101

H

Haematoma..... 86,146,181,315,348,353
Haemoglobin..... 8,11,5599,13,143,292,303
Haemolysis..... 175,253,256,303,304,305,370
Haemorrhage..... 3,66,68,84,87,116,118,140,142,
243,343,356,361

Hepatitis..... 11,13,43,101,388
Heparin..... 60,194,306,360,371
HIV..... 12,38,42,373
Hormone..... 1,2,4,220,317,358,361,382,387
Hypertension..... 5,8,10,12,17,20,30,94,119,
173,188,222,261,363

Hypoxaemia..... 17, 21,22,28,65, 105
Hypoxia..... 22, 15,19,21,22,65,133,138

I

Iatrogenic..... 15,19,21
Immunologic..... 174,251,304,306,368
Incision..... 101,224,322,323,352,353
Insulin..... 98,221,287,288,289,295
Intrapartum..... 7,15,17,89,137,418
Intrauterine..... 10,17,27,40,121,123,185,211,257
Intravascular..... 146,147,253,307
Intravenous..... 116,133,180,194,209,146
Ischaemic..... 118,148,175,180
IUD..... 375,379,385,388,389,391
IUGR..... 19,20,2123,,25,30,185

J

Jaundice..... 12,253,307,359,391

K

Karyotype..... 2,3,17,23,28,240,277
Ketacidosis..... 77,289,136
Ketamine..... 97,99,100,123

L

Labour..... 76,77,131,137,222,243,268,359
Laparoscopy..... 88,211,214
Laparotomy..... 112,117,214,359
Laryngoscope..... 77,99,364
Leukemia..... 10,14,299
Lochia..... 357,358,359

M

Macrosomia..... 8,11,77,110,123,222,350
Malaria..... 12,16,17,19,55,187,228,
250,252,363,396

Meconium..... 87,105,110,133,137
Membrane..... 62,63,86,133,155,208,372
Midwives..... 10,35,245,306
Myometrium..... 28,37,60,63,433,480

N

Naloxone..... 95,98,120,365
Narcotics..... 95,121,122
Nausea..... 32,33,122,120,135,205,264
Necrosis..... 83,103,254,265,304
neonatal..... 1,8,41,68,137,140,155,289,
314,350,363,314

neurologic..... 173,181,206
Neuromuscular..... 95,96,123,182
Norplant..... 383,385
nulliparous..... 83,105,133,174

O

Obesity..... 3,56,140,219
occipito-posterior..... 77,345,350
Oedema..... 12,77,158,173,221,253,269,
353,354,359

Oestrogen..... 32,34,61,205,307,357,381,385,385
Oligohydramnios..... 3,4,20,22,27,30,107,
138,158,233

Oliguria..... 22,28,148
Ovarian..... 77,140,211,214,378,382
Ovulation..... 135,382,385,391
Ovum..... 28,211,233,258
Oxygen..... 55,99,101,136,308
Oxytocin..... 60,66,70,79,133,136,194,389

P

Paracentesis..... 213
paralysis..... 83,113
Parasite..... 17,23,187,250
Partograph..... 50,64,68,69,83,106,108,136
Pelvic..... 40,83,110,124,328,323
percreta..... 86,110,116,117,145
Perinatal..... 15,20,28,38,87,93,110,112,
140,159,222,229,287,305

peripheral.....22,54,83,100,374
 peritonitis.....89,111,113
 Pethidine.....65,80,95,102,120,121
 placenta.....23,30,59,66,86,94,115,
 140,163

 Plasma.....54,61,105,180,312,383,386
 Pneumonia.....264,303,305,306,447

 Polyhydramnios.....3,4,30,37,116,104,146,208,
 232,147

 Postpartum.....55,66,68,88,115,356,362
 Praevia.....18,23,116,140,187,323
 Pre-eclampsia.....17,20,173,179,374
 Preterm.....4,7,8,15,27,146,160,198,208,525,352
 Progesteron(e).....32,33,61,223,307,372,391
 Prolapse.....84,108,208,328,345,350,354
 PROM.....151,154,359
 Prophylaxis.....12,17,19,78,101,188,255,373
 prostagladin.....61,146,174,194
 Proteinuria.....380
 Prothrombin.....34,116
 Protozoa.....7,11,12,250
 Puerperium.....16,32,88,112,123,252,
 308,356,359,362

 Pulmonary.....5,17,33,143,253,256,345,363
 Pyrexia.....89,228,255,358,359

R

Rectus.....352
 Renal.....17,33,175,265,312,314,315,363,378
 Resuscitation.....42,78,88,95,101,365
 Retardation.....5,6,12,23,30,40,141,199,200,269,363
 Rhesus.....10,12,16,30,36,101,143
 Rheumatism.....268
 Rubella.....10,11,12,17,80,84,86,89
 Ruptured.....62,64,66,68,77,133,211,226

S

Serum.....3,12,18,27,55,65,213,264,299,307
 SGA.....22,23,185
 SHF.....23
 Shirodkar.....125,334
 Sickle cell.....68,100,194,303,304,305,389
 Spinal.....95,100,101
 streptococci.....151,359
 STD.....373
 Sterilisation.....357,385,454
 steroids.....12,61,143,154,178
 subcutaneous.....54,224,324,357
 Succeddaneum.....77,345
 Suxamethonium.....90,99,124,126,126
 Symphysis.....18,19,63,68,79,80,84,136
 Syndrome.....3,93,97,99,306
 Syntocinon.....28,90,431
 Syphilis.....12,17,39,43,187

T

Tachycardia.....106,143,253
 Thalassaemia.....251,303
 Thrombosis.....80,223,307,391
 Thyrotoxicosis.....178,319
 Thyroid.....220,317,345
 Tocolysis.....19,20,143,148,154,199
 Transabdominal.....105,136,336,357
 Trimester.....3,8,12,27,83,135,163,198,264
 Troboplastic.....4,28,37,116,110,149,200,214,
 258,381

U

Ultrasound.....2,3,18,20,23,26,28,30,142,208,213
 Umbilical.....116,133,136,157,324,352,357
 Uterine Vessels.....3,5,15,
 23,59,62,63,87,122,336,357,389

V

Vaccine.....357
 Vasculopathy.....141,285
 Vasectomy.....308,388
 Ventouse.....344,346,350
 Virus.....456

X

X-ray.....125,270,359