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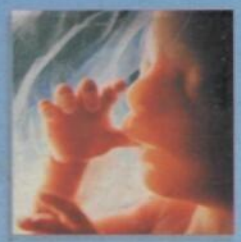
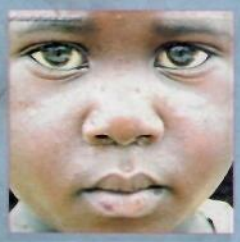
Child Health Edition

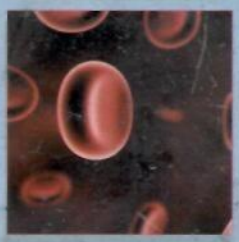













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- Mental Health; The Forgotten Component Of The Child And Adolescent Health.
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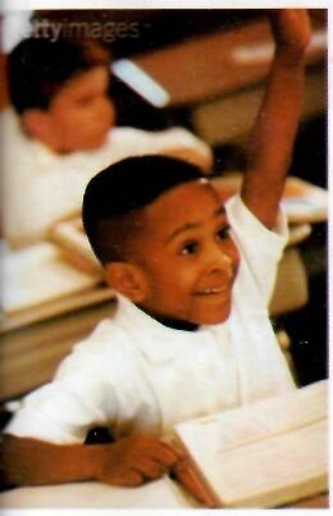

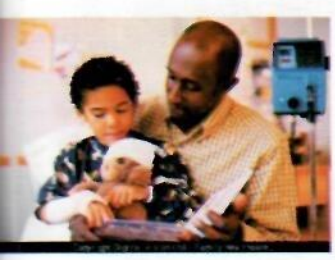

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EDITION

JOURNAL OF UNIVERSITY OF IBADAN MEDICAL STUDENTS' ASSOCIATION

1960

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3. An abstract/summary of 150-250 words stating the objectives, methods, results and conclusion of the paper. Below the abstract, list in alphabetical order three to eight key words for cross indexing using terms from the medical subject headings (MeSH) list of index medicus.
4. Text
5. Acknowledgement
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7. Tables
8. Legends for illustrations

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References are numbered consecutively in the order in which they are first mentioned in the text. Arabic numerals in parenthesis are used. Abbreviations for journal titles should be those adopted by the *index medicus*. 'Unpublished observations and 'personal communications' may not be used as references but may be inserted (in parentheses) in the text. Papers accepted but not yet published may be cited with the journal designated and 'in press' added. Use the style of examples given below. (Note the punctuation and spacing).

1. For standard journal articles: author's name(s) initials, title of article, name of journal, year of publication, volume in Arabic numerals (with number in parentheses) and the number of the first and last pages. E.g. Akinkugbe O. O. Nephrology in the tropical setting. Nephron 1978; 22:249-252. If there is more than one author, list all except they exceed four wherein four are given, followed by et al.
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FOREWORD

I write this foreword for the "CHILD HEALTH EDITION" OF **DOKITA**, with the belief that the use of information contained in the articles will impact positively on the health of Nigerian children.

There are 23 well written articles in this edition, which deal with emergency paediatrics, neonatal medicine, infectious diseases, oncology & haematology, genetics, adolescent medicine, paediatric psychiatry, anaesthesia, radiology and social paediatrics. I was particularly delighted to see adolescent medicine being addressed, because this is somehow neglected in Nigeria.

The article on *Physical Growth and Its Assessment in Children and Adolescents* gives an overview of the important topic: growth. This subject is particularly important because lack of "growth" may be a pointer that something grave is happening to a child.

To achieve reduction in child mortality in Nigeria, it is paramount that reduction of neonatal mortality rate should be vigorously pursued. This opinion stands squarely on the indisputable fact that 40% of the 10 million under-5 deaths recorded annually all over the world occur during the neonatal period. The Millennium Development Goal-4 focuses on reducing under-5 mortality rate by two-thirds of the 1990 figures by 2015. Hence, it is logically sound that emphasis on significant reduction in neonatal mortality in developing countries will drive this goal. This is the reason why the article on: *Neonatal Mortality in Developing Countries A Call to Action* is timely, for it addresses among other important issues, the way forward to reducing neonatal mortality in Nigeria.

The elimination of neonatal tetanus in Nigeria has remained a mirage. Various reasons were adduced to explain this gloomy situation. It may well be that there is a flaw in the way we employ the time tested strategy that is useful in the control of this disease. That this might be the case is revealed in the realization that nine countries in Africa have achieved Maternal and Neonatal Tetanus elimination as at September 2007. The strategies of immunization of *child bearing age women* and of pregnant women, taken together with promotion of more hygienic deliveries which ensured the elimination of the disease in the nine African countries should be applicable in Nigeria.

The campaign against harmful socio-cultural practices has received a boost through an article in this edition. Of no lesser importance are the discussion of the problems of low frequency of initiation of breastfeeding of newborns, the peculiarities of the paediatric patient for the safe practice of anaesthesia, as well as the health and social problems of children in an institution for motherless babies in Ibadan.

One of the easily forgotten health issues is the oral health care. Yet, good oral health is important for child's growth, development and self-esteem. The role of mothers in preventing caries development and maintenance of good health is well addressed in this edition.

Sickle cell anaemia (SCA) is the commonest genetic disease in Nigeria. The most significant problem is anaemia. The management of SCA has been clearly defined, and the article on *Transfusion Requirements in Sickle Cell Disease* is a useful addition in this regard.

This foreword must remain a short piece of writing which introduces the contents of this edition of **DOKITA**. Surely, there can be no substitute for the effort of each individual reading through all the articles and grasping fully all the contents. I wish everybody an informative reading with this edition of **DOKITA**

A.G. Falade
Professor of Paediatrics & Head, Department of Paediatrics

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THE ART OF DESIGN

Editorial

DOKITA, the peer-reviewed journal of the University of Ibadan Me Students' Association, has through the years been a paragon of exce maintaining, still, the reason for its existence. The forty-eight-year old Ed Board was, at inception, founded to produce **DOKITA** which was to serv medium for the publication of research works originating from this sch Medicine and also to improve on our writing skills. However, over the yea Board has evolved to include other scientific programmes like the A Symposium, the Emeritus Professor O. O. Akinkugbe Biennial Interm School Quiz competition and the Professor J. A. Adeleye Biennial National Competition. A tune of excellence is what we strike at any of these progra because by the work, one knows the workman!

Though primarily a medical-student journal, **DOKITA** has enjoyed, and it enjoying, contributions from specialists and professionals in the fi medicine. In our bid to making sure that the journal is widely read and accessed outside the shores of Africa and beyond, the journal is presently through a process of peer-reviewing which will ultimately ensure th journal is accessible and can be indexed on pubmed and other on-line datab

Child Health, in its real sense, starts from the pre-conception period and it is to include genetic couns prenatal screening, and covers the pregnancy period through delivery until the child is nurtured to adulth represents a major part of the developing child as this period is notorious for its vulnerability to insults- orga psychological. The period is one of great joy for the parents, but most times, this season of joy is cut sh childhood-killer diseases especially in tropical Africa.

From the foregoing, you will quite agree that a journal dedicated to these tender beings is necessary fro Editorial Board, most especially as the Board has not published any journal that wholly discusses the issue o Health.

The journal is a splendid collection of articles as we hold it in our very hands. In it, you will find articles on the of neonatal mortality, common paediatric surgical conditions, assessment of physical growth in children, pae anaesthesia, sickle cell disease, critical care of the premature child, child oral health, mental health, infa diabetic mothers, millennium development goals amongst others testifying to the rich nature of this Expectedly, the winning essay of the Professor J. A. Adeleye Biennial National Essay Competition titled '*Change: a ticking time bomb*', and the best student-project in the Ibarapa Community Health posting for th graduating class have been included in the journal. And, of course is the '**DOKITA** extra' section which y definitely find worth going through again and again. All these have been put together in a professional sty have no doubt that this issue will be found interesting by those who take an interest in it.

The Editorial Board is highly indebted to members of the Advisory Council, our Emeritus Consultant a Honorary Board member, and our Editorial Consultants, whom I am protégé to, for their assistance towa publication. Their immense contributions have ensured a better journal for us all to read. My gratitude also g Professor A. G. Falade, Head of Paediatrics Department, University of Ibadan for taking time to write the fore this journal and to all our dear Consultants who assisted in reviewing most of the articles in this issue.

This wonderful edition will not have been possible without the selfless input of the disciplined, dedicat diligent members of the Editorial Board most especially Miss Ayobami Olokode and Mr. Abayomi Oyenu Production Managers that worked tirelessly during the production of this edition.

We appreciate all our sponsors for their kind financial assistance towards this journal. And to God Almighty, all thanks for carrying us through the Board and for the wisdom and strength to combine the burdensome this publication with our primary goal in school as medical students.

Most of all, we are grateful to ourselves for being part of the success story of one of the beautiful and notable to have come out of the Ibadan Medical School- the **DOKITA** journal.

Ajayi Temitope Omotayo
Editor-in-Chief,
February 2008

SHAKEN BABY SYNDROME: A NEUROLOGICAL NIGHTMARE

Oluleye, Oludamilola Wuraola, Final Year Medical Student, College of Medicine, University of Ibadan.
Iwabejire, John Ovuakporaye, M.B; B.S (Ibadan); Corps Medical Officer (NYSC), General Hospital, Bunza, Kebbi State.

...giver, out of anger or frustration vigorously shakes or drops a crying child, the child is immediately put at risk of ...a potentially disabling or even lethal brain injury called the shaken baby syndrome (SBS). The mortality rate of this ...is relatively high, with about one in four affected children dying from it.

...ity of data on the incidence of this condition in Nigeria should not be taken to mean that it is not common. It may well ...is not being seen commonly either because health care givers have not been looking actively for it or many are not ...with the diagnostic criteria.

...idents of Shaken Baby Syndrome are not reported out of fear. It is important to seek immediate and early medical ...as serious complications and even death can be avoided.[9]

...potential SBS, parents and caregivers of infants need help with responding to their own stress.[4]Including talks on the ...associated with shaking an infant and how it can be prevented in the health education sessions in the routine antenatal ...(C) visits would go a long way in preventing this disorder.

...Shaken baby, Syndrome, Prevention, Neurological Deficits, Caregiver.

...CTION

...en baby syndrome (SBS) is a severe form of ...brain injury that results when a baby is vigorously ...It is also known as the shaken impact syndrome ...first described as the *"whiplash-shaken infant ..."* by Caffey in 1974.[1,2] The current definition ...a child who presents with altered consciousness, ...hemorrhages, and evidence on computed ...phy or magnetic resonance imaging of ...spheric subdural hematoma or subarachnoid ...ge.[3] It is produced not only by violently shaking a ...also by throwing or dropping, even onto a very ...face such as the child's bed, usually out of ...ion or anger by the caregiver, often because the ...won't stop crying.[3] As shaking is one of the methods ...suggested by care givers to make a crying baby quiet, ...without saying that numerous babies round the ...Nigeria inclusive, are at risk of this form of severe ...cerebral brain injury.

...advanced countries, shaking a crying baby ...is a form of child abuse and law suits have ...as a result of infant deaths attributable to the SBS. ...ity of the problem is reflected in the drastic steps ...taken by the government and people of many ...ed countries to address it: appropriate legislation, ...ic campaigns, family support, establishment of ...tion programmes and provision of research ...major medical institutions with a view to finding ...ays to treat the condition.[5] However, in Nigeria, ...still exists widespread ignorance of the ...ences of violently shaking a baby, as reflected in ...ce of local data on the subject. Consequently, we ...reading for an epidemic of grave neurological ...in our children, these being nicely labeled ...NCD when the cause is preventable.

EPIDEMIOLOGY

The incidence of shaken baby syndrome is much higher than is reported in the literature. It is estimated to account for about 10% of injuries to children less than 2 years of age. [6,7]The majority of cases occur before the children's first birthdays, with the average age being between 3 and 8 months.[4]

In America every year an estimated 1,200 - 1,400 children are shaken for whom treatment is sought. Of these tiny victims, 25 -30% die as a result of their injuries. The rest will have lifelong complications.[8]Some authorities feel strongly that it is likely many more babies suffer from the effects of SBS than is reported, since the SBS victims rarely have any external evidence of trauma.[8]

The caregivers involved are mainly males either the baby's father, or mother's boy friend, though female baby sitters or the mother could be involved especially if they are experiencing some form of stress.[9]

There is generally a dearth of data on the prevalence of the SBS in Nigeria. Since the SBS shares many clinical features and complications with cerebral palsy, it is possible that many cases may have being misdiagnosed as the later condition. However, studies are needed to confirm this speculation.

AETIOPATHOGNESIS

The distinct anatomy of babies puts them at risk of SBS. This includes their soft brain, weak neck muscles and ligaments and relatively large, heavy heads in proportion to their bodies.[4, 5,9]The head makes up to 25% of the baby's total body weight. Thus the neck muscles are too weak to support such large head.

When a baby is shaken violently, the brain bounces back and

forth against the skull (acceleration-deceleration injury). This can cause bruising of the brain, cerebral swelling, and intracerebral hemorrhage, resulting in an acute rise in intracranial pressure. The force of the head movement can tear blood vessels that bridge the brain and skull (bridging veins), because they are fragile and immature, resulting in an acute subdural hematoma and further increase in intracranial pressure. Permanent, severe brain damage or death can easily result.

There is also associated retinal hemorrhage, a feature that is almost pathognomonic of this type of acceleration-deceleration injury. [10]This underscores the importance of performing a complete neurological (including ophthalmological) examination in suspected cases.

CLINICAL FEATURES

The clinical presentation will depend on the duration and force of the shaking, the number of episodes, and whether impact is involved.[4]In the most severe cases, loss of consciousness, seizures or shock may be the presenting features. Affected patients may not even be brought to the hospital in the absence of such severe clinical manifestations.[4]

In less severe cases of the SBS, the early warning symptoms include lethargy, irritability, vomiting, poor sucking or swallowing, decreased appetite, lack of smiling or vocalizing, rigidity, seizures, difficulty with breathing, altered consciousness, inability to focus the eyes or track movement and inability to lift the head.[4]

Physical examination must include a thorough general and systemic examination, particularly of the neurological system. Skin pallor, tense anterior fontanelle, lacerations and soft tissue injuries may be seen on general examination. Rib, long bone and skull fractures may be evident; poor respiratory efforts, bradycardia and raised blood pressure (features of increased intracranial pressure) may be present.

Head control, level of consciousness, sucking, crying, pupillary function, and reflexes are some of the most important neurological signs to evaluate.[10] Head control may be lost, level of consciousness reduced, pupillary sizes unequal and reflexes depressed.

INVESTIGATIONS

The cardinal investigative modality in any child suspected of having the SBS is the computerized tomography (CT) scan or magnetic resonance imaging (MRI) of the brain. Either of these may reveal an interhemispheric subdural hematoma or subarachnoid hemorrhage. In the absence of these imaging modalities and where a strong clinical suspicion of subarachnoid haemorrhage exists, a lumbar puncture should only be carried out when a raised intracranial pressure has been excluded!

A chest x-ray may also be done to rule out rib fracture, as may x-ray of an extremity if there is clinical suspicion of a fracture

MANAGEMENT

Initial management of the patient with the SBS depends on the presentation. For the unconscious child, endotracheal intubation must be performed to protect the airway and provide adequate ventilation.[10]An anti-seizure medicat should not be given to an obtunded child who has a seiz in order to prevent further decrease in ventilation, incre in P_{CO2}, and increase in intracranial pressure, with ris herniation at the tentorial notch or the foramen magn Instead, the affected child should be anaesthetized wit endotracheal tube in place.[10]

Prior to having a cranial CT scan, a subdural tap ca performed if a child is noted to be in coma, decerebrate posturing or fixed, dilated pupils and a fontanelle. This often causes an immediate decreas intracranial pressure and improvement in the neurolo status.[10]Careful maintenance of intracranial pressur below 15- to 20-cm H_2O may obviate the need for fu surgery.

Since the SBS usually involves total brain injury, *development of language, vision, balance, and m coordination, all of which occur to varying degrees birth, is particularly likely to be affected in any surv child who has had the condition.*[4]Thus, rigorous ph and occupational therapy are usually required to he child acquire skills that would have developed on thei had the brain injury not occurred. Therapists do th providing a sensory-rich environment, which force child to be attentive. Special educational services ma required early in the lifetime of the child in orde improve educational outcome.

PROGNOSIS AND LONG TERM CONSEQUENCES

The mortality rate in children with the SBS who pre unconscious is 25-30%.[8, 10] 30 to 50 per cent show se cognitive or neurological deficits, while the remainder a chance of full recovery.[10]

The long term cognitive/neurological deficits numerous and include partial or total blindness, hea loss, seizure disorders, delayed developmental milest impaired intellect, speech and learning difficu impaired memory and attention deficits, severe m retardation and paralysis. *Herein lies the origin of the t this article:* ***"Shaken Baby Syndrome: A Neurolo Nightmare"****, the innocent, harmless child being plu into a nightmare by a caregiver; one he did not barga and from which he may never recover!*

PREVENTION

The SBS is one condition in which prevention is cheaper and far better than cure, as a "cure" hardly for it.

One of the major strategies for prevention is to increas level of awareness of the populace on the dange shaking a baby.[4]Programmes aimed at helping new pa identify and prevent shaking injuries as well as respon appropriately when infants cry are also useful.

Finding ways to alleviate the parent or caregiver's str the critical moments when a baby is crying can signific reduce the risk to the child.[4] One method that may h Dr. Harvey Karp's "five S's"[4]:

shing (using "white noise," or rhythmic sounds that c the constant whir of noise in the womb, with things acuum cleaners, hair dryers, clothes dryers, a running or a white noise CD)

stomach positioning (placing the baby on the left side help digestion - or on the belly **while holding him or** then putting the sleeping baby in the crib or bassinet **is or her back)**

ing (letting the baby breastfeed or giving the baby a er to suck on)

dling (wrapping the baby up snugly in a blanket to him or her feel more secure)

ging gently (rocking in a chair, using an infant swing, ing a car ride to help duplicate the constant motion aby felt in the womb).

following can also be tried if a baby in one's care will stop crying[5]:

e sure the baby's basic needs are met (for example, he e is not hungry and does not need to be changed).

k for signs of illness, like fever or swollen gums.

or walk with the baby.

or talk to the baby.

the baby a pacifier or a noisy toy.

the baby for a ride in a stroller or strapped into a child seat in the car.

the baby close against your body and breathe calmly slowly.

a friend or relative for support or to take care of the while you take a break.

thing else works, put the baby on his or her back in the close the door, and check on the baby in 10 minutes.

your child's doctor if nothing seems to be helping your et, in case there is a medical reason for the fussiness.

NCLUSION

shaken baby syndrome is indeed a neurological mare for babies and is completely preventable. It is ortant for parents to realize (and let anyone caring for babies know) that it is never okay to shake a baby! To a moment of frustration, anger or carelessness to cut a child's dream (of course, children too have dreams!) aking is never an option. The sooner we realize that is a problem that may be very common in our ronment but has not been given the attention it rves, the better for us.

e care givers should have a strong index of suspicion especially when for a child with the above clinical ses, there is either no history of injury or an propriate history for the degree of injury ewed. This should reduce the rate of misdiagnosis or r diagnosis, especially if ophthalmoscopy is not seen exclusive preserve of the ophthalmologist (which is usual attitude of most none-ophthalmologists, ularly in a teaching hospital setting in this country) the CT scan becomes readily available and affordable.

Managing affected children with the SBS and other challenging neurological disorders, particularly those which require physical and occupational therapy should also be given its rightful place in this country. It is the duty of members of the medical profession to mount pressure on the government to provide the appropriate resources for training such therapists and the materials they need to work with, for *"The principal objective of the medical or dental practitioner shall be the promotion of the health of the patient...."*[12]

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INFANTS OF DIABETIC MOTHER REDUCING MORBIDIT AND MORTALI

By Sobukunola, Oluwadunni E, 500 Level MBBS, College of Medicine, University o
Hwabejire, John Ovuakporaye, M.B; B.S (Ibadan); Corps Medical Officer (NYSC), General Hospital, Bunza, Ke

ABSTRACT

Background: *Lack of routine screening for diabetes mellitus in pregnant women and frequent hyperglycaemic ep mainly responsible for the high morbidity and mortality observed in infants of diabetic mothers (IDMs). This revie discuss the factors responsible for morbidity and mortality in IDMs and to suggest ways of modifying them.*

Method: *A review of relevant literature on the subject was sourced manually and by internet search. The following were used for internet search: Diabetes mellitus, Diabetic mothers, Gestational diabetes mellitus, Infants of diabetic*

Results: *Gestational diabetes mellitus is the most common form of diabetes in pregnancy. Fetal macrosomia, birt congenital malformations, prematurity and neonatal hypoglycaemia are some of the morbidities IDMs suffe antenatal screening for DM in all pregnant women and adequate ante partum glycaemic control in diabetic m some of the ways to reduce morbidity and mortality in IDMs.*

Conclusion: *The effects of maternal conditions on fetal well-being is exemplified by maternal diabetes mellitus in p early detection and adequate control of maternal diabetes mellitus before and during pregnancy, however, give outcomes- reduced morbidity and mortality.*

Key words: *Diabetes mellitus, Diabetic mothers, Gestational diabetes mellitus, Infants of diabetic mothers.*

INTRODUCTION

Diabetes mellitus is a primary metabolic disorder of glucose homeostasis characterized by persistent hyperglycaemia. It results from either an absolute (type 1) or relative (type 2) deficiency of insulin which is an anabolic hormone that promotes the uptake of glucose, storage of glucose as glycogen, lipogenesis, and uptake and utilization of amino acids.[7]

Both pregnant and non-pregnant women could be affected by diabetes mellitus; peculiar to pregnancy, however, is gestational diabetes mellitus which pregnancy-induced. Pre- existing diabetes mellitus (type 1 or 2) is also a cause of morbidity and mortality for both mother and child.

Pregnancy is diabetogenic because there are increased levels of diabetogenic hormones (e.g. human placental lactogen, cortisol and growth hormone) and their diabetogenic property is most marked in the 3rd trimester of pregnancy. The increased secretion of insulin by the pancreas however counteracts the action of these diabetogenic hormones in normal pregnancies.[4]

In pregnancy, glucose crosses the placenta by facilitated diffusion therefore fetal blood glucose level is a reflection of the maternal level.[4] Infants born to diabetic mothers are therefore directly affected by fluctuations in maternal plasma glucose levels.

EPIDEMIOLOGY

The incidence of diabetes mellitus complicating pregnancy and resulting in live births generally depends on the population being studied. In the United States, the 1988 National Maternal and Infant Health Survey [1] showed that diabetes complicated 4% of such pregnancies, with 88% being due to gestational diabetes mellitus (GDM) and the remainder being due to pre-existing maternal DM. Recent estimates in that population has shown little or no difference in that figure. Latin Americans and African-Americans generally have a higher incidence than Caucasians.

Internationally, the average incidence of DM in p is generally put at 2-3% of all pregnancies [2,3], wi of Asian, Indian, or Middle-Eastern descent b higher risk than the general population [1].

In Nigeria, the prevalence of diabetes mellitus in p is 1.7%, with 61% of cases being due to GDM impact of having infants from this population relative to the entire Nigerian population significant.

There is no sex difference in the incidence of diabetic mothers (IDMs).[1]

PATHOPHYSIOLOGY

In the first trimester of normal pregnancies, maternal estrogen and progesterone levels are with a decrease in fasting glucose levels, whi nadir by the 12th week which is followed by an secretion of anti-insulin hormones in the 2nd Higher fasting and postprandial glucose le facilitate transfer of glucose from mother to occur: fetal glucose levels are 80% of maternal le

Human placental lactogen (hPL) is the placenta mainly responsible for insulin resistance and lip net effect is increased placental transfer of glu fetus and reduced maternal use of glucose. The rise steadily during the first and second trimes plateau in the late third trimester.[7]

In diabetic mothers, inadequacy of maternal insulin response results in maternal hyperglycemia which usually manifests as postprandial hyperglycemic episodes and profound effects on the fetus.[8]

This results in pancreatic beta cell hyper increased secretion of insulin and proinsulin fa IGF-BP3) thus creating a state of persi hyperinsulinaemia. Hyperglycaemia is also ac

ncreased fetal amino acid availability and these result in cessive growth (macrosomia) in all fetal organ systems cept the kidneys and the brain.[1]

maternal diabetes complicated by nephropathy, aternal vascular disease compromises uteroplacental od flow and impairs fetal nutrient supply thus causing aired fetal growth.[1]

sistent fetal hyperinsulinaemia causes a restriction in bstrate availability for surfactant biosynthesis. perinsulinaemia also reduces production of fibroblast- eumocyte factor, which normally would stimulate type-II eumocytes to produce surfactant.[1]

condary to parathyroid hormone suppression, pocalcaemia, with or without hypomagnesaemia, may be present: postnatal parathormone response of IDMs decreased compared to their gestationally matched ntrols.[1]

mpared to the adult, the fetus has an increased number receptors which mediate its growth. Because the fetal art is particularly rich in receptors, this may result in rdial hyperplasia and hypertrophy thus causing diomegaly.[1]

version of excess fetal glucose into fat causes depletion tal oxygen levels thereby creating a hypoxic state with equent surges in adrenal catecholamine; cardiac odeling and hypertrophy; stimulation of erythropoietin d polycythaemia. Polycythaemia leads to vascular dging, poor circulation, and postnatal perbilirubinaemia.[8] Excess of red blood cell precursors m the bone marrow may crowd out thrombopoietic ecursors and cause thrombocytopaenia[1]

ious types of congenital malformations (see table 1) y occur within the first 8 weeks of gestation (the period embryogenesis) and the maternal hemoglobin A_{1c} level, ich reflects the blood glucose concentration over the evious 2 months, can predict the risk for malformations en measured in the first trimester (see table 2).[7]

NICAL FEATURES

en of diabetic mothers may present in various ways and ese include:

- macrosomia (fetal weight >90th percentile for estational age or >4000 g in the term infant): when sent, the infant appears puffy, fat, ruddy, and often abby limp.[1]
- Impaired fetal growth (birth weight below the 10th rcentile, when plotted against gestational age on a dard growth curve): these infants are at a greater risk r perinatal asphyxia.[1]
  - Features of neonatal hypoglycaemia such as jitteriness, irritability, apathy, poor feeding, high pitched or weak cry, hypotonia, or frank seizure activity within the first few hours of life. It may also present within the first 3 days of life and infants may be "jumpy," tremulous, hyper excitable, hypotonic, lethargic and may feed poorly. More commonly, however, the neonate is asymptomatic. Early appearance of these signs is more likely to be related to hypoglycemia and later appearance related to hypocalcaemia.[1]
  - Symptoms of hypocalcaemia such as jitteriness or seizure activity[1]
  - Respiratory distress syndrome may present as tachypnea, nasal or intercostal retractions and hypoxia within the first few hours of life.[1]
  - Features of polycythaemia such as a "ruddy" appearance, sluggish capillary refill, or respiratory distress.[1]
  - Thrombocytopaenic haemorrhage.[1]
  - Jaundice.[1]
  - Renal vein thrombosis (due to polycythaemia) may present as a flank mass.[1]
  - Cardiomegaly may be seen in 30% of cases and heart failure occurs in 510% of infants of diabetic mothers. Asymmetric septal hypertrophy manifesting similarly to idiopathic hypertrophic sub aortic stenosis may also occur.[6]
  - Congenital anomalies (see table 1) may be the mode of presentation

LABORATORY AND RADIOLOGICAL DIAGNOSIS

Useful investigative laboratory procedures are as follows:

- Plasma glucose level which may show hypoglycaemia[1]
- Complete blood cell count which may show polycythaemia and/or thrombocytopaenia[1]
- Serum electrolytes estimation may reveal hypocalcaemia with or without hypomagnesaemia.[1]
- Serum bilirubin assay may show total and unconjugated hyperbilirubinaemia[1]
- Arterial blood gas estimation may indicate hypoxia[1]

Useful investigative radiological procedures include:

- X-Ray studies: a chest radiograph may show focal or diffuse atelectasis, presence of interstitial fluid, signs of free air in pleural or interstitial spaces, features of pneumonia, pulmonary malformations, clavicular fractures (in cases of shoulder dystocia), cardiomegaly or other abnormalities of the heart, great vessels/outflow tract.[1]
- Abdominal, pelvic, or lower extremity radiographs may show anomalies such as fusion of the legs, hypoplastic femur, defects of the tibia and the fibula, flexion contractures of the knee and hip, or clubfoot; sacral agenesis may also be present.[1]
- Contrast studies such as barium enema may show congenital anomalies of the gastrointestinal tract. There may be "small left colon syndrome," also known as "lazy colon" which radiologically appears as distal tapering of the colon.[1]
- A cardiac echocardiogram may show a thickened myocardium and significant septal hypertrophy may be present in as many as 1 in 3 IDMs. Myocardial hypercontractility and thickening often with septal hypertrophy disproportionate to the size of the ventricular free walls, may be found as well: cardiac anatomical malformations (see table 1) may be the prominent ECG finding.[1]

MANAGEMENT

A multidisciplinary (obstetric, paediatric, medical) approach to management is usually required for best outcomes.[4]

Table1. Some Congenital Anomalies of Infants of Diabetic Mothers.[7]

Cardiac

- Atrial septal defects
- Ventricular septal defects
- Transposition of the great vessels
- Coarctation of the aorta
- Tetralogy of Fallot
- Truncus arteriosus
- Dextrocardia
- Cardiomegaly

Central nervous system

- Neural tube defects
- Anencephaly
- Holoprosencephaly

Renal

- Hydronephrosis
- Renal agenesis
- Ureteral duplication

Gastrointestinal

- Duodenal atresia
- Anorectal atresia
- Omphalocele

Spinal

- Caudal regression syndrome, s
- Agenesis

Table 2. Relationship between Initial Pregnancy Value of Glycosylated Hemoglobin and Rate of Major Fetal Congenital Malformations.[7]

Initial Maternal Hemoglobin A_{1c} Level	Major Congenital Malformations (%)
7.9	3.2
8.99.9	8.1
10	23.5

In utero, the following should be done:

- Confirmation of gestational age with a first-trimester ultrasound examination[7]
- At 1820 weeks, a fetal ultrasound; a first-trimester nuchal translucency measurement and serum biochemical screening tests for anomalies should be carried out.[7]
- Evaluation of fetal growth and development by ultrasound are essential to detect macrosomia or polyhydramnios[7]
- Surveillance for fetal well-being, especially, in the third trimester should be done by using a twice-weekly nonstress test, the modified biophysical profile and Doppler ultrasound scan: maternal fetal kick counts may also be employed.[7,8]

Consideration for time of delivery should be based on adequacy of maternal glycaemic control during the pregnancy, extent of fetal lung maturity and/or development of complications (e.g. preterm labour).[7]The mode of delivery employed is individualized: spontaneous vaginal delivery or induction of labour may be employed if there is no fetal macrosomia. However, if fetus is macrosomic, elective Caesarean delivery is advised in order to prevent traumatic birth injury.[3]

After delivery, IDMs should have a blood sugar determination within the first hour of life and then every hour for the next 68 hours; if normoglycemic, oral or gavage feedings initially with 5% glucose water, followed by breast milk or formula, should be started at 23 hours of age and continued at 3-hr intervals: breastfeeding is however the most preferred option.[6,3]If an IDM is unable to to oral feeding, glucose should be given by perip intravenous infusion at a rate of 48 mg/kg Hypoglycemia should be anticipated promptly iden and treated, even in asymptomatic infants.[6,3] Intrave infusion of glucose is the mode of treatment; injections of hypertonic glucose should be avo because they may cause further hyperinsulinemia potentially produce rebound hypoglycemia.[6]

MORBIDITY AND MORTALITY

Generally, in mothers with type 1 DM, the per mortality rate doubles and the neonatal mortality triples when compared with that of the ge population[1].

These infants are 3 times more likely to be bo cesarean delivery, twice as likely to suffer serious injury, and 4 times as likely to be admitted to a neo intensive care unit.[1] Therefore, in the absence of suff number of neonatal intensive care units, the consequences are immediately obvious. Not only birth injuries can produce permanent physica neurodevelopmental impairment, with serious s educational and economic implications for the surv infants later in life.

Major congenital malformations are found in 5-9% account for 30-50% of perinatal deaths of infa mothers with gestational diabetes [1]. The terato insults produced by hyperglycaemia and assoc factors tend to occur during that critical perio organogenesis (3rd to 8th weeks of intrauterine emphasizing the need for good peri-conceptio women with pre-existing diabetes or those at r developing GDM.

The principal causes of increased morbidity and mor

diatrics

DMs include large or small for gestational age infants, poglycaemia, prematurity, respiratory distress drome (hyaline membrane disease) and intrapartum hypoxia[4]. Therefore, these conditions must be anticipated, ked for and appropriate treatments instituted

CTORS THAT MAY ACCOUNT FOR INCREASED RBIDITY AND MORTALITY IN NIGERIA

most developing countries[5], data on the morbidity mortality pattern of diabetes mellitus in pregnancy erally and IDMs in particular are scarce. What follow are sociations made to Nigeria, in view of the prevailing th care situation in the country.

re to detect preexisting maternal diabetes before gnancy or gestational diabetes mellitus (GDM) may ntribute to increased morbidity and mortality in IDMs. s more likely to occur in the rural areas where the vast rity of Nigerians live, without access to skilled enatal care. Also, preconception care is generally ng in the country, urban centres inclusive, with the first gnosis of DM often made during the antenatal visits.

maternal glycaemic control during pregnancy may be ther contributing factor. It is well known that the quency of fetal congenital malformations increases with maternal blood glucose during the first trimester[1]. All e directly proportional to the degree of maternal erglycemia in utero.

t of widespread availability of facilities for prenatal gnosis of congenital malformations in IDMs is mething not to forget; this is because congenital formations are contributory to the increased morbidity d mortality seen in IDMs.

e neonatal intensive care units in the country are adequate relative to the population, with most being ated in tertiary centres. This is not generally good, pecially with poor referral systems that exist in many as of the country

PROGNOSIS

h appropriate care during the perinatal period, the gnosis is very good [1], but with poor maternal diabetic ntrol, there is a significant rise in perinatal morbidity and rtality in the offspring [5]

ies have consistently shown that infants of mothers h poor glycaemic control during pregnancy are at ater risk for neurodevelopmental deficits[1]. This may be e to the unpredictable episodes of hypoglycaemia DMs are prone to, but hypoglycaemia as the sole se of this is still controversial [6].

e also at risk of childhood obesity that may extend adult life [1,6]. Considering the numerous medical blems that obese individuals are prone to, this potential plication is not to be viewed lightly.

portantly, the subsequent incidence of diabetes mellitus ldren of diabetic mothers is increased compared with h the general population [6].

COMMENDATIONS

commend the following as very basic steps that can plemented by health policy makers and medical ctitioners alike in order to reduce morbidity and rtality in IDMs.

Screening for pre-existing DM and GDM in all antenatal clinic visits. Additionally, in other hospital visits, as much as possible, women with any clinical suspicion or risk factor for DM should be thoroughly screened, as early detection and management is the key to preventing complications

When DM is detected prior to pregnancy, pre-conceptional diabetic care should be dutifully offered, as most pregnancies occur without planning.

Routine screening of LGA infants for hypoglycaemia should be carried out after delivery. The benefits far outweigh the costs.

Facilities for prenatal diagnosis should be put in place, to facilitate in-utero and postnatal management.

The policy of random placement of neonatal intensive care units in the country, mainly politically motivated, should be discouraged. Rather, strategic placements in each state of the federation with functional referral systems should be encouraged. The common interest of the children of Nigeria should supersede any political or ethnic interests.

CONCLUSION

The effects of maternal conditions on fetal well-being is exemplified by maternal diabetes mellitus in pregnancy; early detection and adequate control of maternal diabetes mellitus before and during pregnancy, however, give improved outcomes- reduced morbidity and mortality.

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PHYSICAL GROW
AND ITS ASSESSME
IN CHILDREN AND ADOLESCEN

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ABSTRACT.

Physical growth is a complex process of increase in size. An interplay of factors, both intrinsic and extrinsic, affects the g a child. Some of these are nutrition, infections, the education and socioeconomic status of the caregiver, hormones genetic makeup of the child. Seasonal and secular trends may also affect growth. Rapid growth occurs in foetal continues in infancy after which there is some deceleration in the process. An increase in growth then occurs at pu growth assessment, anthropometry, a simple and universally applicable technique is most commonly used. By this measurements such as weight, height (or length) occipitofrontal and mid upper arm circumference, and skin fold thick be made. These measurements expressed as Z scores or percentiles can then be compared with reference or standar chart.

Keywords: *Physical Growth, Growth assessment, Anthropometry, Children Adolescents*

Physical Growth and Its Assessment In Children and Adolescents.

Introduction.

The assessment of growth is essential in child care because one normal characteristic of children is that they grow. Globally, malnutrition is associated with 49% of childhood deaths and underlies many of the health conditions contributing to morbidity and mortality in them particularly in the developing nations of the world[1]. Nutrition is the most important factor affecting growth and its adequacy is clearly reflected in the growth pattern of a child[2, 3] The study of growth is basic to paediatrics and child health as it is the most sensitive index of health in childhood [4] Growth occurs in physical, intellectual, emotional and social terms. In this article physical growth will be dwelt upon.

Physiology of physical growth.

Physical growth is a complex process of increase in length, weight or volume involving the accretion of nutrients and water. It is a continuous process beginning from conception until adult life and is normally accompanied by an orderly sequence of maturational changes [5] It is affected by various intrinsic and extrinsic factors.[6]

At the tissue level, there is a first phase of cell division with rapid increase in the number of cells without a corresponding increase in their cytoplasmic content, a second phase during which division continues more slowly but with an increase in the complexity and size of the cells and a third phase where cell division ceases and only an increase in size occurs.[5]

The age at which one phase changes to the next varies in different tissues. For example, the neuroglia continues cell division for about two years of postnatal life whereas cartilage at the growing ends of bones continues mitosis until the epiphyses close after puberty. In general, most of the growth in late intra-uterine life and early childhood consists of the third phase of increase in cell size.[5]

Growth period and growth curves

There are 2 periods of rapid growth (growth spurts), the first is a continuation of the fetal growth and occurs in infancy while the second occurs at puberty just before general growth ceases. This occurs earlier in girls than in boys[6] The growth or height curve represents distance traveled with time, the line is steep in the first two years, becomes less steep thereafter until the puberta spurt occurs after which it finally flattens. The velocity curve represents the speed of growth an growth per unit time. A growth chart incorpo pattern of longitudinal growth and the normal amongst a population of children. Fig1 shows exa growth (height) and growth velocity curves [6]

Factors influencing the growth of a child

There is interplay of the various extrins affecting the growth of an individual. They e effects from prenatal life and the final outcome growth is well reflected in the birth weight.[5] From the effects are more visible. Nutrition is the most i extrinsic factor influencing the growth of an infant it is needed for tissue synthesis.[5] Protein, carbo fats, minerals and vitamins must be adequate and proportioned for optimal growth.[6]

Infections also constitute an important factor growth. They cause decreased food intake absorption [5] (if involving the gastrointestinal t contributing to malnutrition. Infection also has a effect on metabolism thereby causing tissue b and mobilization of stores. These produce a nega on growth by diverting resources that would h utilized in growth to processes of turnover and re

Malnutrition and infections often occur especially where they are common, as in d countries[5]. During a period of sub-optimal nutri infection, there is retardation or even cessation o however, there is adequate compensatory nutri this period, then this may be followed by a period up" growth.[5,6] [e.g. child A, see Fig.2.).

The growth velocity may increase so rapid original curve is attained and thereafter proceeds In some others, the growth is not rapid enough f child continues to grow at a normal velocity b centile line and because of a prolonged period ultimately reaches the original centile line. In s instances (as occurs in many children of the d nations), constant sub-optimal nutrition and prevent an opportunity for convalescence and growth such that original centile line is not rea

child B, see fig.2.).

The educational level of the care-giver of the child is important in determining the feeding method and the way it is practised[7]. The socioeconomic class may, in addition, dictate the quality of nutrition and general care that the child receives. Emotional problems such as arise when a child is separated from its mother also affect growth.[7]

It has been found that the growth of institutionalized motherless babies is often retarded[8] Hormones constitute important intrinsic factors regulating growth. Growth hormone, the primary hormone regulating growth, acts to accelerate chondrogenesis, widening of he epiphyseal plates with laying down of matrix at the ends of the bones with the resultant effect of an increase in height[26] Furthermore it causes an increase in the size of most viscera and protein content of the body, while the fat content is decreased [6] It is believed to stimulate the secretion of somatomedins and growth factors and act through these or in combination with them [6] Thyroid hormones are also essential for normal growth and skeletal maturation, they exert their effects on the contours of the face and the proportions of the body and they potentiate the effect of growth hormone on tissues [6] Insulin causes increased cell growth via its anabolic effect which is aided by the protein sparing action of adequate intracellular glucose[6] Adrenocortial hormones exert a permissive action that results in a positive effect on growth; glucocorticoids in large doses however may inhibit growth by its direct action on cells and also by decreasing growth hormone secretion. The effects of androgens and oestrogens are seen mainly at puberty when they are responsible for the growth spurt and accompanying cessation of growth that occurs.

The genetic characteristic of the individual is very important as it dictates the blue print for the programme within which the hormones act[7] It also has varying direct effects on growth such as seen in Turner and Klinefelter syndromes resulting in tall and short stature respectively[6] Other important influences on growth are systemic diseases such as congenital heart diseases in which growth is oftentimes retarded It is also important to note that there is a secular trend in growth whereby children in later days grow faster than their predecessors. This phenomenon is probably not simply due to better nutrition in terms of quantity of calories but to a better balance of nutrients Some extrinsic factors such as seasons and climates have also been observed to affect growth in which case growth in height is faster in warmer seasons than in cold and vice versa for

weight. The degree of activity can also affect growth, for example, inactivity is an important cause of obesity in children[4]

Assessment of growth

Growth assessment is an essential part of child care.[4] The best method of assessing the nutritional status of an individual child is by longitudinal monitoring of its growth.[5] This is particularly useful because growth faltering when present, can easily be detected before overt signs of malnutrition become manifest[4]and hence intervention strategies put in place early when they are still relatively easy and inexpensive. It reflects the child's state of health and thus draws attention to the antecedents of faltering and then preventive steps can be instituted. Assessment of growth can be done for the individual child or populations of children. The measures can be utilized in reassuring parents about or establishing normality of a child's growth and can also be used to assess abnormalities of growth and to evaluate treatment. Growth assessment is employed in Growth monitoring and Promotion (GMP) programmes[9].GMP is the regular following up of the physical growth of young children with a view of ensuring adequate growth and development of a child so that he reaches its full growth potential.[9] It is one of the most valuable and feasible ways of improving child nutrition particularly at the community level.[9]

Methods of growth assessment

Anthropometry is the simplest, cheapest most widely used and universally applicable technique for growth assessment[10,12] It involves the measurement of the change in size of some part of the individual.[5] Evaluations of nutrient content, food security and distribution, morbidity, mortality and other elements are also required to understand the causal factors resulting in malnutrition in order to provide the complete picture for problem solving. However, anthropometric measurements are relatively easy and convenient to obtain and use in the clinic and on the field. In practice, anthropometric measurements used include weight which is a composite measure of all the body tissues, length (or height in the older child) which indicates skeletal size and skin fold thickness which is a measure of subcutaneous fat.[5] The skull or occipitofrontal circumference (OFC) reflects a combination of skeletal or brain size and the mid upper arm circumference (MUAC) is affected by bones, muscle, skin and fat.[5] Indices relate the measures to age e.g. weight for age, height for age, OFC for age or a measure to another measure e.g. weight for height.[12] The usefulness of these indices depends on the availability of appropriate references or standards with which to compare them.[10,11] The anthropometric indices can be expressed in terms of Z scores or percentiles which can be used to compare with a reference population[12]. Some references that are in use include the National Center for Health Statistics/World Health Organization (NCHS/WHO) International Reference (1976) for weight, length and OFC based on measurements on

healthy children in the United States of America[13] the standards given by Janes (1974) which have figures for weight, height, OFC and MUAC for children in elite and poor communities in Ibadan, Nigeria[14] and standards for MUAC obtained by Wolanski and modified by Jelliffe (1966) and which approximate quite well with figures obtained by Tanner in healthy British children[15]Others are the British reference and the Boston standards. More recently ,the WHO carried out a multicentre growth reference study to generate new growth reference curves for all the children of the world[16] There are curves of 5 indices for ages 0-24 months and 24-71 months and for both sexes. A powerful instrument in the evaluation of the growth of an individual child is the growth chart.[2] Combined with the growth measurements, usually weight; it provides most of the information needed to assess growth. It is primarily designed for the longitudinal follow up of child so that changes over time can be interpreted.[2] The essential features are a graph on which the weight is plotted against the age, the reference curves running diagonally across the graph, proper identification of the child and important care interventions, notably immunization. The World Health Organization growth chart adapts that originally proposed by Morley in 1959, known as the "Road to Health or Ilesha growth chart.[16]

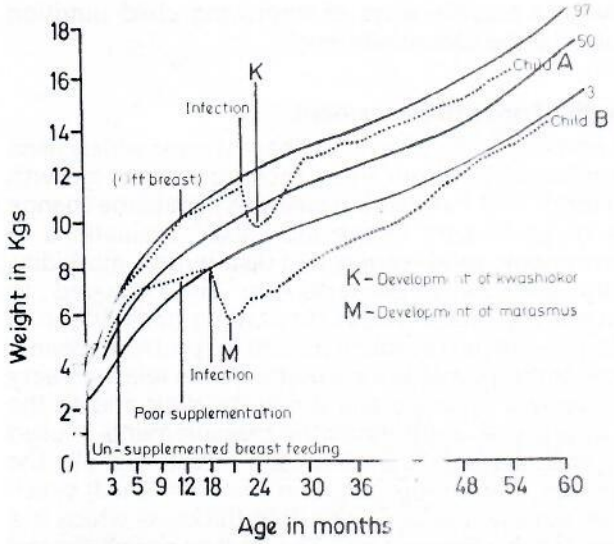
The anthropometric measurements and indices.

Weight for age is a measure most commonly employed in growth monitoring[11].

The relative change of weight with age is quite rapid and it is very sensitive to any change in the state of nutrition of the child, hence significant changes can be observed even in a few days. [2] It is an easy measurement to make, so a high

Fig 2

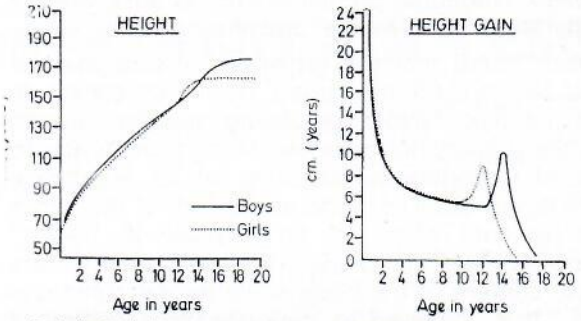
Fig.2: EFFECT OF MALNUTRITION AND INFECTION ON GROWTH



Adapted from A Rathi Growth and development of Children in villages. Postgraduate Doctor - Africa 1983 5 431

Fig 3

Fig.3: GROWTH IN HEIGHT.



Typical individual height attained curve for boys and girls.

Typical individual velocity curve for height.

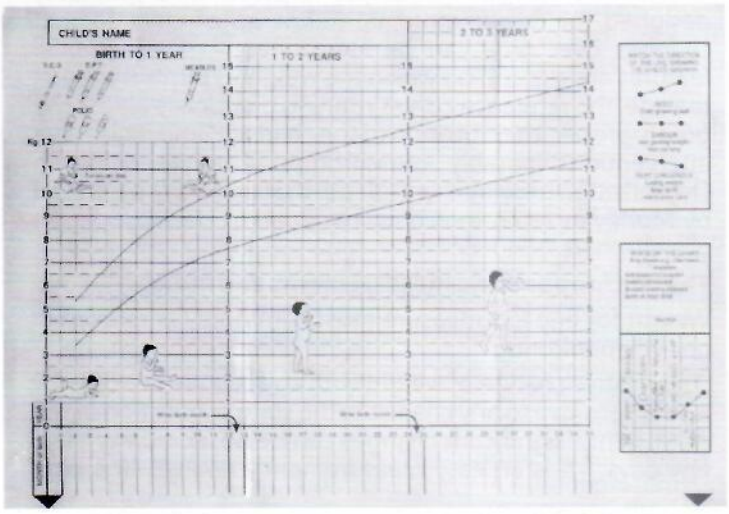
Adapted from A Rathi Growth and development of Children in villages. Postgraduate Doctor - Africa 1983 5 431

presence of oedema.[1] A child that has a low weight for age is described as underweight.

Length or height for age is a stable index. Known as the stunting index, it reflects the total increase in the size of the child from birth[2]. A low height for age in the older

child who appears stunted is frequently associated with poor overall socioeconomic conditions or repeated exposure to adverse conditions and infers a chronic undernutrition[10,11]. The length or height changes more slowly than weight, more so for the older child; accurate measurement is cheap and does not require a sophisticated equipment[11]. However, it is a fairly difficult measurement to make, particularly in infants, and a small error may make a significant difference when interpreting the data, especially that of growth velocities[2]. Furthermore, length does not decrease in a child and therefore cannot indicate deterioration in the state of health.[11]

Relating the weight of a child to its height (weight for height) is an objective measure of the child's degree of thinness and a low weight for height infers wasting[2]. Hence the weight for height index is also known as the wasting index. In monitoring the progress of an individual child, it has no advantage over weight for age but its great advantage is that it can be used in situations where the ages of the children are not known because it is not dependent on age.[2] Its limitation is that in a situation where



for example, a child has both low weight and length, it is regarded as 'normal' whereas it is not and actually requires attention[5].

The occipitofrontal circumference (OFC) directly reflects intracranial volume. Assuming a fairly constant cerebrospinal fluid content, the OFC would be proportional to the volume of the brain and serial determinations in a child assesses its brain growth.[11]

The measurement of OFC is easy, cheap and simply requires a tape measure and little time.[11]

The mid upper arm circumference (MUAC) is an estimate of arm tissues; it is an objective sign of thinness and indicator of current malnutrition[5] It is a composite measurement of several tissues and a high correlation of the MUAC with weight has been found by some workers. Some studies have suggested that MUAC may be more sensitive than body weight in detecting nutritional change but some others have held a contrary view.[2,5] The MUAC is most commonly used as a single measure in children between the ages of

one and five years during which period it remains almost the same and is a simple way of detecting malnourished children independent of the knowledge of their precise age.[5] There are however MUAC standards for infants. Furthermore the MAC/OFC ratio has been used as a simple and reliable method of nutritional assessment even in the newborn[20] The measurement of MUAC can also be made with a simple tape measure.[5] The Shakir strip and the MUAC meter are other instruments that can be used for measurements between the ages of one and five years and one and twelve years respectively.

Measurement of the skinfold thickness is a convenient method for assessing objectively and directly the body fat bulk and in this respect it is superior to weight[11] The large caliper produced in the United States and the Harpenden manufactured in England

are two of the various instruments that can be used in making these measurements, but these they are expensive.[11] The chest circumference (CC) can be easily measured with a tape measure and its application in assessing growth is better when combined with OFC as a ratio OFC/CC. This ratio is expected to be greater than 1 below the age of one year, equal to 1 at the age of one year and less than 1 after the age of one year. The Body Mass Index (BMI) which is also known as Quetelet Index is frequently used to assess adult undernutrition. Anthropometric indices are difficult to interpret during

...ence particularly height / weight indices which ...ected by the adolescent growth spurt and the onset ...ual maturation[22] Only in the last decade has an ...t been made to use anthropometric methods to ...acute under nutrition in adolescents.[22] The BMI is ...commended for use in adolescents as the basic ...or of overweight or thinness.[22] It is calculated as the ...t in kilograms divided by the square of the height in ...[22] The WHO recommends standards using the ...data on U.S adolescents as a reference population.[22] ...Weights and BMI of adolescents are plotted on ...growth charts using these standards .Utilizing these ...as a reference for developing nations has however ...criticized and the use of local reference data is ...ble if available.[24] A few studies have been carried ...the growth of adolescents.[24,25,26] There is a dearth ...data[24] and therefore a need to generate local data ...arly for reference in these regions of the world for ...n and adolescents. Generally in anthropometry, ...t and height are the 2 most commonly used and ...mended measurements.[27] In growth assessment, the ...ements are

...d in combination as no single one can truly reflect ...icate process of growth.[5]

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BURKITT LYMPHOMA

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Abstract

Burkitt lymphoma being a highly aggressive lymphoma, frequently presenting in extranodal sites and having the sh doubling time; creates special challenges for diagnosis and treatment. It is the first tumor to be associated with EB the first type of NHL to be associated with HIV infection. Three major types are recognized; the endemic, sporad immunodeficiency associated. It is a potentially curable malignancy using modern chemotherapeutic combinatio avoiding all factors that minimize good prognostic outcome.

Introduction

Burkitt lymphoma (BL) is a high grade Non-Hodgkins lymphoma(NHL) that is characterized histopathologically by a mass of diffuse small non-cleaved B cell lymphocytes[1,2]. It is a highly aggressive lymphoma that frequently presents in extranodal sites with a very high proliferation rate. It is the fastest growing tumor, characterized by explosive growth with a doubling time of 24 hours. BL is of greatest importance in sub-saharan Africa where it is the most common childhood (2-16years, mean 7years) cancer, accounting for up to 36% of childhood cancers and 70% of childhood lymphomas[3] but , overall constitutes 5% of lymphomas for both adult and childhood populations.

Historical Background

Denis Burkitt, a British surgeon, working in central Africa in Kampala, was the first to describe Burkitt lymphoma in 1956[4]. He noted children with facial swellings involving one or both sides of the face and upper and lower jaws, sometimes accompanied by proptosis. He also observed that some of the children had huge abdominal masses sometimes with facial swelling. There was usually no lymph node involvement. This malignancy was thought to be a sarcoma[5,6] but later established to be a lymphoma and given the name Burkitt lymphoma which happens to be the most common in children in that area[5]. The lymphoma was found to occur throughout tropical Africa with higher occurrence in areas of greater rainfall and altitude greater than 1550M. These geographic and climatic associations suggested an association with falciparum malaria.

In 1961, Burkitt shared samples of the lymphoma patients with Epstein who along with his colleagues, in 1964, identified a virus in culture cell lines of the tumor [7]. The virus come to be known as Epstein-Barr virus (EBV) and was proposed to be oncogenic[5].

Pathogenesis

Burkitt lymphoma belongs to the extended family of Non-Hodgkins lymphoma (NHL) which are solid tumors of lymphoid organs. NHLs are generally clonal malignancies of the multiple cellular components of the normal lymph node, spleen and thymus.

In line with other NHLs; Burkitt lymphoma arise as a clonal transformation occurring at specific stage of normal B cells differentiation during antigenic stimulation in accordance with Murphy's law which holds that for every cell type and stage of differentiation there will occur neoplastic counterparts. The diagram below depicts the differentiation pattern of B cells. A level of immature B cell of small non cleaved cell, occurs a clonal transformation and proliferation o immature B cell.

Aetiological factors

The aetiological factors of Burkitt lymphoma diffe great extent with the clinical variants as described World Health Organization (WHO) classific However, it appears that the major and co aetiological factors are closely and strongly linked geographical location/climate, immunosuppressio chromosomal abnormalities.

The WHO recognizes the Classical (Endemic) Bu and two other variants.:-

The African or Endemic Burkitt Lymphoma

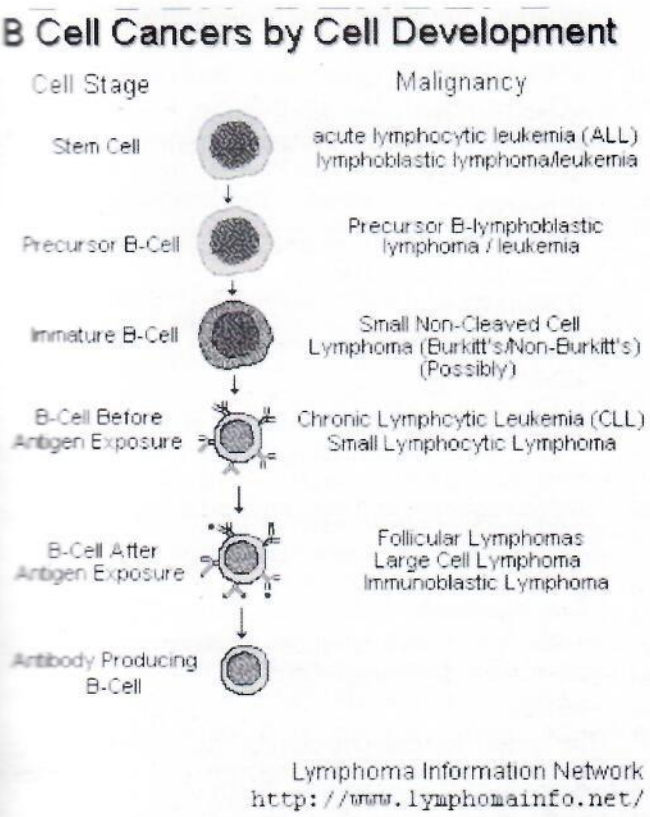
The African type (endemic) is commonly found malaria belt of Africa and Papua New Guinea associated with low socioeconomic st undernourishment, malaria holoendemicity and E Barr virus (EBV) infection. The actual age rang occurrence of endemic Burkitt is 2-16years but the common age of occurrence is 4-7 years with a female ratio of 2:1. The lesion usually involve bon the jaw and other facial bones, as well as kid gastrointestinal tract, ovaries, breast and extranodal sites [8].

Non-African (Sporadic)

This refers to other type of Burkitt lymphoma see where in the world and which do not show rearrangement of the C-myc-proto-oncogene immunoglobulin genes that typically charac endemic Burkitt lymphoma. It accounts for 1-2 lymphoma in adults and up to 40% of lymphoma children in the US and Western Europe [9] . The abd especially the ileocaecal area is the most common involvement. The ovaries, kidneys, omentum, Wald ring and other sites may also be involved. Bila involvement of the breast may occur in association onset of puberty or with lactation[10]. Lymph involvement is more common among adults amongst children[11]. Rarely, patient may also malignant pleural effusion and ascitis [10,11]. EBV pos is 15-30% in some cases or fewer in other series [12]

Immunodeficiency Associated Burkitt Lymphom

This occurs mainly in patients with HIV but also occ



Allograft recipients [13,14] and individuals with congenital immunodeficiency. Several cases of Burkitt lymphoma had been described in homosexual women[15,16].

Ebstein Barr Virus(EBV) and BL

EBV was identified in 1964 in African type Burkitts lymphoma [7] It is present in over 95% of BL in Africa, 50% South Africa, 85% in North African Burkitt Lymphoma and 5-15% Western Countries. Previous studies have shown that endemic Burkitt lymphoma is highly associated with Ebstein Barr virus whereas only one third of non-endemic cases carry the virus [17,18]. It is believed that EBV, analogous to malaria, leads to polyclonal B cell activation and permits poorly controlled proliferation of EBV positive B cells which further leads to a greater risk of c-myc rearrangement, and then to lymphoma.

Chromosomal Abnormalities in BL

The characteristic chromosomal translocation involving chromosome 8 and 14 in Burkitt lymphoma was discovered in 1976[19]. Majority of BL carry translocations between c-myc proto-oncogene and IgH gene t(8;14) (q24.1;q32.3) found in 80% of cases. The remainining 20% of Burkitt lymphoma have presence of translocations between c-myc and the gene for kappa light chain t(2;8) in 15% and lambda light chain t(8;22) in 5% [20] . However specific lymphoma-associated translocations like IgH/bcl-2 and translocations bcl 6 are absent in Burkitt lymphoma. The Break point on long arm chr. 8q is up stream of cmy in endemic whereas it is within in the sporadic.

Evidence linking the Risk of BL to Malaria Infection

The incidence of BL correlates within and between countries with the incidence of malaria and with parasitaemia rates. The age of peak levels of anti-malaria antibodies (5-8yrs) is same as the peak incidence of BL. Individuals living in urban areas where malaria transmission rates are lower have a lower incidence of BL. In regions where death rate due to malaria had declined, BL incidence has also declined. The age of BL cases among immigrants from malaria free area to malarious area is higher than that of the original inhabitants. There is an inverse relationship between the age of onset of BL and the intensity of infection with P falciparum. There is reduced incidence of BL in individuals with sickle cell trait which also protects against malaria. There is an evidence for seasonal variation and for time-space clustering of BL cases.

Incidence

The incidence of Burkitt lymphoma appears to vary widely with geographic locations and climate. The highest incidence of $36.1/10^6$ was found in Kampala,Uganda, followed in descending order by Blantyre, Malawi ($35.8/10^6$). Ibadan, Nigeria ($18.0/10^6$), Harare, Zimbabwe ($2.4/10^6$), Bamako, Mali ($1.7/10^6$) and in the United States (Whites) ($2.5/10^6$).

Clinical Presentation

The clinical presentation of BL varies with the type. The endemic BL afflicts children of age 2-16years with a mean age of 7 years, usually in the jaw and in the retroperitoneum, and commonly with bilateral involvement of the kidneys and ovaries or a para spinal tumor [6,7].

The non-endemic cases show a broader age range (up to 35years) and a higher mean age of 11years. Male cases predominate in both endemic and non-endemic areas and other clinical features (apart from the ones stated above) and prognosis are similar [18]

The outline of the usual clinical presentation of BL includes the followings:-

- Swelling of the mandible or maxillae (1-4 quadrants), which is the commonest presentation in Africa
- Earliest sign is the loosening of child's molar or premolar
- Proptosis may be marked but not usually painful
- Intra-abdominal tumors, especially retroperitoneal lymph nodes or ovaries
- Extradural lesion causing spinal cord compression and paraplegia
- Enlargement of the parotid glands, breasts (usually both), testis, thyroid and kidneys.(all are uncommon)
- Lymph node enlargement is also uncommon except in the abdomen
- However, the child's general condition is usually remarkably good

METHODS OF DIAGNOSIS

Burkitt lymphoma being an aggressive tumor requires a quick and prompt diagnosis and more often Fine Needle Aspiration cytology (FNAC) of facial mass or Ultrasound (USS) guided abdominal mass. Excision Biopsy for histology may also be used in confirmation of diagnosis.

Cytologic appearance

Cells of BL are fairly uniform in size with rounded nuclei and granular nuclear chromatin. The cytoplasm of the cells form a thin rim round the nucleus, it is basophilic, non granular and usually contains some small vacuoles corresponding to lipid droplets. The nucleus is slightly indented, and have 2-5 nucleoli, evenly distributed chromatin and occasionally mitosis

Histological Appearance

Starry Sky appearance indicating sheets of monomorphic small non cleaved cell with bluish cytoplasm interspersed by cellular debris- laden macrophages

Immunophenotyping

The Burkitt's cell is Tdt ve, but CD10, CD19, CD20, CD22, CD24, CD 37, CD 38 and SIgM positive. In most African BL cases, the cells are CD21 positive but negative in American BL cases.

Endemic vs Sporadic BL

Both look alike histologically, have simillar B cell ancestry, share same explosive proliferative potential and exhibit t [8,14] type relocations all through the break points affecting c-myc. However, clinically and phenotypically both are dissimilar. Burkitt lymphoma endemic to Africa and New Guinea is a distinctive syndrome of large extra nodal tumors affecting the jaw bone and abdominal viscera, particularly kidneys, ovaries and retroperitoneal structures; less often it presents as isolated tumor of thyroid, distal long bones, mediastinum, liver or spleen. The vigorous growth rate of retroperitoneal or extradural tumor causes paraplegia, either by vascular compromise or by direct invasion of spinal cord or cranial neuropathy. It rarely involves bone marrow, lymph nodes, lungs, mediastinum, liver or spleen. CNS invasion is often heralded by meningeal involvement with shedding of tumor cells into spinal fluids. The mean age of African patient is 7 years [range 2-16 yrs].

In contrast, sporadic (non African BL) usually originate in payers patches or mesenteric nodes and often home in follicular B cell zone of abdominal and peripheral nodes. Because of the proliferate vigor; patients often present with obstruction of the intestine, airway or ureters. Jaw tumors characteristic of endemic tumors develop in only 15% of sporadic cases compared to 88% in African patients. Marrow involvement occurs earlier and much earlier in non endemic cases than in African Burkitt Lymphoma [20% vs 5%]. It has a broader age range of up to 35 yrs and a higher mean age of 11yrs as against 7 yrs for endemic. The leukaemia associated with sporadic BL has L3 morphology and confers a dismal prognosis.

BL- Staging

Staging of Burkitt lymphoma can be performed using Ann Arbor or, more often, the St Jude/Murphy staging system[8,10,21,22] . Approximately 30% of patients present with limited stage disease(I or II) while 70% present with wide spread disease(III orIV) [8]. The most commonly used staging system is that of Uganda cancer institute originally designed for African patients with BL.

A Single Extra Abdominal Tumor

AR Completely resected intra-abdominal tumor without Extra-abdominal tumor.

B Multiple Extra abdominal tumor

C Intra-abdominal tumor with or without a single jaw tumor.

D Intra abdominal tumor with extra abdominal sites other than a single jaw tumor

Investigations required for Management.

- Physical examinations
- Complete Blood Count
- Liver and Renal serum Serum Chemistries
- Serum LDH
- Serum Uric Acid
- Chest X-ray
- Chest CT scan if CXR are abnormal or suspiciously abnormal CXR
- Chest CT
- Abdominal Ultrasound Examination [including liver spleen kidneys and pelvis]
- Galium 67 scan
- Bone Marrow examination
- CSF cytology

Lab. Findings in BL

Major biochemical findings in Burkitt lymphoma include hyperuricaemia, lactic acidosis, high lactate dehydrogenase and high antibodies to early antigens of EBV.

Management

There is no time for leisurely workup; treatment must be commenced within24-48 hours

- Supportive

In order to prevent/correct tumor lysis syndrome, generous hydration is mandatory and Allopurinol must be given at a dose of 10-20mg/kg daily

- Definitive

Resection of large intra-abdominal mass is no longer favored since the tumor is highly chemo sensitive.

The mainstay of treatment is Chemotherapy .All reported successful protocols include cycophosphamide in doses of at least 1g/m2 and either high or intermediate dose methotrexate. Most also include anthracycline. Short duration, high intensity chemotherapy, often combined with CNS prophylaxis yields excellent survival in children. In localized disease >90%, 5 year survival rate is achievable.

Modified Zeigler Regimen

IV CPM 1000mg/m2 Day 1

IV VCR 1.5mg/m2 Day 1

SC Cytosar 50mg/m2 12hrly x 6 doses OR IV/PO Methotrexate 37.5mg/m2 Day 1

PO Prednisolone 40mg/m2 daily x 5days

Give every 14 days

A more recent, very intensive, highly effective alternating non-cross resistant regimen developed by Magrath et al[24] is CODOX-M/IVAC regimen

CODX-M C = cyclophosphamide, O = oncovin/vincristine, Dox =Doxorubicin and M for High dose methotrexate while IVAC Ifosfomide, Etoposide Cytosar (high Dose) + IT therapy.

The combination chemotherapy is said to give a high cure rate[25,26]. However associated toxicities include frequent myelosuppression, severe mucositis, nausea and vomiting, neuropathy and treatment related death.

Complications

- Tumor Lysis Syndrome, especially with large tumor burden, is usually characterized by lactic acidosis hyperuricaemia, hyperkalaemia, hypocalcemia hyperphosphatemia.
- Haemorrhagic Cystitis which is preventable by maintaining high urine output during the first 24hrs and

alkalinize urine with sodium bicarbonate until urinary pH exceeds 7.

CNS involvement and Intra- Theca [IT] Therapy

The Cranial Nerve palsies in BL are often multiple and paraplegia may occur. Therefore prophylactic IT therapy is mandatory in all cases of BL while CNS IT therapy is required for all patients with CNS disease.

IT drugs being given either as single therapy or in combinations include

IT cytosar 30mg/m2 ± IT Metothrexate 12.5mg/m2 ± IT Hydrcortizone 24mg/m2

IT prophylaxis is given on Days 1 and 5 only per cycle of chemotherapy.

Triple IT therapy is preferred for CNS BL although obvious added advantage is stll controversial.

Prognosis

With modern combination chemotherapy 85-100% of those with early stage disease and 75-85% of those with advanced disease will survive for at least 3 years without the need for treatment [23]. It is a curable malignancy and if there is no relapse a year after combination chemotherapy, patient has a 90% chance of surviving indefinitely. Prognosis with CPM alone is less favorable.

Factors responsible for poor treatment outcome in Nigeria for example include poverty, circulating fake drugs and poor treatment compliance [27,28]. Recently, complete remission rate was observed to be as low as 22.4%[28]

Relapse

Relapse in BL may be early or late

In early relapse, tumor regrowth is usually in the same site and occurs at less than 3months post treatment

Late relapse usually arise from a previously uninvolved site and likely to respond to the same agents. It occurs at greater than 3 months post treatment.

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NEONATAL TETANUS -A REVIEW

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INTRODUCTION

Tetanus, as defined by Stedman's Medical Dictionary (2005), is a disease marked by painful tonic muscular contractions, caused by the neurotropic toxin (tetanospasmin) of Clostridium tetani acting on the central nervous system. It is the only vaccine-preventable disease that is not communicable but acquired through environmental exposures to the spores of Clostridium tetani (World Health Organisation, 2003). In 1885, Nicolaier discovered the anaerobic bacillus Clostridium tetani, and in 1889, Koch's pupil, Kitasato, obtained the bacillus of tetanus in pure culture and associated the disease with animals. During World War 1, tetanus occurred in 1.47 per 1 000 British wounded and in 12.5 per 1000 persons involved in the peninsular campaign (Eleftherios, 2006). Neonatal tetanus, alongside polio, HIV/AIDS e.t.c, is a notifiable disease. Although rare in developed countries, it predominates and is a substantial major contributor to infant mortality (Fair and Sutter,2002). It results from cord contamination during unsanitary delivery conditions, coupled with a lack of maternal immunization (Eleftherios, 2006). A mere $1.20 (approximately N144.00) prevents the death of a mother and her future babies from tetanus. Yet about 180,000 infants and 30,000 women still die each year from the disease which is preventable through immunization and hygienic birth practices (UNICEF, 2004).

The World Health Organisation initiative's aim is to reduce the number of neonatal tetanus cases to such levels that it is no longer a major problem. Unlike polio and small pox, the disease cannot be eradicated (the tetanus spores are present in the environment), but through immunization of child bearing age women and of pregnant women, and through the promotion of more hygienic deliveries, the disease can be eliminated (which is defined as less than one case of neonatal tetanus per 1 000 live births in every district) (World Health Organisation, 2008). As at September, 2007, only 9 countries in Africa; Eritrea, Malawi, Namibia, Rwanda, Togo, Zimbabwe, South Africa, Egypt and Zambia, have achieved Maternal Neonatal Tetanus (MNT) elimination (World Health Organisation, 2008).

AETIOLOGY

The spores of the bacillus are present in soil, animal dung, contaminated instruments e.t.c. (Lewis, Heitkemper, Dirksen, 2004). The disease is caused by contamination of the umbilical stump with spores of clostridium tetani, following childbirth, through cutting the cord with a non-sterile instrument or by application of animal dung to the cut cord (Wikipedia, 2007). Other possible entry sites include circumcision wounds, ear piercing and scarification incisions. Asekun-Olarinmoye and Onadeko in 2003 also identified the following as risk factors for the development of neonatal tetanus:-

Maternal age of less than 20 years

Low socio-economic status

Primiparity

Delivery outside health facilities

Lack of trained attendant(s) at delivery

Type of instrument used to cut the umbilical cord

Presence of livestock within the family compound

Irregular attendance or no attendance at antenatal clinic

Also, maternal HIV and malaria infection during pregnanc reduce tetanus antibody

levels in newborns and mothers thereby exposing them t an increased risk of tetanus

(Egwang, 2007).

PATHOPHYSIOLOGY OF NEONATAL TETANUS

Neonatal tetanus results from infection with Clostridiu tetani, a mobile, spore-forming, anaerobic, gram-positiv bacillus found in or on soil, manure, dust, clothing, ski and 10%-25% of the human gastrointestinal tra (Eleftherios, 2006). The bacillus enters the body most through the umbilical cord (Famakinwa, 2002). Othe possible entry sites include circumcision, ear piercing an scarification incisions.

There are three exotoxins produced which are:-

Tetanospasmin

Nonconvulsive neurotoxin

Tetanolysin.

The effect of tetanospasmin is neurotoxic. It has speci affinity for nervous tissue, especially the anterior horn the spinal cord, where there are motor nerve ending motor nerve cells and cranial nerves. The toxin reaches th nervous system via the bloodstream or by traveling alon the axon cylinders of motor nerves and, eventually,

becomes fixed in the ganglia cells of the anterior horns the spinal cord, and cranial nerves. This leads to refle convulsive activity. The toxin interferes with the reflex ar by blocking inhibitory transmitters at the presynaptic site in the spinal cord and brain stem (Famakinwa,200 Symptoms begin 3 to 14 days after birth following a perio of normal feeding. The infant suddenly fails to su properly and is irritable (Wrong Diagnosis, 2007). The oth two toxins, Nonconvulsive neurotoxin and Tetanolys have haemolytic and cardiotoxic effects on the centr nervous system. The accompanying toxaemia produce headache, fever, malaise, irritability and restlessne

requisites, also called self-care needs, are measures or actions taken to provide self-care.

There are three categories of self-care requisites:-

-Universal requisites are common to all people. They include maintenance and elimination of air, water and food; balancing rest, solitude and social interaction;

preventing hazards to life and wellbeing; and promoting normal human functioning. The nurse should take note of these when caring for a neonate with a medical diagnosis of neonatal tetanus. Respiration is impaired and this impairment can be reversed with the use of oxygen. Fluid intake and nutritional requirements can be maintained through the use of intravenous fluid and electrolytes therapy. Elimination, especially of urine, can be assisted with the use of an indwelling catheter. Further injury, e.g., fracture of the vertebral column, can be prevented through reduction of spasms; this can be achieved by nursing the neonate in a quiet darkened room that is insulated against noise. Judicious sedation should be given and nursing care should be administered with utmost caution. Firm touch should be used if necessary to touch the client (Lewis, Heitkemper, Dirksen, 2004).

-Developmental requisites result from maturation or are associated with conditions or events, such as adjusting to a change in body image. The neonate's development is compromised of the adherent complications of tetanus and lack of adequate nutrition throughout period of illness. The nurse has the responsibility of assessing the infant and his/her environment for risk factors which could lead to severe complications, e.g., lack of ventilation, absence of cot sides e.t.c.

-Health deviation requisites result from illness, injury or disease or its treatment. They include actions such as seeking health care assistance, carrying out prescribed therapies and learning to live with the effects of illness or treatment. The parents of the neonate usually seek health care assistance and are educated on the management and how they can help to improve the prognosis e.g. not allowing visitors which may serve as stimuli to trigger off spasms. The nurse also rehabilitates the family during the course of management, preparing them to live with the effects the infection has on the neonate developmentally.

Therapeutic self-care demand refers to all self-care activities required to meet existing self-care requisites, or in other words, actions to maintain health and wellbeing. Self-care deficit results when self-care agency is not adequate to meet the known self-care demand. Orem's self-care deficit theory explains not only when nursing is needed but also how people can be assisted through methods of helping: acting or doing for, guiding, teaching, supporting, and providing an environment that promotes the individual's abilities to meet current and future demands.

Orem identifies three types of nursing systems:-

-Wholly compensatory systems are required for individuals who are unable to control and monitor their environment and process information. The infected neonate is in such a position because he/she cannot control the internal and external environment. The nurse is then totally responsible for the performance of this action on the neonate's behalf.

-Partly compensatory systems are designed for individuals who are unable to perform some self-care activities. The nurse and the parents assist the neonate in meeting his/her self-care needs.

-Supportive-educative (developmental) systems are designed for persons who need

to learn to perform self-care activities. The nurse educates the parents on the causes,

course and prevention of neonatal tetanus (Kozier, Erb, Berman, Snyder, 2004)

GENERAL MEDICAL MANAGEMENT

The airway should be evaluated. Breathing and circulation should be ensured. Benzodiazepines or Phenobarbital infused intravenously can stop the seizures. If there is reason to suspect hypoglycaemia, even if blood glucose level cannot be quickly established, treat with glucose intravenously. Depending on the aetiology, the infant may stay on Phenobarbital for a varying duration of time (Wrong Diagnosis, 2007). Penicillin G, which has been used widely for years, is no longer the drug of choice. Metronidazole has comparable or better antimicrobial activity, and penicillin is a known antagonist of GABA as is tetanus toxin (Eleftherios, 2006).

PREVENTION

Tetanus generally can be prevented through the use of Tetanus Toxoid (TT). The toxoid is a formaldehyde-inactivated preparation of chemically inactivated toxin, adsorbed onto aluminum salts to increase its antigenicity. Tetanus toxoid is stable and can withstand exposure to room temperature for months and up to 37 degrees Celsius for a few weeks without a significant loss of potency. It is given as a single antigen to immunize pregnant women and women of childbearing age, in order to prevent tetanus in their newborns. Following the administration of tetanus toxoid to the pregnant woman, antibodies pass to the foetus across the placenta to provide protection against neonatal tetanus. They should have at least two doses of tetanus toxoid vaccine at least 4 weeks apart, with the last dose at least two weeks before delivery (World Health Organisation, 2003). For lifetime coverage, individuals are exposed to full regime of tetanus toxoid.

Child delivery in a clean environment and use of sterile instruments during the delivery reduces the risk of neonatal tetanus. Elimination of certain cultural practices e.g. use of cow dung, will also help to reduce the incidence of neonatal tetanus.

COMPLICATIONS

They include spasms of the respiratory muscles th cause interference with breathing. Other complicatio include fractures of the vertebral column or long bon hypertension, abnormal heartbeats, coma, generalize infection, clotting in the blood vessels of the lung(pneumonia and death.

PROGNOSIS

Generally, the longer the incubation period, the mild the illness, the better the prognosis (Lewis et al, 200 Developmental delays are common among survivo (Eleftherios, 2006). About 85% of maternal and neonat tetanus related deaths occurred in newborns (Egwan 2007). Overall, prognosis is poor.

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MANAGEMENT OF CEREBRAL MALARIA IN A TYPICAL TROPICAL PAEDIATRIC EMERGENCY WARD

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ABSTRACT

Cerebral malaria is the most fatal complication of P. falciparum infection in children. It accounts for one-fifth of severe malaria cases in a typical children emergency ward in Africa. Mortality remains high in spite of the use of newer drugs and improvement in treatment modalities. It is responsible for more than one million deaths per year particularly children in sub-Saharan Africa. The exact cause of coma in cerebral malaria remains obscured. A compromised microcirculation, with sequestration of parasitized erythrocytes, is central in the pathogenesis. Intravenous quinine remains the first choice of drug in the treatment of cerebral malaria, possibly because of its broader stage specificity, and high sensitivity to all stages of malaria parasites. Since multi organ involvement is very common, supportive treatment is essential to improve survival in this disease, ideally in an intensive care unit setting. Adjunctive treatments have thus far not proven to be beneficial. Strategies for prevention of malaria and early diagnosis and treatment with effective antimalarial drugs remain the most important measures in the public health sector.

INTRODUCTION

Malaria remains a major cause of morbidity and mortality in sub-Saharan Africa, in spite of several efforts and initiatives in controlling the disease.[1] According to World Health Organization (WHO) estimates, between 300 and 500 million peoples are have clinical attacks of malaria annually[2]. Almost all of the 1.7 2.5 million deaths from malaria are attributable to plasmodium *falciparum* infection. Overall, 90% of all cases of life-threatening malaria occur in African children, especially those in the sub-Saharan Africa.[3] Malaria is among the leading causes of death in children less than 5 years in Ibadan.[4] A more recent review indicated an increasing trend of cases of severe malaria from the year 2000 to 2006 at the children emergency ward in Ibadan.[5] Reports from other centres in Africa also show no significant reduction in the magnitude of severe malaria cases.

Cerebral malaria is the most severe manifestation of *falciparum* infection and it is responsible for majority of the deaths due to malaria. It is defined as impaired consciousness in a patient with peripheral asexual parasitaemia, in whom no other cause of loss consciousness can be found. [6] Other features that usually characterize cerebral malaria include generalized convulsions, anaemia, raised blood urea, and high parasitaemia amongst others.[3] In parts of Africa with high malarial endemicity, cerebral malaria is mostly common in children less than 5 years of age accounting for 10 - 15% of *falciparum* malaria admitted to the hospital and 90% of such deaths.[3] In Ibadan for example, cerebral malaria constituted 2.2% of all paediatric admission, accounted for 19.7% of all severe malaria cases and 12.9% of all deaths.[5]

WHAT ARE THE CAUSES OF COMA IN CEREBRAL MALARIA?

The pathogenic mechanisms underlying CM are not fully understood and it remains a subject of debates. It is not clear why only a small fraction of children with malaria infections develop cerebral malaria. Quite apart from factors related to transmission characteristics, the virulence and sensitivities of the infectious agent P. *falciparum*, numerous genetic, nutritional, and immunological host factors have been implicated. Three pathogenic mechanisms have been proposed for human CM; the theory of permeability, the mechanistic theory and the immunological theory. A combination of many factors from the host and parasites apparently contribute to the acute access of *falciparum* to the brain.[7-9] Generally, *P. falciparum* malaria infection is characterised by development of sticky knobs on the surface of red cells, adhesion of red cells to the endothelial cells of post-capillary venules and formation of *rosettes* with uninfected cells.[10-12] The pathophisiologic feature of CM is characterized by the sequestration of infected erythrocytes (IEs) in cerebral microvascular beds. The precise mechanisms involved in the onset of neuropathology remain unknown, but parasite sequestration in the brain, metabolic disturbances, and host immune responses all play a role.[13]

Cytoadherence is mediated by strain specific, high MW **P. f Erythrocyte Membrane Protein 1 (PfEMP 1)** that is exported to the surface of infected erythrocyte and anchored through the membrane to a sub-membranous accretion of parasite derived **histidine rich protein (Pf HRP).**[14] These accretions cause humps or knobs on the surface of the red cell and these are the points of attachment to vascular endothelium. Some parasites are knob negative and yet show cytoadeherence.[15] A protein called sequestrin has also been identified.[12,16] Altered red cell membrane components may also play a role. On the endothelium, Leukocyte differentiating antigen CD36, intercellular adhesion molecule 1 (ICAM1), Thrombospondin, VCAM and ELAM have been identified as binding proteins.[16-23] Tumour Necrosis Factor (TNF) upregulates binding to ICAM 1 (not CD36) and binding is higher at low pH and high calcium levels. ICAM 1 is abundant in brain microcirculation and CD36 elsewhere. Among adhesion molecules identified in children with CM in Ibadan include ICAM 1, E-Selectin and PECAM.[24] The presence of G allele at the ICAM-1 exon 6 has been associated with 3.6 times increased risk of severe malaria in Nigerian children.[24]

Rosetting is adherence of parasitised red cells with uninfected red cells. It is independent of venular cytoadherence and exhibits 5 times stronger adhesion than cytoadherence. Rosetting causes higher microvascular obstruction than cytoadherence and is associated with cerebral malaria (cytoadherence with other vital organ damage). Rosetting reduces blood flow, encourages cytoadherence to endothelium, enhances anaerobic glycolysis and reduces the pH. As the parasite matures, flexible biconcave disc becomes progressively more spherical and rigid. Reduced membrane fluidity, increasing

sphericity, enlarging and relatively rigid intra-erythrocytic parasites make the red cells less filterable and cause obstruction at mid capillary level itself. Unbridled ***cytoadherence-resetting-sequestration*** results in poor tissue perfusion, organ dysfunction, anaerobic glycolysis in tissues and lactic acidosis, malfunctioning of dendritic cells and T cells due to CD36 binding. While low levels of cytokines may be beneficial, high levels are found to be harmful, contributing to placental dysfunction, suppression of erythropoeisis, inhibition of gluconeogenesis and increased cytoadherence.

CLINICAL FEATURES OF CEREBRAL MALARIA

The earliest symptom of cerebral malaria in children is usually fever (37.541°C), followed by failure to eat or drink. Vomiting and cough are common; but diarrhoea is unusual. The history of symptoms preceding coma may be very brief commonly one or two days. A child who loses consciousness after a febrile convulsion should not be classified as having cerebral malaria unless coma persists for more than 30 minutes after the convulsion.[3] The depth of coma should be assessed according to the Blantyre coma scale[25] or modified Glasgow coma scale for children by observing the response to standard vocal or painful stimuli (rub knuckles on child's sternum; if there is no response, apply firm pressure on thumbnail bed with horizontal pencil).

Severe malaria is a multisystem disease; cerebral involvement is one of the features. It is mainly a disease of the children in the tropics as about 85% of the cases occur in children less than 5 years.[5] The median age of occurrence is 36 40 months. In areas, where transmission is much lower and protective immunity is not acquired, all age groups can get cerebral malaria, but young adults are the most affected group. In all patients with severe malaria metabolic acidosis is a frequent finding and is important to assess since it has a strong prognostic significance.[26] Kussmaul type respiration can be a warning symptom for this.[27,28] Acidosis is mainly, but not entirely, caused by increased lactic acid production as a result of anaerobic glycolysis. In case of renal failure, acid-base homeostasis will be further compromised. Shock is not a common feature of cerebral malaria and should alert the clinician for the possible concomitant presence of septicaemia.

Neurological symptoms in cerebral malaria

The clinical picture is that of a diffuse encephalopathy with unrousable coma; focal signs are relatively uncommon. In young children coma can develop rapidly, with a mean onset after only 2 days of fever, but sometimes just a few hours.[29] It is often heralded by one or more generalized seizures, which cannot be distinguished clinically from febrile convulsions. In adults the onset in usually more gradual, with high fever (mean duration of 5 days) and increasing drowsiness. Occasionally frankly psychotic behaviour is the first manifestation of cerebral involvement. The level of consciousness may fluctuate over a period of hours. Convulsions are present in about 15% of the cases, whereas more than 50% of paediatric cases have convulsions.[29] Convulsions are most frequently tonic-clonic generalized convulsions, but can also be Jacksonian type or focal. In children approximately 25% have subtle or subclinical convulsions, with seizure activity on electroencephalography, but only minor convulsive movements of limbs or facial muscles.[30] These patients often have deviated eyes, excessive salivation and irregular breathing patterns.

On neurological examination the febrile patient has no signs of meningism, although passive resistance to neck flexio is not uncommon and hyperreflexion of the neck m occur in severely ill patients. The eyes often show divergent gaze, with normal oculocephalic reflexes. Pu and corneal reflexes are usually normal. In a study neurological features of cerebral malaria in 103 childr aged 5 years or less in Ibadan, Nigeria, an area of hig malaria transmission; convulsions occurred in 87% wi most cases being generalized tonic-clinic natur abnormalities of posture were observed in 41%, abnorm tone in 70% and abnormal deep tendon reflexes in 74 Absent corneal reflexes were found in about 14%.[31]

On fundoscopy retinal haemorrhages can be observed about 15% of cases.[32] In areas of high transmission (Su Saharan Africa) a high background prevalence peripheral parasitaemia can hamper the diagnosis 'cerebral malaria'. A positive blood slide in a febr comatose child does not always adequately exclude oth possible diagnoses in this setting. The presence of reti haemorrhages can sometimes be useful here because its specificity for malaria.[33] Cranial nerve involvement patients with cerebral malaria is rare. Bruxism wi grinding of the teeth and a positive pout reflex a common in cases with deep coma. Muscle tone an tendon reflexes are often increased, but can also be norm or reduced. An ankle and less frequently a patellar clon can sometimes be evoked. Abdominal reflexes are abse and the plantar reflexes are extensor in approximately ha of the cases. Various forms of abnormal posturing can b present, with either a decorticate pattern with flex rigidity of the arms and extension of the legs or decerebrate pattern with abnormal extensor responses arms and legs with or without opisthotonos.[3] In survivi patients the median time to full recovery of consciousne is approximately 24 hours in children, compared to hours in adults. In children neurological residu abnormalities are more common, with approximately 12 still having symptoms at the moment of discharg including hemiplegia, cortical blindness, aphasia an cerebellar ataxia.[34] These symptoms will complete resolve over a period from 1 till 6 months in over half of th children, but a quarter will be left with a major residu neurological deficits. More subtle cognitive impairmen as a late consequence of cerebral malaria is common children, especially in those cases presenting with combination of coma, hypoglycaemia and seizures.[35]

DIAGNOSIS OF CEREBRAL MALARIA

Accurate diagnosis is essential to proper management an favourable response in cerebral malaria. The diagnosis cerebral malaria is that of exclusion. The most importa element in the clinical diagnosis of malaria, in bo endemic and non-endemic areas, is to have a high index suspicion. Severe malaria can mimic many other disease that are also common in malaria endemic countries. Th most important of these are all types of meningit typhoid fever and septicaemia. In the majority of cases cerebral malaria, examination of thick and thin films of th peripheral blood will reveal malaria parasites. Thick film are more useful than thin films in the detection of a lo density malaria parasitaemia. Facilities and equipment fo microscopic examination of blood films can be easily se up in the side-room of a clinic or ward, and films can b read by trained personnel on site. This reduces the dela that commonly occurs when samples must be sent to distant laboratory. In general, the greater the parasit density in the peripheral blood, the greater the likelihoo that severe disease is present or will develop. This

true among "non-immune" people. It is to note, however, that some individuals develop and even fatal malaria with a very low peripheral aemia. Very rarely the blood film may actually be in a patient who is then proved at autopsy to have tissue sequestration of parasites.

may be a marked difference between the number of cells in the peripheral blood and the number moreover, rapid changes may be expected in infections. Frequent monitoring of the aemia (every 46 hours) is very important for the first of treatment. The prognostic value of the parasite may be improved considerably by assessing the parasite development in the peripheral blood film. parasitaemia, prognosis worsens if there is a ance of more mature parasite stages. In general, than 50% of the peripheral blood parasites are at ring stage (diameter of the nucleus <50% of the of the rim of cytoplasm), the prognosis is good. If more than 20% of parasites contain pigment (i.e. mature trophozoites or schizonts), the is relatively bad.

ence of malaria pigment in polymorphonuclear (neutrophils) is another useful indicator of the of malaria, especially in anaemic children and in with cerebral malaria associated with absent or parasitaemia. Assessment of peripheral blood phonuclear leukocyte pigment is an extremely relatively accurate prognostic index whereby if than 5% of the polymorphonuclear leukocytes visible pigment the prognosis worsens. Several more costly rapid diagnostic tests are now However, these do not replace microscopy as the standard method for the diagnosis of severe malaria monitoring of its management as they do not the valuable observations mentioned above.

blood smear makes the diagnosis unlikely, but if uncertainty the test should be repeated every for 48 hours. Microscopy with fluorescent the buffy coat (quantitative buffy coat analysis has a higher sensitivity to detect low parasitaemia, is seldom needed. Dipstick detection of the *P.* antigens *Pf*HRP2 and *p*LDH (Parasight-F, ICT OptiMAL), have a diagnostic sensitivity similar microscopy, but do not require an experienced However, parasitaemia and parasite stages assessed in this way. *Pf*HRP2 remains circulating after cure, which can result in false positive results in settings in patients with a recent malaria

aemia, a common feature of severe malaria, ruled out. The principal differential diagnosis in is of a bacterial or viral meningoencephaltits. patient presents with any sign of meningeal a lumbar puncture must be performed. This that in most cases a lumbar puncture will be especially in infants. In some centres, lumbar is postponed because of the fear of herniation raised intracranial pressure which is present in a of their cases. These centres start empiric coverage in all children until results of lumbar become available.

OF CEREBRAL MALARIA

of a child with malaria includes antimalarial therapy, supportive therapy and management of complications that are present at admission or develop during treatment period. The following measures should be applied to all patients with clinically diagnosed or suspected cerebral malaria:

1. The physician needs to make a rapid clinical assessment with special attention to level of consciousness, blood pressure, rate and depth of respiration and pallor.
2. Admit patient to a ward where adequate care and monitoring could be achieved or in an intensive care unit if this is available.
3. If parasitological confirmation of malaria is not readily available, make a blood film and start treatment on the basis of the clinical presentation.
4. Give antimalarial chemotherapy intravenously. Oral treatment should be substituted as soon as reliably possible (once patient can swallow and retain tablets).
5. Calculate doses as mg/kg of body weight. Therefore, weigh the patient. This is particularly important for children.
6. Provide good nursing care. This is vital, especially if the patient is unconscious .
7. Pay careful attention to fluid balance, if fluids are being given intravenously, in order to avoid over- and underhydration.
8. Make a rapid initial check of the blood glucose level, and monitor frequently for hypoglycaemia. If this cannot be done, give glucose
9. Examine the optic fundi by ophthalmoscope. This may help in differential diagnosis, and rarely will reveal papilloedema, which is a contraindication to performing a lumbar puncture. Retinal haemorrhages may be seen but these do not influence management.
10. Record urine output and look for the appearance of black urine (haemoglobinuria) or oliguria which may indicate acute renal failure
11. Monitor the core temperature (preferably rectal temperature), respiratory rate and depth, blood pressure, level of consciousness and other vital signs regularly.
12. Reduce high body temperatures (>38.5°C) by tepid sponging and fanning. Administer paracetamol as an antipyretic if necessary.

Antimalarial treatment in cerebral malaria

The mainstay of the treatment of severe and cerebral malaria is the immediate start of

parenteral antimalarial treatment. Available drugs are injectable quinine, artesunate, and artemether. Intravenous Chloroquine has become obsolete in Africa and Asia and almost the whole of the world because of widespread resistance of the parasite. The artemisinins are currently the most rapidly acting and potent available antimalarial drugs. Unlike quinine they not only act on the mature form of the parasite, but also on the younger ring forms, preventing their maturation and sequestration.[37] Quinine has a narrow therapeutic ratio and should never been given by bolus injection, which can lead to fatal hypotension. Quinine should be given at a dose of 30 mg base/kg/24hours, as a continuous infusion or in 3 divided doses of 10 mg/kg, each given over 4 hours time. The pharmacokinetic properties of quinine alter in severe malaria: the volume of distribution is reduced, whereas the binding to plasma proteins is increased. Plasma clearance is reduced proportional to severity of disease.[38,39] Consequently, doses of intravenous

quinine should be reduced by 30 to 50% after the third day of treatment to avoid accumulation of the drugs in patients who remain unconscious. In the presence of severe kidney or liver failure, the dose reduction should be effectuated after the second day of treatment.[3] The total course is seven days of quinine. Quinine can be switched to the oral formulation, using the same dose of 10 mg/kg t.i.d., if the patient is able to eat. Alternatively quinine can be discontinued and a full course of artemether-lumefantrine can be given after the patient is able to eat. In some centres, administration of a loading dose of 20mg/kg/dose stat is usually given. However, there is no sufficient evidence in support of the superiority of giving a loading dose.[40] Minor adverse reactions are common with quinine therapy and consist of the symptom complex known as cinchonism, with tinnitus, high tone deafness, nausea, uneasiness, malaise and blurring of vision. A side effect that has important consequences for patient management is the induction of hypoglycaemia by quinine through its potent effect on pancreatic insulin secretion. Monitoring, at least every 3 hours, of plasma glucose levels is therefore indicated during treatment with quinine. Severe life-threatening adverse effects, such as hypotension, myocardial conduction disturbance, blindness, deafness, and coma are rare and related to plasma doses above 20 mg/l, which should not be reached during treatment with the recommended scheme.

Intramuscular artemether is given in a dose of 3.2 mg/kg on admission, followed by a daily dose of 1.6 mg/kg. Clinical trials comparing artemether with quinine show that artemether is safe and easier to use than quinine but overall survival is similar and not statistically significantly better than quinine.[41,42] Artemether is an oil-based formulation, which releases drug slowly and erratically from the injection site.[43] In some patients, particularly in those who are severely ill, artemether may not be absorbed for up to 12 hours after injection. Because of these pharmacokinetic disadvantages parenteral artemether is not the artemisinin derivative of choice for the treatment of severe malaria.

A third alternative in the treatment of cerebral malaria is artesunate which is given as 2.4 mg/kg on admission, followed by the same dose after 12 and 24hours, and then daily until the patient is able to take oral medication. To prevent recrudescence of the infection, follow on medication should be given. Several regimens are possible, such as a full course of oral artemether-lumefantrine (Co-artemR), or a combination of oral artesunate (2 mg/kg per day, total course 7 days including the parenteral form) and amodiaquine (10 mg/kg per day for 3 days). Mefloquine is not recommended as maintenance antimalarial drug, because of its association with post-malaria neurological syndrome.[44]

Supportive treatment

Patients with coma may need endotracheal intubation and mechanical ventilation to protect the airway, if this facility is available. The usual nursing care for the unconscious patient should be applied (such as regular turning, nasogastric tube, eye care, urethral catheter). Convulsions are very common in children with cerebral malaria, but choice and dose of a seizure prophylactic drug has not been well established and is currently not recommended.

Nursing care

Good nursing care of the patient with cerebral malaria is vital, it is important to ensure meticulous nursing care. This can be life-saving, especially for the severely unconscious patient. Maintain a clear airway. Nurse the patient in the lateral or semi-prone position to avoid aspiration of fluid. Insert a nasogastric tube and suck out the stomach co minimize the risk of aspiration pneumonia. A pneumonia is a potentially fatal complication tha dealt with immediately. Turn the patient every 2 h do not allow the patient to lie in a wet bed. Pay attention to pressure points. Keep a careful reco intake and output. If this is not possible, weigh t daily in order to calculate the approximate fluid ba necessary to note any appearance of bl (haemoglobinuria) and check the speed of i fluids frequently. Too fast or too slow an infusi dangerous. Monitor the temperature, pulse, re blood pressure and level of consciousne observations should be made at least every 4 hou patient is out of danger. Report changes in th consciousness, occurrence of convulsions or c behaviour of the patient immediately; all such suggest developments that require additional tre the rectal temperature rises above 39 °C, re patient's clothes and start tepid sponging and fa paracetamol (the rectal route is usually best).

PROGNOSTIC FACTORS IN MANAGEM CEREBRAL MALARIA

The major indicators of a poor prognosis in ch adults with cerebral malaria are listed below.

Clinical indicators of poor prognosis

1. Age under 3 years
2. Multiple convulsions, more than twice in
3. Absent corneal reflexes
4. Decerebrate/decorticate rigidity or opist
5. Clinical signs of organ dysfunction (e.g. re pulmonary oedema)
6. Respiratory distress (acidosis)
7. Circulatory collapse
8. Retinal haemorrhages
9. Concomitant bacterial infections

Laboratory indicators of poor prognosis

1. Hyperparasitaemia (>250 000/µl of bloo RBC infected)
2. Peripheral blood polymorphonuclear le (>12 000 µl)
3. Mature pigmented parasites (>20% of pa
4. Packed cell volume less than 15%
5. Haemoglobin concentration less than 5 g
6. Blood glucose less than 2.2 mmol/l (<40
7. Blood urea more than 60 mg/dl
8. Serum creatinine more than 265 µ mg/dl)
9. High CSF lactic acid (>6 mmol/l) an glucose
10. Raised venous lactic acid (>5 mmol/l)
11. More than 3-fold elevation of serum (aminotransferases)

Table I: Blantyre coma scale

		Score
Best motor response:	localizes painful stimulus	2
	withdraws limb from pain	1
	nonspecific or absent response	0
Verbal response:	appropriate cry	2
	moan or inappropriate cry	1
	none	0
Eye movements:	directed (e.g. follows mother's face)	1
	not directed	0
Total		**0–5**

A state of unrousable coma is reached at a score of <3.

12. Increased plasma 5'-nucleotidase
13. Low antithrombin III levels
14. Very high plasma concentrations of tumour necrosis factor (TNF)

Common sources of errors in diagnosis of cerebral malaria

The common errors in the diagnosis and management of severe malaria are listed below.

1. Failure to think of malaria in a patient with either a typical or atypical illness
2. Failure to elicit a history of exposure (travel history) including travel within a country with variable transmission
3. Misjudgement of severity of the illness
4. Failure to do a thick blood film in a non-immune patient
5. Failure to identify *P. falciparum* in a dual infection with *P. vivax* (the latter may be more obvious)
6. Missed hypoglycaemia
7. Failure to diagnose other associated infections (bacterial, viral, etc.)
8. Misdiagnosis (e.g. influenza, viral encephalitis, hepatitis, scrub typhus, etc.)
9. Failure to recognize respiratory distress (metabolic acidosis)
10. Failure to carry out an ophthalmoscopic examination for the presence of papilloedema, and retinal haemorrhages in adults.

Common sources of errors in management of cerebral malaria

1. Inadequate nursing care
2. Delay in starting antimalarial therapy
3. Use of inappropriate therapy:
   a. Chloroquine in areas of resistance
   b. unjustified withholding of an antimalarial drug
   c. dosage not correctly calculated
   d. inappropriate route of administration
   e. unjustified cessation of treatment
   f. failure to prevent cumulative effects of antimalarial drugs
   g. failure to switch patients from parenteral to oral therapy as soon as they can take oral medication
   h. unnecessary continuation of chemotherapy beyond the recommended length of treatment
   i. use of unproven and potentially dangerous ancillary treatment
   j. failure to review antimalarial treatment in a patient whose condition is deteriorating
   k. Errors of fluid and electrolyte replacement
   l. failure to control the rate of intravenous infusion
4. Failure to elicit a history of recent chemotherapy
5. Failure to identify or treat metabolic acidosis
6. Unnecessary endotracheal intubation
7. Unduly delayed endotracheal intubation (where this is indicated and possible)
8. Failure to control convulsions
9. Failure to recognize minor ("subtle") convulsions
10. Failure to recognize and treat severe anaemia
11. Delay in considering obstetrical intervention in late pregnancy
12. Failure to recognize and manage pulmonary oedema
13. Undue delay in starting peritoneal dialysis or haemodialysis
14. Failure to pass a nasogastric tube to prevent aspiration pneumonia
15. Failure to give antibiotics as a covering procedure if the decision is made to delay lumbar puncture.11

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CRITICAL CARE OF THE PREMATURE INFANT

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Abstract

Pre-term birth constitutes a significant cause of perinatal morbidity and mortality worldwide. It is known to contribute about 38 to 52 percent of all perinatal mortality worldwide. Half of all neonatal deaths occur in the pre-terms. Most of these morbidity and mortality occur in developing countries.

Severe neonatal conditions that are usually associated with increased morbidity and mortality are commoner in the pre-term than term babies. Such conditions include respiratory distress syndrome, necrotising enterocolitis, intraventricular haemorrhage, septicaemia and broncho-pulmonary dysplasia. The pre-terms are also at higher risk of developing long-term impairment such as cerebral palsy, hearing and visual impairment as well as learning disabilities. The medical care of the premature infant is a complex combination of developmental physiology, evidence based interventions and clinical experience. *Wide variability in approach to care of these infants exists among practitioners and neonatal intensive care units, as does variability in outcomes. High technology characterises preterm care in the developed world with nil or low availability of such in the developing world like Nigeria.*

INTRODUCTION

Infants who are born before 37 weeks of gestational age are defined as premature.

These infants usually weigh less than 2500g [1].

Preterm birth is a major cause of perinatal morbidity and mortality worldwide. A large proportion of these morbidity and mortality occur in the developing countries. In the developed world, survival rates for premature infants have greatly improved over the past two decades. Care of premature infants with birth weights between 1000 and 1500g has become almost routine in Newborn Intensive Care Unit (NICU) [2]. The newest frontier in neonatology is the care of premature infants of less than 1000g birth weight, also known as Extremely Low Birth Weight (ELBW) infants. These infants present one of the greatest medical and ethical challenges to the field. Although they represent a small percentage of overall births and NICU admissions, ELBW infants are the most critically ill and at highest risk of mortality and long term morbidity of any NICU patient.

Critical care of these infants are in constant evolution, owing to new discoveries in

both basic and clinical research as well as growing clinical experience.

PERINATAL MANAGEMENT

Short-term outcomes of extremely premature infants are improved if they are delivered in a high-risk centre rather than being transported after birth [3]. Therefore, if clinically feasible, the pregnant mother who seems likely to deliver an extremely premature infant should be transferred to a high-risk perinatal centre for expertise in obstetrics and neonatology. Upon arrival, she should be evaluated for factors that may have predisposed to preterm labour and assessed for the status of the foetal membranes and the presence or absence of chorioamnionitis.

In addition, best obstetrical estimate of gestational age (by date of last menstrual period and early ultrasonographic dating, if available), ultrasonographic assessment of foetal size and position, and the presence of other medical and obstetrical complications (preeclampsia, placenta previa, abruption placentae, etc) should be documented. Specimens for cultures should be obtained on admission, based on findings on history and physical examination. Prophylactic treatment should be considered to prevent premature rupture of foetal membranes, endometritis and chorioamnionitis as they may lead to premature delivery [4]. Mothers with potential cases of preterm delivery should be treated with either betamethasone; 12mg daily for two doses or dexamethasone 6mg every 12hours for four doses. This has been shown to significantly reduce the incidence and severity of respiratory distress syndrome, necrotising enterocolitis and intraventricular haemorrhage [5].

GENERAL PRINCIPLES OF CARE SPECIFIC TO THE PRETERM INFANT OF EXTREMELY LOW BIRTH WEIGHT

DELIVERY ROOM CARE

The first few hours of admission to the NICU are critical to the survival of the preterm infant. Careful adherence to details in the delivery room and during the first two hours after birth is essential to help avoid some of the immediate and long-term complications of the preterm infant.

At delivery strict attention to maintenance of body temperature by means of rapid, gentle drying of the infant and use of adequate heat sources is paramount to avoid cold stress. Intubation of preterm infants for respiratory support and prophylactic administration of natural surfactant in the delivery room within the first 15 minutes of birth has been effective in preventing respiratory distress syndrome. It is as well considered as a rescue strategy for established respiratory distress syndrome when given within 30 to 60 minutes of birth. At delivery room ensure good thermoregulation, and 'gentle' ventilation as required (while avoiding hyperventilation and hypoxia) [4].

MISSION TO THE NEONATAL INTENSIVE CARE T (NICU)

nfants should be weighed upon admission; frequent mination of subsequent weights is an invaluable managing fluid intake.

MPERATURE AND HUMIDITY CONTROL

nstant thermoneutral environment is essential to rvival of the highrisk preterm infant. This is as a of a relatively larger surface area to weight ratio ounting for the excessive evaporative heat loss. To ain minimal evaporative heat loss, it is best if the nmental humidity is 40-60%. Low ambient dity requires higher ambient temperature to ain infant temperature between 36.4 and 37.1°C. m infants can be managed initially either in ble-walled incubators or under radiant heaters with ective plastic sheeting or covers. Radiant warmers e accessibility to the baby but produce larger nsible heat and water losses and slightly higher ermetabolic rates than incubator[4].

ll infants receiving mechanical ventilation ecially those weighing 1000g, humidification and ming of administered respiratory gases are ortant to minimize insensible fluid losses. In-line ming of respiratory gas circuits minimizes 'rain-out' re-humidified air and oxygen and maintains airway perature, which should be as close as possible to

ses of increased insensible water loss and mature renal function, these infants have increased d requirements, necessitating intravenous fluid n some cases, infusion of fluids through the bilical artery or percutaneous catheter. The fluid irement for a preterm is 80-90mls/kg/day for the day of life with additional 20mls/kg/day until the of first week. By the end of first week, 160-ml/kg/day is usually recommended.

herapy should be evaluated at least twice daily the fluid intake should be adjusted accordingly. ring of fluid status is done by measurements of y weight, urine output, urine specific gravity, emodynamic measurements, serum sodium and more reliable parameters, such as haematocrit and examination.

Daily weight: this is the most important method itoring fluid therapy. Preterm neonates lose 0-3% heir birth weight in the first week of life. Greater ght loss is considered excessive, and fluid ement must be carefully reviewed.

(b) Urine output and specific gravity: monitoring urine output is the second most important method of monitoring fluid therapy. In addition to urine volume, urine specific gravity should be determined to check the renal function and state of hydration.

(a) First 12hours: Any amount of urine is acceptable.

(b) 12-24 hours: the minimum acceptable urine output is 0.5ml/kg/h

(c) Second day and beyond: Normal urine output for the second day of life, may increase to 3.0-3.5ml/kg/h, 4.5ml/kg/h is excessive and indicates early fluid overload which may lead to electrolyte loss. Urine specific gravity should fluctuate between 1.006-1.015.

Values outside this range warrant re-evaluation of fluid management and environmental humidity control[6].

Haemodynamic monitoring: is a valuable tool in assessing fluid status in the preterm neonate. Accelerated heart rate of the preterm which averages 140-160 beats per minute (bpm), is generally considered within normal limits.

Tachycardia with a heart rate in excess of 160 bpm may be a sign of hypovolaemia (Normal Heart Rate of a preterm neonate is 120-140 bpm). An arterial blood pressure (ABP) of 85-90/55-65mm Hg and a central venous pressure (CVP) of 4-6mm Hg are considered to be normal ranges[6]. If the CVP value is consistently lesser than 4mm Hg, the infant is volume depleted, and a transfusion may be necessary. Increased CVP readings indicate hypervolaemia or cardiogenic shock. CVP should be used in combination with Mean ABP and other parameters to assess haemodynamic stability[6].

ELECTROLYTES

Serum electrolyte levels should be monitored at least twice daily or every 4-8 hours for the most immature infants. Sodium is added as diuresis begins and potassium is added after urination has been established.

(a) Sodium: Initially infants have sufficient sodium level and require no sodium intake. However, when the serum sodium level begins to decrease (usually on the 3rd to 5th days of life), sodium should be added to the intravenous fluids (3-8 meq/kg/day of sodium).

(b) Potassium: During the first 48 hours after birth, preterm infants are prone to the development of increased serum potassium levels of 5meq/L and above. The increase is mostly as a result of immature renal tubular function. It is recommended that clinicians do not give potassium at this time.

Usually by third to sixth day, the initially elevated serum potassium level begins to decrease. As potassium levels approach 4meq/l, add supplemental potassium to intravenous fluids. Begin with 1-2 meq/kg/day. Measure serum potassium every 6-12 hours until the level is stabilized at 3.5-4.5 meq/L[6].

BLOOD GLUCOSE

Preterm infants should be supported with 4-6mg/kg/min glucose infusions. This support can usually be achieved by starting with 5% dextrose solution. Meter testing of blood glucose should be performed every 2hours until a stable blood glucose level (50-90mg/dl) is established. All urine samples should be checked for glycosuria. Trace of glycosuria is acceptable and may occur with a blood glucose level as low as 120mg/dl but higher levels of serum glucose and glycosuria require recalculation of glucose administration and total fluid administration[6].

CALCIUM

Serum calcium should be monitored once or twice daily. When serum calcium decreases below 7.5mg/dL, treatment with calcium gluconate should be instituted[6].

NUTRITIONAL SUPPORT

Table 1.0; use of parenteral alimentation solutions[8].

(ml/kg/d)	Volume	Carbohydrate (g/dL)	Protein (g/kg)	Lipid (g/kg)	Calories (kcal/kg
Peripheral: short term(7-10 days)					
Starting solution	100-150	D10W	2	1	46-64
Target solution	150	D12.5W	3-3.5	3	102
Central: long term (>10days)					
Starting solution	100-150	D10W	2	1	46-64
Target solution	130	D20W	3-3.5	3	123

The average caloric requirement for a growing preterm is 140cal/kg/day. Expected weight gain for the adequately nourished preterm infant is 10-30g/day. If infant is metabolically stable, parenteral nutrition should be started on the second or third day of life and continued until infant is receiving sufficient enteral feeding to promote growth. Parenteral alimentation solutions can be given either peripherally or centrally via an umbilical vein line or percutaneous catheter (Table 1.0)[8].

There are now available formula feeds for premature infants. Iron supplementation (2-4mg/kg/day) is recommended for premature infants starting from about age 2 months. Vitamin supplementation is also important (vitamin A: 1200 units/d; vitamin C: 20mg/d, vitamin D: 400 IU/d, vitamin E: 3mg/d).

RESPIRATORY SUPPORT.

The smaller the infant, the weaker the muscles of respiration. Virtually all of these infants initially require mechanical ventilation but it is prudent to electively intubate these infants in the delivery room. It is of paramount importance to monitor the ventilatory status of the infant by blood gas sampling, continuous oxygen monitoring using pulse oximetry, infant's appearance (presence of cyanosis), X-ray studies e.t.c. The use of Apnoea monitors will assist in early detection and monitoring of prolonged cessation of respiration.

PECULIAR PROBLEMS OF PRETERM NEONATES

Problems peculiar to preterm neonates include respiratory distress syndrome, bronchopulmonary dysplasia, apnoea of prematurity, necrotizing enterocolitis, neonatal sepsis, patent ductus arteriosus, anaemia, jaundice e.t.c.

Prophylactic administration of natural surfactant or use of glucocorticoids may be given as a prophylaxis for or treatment of respiratory distress syndrome. Strict aseptic procedures should be maintained and rational use of antibiotics should be considered if there is sepsis. When features of apnoea are seen, bag and mask ventilation should be used to relieve cyanosis and bradycardia. The first line treatment is the use of methylxanthines e caffeine. Fluid overload, metabolic acidosis, hypoxia a anaemia should be prevented as they may cause pa ductus arteriosus. If present treatment with oxygen, f restriction and Indomethacin (if less than 2 we postnatal) is recomended. There also seems to be mi moderate hyperbilirubinaemia in these infants. S efforts should be made to keep the serum bilir below 10mg/dL. Phototherapy can be used to red serum bilirubin, but in cases where serum bilirubin r to ten times the body weight, exchange bl transfusion (rule of ten) should be performed. If feat of anaemia are present, treatment is done accordin severity. If anaemia is severe and shock is pres transfusion of 15-20ml/kg of whole blood over 5 minutes should be done and if anaemia is severe shock is absent, 10-15ml/kg of packed cells at a rate o 3mls/kg/hr should be transfused.

The above series of management are excellent a yield great successes in centres where health ser delivery is optimal. The situation in most develo countries like Nigeria is not so to support such deli of optimal care.

Basic equipments like functioning incubators, rad warmers, phototherapy lamps and some NICU esse drugs e.g. surfactant, caffeine etc, may not be avail In such centres, adaptation of the above manageme usually done. For instance, locally made photothe stands with local fluorescent lamps are used in management of neonatal jaundice. Such lamps are used as alternatives to radiant warmers.

Most centres do not have parenteral nutrition; he use available intravenous fluids in 10% dextrose. Pre formulas or breast milk is commenced as soo possible. There is also increasing awareness of th 'kangaroo' mother care in some centres.

Detailed haemodynamic assessment may also limited by non-availability of CVP measu equipments. The basic clinical cardiovasc assessment (pulse volume, pulse rate, capillary refi then crucial in such situations. The starring proble

poverty is also a major limiting factor in rendering adequate biochemical assessment for such babies. Antibiotic therapy is also hampered because of such lack of funds. All these play significant role in the eventual outcome of such babies.

CONCLUSION

The critical care of the premature infant requires a combination of knowledge of developmental physiology, evidence based interventions, clinical experience and availability of neonatal care facilities.

In Nigeria, the few available neonatal care facilities and personnel are concentrated in the tertiary centres that take care of a very small proportion of our deliveries. Ignorance, poverty and incessant power failure impact negatively on the critical care of these preterm infants. The reduction of perinatal morbidity and mortality from preterm births lies on the prevention of preterm delivery, early identification and prompt management of their critical problems.

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INITIATION OF BREASTFEEDING AND THE FIRST MEAL OF NEWBORNS IN A NIGERIAN TEACHING HOSPITAL

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Abstract

The promotion of breastfeeding is a key child survival strategy. About 4 million babies die in the first month after b across the developing world according to estimates and a lot of this could be prevented if all mothers of newborns wo initiate breastfeeding within one hour of giving birth.

A cross-sectional study of all mothers of newborns in a randomly selected lying-in ward in the University College Hosp Ibadan, Nigeria was conducted to find out the initiation time of breastfeeding in newborns and their first meals.

In all, 26 mothers of newborns were interviewed and they had a mean age of 29.9 ± 4.1 years. Twenty five (96.2%) h antenatal care, 17 (65.4%) had operative delivery and 9 (34.6%) had spontaneous vaginal delivery. The vast majority (92.3%) of the newborns had infant formula as their first meals and the decision was initiated by a nurse.

The mean initiation time for breast feeding was 24.0 ± 16.9 hours and 58.0 ± 23.9 hours for babies born per vaginum Caesarean section respectively.

Breast feeding initiation time in the University College Hospital, Ibadan appears to be much longer than what has b recorded in many literatures from other health institutions and the commoner first meal of newborns was infant formu However, a larger study is highly indicated to confirm the findings of this study.

Introduction

The promotion of breastfeeding is a key child survival strategy and a major component of at least two (#1 and #4) of the eight Millennium Development Goals (MDGs) of the United Nations[1]. Breastfeeding has been with us since antiquity but its practice has been fraught with a lot of misconceptions. In 1990, *participants at the WHO/UNICEF policymakers' meeting in Innocenti, Italy made a declaration that:*

"As a global goal for optimal maternal and child health and nutrition, all women should be enabled to practise exclusive breastfeeding and all infants should be fed exclusively on breastmilk from birth to 4-6 months of age. Thereafter, ***children should continue to be breastfed****, while receiving appropriate and adequate complementary foods,* ***for up to two years of age or beyond"***[2].

This was followed by the Baby-friendly Hospital Initiative of 1991 and then the Global Strategy for Infant and Young Child Feeding in 2002. The initiation of breastfeeding was proposed to be started within few minutes to one hour after birth. It is estimated that every year, in the develop world, about 4 million babies die in the first mon after birth and if all women commen breastfeeding within one hour of putting to b about 1 million of these babies could be save Late or delayed initiation of breastfeeding (usua after one day) is reported to be associated with 2.4-fold increase in risk of neonatal death Therefore, the early initiation of breast feeding is utmost importance in child survival.

Exclusive breast feeding is traditionally rare Nigeria as water and concoctions are usually giv to babies and sometimes this is done very soo after birth. It has also been observed that ma mothers are told to rest after childbirth and the babies are fed with glucose drinks or infant formu milks while they rest. This study therefore set out document the first meal given to newborns in Nigerian tertiary hospital and the time of initiati of the meal.

Methodology

The University College Hospital (UCH) is Nigeri

foremost teaching hospital and a Baby-friendly hospital. It is an 800-bedded hospital with 3 lying-in wards. As a pilot study to a much larger study, one of the lying-in wards in UCH was selected by simple random sampling and a cross sectional study was conducted with total sampling of all the mothers of newborns on the wards after informed consent was sought and obtained from them. All the 26 mothers of newborns on the selected ward consented and were interviewed with the aid of an interviewer administered semi-structured questionnaire. The questionnaire was developed in English, translated into Yoruba and back-translated to English to ensure exactness. The Yoruba version was administered on the Yoruba speaking respondents. The questionnaire contained questions on socio-demographic characteristics, antenatal care attendance, age of pregnancy at booking, baby's first meal, time of initiation of first meal, initiator of first meal and baby's current meal(s) among other things.

Result

Socio-demographic characteristics

Twenty six mothers of newborns were interviewed and their ages ranged from 23 to 41 years with a mean of 29.9 ± 4.1 years. All the respondents had some form of formal education with 15 (57.7%) having post secondary education. Their occupations varied from trading, 8 (30.9%) to fashion designing, 1 (3.8%). Please see Table 1.

Antenatal care and delivery

Only 1 (3.8%) did not attend an antenatal clinic (ANC) at all. Others attended ANC ranging from 3 to 20 times. Nine (34.6%) had spontaneous vaginal delivery while 17 (65.4%) had Caesarean section. Please see Figure 1.

Infant feeding choices

The majority, 24 (92.3%) of the newborns were fed on infant formula while only 2 (7.7%) had breast milk as their first meal. All the 17 (100%) newborns delivered through operative procedure and 7 (77.8%) of the ones delivered per vaginum were given infant formula as their first meals. In all the 24 cases where infant formula was given as first meal, the decision was initiated by a nurse. At the time of the study, breastfeeding had been initiated in only 5 (55.6%) of the 9 newborns who were delivered per vaginum. Two (50%) of the remaining 4 were less than one day old (4 hours and 19 hours old) and the other two were 3 days old. One of the two 3-day olds was born to an HIV positive mother and was on formula feed only. The mothers of the remaining 3 claimed that they were not yet lactating and hence formula feeds were commenced.

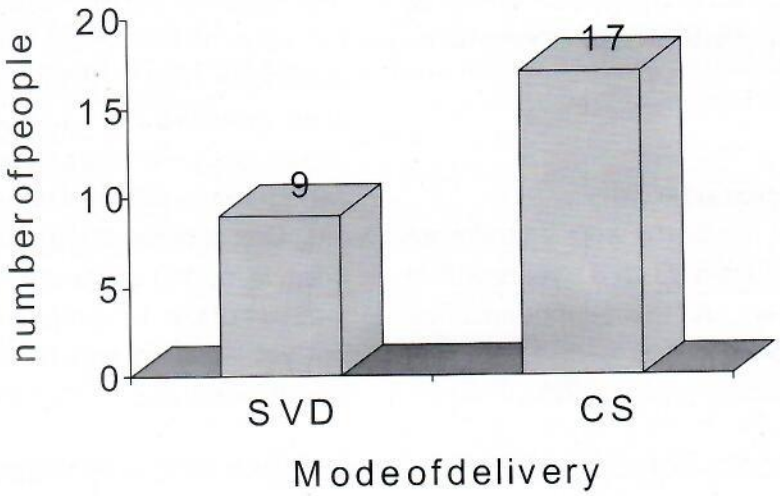
Table 1: Socio-demographic ch aracteristics of the mothers

Variables	n = 26
	n (%)
Age	
≤ 30	16 (61.5)
≥ 31	10 (38.5)
Education	
Primary	2 (7.7)
Secondary	9 (34.6)
Post secondary	15 (57.7)
Occupation	
Unemployed	3 (11.5)
Student	3 (11.5)
Trading	8 (30.9)
Fashion designing	1 (3.8)
Teaching	3 (11.5)
Civil service	4 (15.4)
Professional	4 (15.4)

Table 2: Baby's first meal and the initiator

Variable	Number	
	n (%)	
Baby's first meal	Mode of delivery	
	C/S	SVD
Breast milk	0	2 (22.2)
Infant formula	17 (100)	7 (77.8)
Initiator of first meal	n (%)	
Nurses	24 (92.3)	
Mother	2 (7.7)	

Figure 1: Mode of delivery



No mother who had Caesarean section initiated breast feeding early (within 24 hours). The mean time for initiation of breast feeding was 24.0 ± 16.9 hours and 58.0 ± 23.9 hours for babies born per vaginum and Caesarean section respectively. The difference between the two means was statistically significant (p = 0.012).

Discussion

The mean age of the mothers is comparable to what was found by Nakao et al in Japan[5]. The sample used for the study had an exceptionally high proportion of Caesarean section contrary to what obtained in most other studies reviewed in Nigeria and elsewhere[5,6,7]. This may be due to the fact that UCH is a tertiary hospital and a referral center.

Majority of the newborns were first fed with infant formula. This was surprising especially as UCH has a baby friendly policy which has been in place since 1992.

The fact that nurses initiated the use of infant formula in all babies who had it attested to the fact found in other studies that health workers have great influence in the choice of infant feeds and initiation of breast feeding in newborns[8,9,10]. Comparably with similar studies, initiation of breast feeding was delayed in babies born via Caesarean section more than babies born per vaginum[6] but the mean initiation time found in this study for babies born through the operative route was much higher than what was found in Port Harcourt by Awi and Alikor[11].

In conclusion, the initiation of breastfeeding was delayed in the study population and the first meal of most newborns was infant formula. These two points are not in consonance with the Baby-friendly hospital initiative to which the study site is signatory. It is therefore recommended that re-training of the

medical and nursing staff at the study site should be undertaken and conducted at regular intervals. A much larger study is also highly indicated to confirm the findings of this study

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NEONATAL MORTALITY IN DEVELOPING COUNTRIES – A CALL FOR ACTION

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Abstract

That the mind exerts a number of influences vis-à-vis disease manifestation is no longer an issue; however, there is need for further elucidation in light of the mechanisms involved. Against the conceptual background of Pavlov's conditioned experiments which accentuated how stimuli of purely psychogenic nature mediate and affect cognitive state, hence, shaping physiological function, we seek to understand how the triad of the brain, the mind and our human experiences (in the context of disease) interact. With the methodological enquiry of archival research, we attempt a systematic examination and explication of how this interaction occurs in the context of gastric ulceration. On this note, we argue that this psychocognitive frame of reference would offer creative insight into the feasibility of employing alternative means in the clinical palliation of forms of gastric ulceration that have hitherto been inamenable to organic aetiology.

Keywords Gastric ulceration, Thought, Classical conditioning, Gastric secretion

Introduction

The neonatal period which is the first 28 days of life is a very vulnerable period in the life of an individual when newborn infants readily succumb to illness and death[1]. Up to 40% of the 10 million under five deaths recorded annually occur during the neonatal period.[2] This is twice as high as the number of deaths attributable to Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS)[1]; whereas HIV/AIDS receives so much attention as a global emergency it is not so with neonatal deaths.

Almost all (99%) of these neonatal deaths occur in the developing countries with two-thirds occurring in Africa and South East Asia.[3] High neonatal mortality rates remain a problem in developing nations of Sub-Saharan Africa because of poverty and poor planning of health services.[4] Nigeria's current neonatal mortality rate (NMR) is 48 per 1000 live births while infant and under five mortality rates are 100 and 210 per 1000 live births respectively.[5] The fourth Millennium Development Goal (MDG-4) commits the international community to reducing under five mortality rate by two-thirds of the 1990 figures by 2015.[6] But this cannot be achieved without a significant reduction in neonatal mortality, the causes of which are largely preventive.

Majority of the neonatal deaths in developing countries are not recorded in any formal registration system hence global analysis are extrapolated from national demographic surveys.[7] Population-based information about neonatal deaths in high-mortality settings is largely dependent on verbal autopsy.[8] There is paucity of data on neonatal mortality in Nigeria, most available studies are institution-based and retrospective.

The Burden of Neonatal Mortality

Neonatal mortality rate is the number of deaths in the first 28 days of life per 1000 live births.[1] It is a reflection of the effectiveness of obstetric and neonatal services in any particular community. The burden is greater in developing countries as most of the developed countries have made significant and sustained efforts at reducing neonatal deaths to the barest minimum. Neonatal mortality accounts for about 40% of all mortality in children under the age of five years globally and as much as two-thirds of infant mortality which translates to about 4 million deaths yearly.[3]

Globally, countries are rated as low-mortality countries when NMR is less than 15 per 1000 live births, moderate-mortality when NMR is greater than 15 but less than 45 per 1000 while high-mortality countries are those with NMR greater than 45 per 1000. In most of the industrialised countries NMR is less than 5 per 1000 live births. For example the NMR in Singapore is 1 per 1000. Finland and Sweden (2 per 1000), Australia, Norway and France (3 per 1000), Canada, Netherlands and United Kingdom (4 per 1000) while in the United States of America and United Arab Emirate (UAE) it is 5 per 1000. These countries were able to achieve this great feat because of the widespread provision and utilization of antenatal care, good obstetric practice and neonatal care. The technological advancement of the developed nations in neonatal intensive care, perinatal monitoring of maternal and fetal well-being and good thermal care has led to further reduction in neonatal deaths even among the very premature babies.[9] This is in contrast to what obtains in developing countries.

Of the 20 countries with the highest NMR, 80% are in Sub-Saharan Africa. These countries include Nigeria, Ethiopia, Mozambique, the Sudan and Tanzania.[3]

Generally, NMR in Sub-Saharan Africa ranges from 13 per 1000 live births in Kenya to 108 in Senegal.[3] In Southeast Asia, the countries with the highest NMR include India (where 25% of the global neonatal deaths occur), China, Pakistan and Bangladesh.[10] The high NMR in Sub-Saharan Africa and Southeast Asia is largely due to the prevailing poverty, national economic instability, illiteracy, inequitable health services and harmful cultural practices.[11]

It is also worthy of note, that not only is the burden of neonatal deaths high in developing nations, the conditions that contribute to mortality may also cause severe and lifelong disability. For example, it is estimated that over one million children who survive perinatal asphyxia each year develop problems such as cerebral palsy and learning difficulties.[12] Low birth weight and preterm infants in later childhood often experience impaired cognitive development affecting their long term opportunities and life-chances.[13]

The burden of neonatal deaths has been reported by some researchers. Meme[14] in a hospital-based prospective study in Kenya in 1977 reported a NMR of 33.3 per 1000 live births while Greenwood *et al*[15] in 1986 reported a figure of 65 per 1000 in the Gambia. These two studies identified socioeconomic factors and poor obstetric care as being contributory to the neonatal deaths.

While studies in Zimbabwe[16] showed a rising trend in NMR between 1980 and 1989 probably as a result of maternal demographic changes and rising prevalence of HIV/AIDS in that country, there was a decrease in Ghana[17] between 1995 and 2005. The reduction was attributed to the collateral effect of multiple health research activities undertaken in the area which improved health education campaign, access to health services and antenatal attendance. Dawodu *et al*[18] found a very low NMR of 6.7 per 1000 in the United Arab Emirates which is still higher than those obtained in industrialized countries such as United Kingdom and Japan.

In Nigeria, Okolo and Omene[19] in Benin over an 8 year period (1974 – 1981) reported a decline in NMR from 49.5 per 1000 in 1974 to 16.4 per 1000 in 1981. The decrease was attributed to reduction in deaths resulting from perinatal asphyxia and also the improved national economy as a result of the oil boom. Barely ten years later and in another locality, Ogbonna and Olanrewaju[20] (1991 – 1992) in Sagamu recorded a high rate of 50.88 per 1000. The downturn in the national economy at that time could be partly responsible for this increase.

Causes of Neonatal Deaths

Data on Causes

Only about 1% of the global neonatal deaths occur in countries with good records that are reliable for cause-of-death (COD) analysis. In developing countries where majority of the deaths occur, population-based enquiry is largely dependent on verbal autopsy. The Child Health Epidemiology Reference Group[21] estimated the major causes of death as preterm birth (27%), sepsis/pneumonia (26%), perinatal asphyxia (23%), tetanus (7%) and diarrhoea (3%). Of the remaining 14%, 7% were related to congenital malformations (Figure 1).

Variation in causes of neonatal deaths is seen between and within countries and closely associated with NMR level. Where the NMR is high, more than 50% of the deaths are due to infections, whereas in countries with low NMR, prematurity and congenital malformations are the leading causes of deaths.[1]

Neonatal Infections

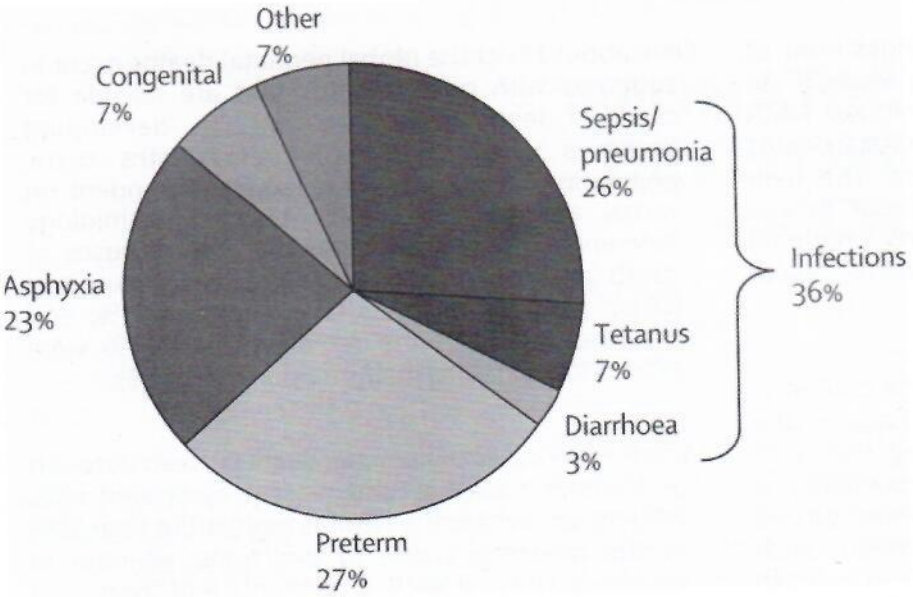
Neonatal infections are the single most common killer of newborns.[11] In countries like India and Nigeria, infections account for 20 – 40% of the neonatal deaths.[20] Apart from intrinsic predisposing factors such as relatively immature cellular and humoral immunity of newborns, factors such as maternal peripartal infections, prolonged rupture of membrane, antepartum haemorrhage, unhygienic delivery, poor hand-washing techniques, poor cord hygiene, and contaminated feeds among others place the newborn at higher risk of developing infections. It is important to note that neonatal tetanus, a totally preventable condition still accounts for about 7% of the annual deaths globally, mostly in developing nations. This is unacceptable as tetanus has ceased to be a major killer of babies in the developed world, even before tetanus toxoid vaccine was developed.

Low Birth Weight

The contribution of low birth weight (LBW) to neonatal mortality is enormous. An estimated 20 million LBW (i.e. weighing less than 2,500g) are born annually.[22] LBW may be due to preterm birth, intrauterine growth restriction (IUGR) or both. NMR is known to be inversely proportional to birth weight.[18,19] The proportional contribution of LBW to neonatal deaths ranged between 50 and 85% in studies from Nigeria.[19,20] The regional estimate for preterm birth for Africa is about 12 percent which is almost double the frequency in European countries.[11] LBW babies are prone to several complications such as infections, hypoglycaemia and hypothermia and are 13 times more likely to die than normal weight babies.[1]

Perinatal Asphyxia

This is responsible for 280,000 deaths in Sub-Saharan Africa annually.[11] Babies born in Africa have a very high risk of perinatal asphyxia and of intrapartum stillbirth.[9] This has been attributed to lack or poor



antenatal care, unskilled birth attendance, home delivery, non-availability of emergency obstetric care, poor neonatal resuscitation skills which are prevalent. This increases their chance of dying on the first day of life.[1] In technologically advanced countries, the risk of perinatal asphyxia is minimal because of adequate monitoring of labour, prompt interventions at delivery and effective neonatal resuscitation.[1]

Congenital Malformations

Lethal congenital malformations are responsible for about 7% of the global burden of neonatal deaths.[21] Major malformation involving the cardiovascular, respiratory, renal and the nervous systems compromise the chances of survival of the affected newborns especially in developing countries where there are little or no facility for their treatment or support.

The percentage contribution to neonatal deaths of congenital malformation is higher in industrialized countries because other prevalent causes have been removed. For example, Dawodu *et al*[18] found that 37% of neonatal deaths in UAE are due to malformations while some other studies in developing countries[14,19] reported values ranging between 2.4 and 8.3%.

Indirect Causes

Maternal Health and Intrapartum Complications

Maternal health and health care are important determinants of neonatal survival. Neonatal outcomes are affected by the health of the woman right from the stage of girl child through adolescence and into pregnancy.[23] Complications during labour have also been documented as important determinants of fetal and neonatal health.[24] In general, intrapartum risk factors are associated with greater increases in risk of neonatal deaths than those identified during pregnancy, which are in turn associated with greater increases in risk than pre-pregnancy factor.[16,24] Onayade *et al*[25] reported high occurrence of neonatal deaths in spontaneous vaginal delivery compared with Caesarean section. This was due to deliveries at home or centres with no facility for Caesarean section leading to traumatic vaginal deliveries. However, Okolo and Omene[19] found neonatal deaths to be twice as common in operative delivery compared with spontaneous vaginal delivery. This was attributed to poor neonatal resuscitation skills. Greenwood *et al*[15] reported more deaths in multiple pregnancies than singleton in the Gambia. More neonatal deaths have been reported in first and fourth or more birth orders.[14,15] This is a result of the peculiar obstetric problems associated with these extremes.

Maternal and Neonatal Health Care Coverage

Early booking and regular attendance at antenatal care services is an essential determinant of pregnancy outcome. It allows for skilled supervision of pregnancy, early detection and management of complications of pregnancy, tetanus toxoid immunization, monitoring of fetal growth and well-being and objective assessment for route and timing of delivery.

Globally, only about 56% of women deliver with skilled attendant while 50% of neonatal deaths arose after a home delivery with no skilled care. However, the variation between countries is very great (5-99%).[26] In Sub-Saharan Africa, less than 40% of women deliver with skilled care while in Southeast Asia it is less than 30%.[1] This shows that the coverage of skilled attendance and the proportion of births that take place in a health facility are very poor in such high-mortality regions. For example, in Nigeria antenatal care coverage of at least one and four visits are 61% and 47% respectively, while skilled birth attendance by health personnel was 35%;[27] Implying that most of the deliveries take place unsupervised at home, consequently leading to neonatal deaths. The poor and dwindling utilization of orthodox prenatal and delivery services in Nigeria is mainly due to religious and financial reasons.[25] Prenatal care and delivery in churches and other unorthodox places are often attended by untrained persons.[28] High stillbirth and early neonatal mortality rate have been associated with such compared with hospital-based care. These losses follow perinatal asphyxia, severe infections and hypothermia.[29]

...birth attendants to be the main provider of ...natal and delivery care (84%). Majority of them ...illiterates who acquired the skill from their ...family members and charged no fee for their ...services. Although the outcome of the deliveries ...not determined in the study, only 4% of the ...babies were taken to hospitals immediately after ...birth for review. Hence early neonatal problems ...which could lead to death in such cases are not likely ...detected and treated on time.

...have shown that death of the mother ...commonly result in death of the child. For example ...et al[32] (1986) in Bangladesh found that of the 21 ...live-born to women who subsequently died, ...were dead by the 28th day of life. In another study ...et al[32] (1988) in Bangladesh, 65% of infants ...alive to mothers who died survived until one ...month, compared with 94.4% who survived in the ...group of infants with living mothers.

...economic Factors

...is an underlying cause of many neonatal ...through increase in the prevalence of risk ...such as maternal infections, malnutrition or ...reduced access to effective care. It is ...that about 1.2 billion people survive on ...one dollar a day in developing countries ...of population in Sub-Saharan Africa live ...Over 60% of Nigerians are below the ...line.[6]

...a study in Ilesa documented the influence ...economic and cultural factors on childhood ...and mortality. The influence of socio-...status on the utilization of maternity and ...services has also been documented; ...the higher social rung are more likely to ...the value of and adequately utilise such ...services.[33] Women in higher social class are also less ...to suffer prenatal morbidities like malaria, ...and pre-eclampsia, which may predispose ...and neonatal losses. On the other hand, ...the lower social classes have been ...to be socially disadvantaged,[35] they are ...to recognise illnesses and take prompt and ...cost-effective health decisions, hence ...inadequately utilise available health care ...even when they do, may not be able to ...fees. Peterson et al (2004)[36] in a study in ...found that almost 80% of the caregivers of ...ill young infants did not comply with ...referral to a health facility. The ...given in 90% of the cases was lack of money. ...the need for user-friendly financing ...to care for the less privileged and ...of appropriate health care seeking ...

...education, occupation and marital status also influence neonatal deaths. High NMR has been reported in teenage unmarried and elderly (more than 35 years) mothers.[14,15] Studies have shown that having an educated mother reduces a child's risk of dying.[24]

Gender

Females have a well described biological survival advantage in the neonatal period. In societies where care is equal for males and females, the ratio of neonatal mortality is usually 1.2 or higher in males.[37] Some studies in Africa also corroborate the survival advantage for female neonates.[11]

Lawoyin[38] reported more female neonatal deaths but it was otherwise in the study by Okolo and Omene.[19] Gender did not significantly affect neonatal deaths in some other studies.[25] It is not known if preference for male child leading to better health care seeking could have affected the result. A number of studies from Southeast Asia have reported reduced care seeking for girls and even female infanticides.[39]

Birth Interval

Short birth intervals have been reported to adversely affect the outcome of pregnancies. Studies comparing birth-to-pregnancy intervals of 18 to 23 months showed that pregnancy occurring less than 18 months after the last birth, and especially within six months after the last birth or longer than 59 months are associated with a significantly increased risk of LBW and are associated with newborn death.[11] Experts at a 2005 WHO technical consultation on birth spacing recommended a birth-to-pregnancy interval after a live birth of at least 24 months before another pregnancy is attempted.[11]

Millennium Development Goals (MDG) and Neonatal Mortality

At the close of 1990s, the international community decided on some measurable goals and targets to combat global poverty, hunger, disease, illiteracy and environmental degradation. At the United Nations Millennium Summit in September 2001, heads of the 147 nations endorsed the MDGs, about six of which directly or indirectly concerns different aspects of health.

The fourth goal (MDG) aims to reduce mortality in children younger than five years by two-thirds between 1990 and 2015.[6] Between 1960 and 1990, the risk of dying in the first 5 years of life was halved.[40] This is a major achievement in child health but greater reduction in mortality than those obtained in the past is essential to achieve MDG-4. With less than a decade to 2015, no remarkable achievement has been made. Challenges including HIV/AIDS,[1] increasing poverty especially in Africa as well as a lack of global investment in child survival[10] are largely responsible for the poor progress.

A major challenge, often ignored in policy analysis and intervention, is the slow progress in reducing global neonatal mortality which accounts for 40% of under-five mortality. The various child survival programmes have largely focused on conditions like diarrhoea, malaria, pneumonia and vaccine preventable conditions most of which are largely relevant after the first month of life, hence the relatively slower reduction in neonatal mortality rate compared with post-neonatal mortality rate.

So far there has been limited progress in reducing deaths in the first month of life especially the first week of life in Africa.[11] However, six African countries – Eriterea, Malawi, Burkina Faso, Madagasca, Tanzania and Uganda – have achieved neonatal mortality rates between 24 and 32 per 1000 live births despite their adverse economic situations. The experiences from these countries provide valuables example of how leadership, district-based management, scaling up of essential interventions and abolition of user fee for maternal, newborn and child health care services[1] lead to reduction in mortality rates.

To meet MDG–4, there must be a substantial reduction in NMRs particularly in high-mortality countries. Specific effective interventions that have been shown to reduce NMR include breastfeeding, attention to hygiene (hand washing), prenatal care, tetanus toxoid immunization, intermittent presumptive therapy for malaria in pregnancy, newborn temperature management, antibiotics for prolonged rupture of membrane, clean delivery practices, and training on newborn resuscitation skills.

It is crucial for every region or community to identify specific problems peculiar to them in order to define their own priority areas. Most of these interventions targeted at reducing neonatal deaths will also help in improving maternal health and thereby help in achieving the MDG-5 of reducing by three-quarter between 1990 and 2015 the maternal mortality rate. In developing countries, it has been shown that the death of the mother in childbirth may lead to the subsequent death of the infant.[11,32] The achievement of other MDGs – e.g. eradication of extreme poverty and hunger (MDG-1), universal primary education (MDG-2), promotion of gender equality and women empowerment (MDG-3), combating HIV/AIDS, malaria (MDG-6) etc will indirectly impact on neonatal survival.

Reducing Neonatal Mortality

Specific and cost effective interventions within the maternal, newborn and child health care are necessary in order to meet MDG-4. An effective continuum of care that connects essential maternal, newborn and child health (MNCH) pa throughout adolescence, pregnancy, ch postnatal and newborn periods an childhood is required. It should also streng links between the home and the first leve and the hospital, ensuring the availab appropriate care in each place. If effective M available for 90% of mothers and newborns i then the NMR could be reduced by two thir present level.[11]

Pre-pregnancy Care

The well-being of women and girls is closel their education, nutrition and health servi receive throughout the lifecycle. Early s sexual activity and adolescent and u pregnancies have serious consequences f health of women and babies. Gender-based v and female genital mutilation are also delete their reproductive capabilities. Therefore, edu with equal opportunities for girls, promot good nutrition, prevention of female mutilation and other harmful traditional pr family planning and prevention and manage HIV are highly beneficial.

Antenatal Care

The coverage of at least one antenatal visit is high in Sub-Saharan Africa (69%) compa South Asia (54%).[11] This presents an opport strengthen MNCH through delivery of ke interventions during routine antenatal Focused antenatal care should include nutrition, prevention of mother-to transmission of HIV/AIDS, malaria pre through promotion of insecticide treated (ITN) and intermittent presumptive treatm pregnancy, tetanus toxoid vaccination, co on maternal and infant feeding and also emergency preparedness at home effectiveness of these interventions rely than one antenatal visit. So, in Africa where antenatal attendance is low, efforts need to to encourage antenatal clinic attendance mothers to benefit from these services.

Childbirth Care

The availability and quality of skilled care at immediately after birth is a major determ immediate survival and health of both mothers babies. Skilled attendance at birth, acc emergency obstetric care and improved h home and health facility are very effec preventing neonatal deaths. Essential new e.g. drying of the baby, provision of w cleanliness etc should be taught to traditional attendants, midwives and community workers as well as training to give basic resuscitation to newborns. Complex inter such as intubation, chest compression and rarely needed, hence, even at the lowest health care, potential cause of newborn de be prevented.

Postnatal Care

Good care during the postnatal period at home and efficient referral facilities is crucial for reducing maternal and newborn deaths. It also helps to reinforce the gains of antenatal care.

Integrated Management of Childhood Illnesses (IMCI)

The IMCI strategy is to reduce child morbidity and mortality in developing countries by improving case management skills of health workers, strengthening the health system and supporting families and communities to take better care of sick children. Thus, IMCI provides a major opportunity for integration of newborn services within health facilities. While the current WHO recommended IMCI protocols excluded management of the sick newborn during the first week of life, a number of African countries such as Malawi and Ethiopia have begun to adapt IMCI to newborn care.[41] Management and care of LBW babies including Kangaroo Mother Care and Emergency neonatal care for illnesses can be included. Nutritional assessment, counselling and home care for newborns to promote exclusive breast-feeding, prevent hypothermia and improve illness recognition and timely care seeking are other simple interventions that will go a long way in reducing neonatal mortality.

In India IMCI is adapted to include the first week of life. All newborns are visited within 24 hours of birth, days 3 and 4 and days 7 and 10 while low birth weight had additional visits on days 14, 21 and 28.[42]

Political Commitment

The commitment of the various governments of the developing countries where most of neonatal deaths occur towards more rapid and progressive reductions in these deaths is highly essential. Adequate funding of neonatal survival programmes and regular research are required. The government should also have a system of ensuring that the various policies are implemented at all levels of health care. Maternal and newborn heath care should be subsidised in order to make the services available, affordable and accessible to all.

CONCLUSION

In view of the high burden of neonatal deaths in our environment, there is urgent need for collective and sustained action at all levels of health care delivery in order to scale down considerably the number of neonatal deaths through accessible and affordable maternal, newborn and child health care services. This will in effect help to achieve the fourth MDG sooner than expected.

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MOTHERLESS BABIES AND ORPHANS

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children should be born without parents—if born they must be" -Langston Huges
born into the world is a finer one than the last" -Charles Dicksons

than 7 million orphans in the world[1], and the figure still rising, issues relating to motherless babies and orphans are child health concern. Poor obstetric practice, epidemics, wars, teenage pregnancy, poverty, HIV/AIDS, social and assault on mothers amongst others, are leading causes of demise of mothers, and\or abandoned babies.

are cared for by foster parents, extended family members, orphanages, adopted parents and child welfare they are not abandoned on the street. If they are dumped on the street, they would be at the mercies of a humane die from neglect.

a lot of medical problems from malnutrition, anaemia, rickets to mention a few, social and psychological limited or no access to adequate education

help curb increasing number of motherless babies and orphans include favourable government policies on practice, medical service, child welfare system, orphanages, HIV AIDS

motherless babies, orphans

refers to a child whose parents are dead or has by parents, while motherless baby refers mother is dead or has been abandoned by

million orphans worldwide[2], this is a stress to a and health sector. The needs of this group of include health, social, educational, psychological done by famous psychologist Dr. Ainsworth the largest number of studies worldwide on children) showed that the long-term effect of mother figure to bind during the first year of leads to failure to thrive, feelings of insecurity amongst the other challenges.[3]

practice, war and epidermic, teenage poverty, HIV/AIDS, social factors all have their in increasing number of orphans and babies. Prompt attention should therefore be problem to reduce the number of these

PERSPECTIVE

motherless babies dates back to biblical times was abandoned by his mother.[4] During the and before then, foster care was done by neighbors, "masters" who would teach them a cobbler, fisherman, hunter and so on) .At different institutions built more rigid for these children.[5] This has continued with Nigeria having more than 200 registered institution owned by churches, philanthropists, and Non-Governmental Organization (NGOs).[6]

In America, 2 white house conferences held in 1909 and 1919, favoured anti-orphanage direction; so there is a shift to the pre-colonial era foster care method to facilitate bonding between these children and a motherly figure.[5] Nigerian mainstays of care is orphanages and foster care by family relatives[6]

CAUSES OF MOTHER'S DEATH AND ABANDONMENT OF CHILDREN

A. Poor Obstetric Practice: Maternal mortality from childbirth stands as one of the top reasons for increasing number of dependant children. Twenty-five percent of mothers die from bleeding and 15% die of infection.[7] There has been a global increase in the yearly maternal mortality figures from half a million deaths in 1987 to between 600,000 to one million deaths annually in the year 2000,ninety seven percent of such deaths occurred in developing countries, these also include deaths from indirect causes like anemia, malaria, heart disease and hepatitis.[7] In Nigeria, 54,000 mothers die annually from child birth and children who survive their mothers death are up to ten times more likely to die within two years than children with two living parents.[6]

B. Teenage Pregnancy: Worldwide, close to 20 million teenagers get pregnant.[8] In Nigeria teenage pregnancy accounts for nearly 1million births. With steady decline in the age of sexual debut, girls who get pregnant don't want to keep the pregnancy because of disapproval (especially

outside wedlock) or due to unavailable resources (financial, maternal, psychological) ,leading to unsafe abortions. This is another cause of maternal death which can involve a teenager with a previously viable pregnancy, or following delivery, a mother abandons her child on the street to continue her education or trade.

C. **Poverty:** Half of the world's population, nearly three billion people live on less than 2 dollars a day[9].A poor family in Nigeria struggling to feed is not likely to want to keep a lot of children ,so children might be abandoned after delivery. A pregnant woman with a low financial status might not receive adequate medical attention during pregnancy, so she can die during pregnancy, at or shortly after delivery from medical complications.

D.HIV/AIDS: A study carried out in Port Harcourt, Nigeria showed the sero-prevalence of HIV/AIDS among abandoned babies that are in motherless babies home and orphanages to be 13.6%.[9] This suggests a high maternal infection in this group of children. It is therefore possible that maternal death from the viral disease and social stigma in nursing such babies be major factors contributing to newborn babies being abandoned.

E.**OTHERS:** Other causes of child abandonment include: Congenitally deformed or malformed babies, rape, assault, substance abuse, effect of alcohol and other social drugs, and domestic violence.

Structure of care

A. Motherless babies' homes & orphanages: In Nigeria alone, there are over 300 registered motherless babies' homes and orphanages[10] .The structure depends on available finances but on the average, each home caters for 50-100 children, usually with a nursery and primary school to cater for the basic educational needs of these children. Finance is from philanthropists, clubs, religious organizations, humanitarian societies and other groups.

B. Foster care: Over 500,000 children in the U.S. reside in some form of foster care.[11] Placements have been inadequate over the past 10 years because there is a higher proportion of increase in this group of children as compared to individuals ready to provide foster care services. Children are usually placed in foster care by the child welfare system. Foster care by neighbors and relatives apply in Nigeria.

C. Adoption: Child adoption is done by well meaning individuals who cannot have children, want more children have been foster parents or are related to the child in question or for several other reasons[12] More than half a million dependent children are adopted in the United States.[12]

Problems and Challenges

A. Medical and Health Needs: Malnutrition, rickets, aneamia, HIV, lead poisoning, asthma, tuberculosis, hepatitis B, eczema, bacterial and parasitic infections are common problems of children living in institutions. These problems are compounded by lack of ready acc adequate medical facilities[13].

B. Age determination: This might not sound lik problem but it may pose a major problem to the that is about to adopt the child. Current ways used solve the puzzle include general appearance of the multi disciplinary approach involving pa pediatricians, radiologists, dentists, teachers, lawye development specialists[14]. This is important for educational monitoring and the child's sense of and security.

C. Education: Most of these children don't have ac it, majority of the orphanages have a make shift school with orphanage staff as teachers.

D. Social and psychological problems: Des children are exposed to a lot of psychological di such as post traumatic stress disorder, multiple per disorder, depression, anxiety, reactive attachment behavioral risk (cigarette, alcohol, substance abuse

E. Absence of bonding with mother: The import bonding can not be over emphasized the mother of the child's early psychic apparatus (Bowlby unfortunately abandoned babies do not have the bonding with their own mothers.

F. Legal issues: Who defends a dependent child is abused, neglected, assaulted even by care provid

These are some of the problems of motherless and orphans.

The Way Forward

There is still hope and a future for the manage motherless babies especially in Nigeria. The follo recommendations to that e

Legislation: A bill to protect the rights of orphan be pursued and passed into law by the nation's le arm of government. Government policies shou poverty and control of HIV should be instituted.

Safe obstetric practice: Since maternal mortality one of the leading causes of increasing orphans factors associated with maternal mortality sh reduced to the barest minimum, through good an care and prompt obstetric attention in emergencies like ante-partum heamorrhage[16].

Discourage teenage pregnancy: The school religious organizations/bodies, governmental an governmental organizations, the family system, an stakeholders must join in the campaign agai marital sex and teenage pregnancy, knowing tha lead to child abandonment and an increasing nu motherless babies.[17]

Protection of female adolescent reproductive rights: Many of the abandoned and hence "mo babies" we see on streets, in refuge dumps, bushes etc, are products of sexual violence and reduce this trend, the rights of females, especia regards their reproductive health, must be up

enforced by both governmental and non governmental agencies.

Standardization of motherless babies homes and orphanages: The state of many motherless babies homes and orphanages in Nigeria is not favourable for healthy growth and development of the children. Some of them are poorly managed and the health of the children is given little or no attention. This must be addressed by the child welfare system if our orphanage system must serve our children effectively.

Also the feeding of these children is of paramount concern. Well formulated feeds and food items that will meet the growth and developmental needs of these children must be prepared and served to these children in the orphanages and homes, the child welfare system in each state should ensure this.

Training of orphanage and home staff: Because of the peculiar nature of orphans and motherless babies, caregivers must be trained to meet their needs for love, care and comfort, as much as a mother would give to her own child. This will ensure that the child develops in a near-normal family setting and has equal opportunities later in life when compared with children who grew up in their normal family setting.

Medical Services: A trained nurse should be attached to each orphanage or home so that he\she attends to their health needs as soon and as promptly as they arise and make referrals to a doctor or hospital as appropriate. This will keep the orphanages and homes diseases-free, for proper development of the child.

We can all work hand in hand to give these children a new hope, ensuring that they live with the same love and comfort as their colleagues.

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HEALTH AND SOCIAL PROBLEM OF CHILDREN IN AN INSTITUTION FO MOTHERLESS BABIES IN IBADAN, NIGER

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Abstract

Objective

This study was conducted to describe the health and social problems of children in an institution for care of mother abandoned babies.

Methods

A descriptive study was conducted and information on socio-demographic characteristics as well as health and social pr of the children in the institution was obtained from their carers and from their case files. The children's weight and length were also measured.

Results

There were 19 children aged 11 days to 42 months, (median: 9 months) in the home, 15 (78.9%) were females and 4 Seventeen (89.5%) were admitted on account of maternal deaths and 2 (10.5%) because they were abandoned. They ha in the home for 7 days to about 38 months (median = 8 months). Seven (36.8%) of the children had stunted growth, were wasted and 10 (52.6%) were underweight. They had all commenced the routine immunizations and all those a months and above were fully immunized. Six (31.6%) had been ill in the 1 month period prior to the study and one child d before the study was concluded. On their social problems, only 4 (28.6%) were visited by their father or other relations re i.e. at least once a week. Most fathers had paid the initial admission deposit but only 5 (26.3%) had since paid any additio though additional upkeep fees were not yet due for 5 (26.3%) children.

Conclusion

The study confirmed that children in institutions have a number of health and social problems. Prevention eff government, health workers and the general populace are needed to reduce maternal mortality as this was the com reason for admitting children into the institution. In addition, carers of these children need to pay attention to their intake in view of the high prevalence of undernutrition reported. Relations of children in homes should also be more involved in their care by paying them regular visits and taking them out of the homes at the stipulated time.

Key words: Children, Health and Social problems, Institution, Motherless babies' home, Ibadan

Introduction

The family is the fundamental unit of society and the natural environment which ensures the optimal growth and well being of all its members. Within the family, the children's needs for stimulation, recognition and security are in place and this enhances children's growth and development to adult life. If however, the child has no family, is abandoned or lives in a family where abuse and neglect occur or in a situation when the family is experiencing the shock of a maternal death the onus rests on society/ government to develop an effective mechanism of intervention to take care of such children.

Options of care for motherless babies include care within the extended family with or without supervision, fostering, adoption and care in institutions. In Nigeria, the responsibility of caring for motherless babies usually falls on the extended family; however, in the face of increasing economic hardships, the role of institutional care is becoming increasingly important in the care of these children.[1] This, coupled with the spate of abandonment of babies has led to an increasing demand for institutional care.

The development of residential institutions for children is not peculiar to Nigeria. In Europe this practice commenced with industrialization and urbanization.[2] The industrial revolution brought about changes within family structures as parents took on new roles, moving away from production within the household to production for an employer outside the home. Large-scale poverty a resulting inability of families to care for their c demanded social reaction. Hence, residential institu children came into being as a positive measure a taking care of vulnerable children all over Europe.

A number of disadvantages are associat institutional care for children. Generally, children br in institutions have higher rates of morbidity and m compared with those brought up in the extended **Research has also demonstrated that children br up in institutions experience developmental del potentially irreversible psychological damage w especially evident in the earliest stages o development (birth to 4 years). This is the period which the child learns to make psycho attachment to parents (or substitute pa** Psychological and developmental problems occur b children brought up in institutions seldom receive a attention. This has been attributed to the high staf over rate as well as the high child-to-staff ratio charac of many institutions.[1] **Even in well staffed institu child rarely gets the amount of attention he or she receive from his or her own parents. Also, c temporarily placed in an institution often lose co with family or friends in the community. The e these contacts makes return to the commun difficult over time and once family ties are se child may lose hope of ever returning to the com**

despite of the disadvantages associated with care of children, these institutions also offer advantages for instance, for centuries, they have the responsibility of bringing up millions of Europe.[1] Most of the states in Central and Eastern have since developed important policies and specific measures in order to improve the children in institutions.[2] In Nigeria, institutional children has some advantages as it provide a place for motherless babies to be taken care off family adjusts to the shock of the maternal demise provides shelter for abandoned children. A search did not reveal any recent studies which the health and social status of institutionalized This study was thus conducted to describe the social problems of children in an institution for and abandoned babies in Ibadan, Nigeria.

conducted in an institution for motherless babies located in Ibadan, Oyo State. The capacity for up to 50 babies, has a well kept and up to 5 wards with adequate facilities. running the home come from philanthropists, other government and non-governmental and the token amounts relations of children pay. running of the home is administered by a The children in the MBH are fed with infant weaning foods (pap and soya beans) are they are 4 months old. The older children a day in addition to which they are given a Meals are prepared in the home and a meal lowed. The home admits and cares for the are about 2 - 3 years old, after which the motherless babies are expected to come back they can be re-integrated into the family. babies could be adopted after following laid or sent to the SOS childrens' villages.

study was conducted and information on the status and social factors for all 19 babies in time of the study was obtained from the staff as well as from each child's case file using a proforma. Each child was weighed without a Salter weighing scale for those aged 23 and weighing scale (bathroom) for those Weights were recorded to the nearest length of children aged 0 23 months was height of those aged 24 months and measured. Each child was bare-footed and his/ her length/ height was measured. were taken to the nearest 0.1 cm. The for-age (WFA) and height-for-age (HFA) weight (WFH) were calculated using EPI nut compared with the National Center for (NCHS) median values.[3] Children whose were below the 10th percentile were moderately underweight, stunted or with WFA, HFA and WFH below the 5th the median value were classified as being weight, stunted or wasted respectively. Data the Statistical Package for the Social version11. Data on anthropometric analyzed using EPI-NUT (EPI-Info 2000).

children in the institution at the time of the females and 4 (21.1%) males. Their ages 42 months (median: 9 months). Nine (47.4%) had been living in the home for more than a year while the rest had been living in the home for a week to 8 months. Seventeen (89.5%) were admitted into the home on account of maternal death and were all brought to the home by their fathers who were mainly in unskilled, semi-skilled and skilled occupations (Table 1). Two (10.5%) children were abandoned.

Health problems

Anthropometric measurements

The mean weights and heights/lengths of the children in the home ranged from 3.4 ± 0.3 kg to 12.5 ± 2.4 kg (Table 2). Nine (47.4%) of the children had an appropriate WFA, while the rest: 10 (52.6%) were severely underweight. Ten (52.6%) children had a normal weight for their height/ length, 3 (15.8%) were moderately wasted while 6 (31.6%) were severely wasted. In all, 12 (63.2%) had a normal height/ length for their age and 7 (36.8%) had severe stunting. For all anthropometric indices, higher proportions of the male children were underweight, stunted and wasted compared with the females (Figs. 1 3).

Immunization status

Regarding their immunization status, the 9 children aged 12 months and above were fully immunized. The other children had all commenced their routine immunizations and these were up to date. It was observed that majority of the children were admitted when they were over 4 weeks old, thus they received the first and thereafter subsequent doses of the antigens at a later date than the age stipulated in the routine immunization schedule. Immunizations were received at the Institute of Child Health, University College Hospital, Ibadan.

Morbidity pattern

Since the children started living in the home, 4 (21.1%) had been ill and required admission in the hospital - for treatment of neonatal sepsis (2 children), meningitis (one child),) and prematurity (one child). The number of days on admission ranged from 1 week to a month. Six (31.6%) children had been ill in the month preceding the study. Three of these children had fever; while one of them had septic rashes. They were treated in the home and they all recovered. Five (26.3%) children were ill at the time of the study and their symptoms included conjunctivitis, furuncle, failure to thrive, fever and pustular rash. They were being treated in the home and were responding well to home-treatment. One child, a 2 month old female child who had previously been healthy developed an occipital abscess during the course of the study, had incision and drainage done in the hospital and subsequently died.

Social problems

Sixteen (84.2%) were brought to the home by their fathers. One was brought by staff of a television station in Ibadan because the staff realized that the child's father could not afford to take care of the child following the demise of the mother, 2 were abandoned. All the fathers who brought their babies to the hospital paid the initial mandatory fee for upkeep of the children. Only 5(26.3%) relations had since paid additional funds for upkeep of their children on a regular basis. For 5 (26.3%) children, additional funds were not yet due to be paid. Fourteen (73.7%) children had been visited by relations since they were admitted in the home, 4 (28.6%) of these children were visited by relations in the week preceding the study. One child was last visited 4 years prior to the study. On the arrangements being made for the children after leaving the home, the staff

Table 1: Socio-demographic characteristics of children in the study

Socio-demographic characteristics of children	N (%)
Sex	
Female	15 (78.9)
Male	4 (21.1)
Age group	
< 12 months	10 (52.6)
12 – 24 months	3 (15.8)
25 – 36 months	2 (10.5)
37 – 48 months	4 (21.1)
Father's occupation* (n = 17	
Trader	4 (23.5)
Artisan	3 (17.6)
Clergy	3 (17.6)
Soldier	2 (11.8)
Driver	2 (11.8)
Secondary school teacher	1 (5.9)
Porter	1 (5.9)
Contractor	1 (5.9)

* not known = 2

Table 2: Mean anthropometric measurements of children studied by age group (months)

Age (months)	Frequency	Weight (kg) Mean ± standard deviation	Height/ length (cm) Mean ± standard deviation
< 1 month	4	3.4 ± 0.3	50.6 ± 1.1
1 – 4 months	2	3.0 ± 0.7	52.5 ± 2.1
5 – 8 months	2	2.9 ± 0.1	51.8 ± 1.1
9 – 11 months	2	8 ± 0	68.5 ± 0.7
12 – 23 months	3	8.3 ± 0.9	76.4 ± 4.5
24 – 35 months	2	10.8 ± 0.4	84.5 ± 0.6
? 36 months	4	12.5 ± 2.4	92 ± 3.8

Table 3: Morbidity pattern among children in the study

Symptoms	When last was child ill		
	< 1 month ago n = 19 (90.9) n (%)	1 – 2 months ago n = 1 (10) n (%)	> 3 months ago n = 1 (50.0)
Fever & septic rashes	1 (0.1)	-	-
Failure to thrive	-	-	1 (50.0)

Table 4: Most recent visit by relations of children in the home

Most recent visit by relations	N	%
? 1 week ago	4	21.1
> 1 week – less than a month	3	15.8
1 – 2 months	5	26.3
> 2 months	2	10.5
No visit	5	26.3

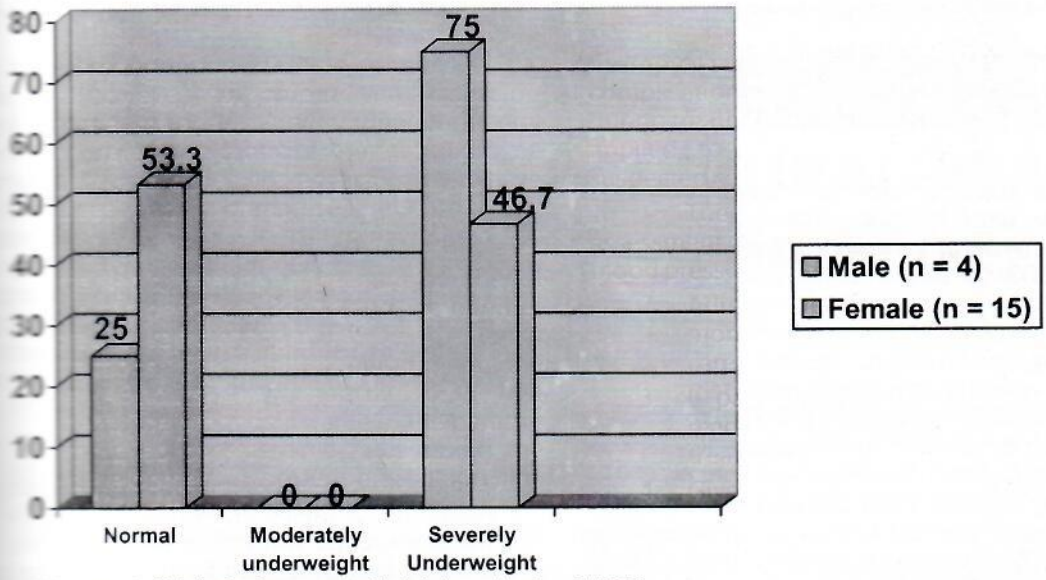

Figure 1: Weight for age of children in the MBH

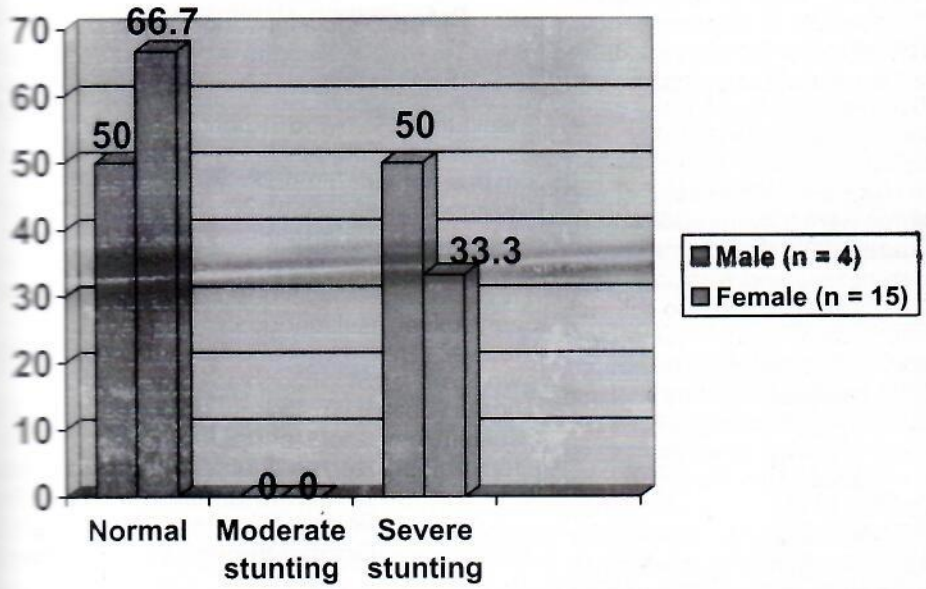

Figure 2: Height/ length for age of children in the MBH

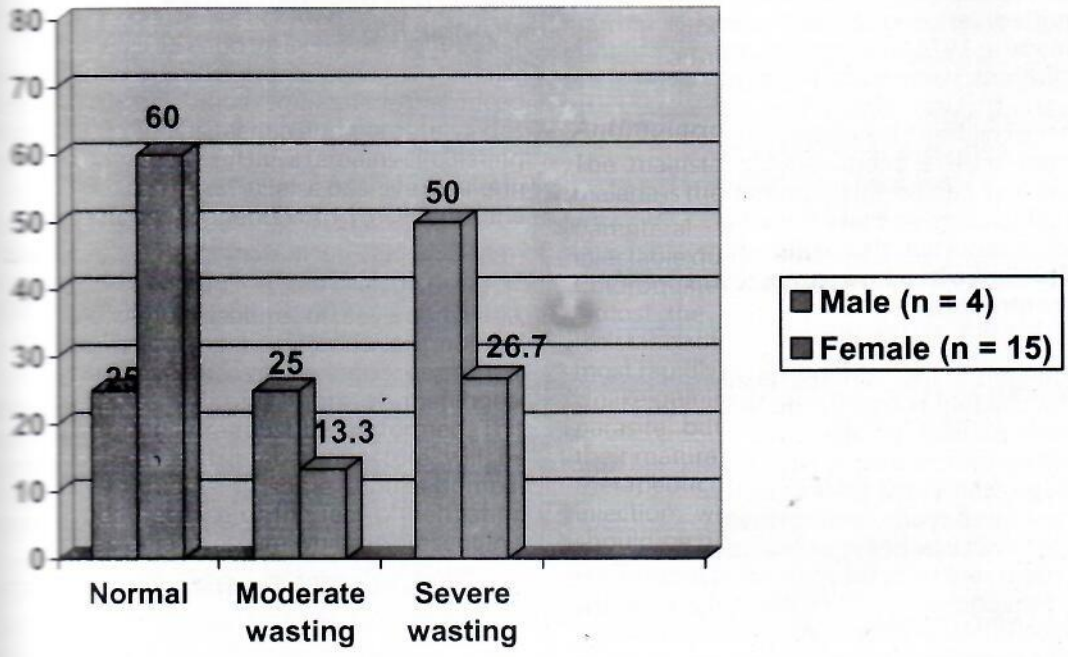

Figure 3: Weight for height/ length of children in the MBH

could only vouch for one family who had made arrangements for their twin children to be re-integrated into the family when they clocked 2 years old.

Discussion

This study revealed that the majority of the children were admitted into the home following maternal death. The current maternal mortality ratio in the country which is about 1,100 per 100,000 births [6] suggests that maternal deaths still need to be given urgent attention. More than half of the children were malnourished with 52.6%, 33.3% and 66.7% being underweight, stunted and wasted. These figures are worse than national rates documented by the 2003 National Demographic and Health Survey[6] and rates reported by UNICEF[7] and Asekun-Olarinmoye and colleagues in Ibadan[8]. The Nigerian Demographic and Health Survey (NDHS), 2003 showed that 57.5% of children under-5 were stunted and 11.4% of them were wasted[6]. Asekun-Olarinmoye et al reported that 16.5% of children under 2 years taken care of in day-care centres and 18.5% of those taken care of in their home environment were underweight; 42.1% of those taken care of at home and 24.2% cared for in daycare centres were stunted and 12.1% of those in the daycares and 5.5% of those cared for at home were underweight[8]. The State of the World's Children, 2008 reported that for the years 2000 2006 29%, 38% and 9% of Nigerian children less than 5 years old were underweight, stunted and wasted respectively[7]. All the children aged 12 months and above in the motherless babies' home were fully immunized this is much higher than figures reported by Asekun-Olarimoye[8] (87.9% fully immunized) and National rates reported by UNICEF[9]. This might be attributed to the fact that the activities in the home are being overseen by a retired nurse/mid-wife and as well as the proximity of the home to the Institute of Child Health, University College Hospital, Ibadan. For those who had not completed their immunizations for their ages, examination of their case cards revealed that they commenced the immunizations late because they were admitted in the home when they were older than the stipulated ages (4 weeks) for commencement of routine immunization according to the National Programme on Immunisation (NPI) schedule, hence the first and subsequent doses of vaccines were late.

Most of the children had been visited by relations since they were admitted; however in most cases, visits were irregular. Oyemade in 1974 also noted that most children reared in institutions were visited irregularly by their relations[1]. Even though the home is meant to accommodate the children till they are 2 3 years old when they are expected to be re-integrated into their families, about one third had passed this age and their fathers/ relations were yet to come and take them home. Pashkina in 2001 had also reported that **children temporarily placed in institutions often lose contact with family or friends in the community.**[4]

Conclusion

The study highlighted the various health and social problems of the children reared in the MBH. The home appeared to have fulfilled it's objectives of providing a temporary place for the children to stay pending the time that the family adjusted to the grief caused by the death of their mothers and made arrangements for them. However, this goodwill was being exploited as 6 children were yet to be integrated since they turned 24 months old. We recommend that government, healthworkers and the general populace increase efforts at reducing maternal mortality in the nation since this was the portal through which majority of the children ended up in the institution. Nutritional interventions targeting carers of institutions should be conducted to imp nutritional status. Relations of children bro home should be encouraged to give mor attention to their children and not just dump home. Other options of care e.g care within t family with supervision be explored and reco families faced with the decision of care for c lose their mothers. Further studies incorpor institutions caring for other special groups residing in institutions need to be conducte their problems and proffer feasible solutions.

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A REVIEW OF HARMFUL SOCIO-CULTURAL PRACTICES AGAINST CHILDREN IN NIGERIA

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ABSTRACT

...ection of children in Africa to certain socio-cultural practices is widespread and Nigeria is not an exception. ...parts of the country, some traditions including respect and courtesy to the elderly and mutual solidarity ...with extended family system promote social cohesion and unity. However, a lot of harmful socio-cultural ...still exist among various ethnic groups in the country. These have deleterious effects on the health of our ...and adolescent girls and also infringe on their human rights.

...view, these Harmful Socio-cultural Practices (HSCPs) have been grouped into Unwholesome Traditional ...(Female Genital Cutting (FGC), Child Marriage, Son Preference, Traditional Marks, Female Infanticide and ...Nutritional Taboos), Exploitation of Children (Child Labour, Begging and Trafficking, Sexual Exploitation ...) and Violence against Children.

...HSCPs pose great threats to the development and survival of affected children in Nigeria and cause ...of their physical and psychological health.

...of these HSCPs against children in Nigeria requires the commitment of all, from government through ...governmental organizations, opinion and religious leaders to the community at large. This can be brought ...by community mobilization, health education and advocacies which can unite communities, reinforcing ...which benefit all citizens and at the same time confronting those which damage the integrity and diminish ...of our children and young women without necessarily disrespecting our age-long traditions.

Harmful Socio-cultural Practices, Children, Violence, Children's Right, Nigeria.

...UCTION

...Charter of the United Nations includes among its ...principles the achievement of international ...tion in promoting and encouraging respect ...rights and fundamental freedoms for all ...distribution as to race, sex, age, language or ...(Art. 1, paragraph 3,UN). The article 1 of the ...on the Rights of the Child (CRC) describes ...under the age of 18 as being children.[1]

...the subjection of children to Harmful Socio-...Practices (HSCPs) is widespread and Nigeria is ...ception.[2] These inhuman, discriminatory and ...dangerous practices reflect values and ...held by members of a community for periods ...spanning generations. These practices often ...verse effects on the health status of the victim ...fringe on his /her human rights. Many ...promote social cohesion and unity. For ..., the healthy post-partum practices based on ...framework, including rest, cleanness, love ...nutrition is prominent among people of the ...of Korea; long period of breast feeding ...in many parts of Africa, Latin America, and ...beneficial. Also, the low homosexual practice ..., unalloyed respect and unfailing courtesy to ...among the Yorubas in the South-western ...Nigeria and values of mutual solidarity ...with the extended family system which ...mechanisms to assist needy family members ...examples of positive practices that uphold ...alues and inculcate in people the expected ...orms and values. There are however several ...socio-cultural practices (HSCPs), such as Female Genital Cutting (FGC), Early Marriage, Son Preference, Female Infanticide, Child Labour, Child Abuse, Early Pregnancy and Nutritional Taboos that erode the physical and psychological health and integrity of individuals.[2]

Millions of Nigerian children, especially girls face special problems of disadvantage, discriminations, abuse and exploitation, sometimes in appalling circumstances. These problems not only compound the risks of survival and create formidable obstacles for the well being and development of children, but are major challenges in their own right requiring special protection measures if they are to be addressed effectively.[3,4] This review critically assesses the nature and extent of harmful practices affecting children in Nigeria. It provides recommendations on how to address the problems.

Forms of Harmful Socio-Cultural Practices against Nigerian Children

There exists a long list of harmful practices against children among various ethnic groups in Nigeria. HSCPs may be traditional or modern. While female genital cutting is traditional, trafficking girls for prostitution is modern. They are also categorized into three broad subdivisions vis-a-vis: Unwholesome Traditional Practices, Exploitation of Children and Violence against Children.

UNWHOLESOME TRADITIONAL PRACTICES

The Unwholesome Traditional Practices (UTPs) against children include, but not limited to the

following: Female Genital Cutting, Child Marriage, Son Preference, Traditional Marks, Unequal Access to Health and Education.

Female Genital Cutting

Female Genital Cutting or female genital mutilation (as it is otherwise called) constitutes all procedures which involve partial or total removal of the external female genitalia and /or injury to the female organs, whether for cultural or any other non-therapeutic reasons.[5]

It is estimated that 85-114 million girls and women have undergone genital cutting around the world, and at least 2 million girls are at risk of FGC each year.[6] Female Genital Cutting is practiced in 27 of the 46 African countries.[7,8] It is estimated that about 60% of Nigerian women and girls are genitally mutilated in one form or the other.[9] Female circumcision, as it is erroneously referred to in Nigeria, has been condemned in recent times as the most barbaric and unprogressive cultural practice of our time, yet the practice continues to thrive.[10] The form practiced varies by ethnic group and geographical location. (Fig. I). Female genital cutting crosses the lines of various religious groups. It is found among Christians, Muslims and Animists alike.[11]

The origin of FGC has not been established but records show that the practice predates Christianity and Islam practicing communities of today. The age at which the mutilation is carried out varies from area to area. FGC is performed on infants as young as few days old in some parts of Nigeria such as in Edo State.[11] It is also performed on children from age 4 to 10 years and on adolescents in other parts of the country. Adult women also undergo the operation at the time of marriage and during pregnancy.[2,12]

The following are WHO's classification of FGC.[13]

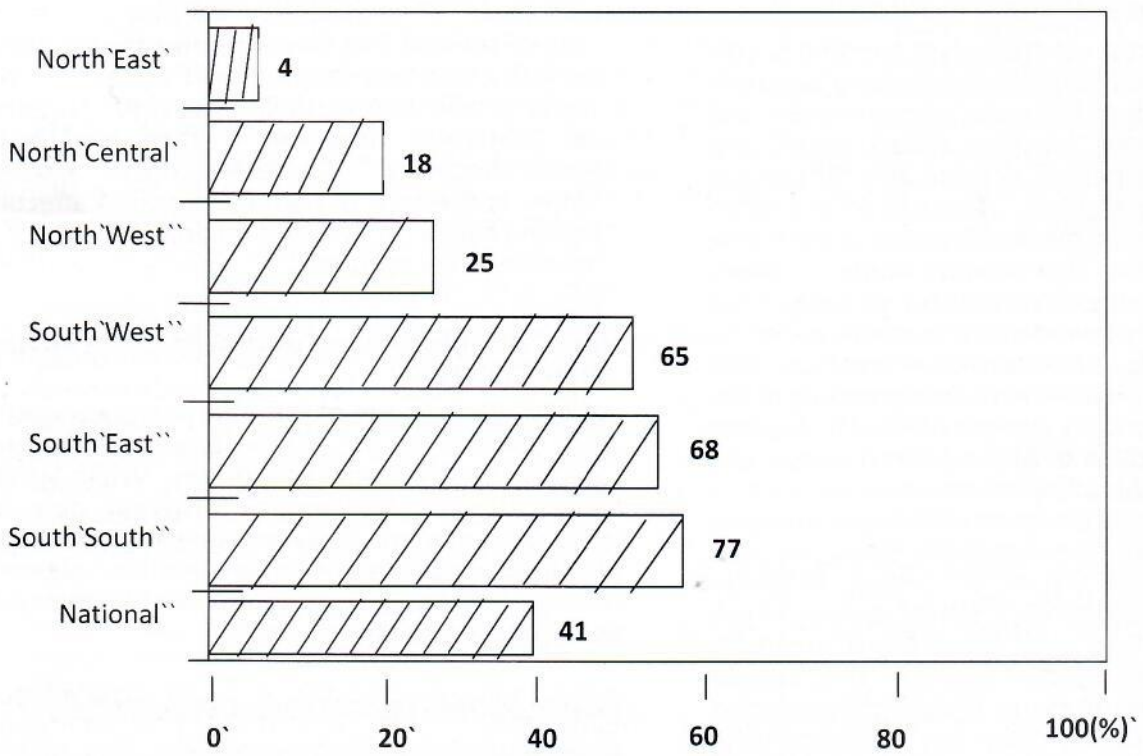
Type I Excision of the prepuce with or without excision of part or the entire clitoris. This is also called *Sunna* (traditional) circumcision and it is practiced across Nigeria.

Type II Excision of the prepuce and clitoris (clitoridectomy) together with partial or total excision of the labia minora.

Type III Infibulation: This involves the removal of the clitoris, the labia minora and part of the labia majora. The tips of the labia majora are then sutured together, leaving a tiny passage for urine and menstrual blood.

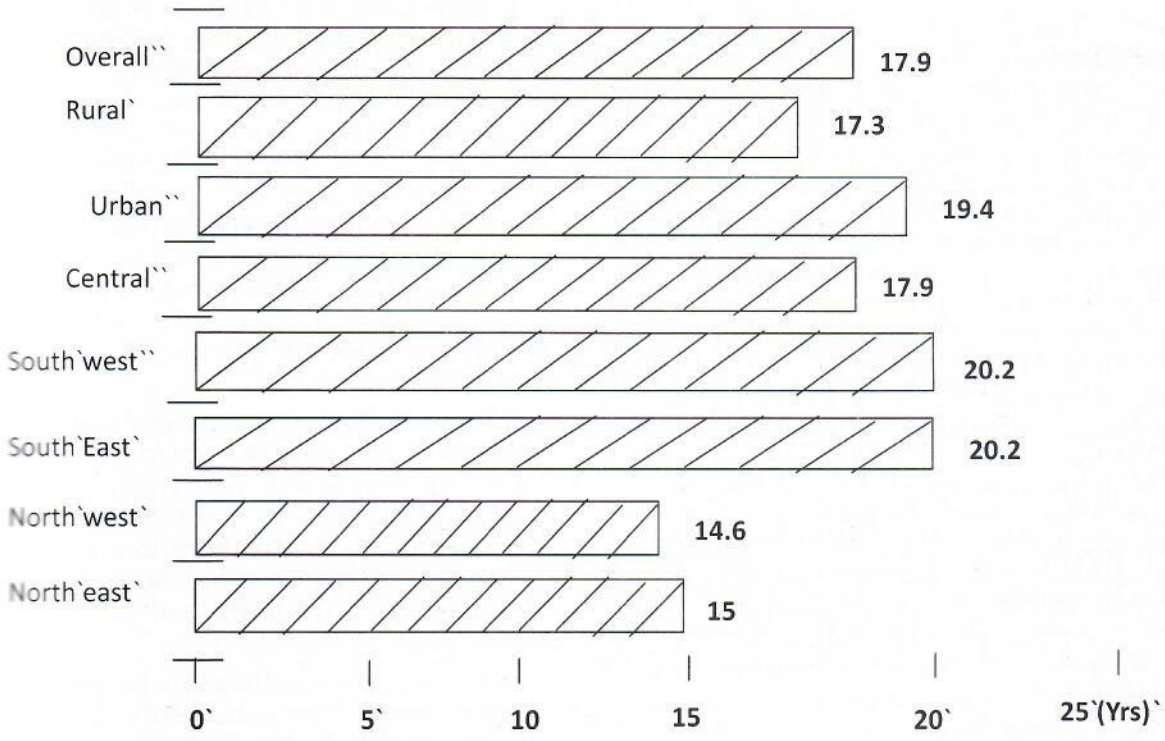
Type IV Unclassified: Include pricking, piercing or incision of the clitoris and /or labia; stretching of the clitoris and surrounding tissues; scraping (*angury* cuts) of the vaginal orifice or cutting (*gishiri* cuts) of the vagina. The latter is commoner in some Northern States of Nigeria where it is used to treat infertility or to facilitate the delivery of

FIGURE 1: Prevalence of female genital mutilation among adult women, by geo-political zones in Nigeria.



Source:`United`Nations`Development`Programme,`1998`

FIGURE 2: Median age of females' first marriage



Source: `1999`Nigerian`Demographic`and`Health`Survey`

a foetus in obstructed labour.

Type I and II are the commonest types, constituting about 80% of FGC operations in Nigeria while Type III, which is the most severe form in terms of health consequences, comprises about 15% of all.[7, 10, 14] Also, Type II and Type I are more predominant in the South, while Type III has a higher incidence in the northern states. Of the six largest ethnic groups, the Yoruba, Hausa, Fulani, Ibo, Ijaw and Kanuri, only the Fulani do not practice any form. The Yoruba practice mainly Type II and Type I. The Hausa and Kanuri practice Type III. The Ibo and Ijaw, depending upon the local community, practice any one of the three forms. Type III form of FGC is most practiced in Adamawa, Bauchi, Borno, Jigawa, Kaduna, Kebbi and Plateau states in the Northern part of the country.[11]

The reasons for practicing FGC vary, ranging from being a custom and tradition to improving marriage prospects and avoiding potential health risks to themselves and their children, especially during childbirth.[10] For instance, one traditional belief is that if a female child's head touches the clitoris during childbirth, the child will die.[11]

Female genital cutting causes grave damage to girls and women, and in many cases results in serious reproductive, health and psychological consequences. Complications following the procedure can be of short or long term duration. Usually performed without anaesthesia and using unhygienic methods and instruments, the procedure becomes a portal of infections, e.g. hepatitis, HIV, septicaemia. Other health complications of FGC are haemorrhage, shock, tetanus, failure of the wound to heal leaving a weeping or chronic infected ulcer, urinary retention due to pain and inflammatory injuries to adjacent tissues and organs like urethra, bladder and rectum. Recurrent urinary tract infections (UTIs), urinary incontinence and chronic urinary tract obstruction may result from tight infibulations, accidental injuries or urethral stricture.[3, 13, 14]

The reproductive consequences of FGC are myriads and may include the following: loss of sexual sensation, acquired gynaetresia, dysmenorrhoea, haematocolpos, dyspareunia, pelvic inflammatory disease (PID), infertility, keloid formation, vulval abscesses, clitoridal cysts, etc.[2, 14, 15] The earlier in life FGC is performed the greater the chance that it would be associated with scaring of vagina and difficulties during childbirth.[16]

Psychological effects of FGC on victims may be profound and permanent.[10] Since the procedure is usually performed on very young girls, some may not understand what is being done to them or why. The psychological effects of this painful experience are similar to those of post-traumatic stress disorder. Although very rare, girls and women who have had FGC may have problems sleeping, have more anxiety, and become depressed[17]

Child Marriage

Early arranged marriage is a widespread socio-cultural practice in Nigeria. A large number of girls are married off by their parents in their early teens, when they are too young for their consent to be sought or

given. The 1999 NDHS revealed large zonal variations in the mean age of marriage in Nigeria with the females in the North marrying an average of about 5years earlier than those in the South (Fig. 2). Also, in the North-West and North-East, over 50% of girls aged 15-19 were either already mothers or pregnant compared with about 8% in the southern zones.[4] In most cases, these young girls are married to much older men and in such marriages, the female partners have little power and sense of self determination. Some cultures believe early marriages guarantees a long period of fertility; very young brides may need a smaller dowry and young virgins are considered less likely to be infected with HIV/AIDS. Early marriage usually results in early childbearing with severe consequences for the health of the young mothers and their babies. Infants born to teenage mothers are up to 80 percent more likely to die within their first year than infants born to mothers ages 20 to 29.[18]

Maternal mortality rates are twice as high for women of ages 15 to 19 as against women of ages 20 to 29.[19] Zabiu and Kiragu reported in a research conducted in Zaria, Kaduna State that maternal mortality among women younger than 16 is six times higher than for women aged 20-24.[20]

Medical problems associated with early childbearing in child marriages include poor maternal weight gain, anaemia, prematurity (birth at <37 weeks' gestation), pregnancy-induced hypertension, toxaemia and STDs. Common obstetric complications that could occur in child marriage are prolonged obstructed labour, ruptured uterus, and obstetric fistulae (vesico-vagina and recto-vaginal fistulae) which leave a life-long debility[10,21]

Furthermore, early marriage and child bearing are closely linked to low educational attainment. In many instances, the teenage mothers are withdrawn from schools. In same vein, they are neither physically nor mentally mature to carry out the marital and reproductive responsibilities this harmful practice thrust upon them.[3,4].

FIGURE 2: Median age of females' first marriageSon Preference

The preference for male child is universal; however the attendant neglect of the girl child is more frequent in Asia, Latin America and parts of Africa including Nigeria. Son preference refers to a whole range of values and attitudes which are manifested in many different practices, the common feature of which is preference for the male child, often with concomitant neglect, deprivation and discriminatory treatment of daughters to the detriment of their physical and mental health. This practice adversely affects the girl child through inequitable allocation of food, healthcare, education and economic opportunities; a disparity frequently reinforced throughout life.

The practice of male preference is deeply rooted in culture and socio-economic climate. This is because a son is regarded as the one who will perpetuate the family name as well as being the bread winner and decision maker in the family.

Scarification

This is a cultural practice which often gene controversy as to whether it is harmful or not widely practiced across Nigeria especially in so parts of South-west and North-central zo Traditional facial marking is one of the indige surgery; others being circumcision, tattooing medicinal incision.[22] These markings are o performed when the child is young and therefor child has no say in it. It involves inflicting an injur making a deep cut on the face or the parts of the bo It causes bleeding, pain and sometimes if it is infec with consequent delayed healing and se scarification.[10]

It is certain if people with tribal marks were given option of choice, many would wish that they did have such defacing and denigrating marks on th faces, palms or body. Traditionally, they are inten to be marks of identification for lineage of individual or his/her tribe. Some marks are mad denote date of birth and sites of incision therapeutic purpose.

Most of these procedures are performed under se conditions and the instrument usually knife is used many children by the *Olola* (the traditional surge thus making the children prone to tetanus and o infections such as Hepatitis B and the deadly HI These infections are responsible for a numbe infant deaths but are often not documented. practice has severe psychological effects on chil as they find these marks highly embarrassing and been associated with low self-esteem and depres later in life.

OTHER UNWHOLESOME TRADITIO PRACTICES

Other unwholesome traditional practices a against children and adolescent females that ne be done away with in our society include fe infanticide and female foeticide, nutritional ta harmful practices related to child delivery e.g. Gis Cut and *Zur zur,*[2] introduction of corrosive substa or herbs into the vagina with the aim of tighten narrowing the vagina,[10, 13, 14] traditional practi dowry and bride-price, dry sex (removal of va fluid with absorbent materials).[23,24]

EXPLOITATION OF CHILDREN

While children have always worked in Nigerian so the increasing poverty since the end of the oil bo in the 1970s has driven millions of children into of labour that are exploitative, hazardous prejudicial to their welfare and developmen instance, street hawking is very prominent in ma urban centres across the country. Poverty alon certain cultural traits has also resulted in the sp child begging.[4] Furthermore, middlemen h exploited the desperation and ignorance of pa particularly in the rural areas to procure childr commercial trafficking. There is a growing tra young girls for the purpose of prostitutions, m by international traffickers who transport Nig

to Europe as debt-bonded sex workers.[4]

Child Labour

Child labour refers to any work which by its nature or employment conditions is detrimental to a child's physical, mental, moral, social or emotional development.[25] It occurs when children are exposed to long hours of work in a dangerous or unhealthy environment, with too much responsibility for their age and at the expense of their schooling. This should be distinguished from child work which means work in which the primary emphasis is on learning, training or socialization. The schedule of child work is flexible, tends to be responsive to the developing capacity of the child and encourages his or her participation in appropriate aspects of the decision-making process.[4]

An estimated 8 million Nigerian children are engaged in child labour.[26] Section 59 of the Labour Act of 1974 prohibits a child under the age of 12 from all work except where he is employed by the family in light work of an agricultural, domestic or horticultural character. It allows apprenticeships from age of 12 upwards with the consent of the child's parents, but forbids any child under the age of 15 from working in any industrial undertaking.[27]

Most child labour occurs in agricultural and in the informal sectors of the economy,[4, 28, 29] which consist of small businesses that lack government recognition, registration, or support. Persons operating in the Nigerian informal economy do not have access to commercial sources of credit, earn low incomes, and have no employment security or minimum wage.[29] Child labour in agricultural sector is mainly within household economy and involves helping the family farming, fishing and cattle herding. Also, Nigerian children are engaged in commercial plantations across the country and the neighbouring countries Cameroun[30]. Children work in the informal sector throughout the country, particularly in the cities. There are three broad categories of work in the informal sector in which they are engaged, namely: work in public places (such as markets and streets), work in cottage industries and mechanical workshops, and domestic services in private households[4].

Those who work in public settings include street vendors, shop and market stall minders, beggars, shoe-shine boys, car washers/watchers, scavengers and head loaders in markets. Among these, street hawking is by far the largest single form of child labour accounting for well over 50 percent of total urban child labour.[4] Apprentices, mechanics, hairdressers, bus conductors, hair dressers, and workers in catering industries are among those who work in cottage industries and workshops. Apprentices in various local vocations are exceptionally vulnerable to exploitation. They work for instructors for long hours, but are paid little or nothing, often run personal errands and carry out domestic chores for their instructors as well.[29] There are hundreds of thousands of boys and girls employed as house-helps, most of them originating from Akwa-Ibom, Cross Rivers, Delta, Imo, Anambra, Kwara and Ondo states. Increasing numbers of domestic servants have been migrating to Nigeria from neighbouring countries such as Benin and Togo.[4, 31]

The hazards of child labour depend on the age and gender of the child as well as the conditions and characteristics of different types of work.[32, 33] These hazards among other things include falls, injuries, assault from adults, road traffic accidents, emotional deprivation, poor health, disability, chronic illness, sexual harassment, substance abuse, denial of educational opportunity and poor academic performances.[4, 34, 35]

Child Begging

The children involved in begging are among the most vulnerable in Nigeria, coming from families that are among the poorest of the poor. Although a form of child labour, the negative psychological, social and health consequences are grave enough to necessitate a separate discussion. Three categories of child beggars are found in urban centres across the country: those who lead blind parents or relatives, those who beg entirely on their own and those who act as fronts for parents especially mothers, who are usually hidden from public view but supervise them from a close distance. [4] In all three cases, children involve in begging run enormous risks e.g. road traffic accident, often darting between cars in heavy traffic begging for alms from motorists. Child beggars also suffer the severe psychosocial consequences of engaging in a demeaning type of activity and being exposed to constant abuse and aggression from the general public. Child begging is not traditionally significant in the Southern Nigeria; however, it is much more widespread in the North where alms giving are widely regarded as a religion duty.[4] The largest category of beggars in the northern part of the country is associated with *almajinanci* system, a semi-formal system of Qu'ranic education, in which children, mostly boys are sent by their parents to take up residence with Islamic teacher or *mallamai,* for instructions in the Qu'ran and other Islamic text.[36] They would beg for alms or serve in their teacher's farms as a means of compensation for their religious education and upbringing.

Child Trafficking

Trafficking in children and women, mainly for the purpose of domestic services or prostitution is a relatively new phenomenon that has received wide media coverage in Nigeria but has been the subject of few research studies.[4] Trafficking has been defined as the relocation of children from their communities of origin by middlemen or agents for economic gain.

Globally, an estimated 1.2 million children are being trafficked every year while 200,000 of these children population are reported being trafficked into agricultural, domestic, restaurant, or market work as well as plantation labour, diamond mining and sex work in West and Central Africa,[37] where Nigeria occupies a strategic position as the most populous country and major child trafficking route in the region. According to a religious institution in Italy, the trafficking of Nigerian girls and women to Italy and other parts of Europe has slave-like characteristics,

and it indicated that there are about 20,000 Nigerian girls engaged in commercial sex work in Italy, including 3,000 in Turin alone.[4, 38]

Child trafficking involves several actors; village recruiters, family members, friends, acquaintances and agents that prompt the child to move sometimes by deception or through an agreed payment. Different patterns of trafficking have been identified which include internal, cross border, inter-regional and international forms.[28]

Locally, Lagos, Owerri, Port-Harcourt, Calabar, Kano, Sokoto, Maiduguri, and Ibadan are locations noted to have high populations of trafficked children.[39]

Across international frontiers, Nigerian girls are trafficked by criminal rings and smugglers to developed countries e.g. Italy, Belgium and Netherlands for commercial sex work.[4, 40] Also, children between the ages of 7 and 16 have been transported to Gabon and Cameroon, from various points in the Eastern part of Nigeria; in the states of Abia, Akwa-Ibom, Cross River, Rivers and Imo.[41]

Factors promoting trafficking of children include poverty and the desire to earn a living or help support the family, demand for trafficked children for sexual exploitation and cheap migrant labour, porous border and lax regulatory environments, traditional migration pattern, lack of education and access to schools, conflicts, political unrest and natural disasters that devastate local economies and infrastructure. Others are trends associated with a rapidly globalizing world - use of internet facilities, child pornography, and scourge of HIV/AIDS in the sub-Saharan Africa, leaving millions of children orphaned, forcing children to earn money to care for a sick or dying parent or pressure on children to leave their village as a result of the stigma associated with having AIDS in the family.[28, 40, 42]

The consequences of trafficking are always devastating on victims whatever their age. They are especially pernicious and multiple. These include isolation from family and community, fear and psychological trauma as a result of their illegal status, physical and emotional harm, loss of childhood and education, therefore a blighted future. Also, in worst cases trafficking can result to drug dependence, permanent physical and mental health impairment or death. They also suffer food and sleep deprivation, poor accommodation and lack of access to good health services. If trapped in commercial sex exploitation, they may suffer violence at the hands of clients, sexually transmitted diseases including HIV/AIDS, early pregnancy and reproductive illness which may affect their ability to have children later in life.[28, 40]

Sexual Exploitation of Children

Sexual abuse of children and young adolescents is wide spread in all societies. The World Health Organization (WHO) estimates that overall prevalence is 25 percent for girls and 8 percent for boys.[43] A father or other male relative is the most common perpetrator, but abuses by peers, teachers, child caregivers, family friends, religious leaders, and neighbours also occur. Fifty-seven percent of sexually active female apprentice tailors in a peri-urban community in Ibadan reported that their sexual debut was with an instructor.[29] Boys and girls between the ages of 7 and 13 years are at greatest risk.[44] The sexual exploitation of children and adolescent girls constitutes a grave abuse of rights and is consequently deplored by both the Convention on the Rights of the Child (CRC) and Convention on the Elimination of all forms of Discrimination against Women (CEDAW). Forms of sexual exploitations against children can range from fondling and kissing of child's genitals through rape to child prostitution. Child prostitution is found to be common in towns such as Port Harcourt, Calabar, Owerri, Makurdi, Ilorin, Maiduguri, Lagos and across the geo-political zones in the country.[26, 45, 46] On the streets, in fostering homes, students in secondary and tertiary education experience one form of sexual exploitation or the other. Factors that put Nigerian children and adolescent girls at risk for sexual abuse include family instability, conflict, parental psychopathology, childhood neglect and physical abuse. Others are low socio-economic class, unemployment, culture of silence, gender inequality, poverty etc.[47] Traditional gender norms (societal assumptions and expectations about what it means to be male and female) have also been placed at the centre of factors underlying sexual coercion. Ajuwon (2003) showed that young men often feel entitled to sex while young women frequently agreeing that sex is a man's right illustrating one of the traditional gender norms among the Yoruba speaking part of Nigeria.

Association between childhood sexual exploitation and many short- and long- term adverse mental and physical health effects abound. These include adolescent pregnancy, HIV infection, physical trauma, tendency for victim to later force someone else to have sex, reproductive health problems such as chronic pelvic pain, premenstrual distress and inadequate or excessive prenatal weight gain, emotional problems such as depression and several anxiety disorders, sexualized behaviour and substance abuse. Vesicovaginal and rectovaginal fistulae have also been reported in cases of rape.[48]

VIOLENCE AGAINST CHILDREN

Child battering refers to acts of physical violence on children. In practice, the beating of children is almost universal in Nigerian homes and is applied frequently, as a mode of discipline for almost any type of misdemeanours, however trivial.[50] Corporal punishment is also widely used in schools.

Many a time, it is taken to the extreme and children are battered in the process of correction. Two groups of children - foster children and young house help tend to be especially vulnerable to the risk of abuse and negligence within the house.[47] There are other several forms of punishment meted out to children which are extremely harsh and are both physically and emotionally dangerous. These include forcing children who wet their bed to urinate by standing

at risk of burning; making incisions on the children's hands as punishment for petty within the household resulting sometimes in and leaving permanent mark of shame; of pepper to incision or to sensitive part of such as the eyes or genitalia to cause pain.

children usually have few friends, low self and wear clothes to cover up scars from injury. up with hatred for parents, become into violent behaviours and usually have dealing with their own children later in life.[9] deepened poverty, urbanization and changes, parents' frustration due to earnings and high inflations and polygyny the various factors that lead to loosening of restrictions on the forms, intensity and beating and cause an increase trend of child in Nigeria.[4]

dation

and traditions have been with us from time and are in a dynamic state; they are and can be revised to accommodate new and new values that emerge in human

many traditions and socio-cultural practices social cohesion and unity, others like UTPs, itative practices and other forms of child the physical and psychological health of individuals, particularly children and men in our society. Hence, there is urgent inate them to ensure the protection and of human rights of Nigerian children their right to bodily integrity and to the standard of physical, mental and social well

ns needed to eliminate these harmful must involve the three tiers of governments, al agencies, civil society and the various community.

ted Nation's plan of action for the of harmful traditional practices [51] as a following are advocated to bring an end to mful practices highlighted above:

The Nigerian government must express a political will and an undertaking to put an end al practices affecting the health of our particularly FGC.

There is a need for cooperation and understanding among community leaders, olicy makers, and the people who have perienced or witnessed the hardships these ractices cause.

Community mobilization and education is a powerful strategy that can be used to bring about change in attitudes and perceptions. It has the potential to influence and persuade religion and opinion leaders and heads of individual households to change their attitudes towards female subordination and abuse of children. It is critical to increasing public awareness of the negative consequences of these practices and changing societal norms.

- Legislation prohibiting various HSCPs should be enacted. The Child Rights Act should be passed into law by all State of Assemblies in the country, implemented and enforced.
- National committees and state governmental bodies should be established to combat HSCPs against children and young women and implement official policies adopted.
- As recommended by the WHO, the 18 years minimum age for girl marriage should be adopted through legislative process and re-enforced with necessary mechanism for its implementation.
- The media should be mobilized to raise public awareness on the consequences of HSCPs against children.
- Since the family is the basic institution from where gender biases emanate, wide ranging motivational campaigning should be launched to educate parents to value the worth of a girl child, so as to eliminate such biases.
- To discourage the early marriage of girls, governments should implement compulsory primary education, free secondary education and increase the access of girls to technical education. A certain percentage of places in existing training institutions should be reserved for female teenagers.
- Government should encourage by all means the activities of non-governmental organizations concerned with the problems of HSCPs in our society so as to bring about a joint effort in combating these problems.
- Efforts should be made to domesticate international instruments of change such as Convention on the Rights of the Child (CRC) and the Convention on Elimination of Violence against Women (CEDAW).
- Since the HSCPs are well entrenched in our culture, there is need for well-structured multidisciplinary research to adequately document the prevalence of such practices, explore reasons for perpetuation and document the immediate and long-term consequences. This information would help to inform government, custodians of our culture, and entire populace of the adverse effects of HSCPs against our children, and motivate and promote appropriate legislation to eliminate them.
- There is an urgent need to build national capacity for effective law enforcement and strengthening regional and international cooperation to combat organized trans-border criminal activities including children and women trafficking.
- The social welfare departments and Child Rights Implementation Committee for the

federal, state and LGAs should be provided with resources and capacity to carry out sensitization activities and the task of identification, reporting, referral, investigation, treatment and follow up of instances of child maltreatment, as spelt out in the article 19 of the CRC [1].

CONCLUSION

Elimination of HSCPs against children in Nigeria requires the commitment of all, from government, through non-governmental agencies, opinion and religious leaders, to the members of each community at large. Sentiments, political and religious ideology which help to perpetrate these harmful practices should be abrogated to make room for realistic examination of the threats posed by HSCPs to Nigerian children.

When respectful of tradition, advocacy can unite communities, reinforcing practices which benefit all members while at the same time confronting those which damage the integrity and diminish the humanity of our children and young women.

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CARDIOPULMONARY RESUSCITATION IN PAEDIATRICS- A STEPWISE APPROACH.

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ABSTRACT

It is imperative that guidelines be developed for paediatric resuscitation that address the unique needs of new born, infant and young adult.[1]

The 2005 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation and emergency cardiovascular contain recommendations designed to improve survival from sudden cardiac arrest and acute life threatening cardiovascular problem.[2]

These recommendations confirm safety and effectiveness of many approaches and acknowledges that others may not be & recommend new treatments that have undergone evidence evaluation.

Since children differ from adult as to the cause & pathophysiology & cardiopulmonary problems, so pre-hospital and resuscitation teams must be knowledgeable in this regards.

The purpose of this article is to present current views on this all-important topic in paediatric age group.

INTRODUCTION

Sudden collapse of individuals is common in our environment but most people lack the skills & knowledge of emergency cardiovascular care which include all responses either pre-hospital or in-hospital needed to stabilize victim or patient who suddenly developed life threatening events affecting cardiovascular, cerebrovascular & respiratory systems.

Any member of a community may have to provide immediate resuscitation because of the need for immediate treatment in various settings ranging from home, market, shopping mall etc. Basically, paediatric cardiopulmonary resuscitation (CPR) is the attempt to restore spontaneous circulation by using chest-wall compressions and pulmonary ventilations through any of a broad range of manouvers and techniques.

The goal is to maintain adequate oxygenation and perfusion of blood throughout the body while steps are taken to stabilise the child and establish long-term homeostasis.

INDICATIONS: CPR is highly recommended & indicated in foreign body aspirations in children, an electrocuted child, burn child, child in road traffic accident, near drowning, cardiac arrest etc.

COMPONENTS

CPR is one of the components of paediatric chain of survival, namely prevention of cardiopulmonary arrest, early CPR, early access to emergency medical services (EMS) and early access to paediatric life support (PALS).

When a child develops respiratory or cardiac arrest, immediate bystander CPR is crucial to survival. In respiratory arrest, rescue breathing with oxygen provides oxygen delivery to the brain & other vital organs. CPR also results in resumption of cardiac activity.

Components of CPR include; Basic life support (BLS), Advanced Life Support (ALS) & prolonged life support.

CHILD CARDIOPULMONARY RESUSCITATION

SEQUENCE

- Ensure safety of rescuer and victim
- Assess responsiveness
- Shout for help
- Activate emergency medical services (EMS) second rescuer is available

  A= Airway, B= Breathing C= Circulation
- Activate EMS after 5 cycles of CPR rescuer
- Continue CPR until PALS is available

CPR FOR NEWBORN

How do you determine whether the baby needs resuscitation?

- Is the body clear of meconium? If meconium present in the amniotic fluid or on the body's skin and not vigorous. You should intubate & suction the before performing other resuscitation steps. No more few seconds should elapse while you make determination.
- Is the baby breathing or crying? Breathing evident by watching the baby's chest. Gasping series of deep single or stacked inspirations occur in the presence of hypoxia and/or acidosis is indicative of severe neurologic and depressions.
- Is there good muscle tone? Healthy term have flexed extremities and are active.
- Is the baby pink? A baby's skin colour change blue to pink following delivery can provide rapid visible indicators of adequate circulation. Cyanosis caused by too little the blood will appear as a blue hue on tongue & central trunk.

INITIAL STEPS

- Place baby on pre-heated warmer
- Position baby with neck slightly extended

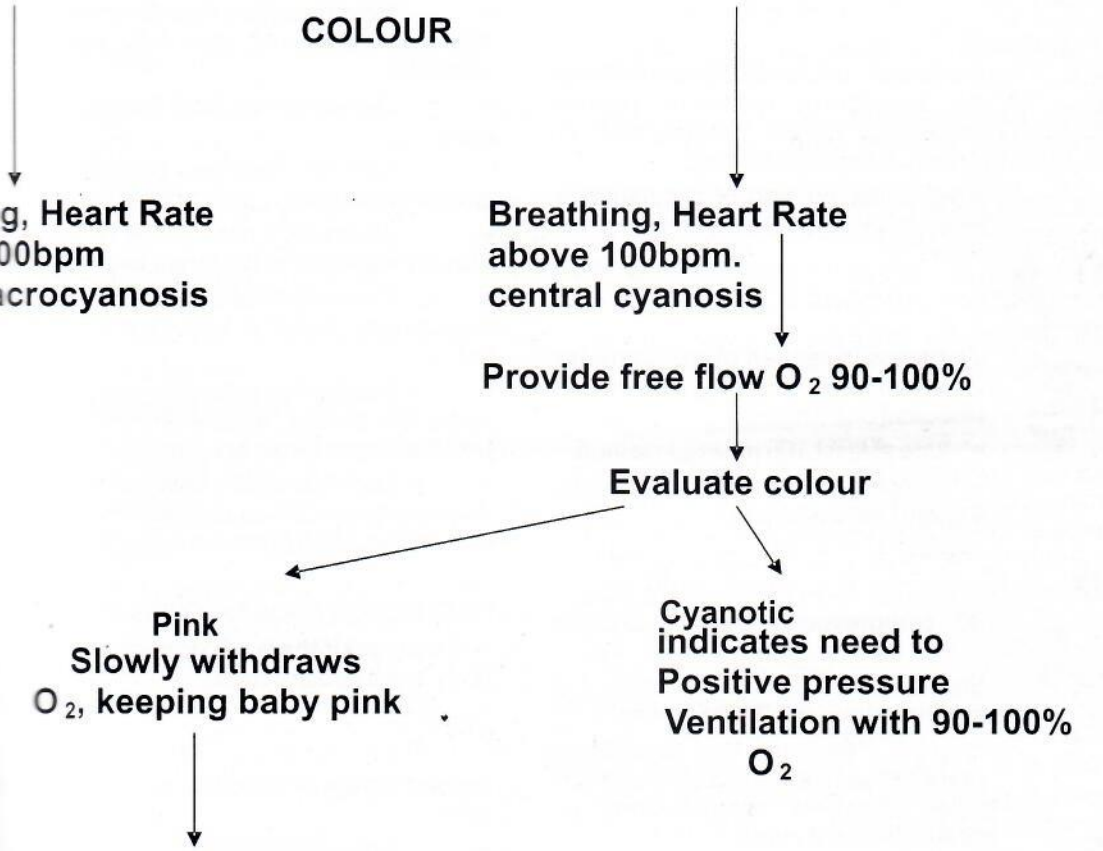
...tion mouth then nose
...amniotic fluid from the body & head, stimulate ... to breathe
...ove wet linen from contact with baby
...sition baby with neck slightly extended
...inister Oxygen as necessary.
...plete initial assessment, & steps evaluation in ...x 30 secs.

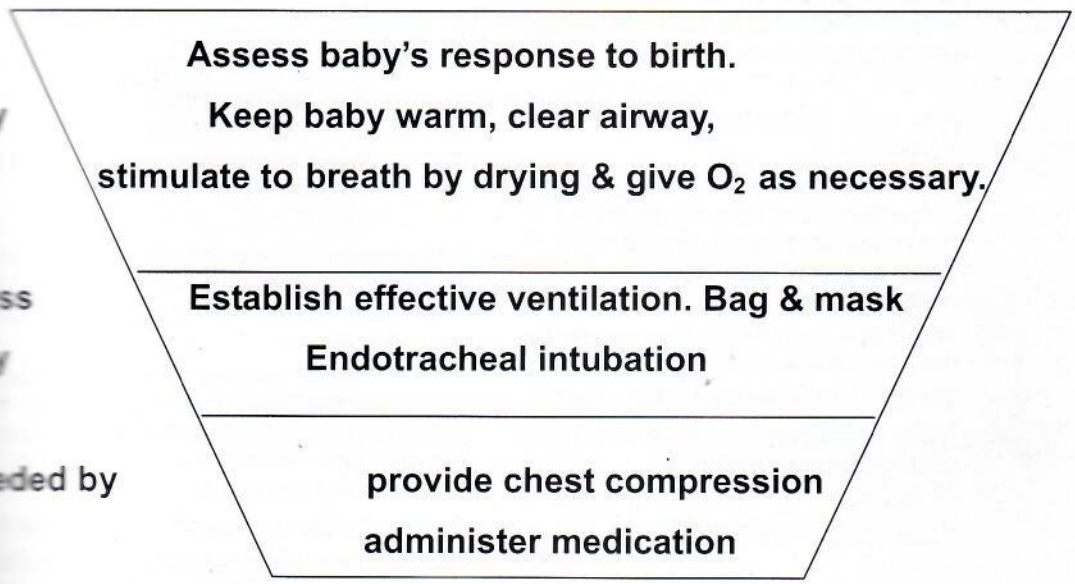
CPR FOR INFANT STEP WISE APPROACH

If you find an unresponsive infant, place flat on back on a firm surface. If infant is injured, moved only if necessary and turn head, & body as a unit.

Step 1: A- Open the Airway: Head tilt-chin lift (if head or neck injury is suspected, use jaw thrust)

FIGURE 1: EVALUATION OF RESPIRATION, HEART RATE & COLOUR





Step 2: B- Check for breathing (look, listen, feel). 1breathing is not adequate, provide 2 effective breaths at 1 second /breath

- Use bag-mask with oxygen if available
-

Be sure the chest rises with each breath

- If chest does not rise, re-open the airway, try again.

Step 3: C- Check for signs of circulation Pulse (brachial artery), breathing, coughing or movement.

If signs of circulation are present but no breathing; provide rescue breathing 12-20 breaths/min (1 breath, every 2-5 seconds).

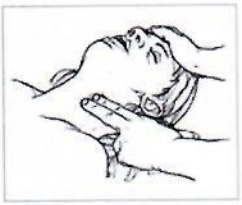
If no pulse, no sign of circulation is present, perform chest compressions. How?

- Compress lower half of sternum with 2 thumbs - encircling hands (2 rescuers) or 2 fingers
- *Rate at least 100 times per minute*
- 15 compressions, then 2 breaths (repeat) for 2 rescuers

30 compressions then 2 breaths - for 1 rescuer

Step 4: CPR: Provide 15 *compressions and 2 ventilations*

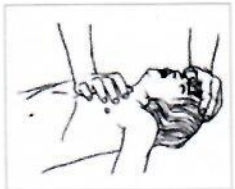
Step 5: Be sure the emergency number has been called after about 5 cycles of chest compressions or breaths (about 2 mins).



Also emphasise what not to do: Do not leave the victim alone, do not try to make the victim drink water, do not throw water on the victim's face, do not prompt the victim into a sitting position. Do not try to revive the victim by slapping his face.

CPR FOR CHILD BETWEEN 1 TO 8 YEARS

If you find an unresponsive child, place flat on back on a firm surface. If the child is injured, move only if necessary, and turn head, neck and body as a unit.



Shout for help send someone to phone emergency response number.

SAME: STEP AS WRITTEN ABOVE

TABLE I: Chest compression: ventilation relationship

	Neonate	1-8 yrs	> 8 yrs
Compression rate	120	At least 100	100
Compression to ventilation ratio	3:1	5:1	15:1

FIRST AID FOR THE CHOKING INFANT BIRTH TO 1 YEAR

You must act if there is any sign of severe or complete body airway obstruction

Signs of severe or complete airway obstruction in responsive infant

- Infant cannot cry forcefully
- Breathing attempts may produce high sounds
- Cough and voice are weak or silent
- Lips and fingernails may become blue

Actions to relieve foreign body obstruction

- Hold the infant face down on your forearm, infant's head in your hand (rest your arm on leg support).
- Deliver up to 5 back blows with the heel of hand
- Turn the infant over & give up to 5 chest thrusts below nipple line)
- Alternate 5 back blows & 5 chest thrusts object is expelled or the infant becomes unresponsive
- If the infant becomes unresponsive, open the with tongue jaw lift & look for a foreign object seen)
- Attempt to provide ventilation. If unsuccessful open the airway, re-attempt ventilation. If unsuccessful provide 5 back blows & 5 chest thrusts.
- *Continue steps 5&6 until ventilation is successful.* Then perform CPR as needed. Phone emergency after about 1 min of rescue support.

FIRST AID FOR CHOKING CHILD (APPROX. 1-8 YRS)

You must act if there are signs of severe or complete body airway obstruction.

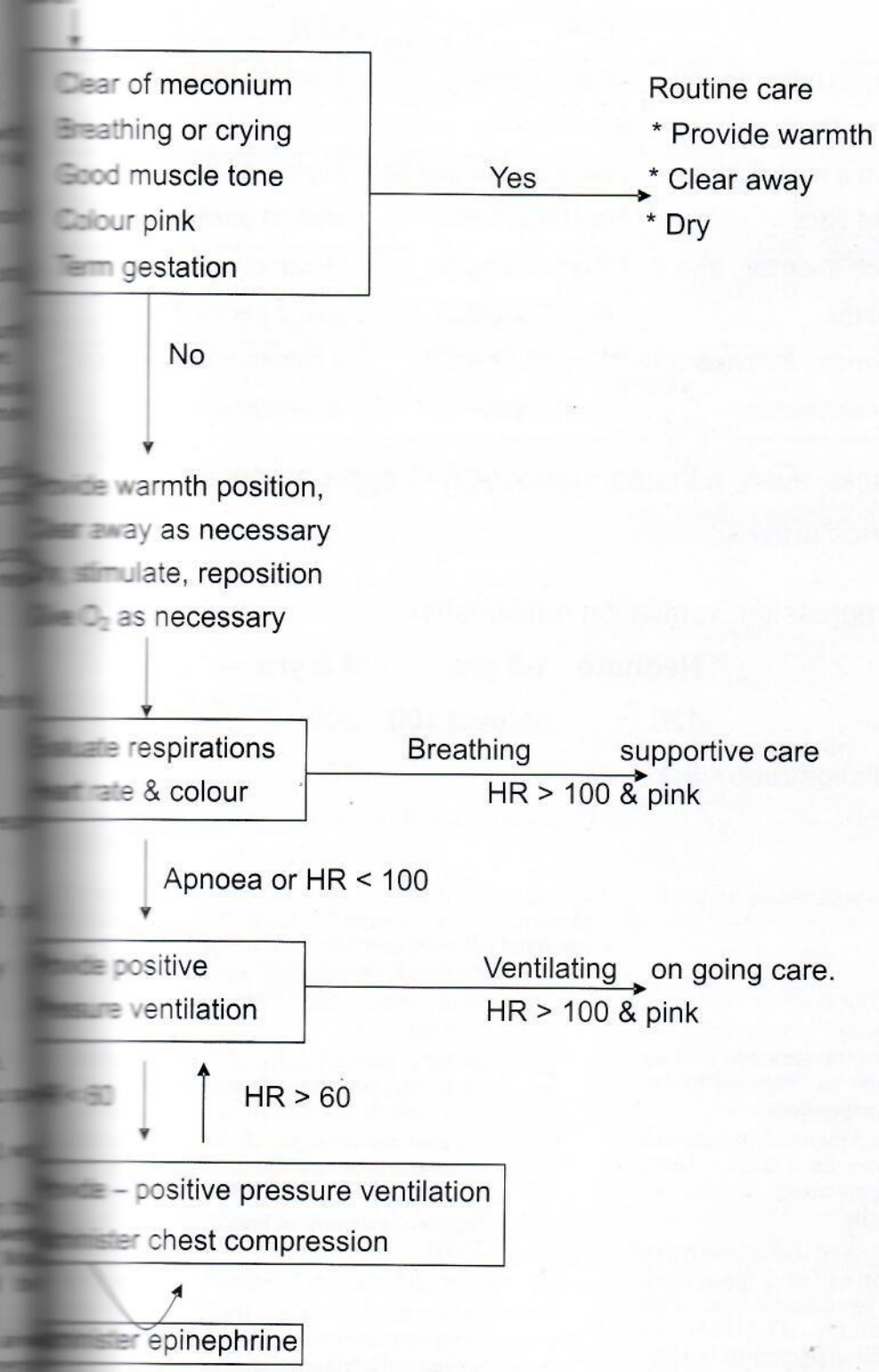
Signs of severe or complete airway obstruction in responsive child

- Universal choking signs
- Breathing attempts may produce high sounds.
- Child cannot cry, cough or speak forcefully
- Lips & fingernails may become blue.

Actions to relieve foreign body airway obstruction

1. Ask "Are you choking?" if yes ask; "can you" no, tell the child you're going to help.
2. Kneel or stoop or stand behind the child you arms around the child.
3. Make a fist with one hand; hold it with hand against the centre of child's abdomen, between and ribs. Provide abdominal thrusts (Heimlich) until the object is expelled or the child unresponsive
4. If child becomes unresponsive, open the tongue-jaw lift and look for a foreign object seen). Attempt ventilation. If unsuccessful, re-airway and re-attempt ventilation. If unsuccessful, see step 5.
5. Perform a series of 5 abdominal thrusts
6. continue steps 4&5 until ventilation Then provide CPR as needed. Phone the

FIGURE2: POST RESUSCITATION CARE IN NEONATES



Endotracheal intubation may be considered at several steps.

TABLE II: CARDIOPULMONARY REVIEW

	ADULT over 8 years of age	CHILD 1-8 years	INFANT < 12 months
Check for Responsiveness	By shaking & shouting	By shaking & shouting	By patting feet & chest
DIAL 9-1-1	If unresponsive	After 1-2 minutes of CPR	After 1 to 2 minutes of CPR
Pulse location	Carotid artery (neck)	Carotid artery (neck)	Brachial artery (arm.)
Airway	Lift the neck & tilt the head back	Lift the neck and tilt the head back	Slightly tilt the head into sniffers position
Breathing	Pinch the nose: give 2 breaths	Pinch the nose: give 2 breaths	Mouth over mouth &nose give 2 gentle puffs
Circulation	2 hands; 2 inches 30 compressions	1 hand, I inch 30 compressions	2 fingers, ½ chest depth 30 compressions

Check for pulse every minutes, repeat A-B-C cycle as necessary until ambulance arrives.

TABLE I: Chest compression: ventilation relationship

	Neonate	1-8 yrs	> 8 yrs
Compression rate	120	At least 100	100
Compression to ventilation ratio	3:1	5:1	15:1

responsive number after about 1 minute of rescue support.

COMPLICATIONS OF CPR

- Gastric insufflations: distension of the stomach with air could lead to regurgitation, vomiting & aspiration
- Chest injuries: rib fractures, pneumothorax, lung contusion etc.

CONCLUSION

Prompt cardiopulmonary resuscitation can save many lives. A,B,C of resuscitation is very important and must be learnt not only by medical personnel but lay-persons so that anybody can administer it before qualified help/hands arrives and patient is transferred to ICU.

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PAEDIATRIC ANAESTHESIA

ADEYANJU O.A , ATUCHUKWU O.N.

At the time of writing, the writers were second year clinical students at the College of Medicine, University of Ibadan.

This article gives an overview of the practice of anaesthesia in children. It outlines relevant anatomical and physiologic features of the paediatric patient with regard to safe delivery of anaesthesia. It also describes the preoperative assessment, intraoperative monitoring and post-anaesthetic complications in paediatric patients.

INTRODUCTION

Paediatric anaesthesia, as a specialty, utilises the knowledge of the physiological and anatomical peculiarities of the paediatric patient for the safe practice of anaesthesia. The paediatric patient includes the neonate, a child in the first 28 days of life, and the infant, a child in the first year of life. Children between ages 1 and 12 fall within this group.

Paediatric anaesthesia clinically categorises patients into:

- Premature neonates[born before 37weeks of gestation]
- Full-term neonates[37-40 weeks and less than 1 month of age]
- Infants[>1month - <1 year]
- Preschool[1-4 years]
- School age[4-8 years]
- Older child[8-12 years]

The risks of anaesthesia are greater in neonates and infants than in expert hands.[1]

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

Respiratory system

The airway, the passages of the nose, glottis and trachea are narrower. The epiglottis and tongue are also relatively larger in the small buccal cavity. This may predispose to airway obstruction. They are also obligate nasal breathers, hence a patent airway is mandatory. The diaphragm is almost entirely diaphragmatic with the soft, cartilaginous ribs contributing little to gas movement. The respiratory rate is also very rapid, about 30-40 cycles per minute. The average normal PCO_2 is 35mmHg and the normal PO_2 is 80mmHg.As a result, higher concentrations of oxygen is often needed in anaesthesia .At approximately 8 years of age, airway features are similar to that of the adult but smaller.

Cardiovascular system

Heart rate is an important determinant of cardiac output and must therefore be maintained in the normal range. The heart rate at birth is about 140 beats per minute and decreases gradually as shown in Table 1 below. The blood pressure at birth is 80/50 mmHg. This rises to 100/60 mmHg by the end of the first year and 120/70 mmHg at 10years of age. Table 2 shows the blood volume of a neonate in relation to birth weight. Blood volume is about 95ml/kg in the neonate, 85ml/kg in the infant and 75ml/kg in the older child. This is proportionally greater as compared to that of the adult. Haemoglobin concentration at birth is 20g/dl. This drops to about 8g/dl at the end of the first year.

Central nervous system

At birth, the spinal cord extends to the third lumbar vertebra and it takes its permanent position at the level of the first lumbar vertebra by the end of the first year. Neurones are developed at birth but myelination is incomplete .Thus, lipid-soluble drugs reach high levels more rapidly than in the adult. The blood-brain-barrier is also more permeable than that of the adult.

Liver

The liver is partly immature at birth. Thus, there is less protein available for binding to drugs, allowing more drugs to remain active.

Body temperature control

In infants, the temperature regulation centre in the hypothalamus is active but not well developed. The risk of developing hypothermia or hyperthermia is therefore higher.

PRE-ANESTHETIC ASSESSMENT AND PREPARATION

A pre-anaesthetic visit to the patient is important in order to establish a rapport with the patient, assess the patient's fitness for anaesthesia and where indicated, prescribe pre-medication. A rapport is established with the patient and his/her parents/guardian and the anaesthetic components of the procedure are explained.

Adequate history is obtained and should include perinatal and neonatal history, previous administration of anaesthetics, drug allergies and a review of systems to identify problems especially in the cardiovascular and respiratory systems. A physical examination which includes measurement of the child's weight preoperatively in order to assess the appropriate drug dosage is also carried out. The airway should also be examined for risk factors for possible difficult intubation which can be predicted in the presence of craniofacial malformation or tumours, trauma, foreign body aspiration, infections like retropharyngeal abscess. The child should be asked to open his/her mouth wide and to extend the neck to exclude trismus and cervical spine problems. Relevant laboratory investigations may be required. These include haemoglobin levels; blood grouping and cross-matching in view of possible blood

transfusion; electrolyte, urea and creatinine levels. The child is then resuscitated as indicated.

Premedication may be prescribed as necessary and are administered about 1-2 hours before the patient is brought to the operating room. Sedative premedication may not be required for neonates and infants however, for the older child, sedatives may be prescribed as required. These include:

-*Tranquilisers* such as diazepam[0.2mg/kg] or trimeprazin [2mg/kg by oral route].

-*Narcotics* such as pethidine [1mg /kg] or morphine [0.25mg/kg] may also be given intramuscularly 1 hour preoperatively.

-*Anticholinergics* such as atropine [0.02mg/kg] or hyoscine[0.015mg/kg]is given intramuscularly an hour before induction of anaesthesia to prevent excess salivation and mucous secretion which may be induced by some anaesthetic agents such as ketamine.

EQUIPMENT

- Anaesthetic circuit
- Ayre's T-piece

The use of this apparatus helps to reduce dead space to a minimum as any increase from anaesthetic apparatus is more significant in the infant.

- Jackson-Rees modified T-piece

This facilitates controlled ventilation and monitoring of spontaneous breathing due to the addition of a 500mlbag with an open tail to the respiratory limb.

- **Monitoring device**

These include: *pulse oximeter* to monitor arterial oxygen saturation; *sphygmomanometer* with appropriate sized cuff [the width of the cuff should be appropriately equal to half the circumference of the limb; *capnograph* for end tidal CO_2; *electrocardiograph* to monitor the electric activity of the heart; *catheter* for monitoring urine output ; *peripheral nerve stimulator* when muscle relaxants are used etc.

INDUCTION OF ANAESTHESIA

Anaesthesia may be induced by:

- Inhalational route: using halothane, sevoflurane, etc
- Intravenous route: using thiopental, ketamine, propofol (in those above 3years), etc
- Intramuscular route: using ketamine, or
- Rectal route: using thiopental suppositories.

Inhalational induction is generally preferred due to avoidance of venepuncture although it is associated with increased pollution of the operating room .Intravenous induction is however the anaesthetic technique of choice in emergencies especially in patients at risk of aspiration.

MAINTENANCE OF ANAESTHESIA

Maintenance can be achieved by:

- Inhalational anaesthetic agents.

The minimal alveolar concentration (MAC), that is, the end tidal concentration of a gas or vapour at which 50% of patients move in response to surgical skin incision[2], is higher in children as shown in Table 3 below. Therefore higher concentration of the inhalational agent is required for the maintenance of anaesthesia.

- Muscle Relaxant/Narcotic technique

Neonates are particularly sensitive to non-depolarizing blocker, although this reverts to normal after the neonatal period. They are also more sensitive to narcotics especially morphine.

ENDOTRACHEAL INTUBATION

Due to the narrowness of the airway of the paediatric patient, endotracheal intubation is indicated to secure the airway. The endotracheal tube used is usually non-cuffed because the narrowest portion of their airway is sub-glottic . Wet gauze is used to pack around the tube to prevent aspiration and gas leakage.

The internal diameter in millimetres of a tracheal tube for a child can be calculated using the formula:

$$\frac{\text{Age (in years)}}{4} + 4$$

FLUID AND ELECTROLYTE BALANCE DURING OPERATION

Total body water constitutes 75-80% of body weight in neonates and 70% of body weight in infants. For every 100mls of water, an infant needs 3mEq of Na^+, 2mEq of K^+, 2mEq of Cl^- and 5g of glucose. This is provided by 5% dextrose in one-quarter strength normal saline and is the reason for its routine use in maintenance.

Hourly fluid requirements based on weight are as follows:

Body weight	<10kg :4ml/kg/hr
Body weight	10-20kg:40ml +2ml/kg/hr for each kg over 10kg
Body weight	>20kg: 60ml+1ml/kg/hr for each kg over 20kg

Intravenous fluids may be administered using the burette type of infusion set allowing rate to be controlled.

BLOOD THERAPY DURING OPERATION

Blood volume represents 8-10% of the child's weight. Thus, replacement is necessary if there is loss of more than 10% of estimated, circulating blood volume and low pre-operative haemoglobin concentration. Blood can be given slowly using a two-way stop-cock syringe, after passing the blood through a blood filter.

POST-ANAESTHETIC CARE AND COMPLICATIONS

Post-anaesthetic care should be in place in order to prevent early post-anaesthetic complications, promptly diagnose and manage the complications that may develop and provide the patient with comfort. It involves continuous monitoring of vital signs and adequate attention to the patient.

Possible complications of anaesthesia and their management post-operative include:

- *Nausea and vomiting*: This may be due to the effect of the use of some drugs and may lead to aspiration. It can be managed by the use of anti-emetic drugs.

Table 1[3]

Normal Heart Rates	[beats per minute]
Term	140
6 months	130
1 year	120
7 years	100
14 years	80

Table 2

Blood Pressures in Normal Neonate [in mmHg][4]

	Birth weight			
	> 1 k g	1 - 2 k g	2 - 3 k g	> 3 k g
Systolic	40-60	50-65	50-70	50-80
Diastolic	15-35	20-40	25-45	30-50

Table 3

Minimum Alveolar Concentration [in %] of Anaesthetic Agents[5]

Age[in years]	Halothane	Enflurane	Isoflurane	Desflurane
0-3	1.08	2.0	1.35	9.0
3-10	0.9	1.9	1.3	8.0
Adult	0.76	1.7	1.15	7.0

nursing them child in the recovery position to prevent aspiration.

- *Pain:* This can be managed by the administration of adequate parenteral narcotics, nerve blocks with local anaesthetic agents, ketamine at sub-anaesthetic doses and inhalational anaesthesia.
- *Laryngospasm:* This is a fairly common peri-operative complication managed with 100% oxygen via face mask or re-intubation and oxygen therapy.
- *Hypothermia:* This may lead to hypoventilation, apnoea or delayed recovery from anaesthesia and should be managed adequately. This can be achieved by maintaining a warm ambient temperature and pressure, warming of intravenous fluids, covering of exposed skin.
- *Post-operative psychological or sleep disturbance:* This can be prevented by avoidance of opiods where possible and encouraging parental presence with patients.

Other post-anaesthetic care includes:

-Oxygen therapy

-Fluid and electrolyte management.

-Management of hypoglycaemia which may cause prolonged unconsciousness after general anaesthesia in neonates and infants.

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MENTAL HEALTH THE FORGOTTEN HEALTH ASPECT OF NIGERIAN CHILDREN AND ADOLESCENTS

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Abstract

Child and Adolescent Mental Health is a field which started to develop in the twentieth century. The growth of this field has been slow because of higher priority given to other causes of childhood morbidity and mortality by policy makers and may have the greatest impact in sub-Saharan Africa which though has the highest population of children, is still battling with the burden of infectious disease and malnutrition. This article reviews the components of child and adolescent mental health, the relationship between developmental stage and presentation of mental illness in children, protective and risk factors for child mental health disorders with particular reference to present day Nigeria.

The United Nations Convention on the Rights of the Child delineates the rights to be accorded to children and their families which include sound mental functioning. There is a dearth of mental health policies and services for children all over the world especially in the developing world. There is also an urgent need for interdisciplinary collaboration among child care professionals to ensure physically and mentally healthy children

Key Words: *Mental Health, Children, Adolescents, Aspects, Services, Nigeria*

Introduction

The World Health Organization defines health as a state of complete physical, mental and social well being and not merely the absence of disease or infirmity[1]. Children and adolescents have been recognized as thinking and feeling beings with varying degrees of mental complexity and a child is mentally healthy when he is able to learn productively, develop his own abilities, cope with the normal stresses of life, and gradually make positive contributions to his own community[2,3].

For several decades, the focus of child health worldwide has been targeted at reducing childhood mortality and disability resulting from infectious diseases and malnutrition, and increasing child survival[4,5]. The field of Child and Adolescent Mental Health only started to develop mid- way through the twentieth century, and has recorded a much slower growth in sub Saharan Africa despite the fact that almost half of the population is less than 15 years of age[2,6,7,8]. This however is not surprising as this sub-continent is still battling with providing basic infrastructure and social services like food, education, housing, roads, portable water, electricity etc[4,7,9,10].

Mental illness in children and adolescents is a poorly understood phenomenon. In some parts of the world, mentally ill children are seen as 'troublesome' or 'not trying hard enough.[11,12] In the Nigerian society they are labeled as 'wicked', 'under a spiritual attack' or a product of generational curses from the gods[13,14]. Epidemiological surveys on child and adolescent mental disorders worldwide reveal prevalence rates of between 10 - 20%[15] and similar findings have been reported in Nigeria. For example, the study by Gureje et al, (1994) in an urban primary care facility in south west Nigeria found a prevalence of 20%.[16] In comparison, Abiodun, (1992) in North Central Nigeria found mental health problems in 15% of children between the ages of 5 -15 years within the community.[17] Lifetime depression rates of 9 and 12% respectively have also been reported among secondary school children in South West Nigeria[18,19].A similar study also found that 20% of children aged 10 -17 years reported suicidal ideation while 12% had attempted suicide in the past one year.[20] Approximately 50% of adult mental disorders are reported to start before the age of 14 years. Thus when mental health problems in this age group are left untreated, they lead to chronicity in adulthood, reduced occupational and economic achievement, poor quality of life, increased burden on family members and have a negative impact on national development.

Components of Child and Adolescent Mental Health

Mental health like other aspects of health involves components. These are mental health promotion, prevention, treatment and rehabilitation[3].

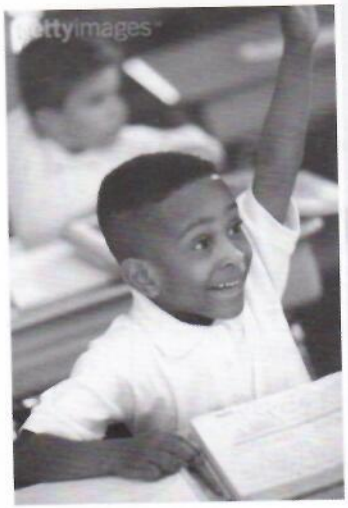
Promotion of mental health involves actions that create living conditions and environments which support mental health such as good nutrition, safe housing, good schools and drug free environments and allow people to adopt and maintain healthy life styles[1]. Prevention refers to the organized effort to prevent occurrence or progression of mental disorders such as providing safe antenatal care for mothers, ensuring immunization of infants, and educating children/ parents on good mental health habits[3,7]. Treatment refers to all clinical and non-clinical care which is aimed at reducing the impact of mental disorders and improving the quality of life of children with mental disorders, while rehabilitation refers to care given to mentally ill children and adolescents to help restore them to their optimum level of social and psychological functioning and be accepted back into their communities.

Developmental perspective of Child Mental Disorders

The mode of presentation of mentally ill children is determined partly by their stage of biological development

Table 1: Factors affecting child and adolescent mental health.
(Adapted from Mental Health Policies and Service Guidance Package; Child and Adolescent Mental Health Policies and Plans, WHO)

Domain	Risk Factor	Protective Factor
Biological	Exposure to toxins in pregnancy, genetic vulnerability to psychiatric disorder, hypoxia at birth, other birth complications, infections including HIV and malnutrition	Age, appropriate physical development, good physical health, good intellectual functioning
Psychological	Learning disorder, physical/ emotional abuse or neglect, difficult temperament	Good self esteem, good problem solving abilities, social skills
Social		
a. Family	Family conflict, poor family discipline, bereavement	Family cohesiveness
b. School	Inadequate/ inappropriate provision of education, academic failure	Opportunities for involvement in school life. Positive reinforcement from academic achievement
c. Community	Exposure to violence, discrimination and marginalization, frequent displacement and migration	Connectedness with community, positive cultural experiences, positive role models



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combined with psychological and environmental factors such as parent and family relations, peer, neighbourhood, school and community factors[22]. Certain disorders are therefore more commonly found within certain age groups. In the first three years of life, children who have suffered abuse or neglect from parents or caregivers may present with Attachment Disorders which are characterized by being withdrawn from family members, or being over-familiar with strangers. From age three upwards, the Disruptive Behaviour Disorders commonly present. These are the Attention Deficit Hyperactivity Disorders (ADHD) where children are persistently more restless and inattentive than their peers, tend to be troublesome, highly distractible, perform poorly in class, and are unpopular with other children. [12]. Conduct Disorder on the other hand, is characterized by children who persistently have great difficulty following rules and behaving in a socially acceptable manner with little or no remorse[12]. Though all children pass through a stage of defiance to laid down rules, and self assertion, persistence or exaggeration of this beyond the expected age should raise the suspicion of a mental disorder. Pervasive Developmental Disorders like Autism present within the first six years of life. These are a group of disorders which severely impair development of a person's ability to communicate, interact with other people, and maintain normal contact with the outside world.

During the school age years (age six upwards), emotional disorders such as Depression and Phobias begin to manifest with mood swings, irritability, social isolation and. suicidal ideas[12]. From adolescence (age 11), experimentation with psychoactive substances like cigarettes and alcohol may lead to substance use dependence disorders, and finally from about age 15 upwards, children may experience adult type of psychosis with its characteristic disorganization in the thinking process, abnormal perceptions and behaviour. In Nigeria, a five year review from of children referred to an outpatient clinic in Lagos opened in 1999 revealed that 25% presented with acute psychosis, 6% with ADHD, 2.9% with Autism, 6% with Conduct disorders, and 1.3% with Depression.[14]

Factors affecting Child Mental Health

Protective and risk factors have been identified in the development of mental disorders in children[3] and these can be grouped into biological, psychological and social (Table 1).

A study carried out in a clinic in Ibadan Nigeria showed an association between obstetric complications and subsequent mental disorders. Children with disruptive behavior disorders (ADHD, conduct disorder and oppositional defiant disorder) had the highest number of perinatal and pregnancy complications. These children also had the highest number of birth injuries[23]. Other biological factors which are equally relevant in this environment include infections and nutrition.

Omigbodun, (2004) reports that two thirds of children attending a clinic in south west Nigeria experienced significant psychosocial stressors like family disruption, dropout of school / lack of appropriate school, having to live on the streets, and extreme poverty within a year before presentation. Cultural practices peculiar to this environment such as separation from parents to live with relatives for financial reasons or to correct behaviour, young children working as house helps, children embarking on prolonged fasts for religious purposes or being locked up in traditional healers homes/ religious centres for years, physical abuse by care givers to get rid of demons responsible for behavioural problems, and abandonment of children with epilepsy, were also identified as potential stressors, but the long term effects of these practices are yet to be determined. [18]

Value systems which promote individualism, weaken social ties, and create ambivalence towards children are also being recognized as harmful to children's mental health, while values such as duty, responsibility, and a community orientation are protective to child mental health [24, 25, 26].

Child and Adolescent Mental Health Policies and Service Provision

The United Nations (UN) Convention on the Rights of the Child recognizes that children and adolescents are a group of individuals with special needs and states the duties of their respective governments towards them. Despite this, the mental health provisions of this convention are yet to be implemented by many world governments[7]. In Africa for example, local child relevant mental health policies are available in only 35% countries as compared to 95% European countries[27]. In Nigeria, the mental health policy formulated in 1991 recognizes children and adolescents as a special group in need of special interventions but there is currently no separate child mental health policy though there are a few other Nigerian policies which impinge on the mental health of Nigerian children such as The National Policy on the Health and Development of Adolescents and Young people in Nigeria (2007), and the Substance abuse policy formulated in 1990.

The organization of mental health services for children may range from community to hospital-based services. Child psychiatry actually started as a community discipline in child guidance clinics but due to practical difficulties in the administration of these clinics, many closed down and were replaced by hospital based services in the 1980's.[28, 29] Mental health services in the last 10 -15 are gradually shifting back to the community based on growing evidence that community services are more accessible, cheaper, and more easily integrated with other sectors of society (social, educational, legal, and religious) in order to achieve a continuous and comprehensive system of care.

Hospital based services can be organized in primary care, general (secondary) or specialist (tertiary) hospitals. Primary care services aim at education of parents/ children about general and mental health issues, screening for mental health problems, and offering counselling and support[30]. General hospital care supports primary care facilities, as well as managing psychiatric presentations in children and adolescents before referral to specialist services if necessary. Specialized services offer highly specialized diagnostic and treatment services through specialized units (Child and family, Adolescent, Substance abuse and Eating disorders units, and Rehabilitation) for those with severe mental disorders[3] In present day Nigeria, mental health care is provided either informally by families and their support network (natural healers and faith-based organizations) or formally by few government tertiary facilities while community services are almost non-existent.[31, 32]

Conclusion

Overall one in five children is at risk for developing mental health problems. However, child and adolescent mental services should focus not only on treating childhood mental disorders but also in preventing, promoting, as well as maintaining good mental health. A well structured mental health system which provides these services and allows integration with other child care services is essential. This will help not only to reduce the distress caused to

families, but will also go a long way in improving the functioning of each community.

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DEATH IN THE FAMILY; IMPULSES AND RESPONSES OF A CHILD

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ABSTRACT

During the first 10 years of their lives, 6% of children lose a parent, children often experience other vital loses, such as uncles, aunts, cousins, siblings, playmates, teachers, and favourite recreation leaders.

Psychiatrists and paediatricians will be useful in this setting as the affected child goes through a lot of psychological stress which could eventually result in conversion disorders.

This article sheds more light into the problems associated with bereavement in the family taking the child into consideration, solutions and presentation of complications.

Keywords: Bereavement, child, family.

INTRODUCTION

Death in the family affects everyone but the transiting period of grief varies from one individual to another and the way we react to these changes in our lives depends on a number of factors. Children most especially need to be thought about even if it is a difficult time for the entire family most especially being around to answer their never-ending questions about the demise of the person as well as helping them through the bereavement.

Nonetheless, in most families, parents don't think about explaining death to their children until a relative dies. Today it is important to be aware that an understanding of death does not enter a child's picture of the world by itself. Children have to be told about death. It will make sorrow and death much easier for a child to deal with if they know something about it beforehand.

When someone in a family dies, most parents attempt to protect their children by not talking about sorrow or death. This is a misguided kind of protection. Sooner or later the child will be confronted with the subject. If a child has some understanding of the meaning of the word 'death', he/she will be better equipped to deal with the situation.

If a child has been protected against sorrow, they will still be hurt when they realise what has happened. Nobody can protect a child, only postpone it. Often, trying to protect a child will only cause them unnecessary anxiety and sometimes even guilt.

It may not be possible for a parent to talk to a child about death when someone in the family dies, because that parent is too upset. It would be beneficial for the whole family if the child had been prepared before the actual death.

FACTORS AFFECTING BEREAVEMENT

Various factors determine how a child, particularly in this age group will go through the process of grief and what the outcome would be. These are discussed below.

The relationship (mostly emotional) of the child with the deceased goes a long way in affecting the process of bereavement. The death of a distant uncaring father may affect the child as it would for a close uncle.

Another point to consider is the child's age and level of understanding. Infants may feel the loss mainly because of the way in which they are looked after and their daily routine. They are very sensitive to the unhappy feelings of those around them, and may become anxious, difficult to settle and more needy of attention. Pre-school children usually see death as temporary and reversible - a belief reinforced by cartoon characters that `die' and `come to life' again. Children from about the age of 5 are able to understand basic facts about death: it happens to all living things, it has a cause, and it involves permanent separation. They can also understand that dead people do not need to eat or drink and do not see, hear, speak or feel. Teenagers are able to understand death much more like adults, and are very aware of the feelings of others.

Most children get angry and worried, as well as sad, about death. Anger is a natural reaction to the loss of someone who was essential to the child's sense of stability and safety. A child may show this anger in boisterous play, by being irritable, or in nightmares. Anxiety is shown in `babyish' talk and behaviour, and demanding food, comfort and cuddles. Younger children believe that they cause what happens around them. They may worry that they caused the death by being naughty. Teenagers may find it difficult to put their feelings into words, and may not show their feelings openly, for fear of upsetting others.

The circumstances of the death also affect the impact on the child. For instance, the reaction of a child to a sudden unexpected death seems quite different from that resulting from a protracted illness because the child would have been psychologically and naturally prepared for such occurrence.

Religion and culture will have an important influence on what happens. So also, the coping skills of other family members and the effect of grief on other family members, especially if they are not able to cope with giving the child the care he/she needs.

RESPONSES BY CHILDREN

Being aware of how children normally respond to death makes it easier for an adult to help. It also makes it easier to identify that a child is finding it particularly hard to cope with.

In the early stages, adults sometimes try to protect children from pain by not telling them what has happened. Experience shows that children benefit from knowing the truth at an early stage. They may even want

to see the dead relative. The closer the relationship, the more important this is. Adults can also help children to cope by listening to the child's experience of the death, answering their questions, and reassuring them. Children often worry that they will be abandoned by loved ones, or fear that they are to be blamed for the death. If they can talk about this, and express themselves through play, they can cope better and are less likely to have emotional disturbances later in life.

Young children often find it difficult to recall memories of a dead person without first being reminded of them. They can be very upset by not having these memories. A photograph can be a great source of comfort. Children usually find it helpful to be included in family activities, such as attending the funeral. Thought should be given as to how to support and prepare a child for this. A child who is frightened about attending a funeral should not be forced to go. However, except for very young children, it is usually important to find a way to enable them to say goodbye. For example, they can light a candle, say a prayer, or visit the grave.

Later, they accept the death; they are likely to display their feelings of sadness, anger and anxiety on and off, over a long period of time, and often at unexpected moments. The surviving relatives should spend as much time as possible with the child, making it clear that they can show their feelings openly, without fear of upsetting others. Sometimes a child may `forget' that the family member has died, or persist in the belief that they are still alive. This is normal in the first few weeks following a death, but may cause problems if it continues.

HOW TO DEAL WITH DEATH IN THE FAMILY

Bereavement is a powerful, life-changing experience that most people find overwhelming the first time. Although grief is a natural process of human life, most of us are not inherently able to manage it alone. At the same time, others are often unable to provide aid or insight because of discomfort with the situation and the desire to avoid making things worse.

The sudden death of a person close to you is always a very painful and difficult experience. You will probably have strong feelings over a period of time, and sometimes they may seem overwhelming. Some feelings you might expect to have are those of shock, sorrow, and loneliness. Others might surprise and confuse you. For instance, many people are not prepared for feelings of denial, anger, or guilt. Yet they are very common reactions to the death of a loved one. The pain can be physical as well as emotional. You may have changes in eating and sleeping patterns, weakness, or other physical symptoms. Altogether, these are the feelings of grief. Working through the feelings toward a meaningful life in the future is called the "grieving process." Although most people experience some or all of these feelings when they lose someone they love, each person is different. The feelings you have, how strong they are, and how long they last will not be exactly the same as for anyone else. The practical things that must be dealt with at this time can be confusing and stressful as well, particularly because the loss is unexpected.

Children grieve over the loss of a loved one, just as adults do. It is important to include children in the grieving process. Tell them the truth, using words they can understand, and be honest about your own feelings. Don't exclude them in order to protect them from the pain of their loss. It will only increase their confusion and fear about their own feelings and the feelings they see around them. Young children may have a hard time expressing their feelings in words, and they may have a limited understanding of what death means. Their fear, anger and sense of loss may be expressed in changes in behaviour. They may have nightmares, or trouble eating or sleeping. They may go back to earlier childhood behaviours, such as bedwetting. Some children might become more aggressive and some might become quiet and withdrawn. It is important that children be able to express these feelings safely. At this time, when you are coping with your own grief, it may be difficult to deal also with the special needs of children.

Pay particular attention to children-they need to grieve as much as adults. However, the grief may show itself in a different way. It is not unusual to see children acting out grief one minute, and then playing happily the next minute. Try not to limit their tears, feelings or even anger.

Above all, children need to feel safe, loved and cared for. Use simple, direct language to explain that a loved one has died.

Some helpful guidelines may include:

- answer their questions in a way that satisfies them and try not to give more information than required-give a brief explanation and answer in a language level that the child can easily understand
- don't be afraid to use words like dead and death
- never tell children anything they will have to unlearn later (e.g., "Grandma has gone away" or "Grandpa is sleeping") the child should understand that death is permanent, and the loved one will not be coming back
- let children know that it is okay to show their emotions, and
- reassure the child that he or she is loved and will be cared for by others.

Children do not need protection; they need competent guidance and satisfactory answers to their questions. The development and age of the child needs to be borne in mind. The parent or guardian knows how the child likes like to talk about things, the sort of language they can use and if there are other ways they like to communicate, through drawing for example.

Children less than eight years of age are often interested in death and have complex concepts about it but are not able to grasp its finality. They, and many adults, have magical beliefs about how life can carry on after death or how many people come back to life. The understanding that this is not the case only comes with greater maturity and then will be affected by the cultural and spiritual beliefs of the child's family and community. By making this preparation a part of everyday life, death will be a natural thing for a child. Flowers that wither and die, or a pet that dies, may provide an opening. It is possible to talk about elderly people whom the child knows and talk about yourselves getting old and dying.

It is important to be prepared for these questions. If they make the parent uncomfortable, the child will notice and stop asking questions. A child will watch to see whether they are allowed these kinds of questions and the reaction they create. Remember children do not sit down and discuss a subject for hours on end. They will come running and ask some of the hardest questions

the world. That offers little time to think answers through. A child may ask a question that a parent cannot answer. It is honest and OK to say 'I don't know'. After a couple of minutes, they might want to go back outside to play. Seizing the moment is important. Talk about the subject when they want to. It is natural for them to change the subject and then return to it later.

When telling a child that someone has died, make sure the word 'died' is used. Children do not understand euphemisms. Some children have waited years for a grandparent to return because they had been told he or she had 'passed away'. Euphemisms may help an adult feel better but they won't help a child understand what has happened.

When mourning, let a child know it. The parent should let him or her see they are truly sad. If grief is hidden, the child will think that grief is not an acceptable feeling.

A funeral is a ceremony that helps people accept death. The child is a part of the family and it is only natural that they take part in the funeral along with everybody else. Prepare them for what might happen at the funeral. Tell them exactly what is going to take place and why. Tell them that some of the mourners may cry. If a parent's own grief prevents them from talking to the child and preparing them for the funeral, another close relative or friend can do it. Whether or not to take part in the funeral should be the child's choice. It is not something a parent should force a child to do. If they don't want to go, ask them why not and let them talk about their feelings.

If your child asks 'am I going to die?' tell them that they will, but not for a long time. If a child asks whether a parent is going to die, they should be told that all people die eventually, but that their parent will not die for a long time. It is always good for everybody to remember their loved one who has died. Through memories, the person is kept alive in our minds. It is helpful to leave a photo album out for the child to look at their pictures whenever they like. Help children hold on to happy memories of the person who died. Say 'Do you remember?' or 'That was how he wanted it' or 'This was her favourite food'. A child will know that it is good to remember.

NEED FOR PSYCHOTHERAPY

Ineffective communication between the parents and their child (or children) during the period of grief may result into some behavioural problems without an organic basis in the child. These may include:

1. a long period of depression, with loss of interest in daily activities and events
2. inability to sleep, loss of appetite, prolonged fear of being alone
3. acting like a much younger child for a long time
4. denying that the family member has died
5. imitating the dead person all the time
6. talking repeatedly about wanting to join the dead person
7. withdrawing from friends
8. a sharp drop in school performance, or refusal to attend school.

These are warning signs and indicate that professional help may be needed. A child and adolescent psychiatrist or child psychotherapist can help the child to accept the death, and also assist the survivors to find ways of helping the child through the mourning process. Your general practitioner will be able to offer you help and advice, and can refer you and your child to your local child and adolescent mental health service. The team includes child psychiatrists, psychologists, social workers, psychotherapists and specialist nurses.

Conclusively, death is an inevitable end for everyone irrespective of the age we own up to the fact. And as such our minds should be well-prepared in this light; most especially children who may feel uneasy with the events following the death of a relative. The main therapy in this regard is by making sure the child understands that death is certain for everyone; only then can he/she smoothly go along with the family in the grieving process without any untoward effect.

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ENURESIS, ITS EPIDEMIOLOGY, OUR BELIEFS PRACTISES AND THE MEDICAL POINT OF VIEW.

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ABSTRACT

It is no secret that most, if not all of us have at one time or the other bed wet even though we probably stopped before the age of 6 years. Enuresis not disabling though could be a cause of great distress, low self esteem and could also interfere with normal activities for instance sleepovers or holidays. Frequently, there may be psychosocial stressors predisposing a child to enuresis thus, making it an important aspect in the assessment of the mental health of children. This article aims to highlight some of the epidemiological patterns, some myths and cultural practises, and the medical point of view of this elimination disorder.

Key Words: *Enuresis, Enuretics, Nocturnal, Bladder, Incontinence.* ***ENURESIS: EPIDEMIOLOGY; BELIEFS; CULTURAL PRACTISES AND THE MEDICAL POINT OF VIEW***

INTRODUCTION

Enuresis is a common problem that can be a source of worry to children and their families[1]. Enuresis is an elimination disorder in which there is repeated voiding of urine into a child's clothes or bed after the age at which bladder control would normally be expected, it may be voluntary or involuntary, nocturnal or diurnal[2].

It could also be defined as a near complete emptying of the bladder in a wrong locality at a wrong time at least twice a month after the 5th year of life[3]. Enuresis can be primary, where the child has not achieved continence before or secondary where the child has earlier achieved continence.

Enuresis though does not kill; it often causes great mental suffering and anxiety to both the enuretic child and his parents. It has been associate with a slight increased risk of conduct problem, attention deficit behaviour and anxiety symptoms[4].

EPIDEMIOLOGY

The prevalence of enuresis decreases with increasing age. The evidence from the majority of studies has shown that enuresis is a rather frequent disorder in children. Eighty-two percent of 2 year olds, 49 percent of 3 year olds, 26 percent of 4 year olds, and 7 percent of 5 year olds have been reported to be enuretic. However, Prevalence rates vary, on the basis of the population studied and the tolerance for symptoms in various cultures and socioeconomic groups[2].

In a study of 7931 children aged 5-15years in the Netherlands the overall prevalence of enuresis was 6%: 15% in the 5-6 years age group and 1% in the 13-15 years age group[5].

While in Italy, an epidemiological multi-centre study of nocturnal enuresis in 7012 school children revealed a prevalence of 3.88%. Bedwetting was more frequent in boys than in girls and the prevalence of enuresis was higher in children from families with low socio-economic status across all age groups[6].

In 1996, a Dutch study among 6009 children reported prevalence of 24.6%, 15.7% and 5.4% at 3-4, 5-6 and 11-12 years age groups respectively[7].

A study carried out in 2001 in Igbo-ora, Nigeria among 633 children found a prevalence of 18.64% with a higher frequency of bed wetting in boys than in girls[8].

All the studies reported a higher frequency of enuresis in boys than in girls. However, the study done in Nigeria shows a higher prevalence, this is likely due to the lower sample size or environmental factors like socio-economic status, culture etc.

The Isle of Wight study reported that enuresis affects about 1 percent of adults[2]. About 20 percent of enuretic children have mental disorders; more of them will be girls, diurnal enuretics or children who will maintain the symptom into adulthood[2].

AETIOLOGY

Normal bladder control, which is acquired gradually, is affected by neuromuscular and cognitive maturation, socio-emotional factors, toilet training, and possibly genetic factors. Disturbances in any of these factors may lead to delay in acquiring urinary continence.

Enuresis is a symptom which can have a multi-factorial origin, the following have been implicated in the pathogenesis of enuresis:

Genetic causes

Family studies have shown an increase in prevalence of enuresis in siblings of enuretics than in controls. There is greater risk of primary enuresis in monozygotic twins than in dizygotic twins and the risk of enuresis have been found to be seven (7) times greater in a child with an enuretic father. This familial tendency suggests a strong genetic or constitutional component, environmental factors e.g. tolerance for enuresis in some families and psychosocial factors may also be involved[2].

Developmental causes

Studies have suggested that enuretics have a functionally small bladder although the anatomic capacity is normal when anaesthetized[2]. This has been further corroborated by the observation that enuretics empty their bladder more frequently during the day than others although they do not actually pass more urine[9].

Psychosocial Causes

Psychosocial stress has been implicated in both primary

secondary enuresis depending on the time of onset stressor. If psychosocial stress occurs during the period for learning bladder control (between 2 and 3 ½ years), primary enuresis will be the result but the child has gained initial bladder control it could precipitate the onset of secondary enuresis. Such stressors include: birth of a sibling, change of home, hospitalization, maternal separation, start of school, break up of family due to divorce or death. Nocturnal enuresis has been associated with both the severity of life stresses in the first four years of life, and the total number of stressful events[9].

Organic causes

These include: major anatomical faults such as ectopic ureter, spina bifida occulta diseases such as diabetes mellitus, diabetes insipidus and urinary tract infection. Urinary tract infections are common in childhood, and a striking feature of surveys on school populations is the high incidence of wetting problems in children with "asymptomatic" bacteriuria. All enuretic children should have their urine cultured to exclude infection; this is particularly important in those with diurnal enuresis[9].

MYTHS AND CULTURAL PRACTISES

In different communities and societies, enuresis has been alleged to be due to a myriad of causes many of which are peculiar to the community that is being studied. For instance in western Nigeria, infection is believed to be the main cause of enuresis, this infection is thought to be in the child's blood and peculiar to him if there might be other siblings cared for by the same parents who might have stopped bedwetting[8]. Therefore the child is seen to be at fault and the consequence is blame and societal discrimination. Other causes include excessive playing, excessive sleeping, overeating, eating late night and drinking too much water[3].

In most African communities enuretics are usually stigmatized, as the society thinks it was the mother's failure to adequately toilet train the child that caused the condition. The mother also joins the community in the stigmatization as she may think this curative. This misconception about the curative measures for bedwetting in some communities led to roasting of moth pupa to be eaten by the enuretic child while humiliating songs are sung for him, this is meant to stigmatize the child so that he stops bedwetting.

Another of these measures include standing astride a pan of hot coal to urinate over the heating system, this causes scalds, burns and eventually scars that make the child remember the punishment, this scares him and he stops bedwetting. Enuretics have been made to drink bitter concoctions that are thought to cure the disorder, kneeling down before a mortar and begging not to wet has been described by some communities to be effective.

There are no scientific proofs of the effectiveness of these measures. The fact that some children with enuresis will be cured as they grow older may erroneously lead some to believe in the effectiveness of these 'curative measures'. In addition, these 'curative measures' usually make the enuretic come down with feelings of shame, guilt and sadness which ultimately affect impairment of the child's self esteem.

ASSESSMENT OF CHILDREN WITH ENURESIS

According to the Diagnostic and Statistical Manual of mental disorders of the American Psychiatric Association (4th edition) enuresis is significant only if it has a frequency of twice week for at least three consecutive months or occurs with a clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning. The chronological or developmental age must be at least five and it must not be due to the direct physiological effect of a substance (e.g. a diuretic) or a general medical condition (e.g. seizure disorder)

The diagnosis of enuresis is mainly clinical, therefore a detailed history is important, and keeping in view the psychosocial factors that may predispose children to this disorder. Physical examination helps to exclude organic causes of enuresis e.g. spina bifida - ankle reflexes and gait abnormality may reveal the condition. The history and the examination of the child will also reassure the parent and relieve the anxiety.

The use of investigations is usually to rule out any organic predisposing factor of enuresis. This includes presence of urinary tract infection, or a structural obstruction within the urinary tract. The tests performed include urine culture and microscopy for white cells and bacteria, Dipstix test for glucose to exclude diabetes. A spine x-ray film is not indicated unless there are neurological signs or symptoms. Sophisticated radiological studies (e.g. intravenous urography) are not indicated unless there is a strong suggestion of a major anatomical fault.

ISSUES IN THE TREATMENT OF ENURESIS

Most of the patients will be healthy children with sterile sugar-free urine, who wet their beds at night. It is the treatment of this important group that will be considered in the following sections.

The Personnel

Enuresis is a cause of doctor's insecurity; because of the apparent inevitability with which many children wet their beds and the way in which, despite energetic management some of them will continue. But while prevention and 100%, cure may not be in a doctor's power he can certainly lessen the parental anxiety and the childhood fears.

The most important things for the doctor to give are concern, involvement, and time to listen and explain. It has been observed that the arrival of a new enthusiastic doctor cures a lot of enuretics. It has been suggested that enuresis be managed by the family doctor, for the large number of enuretic children all of who need the entire doctor's attention they can get. Only the refractory cases should get the child psychiatrist and paediatrician's attention. If there is a specific enuresis clinic, that should be the first choice because it would indicate someone's enthusiasm to solve the problem.[9]

Family Support

The mother's attitude is important, as excessive verbal, physical and other kinds punishment only serve to increase the anxiety at home. The approach should be praise and incentives for dry nights rather than blame and punishment for wet ones. The family should help to monitor and encourage progress; dry nights should go not by without adequate commendation.

The family will also be useful in encouraging the first line treatment which is appropriate toilet training. Record keeping is needed to have a baseline and following progress (not failures) may be a reinforcing factor.

Many at time, the doctor will also have to manage the parental anxiety and child's agony.

TREATMENT

Enuresis is usually self limiting and a child can eventually achieve continence without psychiatric sequelae.

Fluid restriction

Fluid restriction is an effective treatment for enuresis as it requires that the child eats dinner at least 2 hours before bedtime and also drink last at this time. Drastic water deprivation does no good as the child remains thirsty through the night and still wet by the morning. Thus, fluid restriction should be gradual and there should be avoidance of caffeine containing drinks like coffee and colas because of their diuretic action.

Motivation

This involves making the child understand that he has an important role to play in curing the problem even though it is not his fault. A chart or diary known as star charts, recording dry nights should be maintained, some praise or reward offered for them, most importantly for the nights the child stayed dry by waking and using the toilet. The child's esteem must be protected, allowing the child to understand the goal of the treatment and be reassured that he will eventually be dry.

Bladder Exercises

Delaying urine at home helps to increase the functional capacity of the bladder and the treatment of enuresis is based on the premise that children with enuresis have small functional bladder capacity. This is called Retention Control Training (RCT), it reduces wet nights by half in 50 percent of Children and some even achieve total dryness. Also, the practice of stopping the flow of urine midstream strengthens the external urethral sphincter and this has been found to be useful.

Waking Routines

This also known as the child lifting has been found to be effective in achieving dry nights in children with enuresis. It involves waking the child up in the night to use the toilet.

Conditioning Therapy

Classical conditioning with bell and pad apparatus is also an effective treatment for enuresis with more than 60% being cured and relapse can be managed successful with another course of alarm. This bell and pad apparatus is called enuresis alarm, which works on the principle that urine is a good conductor of electricity, it is difficult to use below age 5 and most effective beyond 7 years. Though it is often written that the child is being trained to awaken when he wants to micturate, this is not so, for more commonly after using the buzzer the child is conditioned to putting up with the sensation of a full bladder and sleeping on until the morning when he awakes and passes urine in the pot or lavatory. The difficulties may include: availability of alarms, improper use of the alarms, child and family compliance, and relapse. Giving incentives for dry nights is equally important and sometimes effective but is inferior to the alarm. This method is difficult and time consuming.

Psychotherapy

Although psychoanalytic theories regarding enuresis have been proposed, it has been found that psychotherapy alone is not an effective treatment. It may however be useful in dealing with coexisting psychiatric problems or the emotional and family difficulties that arise secondary to the disorder.

Pharmacotherapy

Drugs are not the first line in the management and may not be needed at all. There is a doubt of the efficacy of drugs used and relapse is a usual event with the use of drugs. The drugs having serious side effects are not used until the problem is so troubling that it interferes with child functioning.

The tricyclic antidepressants (imipramine, amitryptiline) are effective on a short term basis- up to 30% stay dry, 85% wet less frequently[2]. The adverse effects include cardiotoxicity, also sudden death in several children with attention deficit hyperactivity disorder on desipramine have been reported.

Desmopressin (DDAVP) an antidiuretic compound has shown initial success (10-90%) in reducing enuresis. Side effects include: headache, nasal congestion, epistaxis, stomach-ache and the most serious of all is hyponatraemic seizures. Primarily anticholinergics are rarely worth trying for nocturnal enuresis, stimulants like amphetamine produce more bad tempers than dryness.

CONCLUSION

The history of the cause, treatment and beliefs of enuresis must make one skeptical about any particular method of therapy. The skepticism should be tinged by neither cynicism nor despair; nocturnal enuresis is a challenge not a defeat. A lot can be done to help the thousands of children who wet their beds, and the doctor (nurse, pharmacist) who is sympathetic to their problems and has a clear plan of campaign which he carries out enthusiastically will achieve many successful cures.

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ADOLESCENT PREGNANCY: A REVIEW OF THE ASSOCIATED COMPLICATIONS.

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ABASTRACT

Adolescence represents a transitory phase in human development and it is characterised by many adventurous events including sexual experimentation. One of the aftermath of this behaviour may result in unplanned and on many occasion unwanted pregnancy.

Teenage pregnancy has remained a global social enigma with majorities of cases in developing countries including Nigeria.

This article chronicles the associated medical complications unsafe abortion, pregnancy induced hypertension, anaemia, malaria, HIV, preterm labour/delivery, low birth weight, obstructed labour, obstetric fistulae, higher maternal mortality, perinatal morbidity and mortality. Furthermore, various social implications such as physical abuse, stigmatization; emotional trauma and so on that result from conception by this group and the peculiarities of Nigeria as a country with one of the highest burden are discussed.

Holistic solutions with involvement of all parent, health care professional and government including private stakeholders are considered with the sole aim to coalesce effort at reducing the menace.

BACKGROUD

Adolescence represents a transition from childhood to adulthood with features including secondary sexual growth, changes in hormonal milieu, emotional, cognitive and psychological development[1]. This period is characterised with series of adventurous behaviours including sexual practices[2, 3]. Although, there are various definitions of adolescents, the widely accepted is those whose age is less than 19 years. They constitute about 20 percent of the world's population[4] with about 85 percent of them in developing countries. Female adolescents are sometimes further classified for health reason as either young adolescents (10 to 15years) or old adolescent (16 to 19years)[5]. Various researches have shown that both male and female adolescents engage in risky behaviours but, the later usually have worst outcomes[6].

In many communities, adolescent are generally viewed as an "adult child" that is on the verge of forming a career. Therefore, she is expected to engage purely on ventures to actualise her dream in life and any deviation including getting pregnant is seen as an aberration.

Pregnancy is one of the aftermaths of the adolescent behaviour. It usually occurs either following casual unprotected sexual intercourse or as part of her marital responsibilities. Those that engage in casual sex may be due to poverty, peer pressure effects and sometimes a consequent event of other anti-social engagements such as use of hard drugs, alcoholism and so on[2, 7-11]. Several Nigerian studies have shown that the majority of female adolescent first sexual cohorts are elderly[12] and are therefore not in a better position to negotiate for safer sexual practices or refuse outrightly. However, some communities encourage childhood marriage/childbearing and families' take pride in not allowing their daughter for various fears from commencing the second menstruation in her father's house. The adduced reason for this trado-religious norm was to prevent premarital sexual activity and the consequent shame to the family. On many occasions, these young girls are forcefully married out and sometimes threatened to comply. This practice is common in Nigeria especially in the Northern region.

EPIDEMIOLOGY

Globally, adolescent pregnancy is a well known societal enigma but, the incidence and acceptance differs between various countries and communities[13, 14]. Of the over 14 million adolescent pregnancies recorded worldwide per annum, about 12.8 million of these occur in developing countries[15]. Evidence from both community survey and hospital based data have revealed that sub-Saharan Africa share half of the total population of adolescent pregnancies with Nigeria harbouring the majority[16]. Currently, it is estimated that one in five teenage women is a mother and another 4 percent are pregnant with the first child[16].

The adolescent age specific fertility rate is about 54 births per 1000 women worldwide with Africa having the highest regional rate of 115 per 1000[17, 18]. According to the Nigerian National Demographic Health Survey of 2003, the age-specific fertility rate trend was 126 per 1000 women in the four preceding year of the survey[19]. In addition, there are regional variation with both the North -West and North -East having the highest figure and South -West, South -East and South -South having the lowest rates in that order[19]. At the same time, the rate was also found to be higher in the rural communities compared with the urban areas. The reasons advanced for this disparity is the level of education of women and urbanization. Lower level of modern education or none encourages adolescent pregnancy and childbearing and this is further flared up among those in rural communities. These factors have universal effect in both developing and developed countries. Infact, many western countries adopted school enrolment as one of

the key intervention to reduce the level of adolescent pregnancy.

THE ASSOCIATED COMPLICATIONS

Adolescent pregnancy is regarded as "high risk" because of the associated complications that may occur either during the pregnancy, childbirth and or in the postpartum period[8, 20, 21]. These complications are mostly health related to both the mother and her baby. Other consequences could affect the social and economic well being of the young mothers, their children and society at large[4].

The health related complications that are associated with adolescents include abortion, pregnancy induced hypertension, anaemia, malaria, HIV, preterm labour/delivery, low birth weight, obstructed labour, obstetric fistulae, higher maternal mortality, perinatal morbidity and mortality[22].

Unsafe Abortion

The first option that most adolescents tried to adopt is to terminate the unplanned pregnancy irrespective of the law governing their country. This attitude is usually pursued desperately as evidenced from all sorts of methods that have been reported to procure abortion[23]. Conservative estimate have shown that 2.2 4 million illegal abortions are performed per annum by pregnant adolescents with majorities causing permanent disabilities and mortality[23,24]. In Nigeria several reports both from the hospital[25, 26] and community based data have revealed that unsafe abortion are clandestinely performed by mostly adolescents resulting in annual mortality of about 20,000 maternal deaths in Nigeria[24].

Violence against Adolescent

While the adolescent is undergoing this dilemma of whether to keep the pregnancy or not, they sometimes suffer all forms of abuse from their sexual partner who may deny any prior relationship, parents and friends on many occasions compound her ordeal by further subjecting her to various forms of discrimination and physical abuse for her uncharitable and disgraceful act[14, 27]. She is thus subjected to this torture on daily basis.

Pregnancy Induced Hypertension

One of the key risk factors for developing pregnancy induced hypertension is nulliparity or first pregnancy experience from a particular sexual partner (New husband syndrome)[22]. Many reports have consistently shown over the years that pregnant adolescents constitute a significant proportion while there are few others that found no difference in the risk compared to the other age groups[28]. The question then arose whether the reported high prevalence of this medical condition amongst the adolescents is a chance occurrence or not? The plausible explanation is that the majority of adolescent pregnancies are unplanned and unwanted. Therefore, they do not seek care during pregnancy and this poor attitude result in progression to convulsion (eclampsia) with fatal outcome. This scenario is peculiar to most adolescents in sub-Saharan Africa[29]. The problem is further compounded by lack of efficient health care facility and necessary capacity to manage this challenging medical condition in many Nigerian rural communities. It is therefore not surprising that eclampsia is the commonest cause of maternal mortality in Northern Nigeria and second commonest in the south of the country[29].

Burden of Anaemia during Pregnancy

Although there is dearth of scientific evidence that specifically explored the relationship between the severity of anaemia and morbidity/mortality amongst pregnant adolescent women,[30, 31] there are incontrovertible evidences from many hospital based studies in Nigeria that anaemia is strongly associated with adolescent pregnancy compared to others[32-34]. This risk is greater because this young mother's body has to compete for nourishment with fetus, thus, causing a rapid depletion of the nutrient reserves. In addition, majority of the adolescent are not empowered to eat balanced diet that will boost their haematocrit level. In some communities, the cultural taboos of preventing pregnant women from eating food rich in protein could add to the problem[35].

When the anaemia is severe, there is the possibility of failure of the fetus to grow as expected (intrauterine growth restriction) and this may result in preterm labour with a small-for-gestational baby being born or a still birth[36-38]. Small amount of bleeding during childbirth may lead to dire consequences to the woman with likelihood of blood transfusion reactions or even death.

Burden of Malaria in Pregnancy

Malaria is another peculiar medical condition that has devastating effect on pregnant women in general. Several studies have identified first time pregnant women of which adolescent belong to be more vulnerable to malaria infection[39] but, there is limited research that compared the burden between adolescent primigravidae and other primigravidae[40, 41]. This is due to decreased level of immunity in pregnancy that is further worsened by the poor health status of many adolescents. The consequent effects of malaria include anaemia, IUGR, low birth weight, and sometimes intrauterine fetal death. Health interventions such as intermittent presumptive therapy of malaria, use of insecticide-treated bed nets and keeping a clean environment that is devoid of mosquito breeding remains the proven strategy to prevent malaria worldwide[42] but these practices are not widely available in most developing countries including Nigeria. Worse still, there is lack of targeted sustainable and pragmatic interventions aimed to reduce the malaria burden on at risk group in Nigeria. These deficiencies often leave pregnant adolescent women to suffer with damming consequences.

Preterm Delivery and Low Birth Weight Babies

Preterm birth (childbirth before 37 weeks of gestation) is major cause of perinatal and neonatal morbidity and mortality any where irrespective of the available health care facility. However, the greater part of the burden occurs in developing countries where there is lack of adequate facility and care to manage this challenging condition. Most times iatrogenic preterm delivery is usually due to an informed decision to terminate the pregnancy to either save the mother or the fetus life. On many occasions, adequate preparations are made to minimize morbidities and mortality. However, on the contrary the spontaneous preterm labour and delivery usually arise as an emergency and many times the clinicians are cut unaware with dire consequences. Several hospital and population based studies identified younger age of less than 20 years as an independent predictor of spontaneous preterm birth[43-45]. Some researchers have further established that the gynaecological age (chronological age minus age at

arche) rather than chronological age predicts the risk eterm labour and delivery[45]. Findings from the study ving over 1700 young women showed that ecological age of less than 2years strongly predicts elihood of preterm birth[45]. The study concluded he younger the age of the mother the higher the Other scientific evidence that corroborated strong ation between adolescent pregnancy and risk of m birth include the ethnicity being a black escent, evidence of socioeconomic deprivation, lack rmal education and those in rural communities ally in developing countries[46,47].

th weight (birth weight that is less than 2500g) and for gestational age (infant below 10th percentile of eight for the gestational age) infants incidence has eported to be higher amongst pregnant escent women compared to others[9]. An inverse ship therefore exist between the likelihood of and maternal age at delivery but, the evidences for of SGA is not consistent because of confounders aemia, medical illness and so on[30,36,47].

of Cephalopelvic Disproportion

lescent women are faced with possibility of elvic disproportion in labour resulting in cal obstruction due to her immature pelvic of the birth canal[48]. Harrison et al as far as 1985 has to the whole world that very young girls are still [49,50]. He and colleagues demonstrated increase in pregnancy and also a higher incidence of ted pelvis in young (<16) and short (<1.50m) nts[49]. He then advocated for routine clinical y at 36 weeks gestation and those with evidence ted pelvis should be offered elective caesarean The peculiar delay of attaining puberty amongst nts in developing countries as well as poor l support are other reasons advanced to this developmental delay as compared to nts in developed countries[51-53].

Skilled Attendants at Delivery

pregnant Nigerian women are delivered by an birth attendant for reasons such as poverty, beliefs, religious sentiments and host of others[54,55]. study conducted in over by Galadanci and her s in Northern Nigerian states revealed that 5 percent of women delivered at home with portion delivering with those with no verifiable in sanitary birthing techniques[56]. Another dimension is that the pregnant adolescent is to customarily deliver her first child at home for cultural reasons[57]. All these practices expose hanical obstruction during labour with myriads cations such as ruptured uterus, sepsis, haemorrhage and obstetric vesico-vaginal ginal fistula.

loping Obstetric Fistula

epidemiological researches have described nts as young, immature, malnourished and individuals that are victim of sociocultural Available evidence have shown that many are before attaining menarche (age of first) and are therefore described as "child The prevalence of this socially distressing condition has been described as a measure of the quality of maternal health services in any environment[60]. In Nigeria, more than half a million are suffering from obstetric fistula with about annual rate of about 40,000 new cases per annum[61]. The principles of care are restoration of urinary and faecal incontinence, early integration to the family and community as well provision of empowerment skills[62].

Sadly, there are no measurable fistula surgeons to cope with the large available cases in Nigeria. It has been postulated that it will take a century to clear the backlog of cases with the current repair rate of about 2500 cases per annum[63].

Accentuated Risk of Maternal Death

Adolescent pregnant women tend to have a higher maternal mortality rate than older women[64,65]. The risk of dying from pregnancy related causes is twice as high for women aged 15 19 as for women in their early twenties and worst ill, the risk increases to five times amongst those aged 10 14years[66]. The link identified to this risk includes lower socioeconomic status and educational level of adolescents[64]. In addition, lack of antenatal care attendance and obstetric care services may further compound their problem. Adolescents leaving in rural communities are also reported to be 4 6 times at risk of dying in pregnancy or during childbirth compared to their peers in urban areas. Available data from Nigeria that understudy this risk is mainly hospital based but, they were consistent in identifying adolescent as an independent predictor of maternal death[67]. The main causes of these deaths were preeclampsia/eclampsia, sepsis, prolonged obstructed labour, unsafe abortion and anaemia[67].

The Burden of Adolescent Parenthood

Even when a safe delivery is achieved by these adolescents, they are faced with challenges of responsibilities of mothering. In many situations, they are not emotionally and psychologically prepared to fulfil these assignments[68]. Breastfeeding is regarded as the optimal nutrition of the newborn but, many a time babies born suffer severe malnutrition due to lack of developed breast or milk production by these adolescent mothers. One obvious fact is that there is lack of research that specifically examined adolescent mothers' attitudes towards breastfeeding their child and associated health implications.

Other responsibility of ensuring childhood immunizations is deficient among these young mothers thus predisposing them to lots of vaccine preventable diseases.

Apart from challenges of mothering, adolescent fathers are faced with coping with the new and ill-prepared role to his immediate family[69]. There are reports that adolescent fathers are psychologically unstable and stigmatised in the society[70,71]. On many occasions, young fathers are more likely to have economic and employment hardship, reduced educational attainment, anti-social behaviours and possibility of living in neighbourhoods characterised by poverty[72,73]. In addition, many studies, have demonstrated a correlation between teenage fatherhood and childhood delinquency[72-74]. All these constitute a huge burden for adolescents to assume the much expected and desired parental role.

Socio-economic Consequences

The social and economic consequences of adolescent pregnancy and childbearing depend on their particular cultural, family and community settings[68,75,76]. Pregnancy can bring status for married adolescents in cultures where motherhood is a core aspect of woman's identity[77]. On the other hand, unmarried adolescents might be considered an embarrassment for the clan and is either abandoned or chased away from home, and she is therefore left with no guaranteed means of support for the child and herself[74,76].

The adolescent pregnancy has been associated with poor educational achievement, poverty and other related factors[78]. Often, pregnant girls are forced to leave school and abandon career aspirations because of the embarrassment and physical demands of pregnancy and childbirth. All these result in a vicious cycle of a repeat occurrence in those children reared by these adolescents because of inadequate parental guidance and modelling.

SOLUTIONS

The problems associated adolescent pregnancy and childbearing are numerous to exhaust but alas they are surmountable! However, this require dedication of all and putting in place sustainable policies and programmes that will not condemn but complement values that will proactively discourage adolescent pregnancy in our land.

The solution is to prevent pregnancy and for those already pregnant, genuine measures should be put in place to offer good and specific care to them as well as counsel these adolescents on the need to continue pursuing their chosen career after the nursing period[79-81].

Advocacy

Awareness creation to all communities, parents, teachers, civil societies, faith based organizations, community leaders, government all level and the youth themselves through advocacy visit, mobilization activities, provision of jingles and playlets that discourages adolescent pregnancy and also spell out associated dangers[82, 83]. Collaboration with media experts to disseminate information through their outfit will be of tremendous help[84]. This is an opportunity that has not been fully utilized. Above all counselling session should be made freely available and confidential for any young girl that wants to seek information that bothers on her reproductive health. Medical professionals especially obstetrician and gynaecologists should create enabling environment that will encourage these young helpless girls from accessing the correct information within their health care facility[85,86].

Prevention of Pregnancy/Contraception

The prevention of pregnancy among adolescent remains the cornerstone. This involves abstinence and use of barrier contraception for those sexually active[87,88]. Promotion of abstinence via sexuality education or otherwise known family life education (FLE) programme should be vigorously pursued[89] and given the priority it deserves within Nigerian communities. The essence of this concept is to expose a girl child early in life preferably before puberty to know about her body's physiological mechanism[83]. Establishment of youth clubs where moral values and other diversionary activities should be encouraged to minimise the tendency to engage in risky premarital sexual activity[90, 91]. At the same time, they should be thought on various contraceptive methods that could be employed to prevent unwanted pregnancy with emphasises on dual protective strategies and emergency contraceptive techniques[92]. This is to ensure safer sexual practices and prevent the attendant complications.

Currently in Nigeria the champions of this concept are mainly international donor agencies and some non-governmental organization such as Association for Reproductive Health in Nigeria based in Ibadan and Women Health Action Research Centre (WHARC) based in Benin-city. The worrisome aspect now is the growing opposition to this programme by many political, religious and even some policy makers that negatively viewed it as an avenue to corrupt the minds of the so called innocent young girls. Paradoxically, all the available studies from Nigeria have consistently shown that adolescents are sexually active and that there has being decreasing age of sexual initiation with the majority not using any form of protective methods during their first attempt[93]. It therefore behoves us as a nation that we objectively face this reality of adolescent pregnancy by embracing the FLE programmes in our schools and also encourage government at all level to proactively support this initiative.

Liberalization of Abortion Laws

Liberalization of abortion laws has remained a controversial issue in many countries[23, 94] including Nigeria but, there remain questions that are yet to be answered. Who and where do adolescents procure abortion despite the restriction? Who are those that should identify these abortionists and make them face the wrath of the law? Whose right should be respected, is it those against abortion law or those for it? Are people dying in Nigeria on daily basis due to unsafe abortion? Is Nigeria situation currently better that other country where abortion is liberalised? These questions should be answered by all with utmost sincerity that is based on fact but not sentiment. The rejection of reproductive health bill submitted by the Society of Gynaecology and Obstetrics of Nigeria (SOGON) to the national assembly was due to these unanswered questions.

Education of the Girl Child

School enrolment for the adolescent has been reported to reduce the level of adolescent pregnancy in many western countries[95, 96]. It stands to reason that education will provide opportunity for better exposure including family life education, delays the age at marriage and promote empowerment later in life. In Nigeria education of the girl child is not being given the deserved priority as it is being opposed by many community values. Many parents viewed educational investment of the girl child as a waste and rather prefer to encourage their sons. This negative impression needs to be corrected in other to reduce adolescent pregnancy in Nigeria. UNICEF is currently funding a project in many Northern states on the need to educate the girl child. It is therefore desirable that the state government should fulfil their own side of the bargain for the programme to be sustainable. It is plausible to mention that the Nigerian government has established universal basic education but the effect is yet to be felt in many rural areas. Therefore many still regard it as a mere political agenda.

...arriage is another issue that needs to be ...because reports have shown that a significant ... of Nigerian adolescent are in one form of ...ship by the time they are pregnant. The ...s have stated times without number that ...arriage is violation of human right as the ...ese young girls regarded as "minor" are not ...e the marriage. It therefore ruled that the ...e for marriage should be 18 years[97]. The ...ment of Nigeria in 2003 followed this ...ssing a Child right Act in to law putting the ...of marriage to 18 years but, presently many ...to implement the law judging by the high ...arriage in many communities especially in ...Perhaps, communities that demonstrate an ...eduction in childhood marriage should be ...incentive to encourage others. The ...n of this law is crucial as it will prevent all ...tioned complications of pregnant young

...ffordable Reproductive Health Services for

...s have suggested a specialised antenatal ...for the pregnant adolescent where specific ...tions and other expected responsibilities ...with the purpose to minimise complications. ...ence thus far does not conclusively reflect ...ering this so called Teenage Pregnancy Unit. ...s group of expectant mothers could be ...our routine antenatal clinic through their ...ds and offered special health education ...ses on circumstances relating to her ...her intention after delivery, counselling on ...methods and the need to continue on her ...after delivery. This arrangement could be ...into our service rather than creating ...el clinic. This suggestion will need to be ...cientific research in Nigeria.

...ological and emotional support to these ...ung mothers both during pregnancy and ...d be beneficial and this laudable practice ...essed in Nigeria where family ties are ...upheld.

...adolescent pregnancy in Nigeria is public ...that has not received the desired priority. ...is crucial as management of the associated ...constitutes a huge avoidable burden on ...nomic system. Adequate mobilization of ...commitment of all shall provide hope for ...dication of this problem from our land.

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MANAGEMENT OF HYDROCEPHALUS IN THE DEVELOPING WORLD

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ABSTRACT

Hydrocephalus is a disorder of the Central Nervous System (CNS) that has a tremendous impact on its further development and the physical appearance of its victims especially the children. The challenge of accurate diagnosis with radiologic imaging has been largely solved in the developed world with the availability of CT scan and MRI but their routine use is limited by cost in the developing world. The treatment options for this condition vary from shunts to the rejuvenated endoscopic procedures like Endoscopic Third Ventriculostomy. Although the problem is age-long and the advances progressive, the developing world has persistently lagged behind in enjoying the benefits of the progress made so far due to the challenges of poverty, lack of political will and sufficient manpower. How then have we been coping with the management of this condition despite the surrounding limitations? This is the thrust of this paper.

"Hardly any other pathological condition has been accorded more determined attention on the part of the medical profession... than has hydrocephalus. And in hardly a single other conditions have cures been so elusive or so often wrecked on purely mechanical obstacles"

LEO-DAVIDOFF, 1929

Effective shunts came into existence twenty five years after this statement, resulting in significant symptomatic recovery and improvement in development and functional ability. The phrase 'so often wrecked on purely mechanical obstacles' still remains true today.

The management of hydrocephalus has undergone tremendous changes with a variety of options which nevertheless are encompassed with a number of limitations and complications. The advances are progressive but the developing world is still many decades behind because of the peculiarities which have poverty and illiteracy as the common denominator. The pathologies are the same but the available treatment modalities are limited and unevenly distributed in the developing countries such as ours in Nigeria.

Hydrocephalus results from a disturbance of the formation, flow or absorption of the cerebrospinal fluid (CSF), leading to an increase in the volume it occupies in the Central Nervous System (CNS). This eventually leads either to an acute or chronic increase in the intracranial pressure. It can be classified into congenital and acquired hydrocephalus but within each group exists a further classification into obstructive (non-communicating) and communicating (non-obstructive) hydrocephalus which relates to the lack of communication or the presence of it with the subarachnoid space.

- **Congenital hydrocephalus**
  - Primary Aqueductal stenosis
  - Secondary Aqueductal gliosis Due to intrauterine infection or germinal matrix hemorrhage.
  - Dandy walker malformations
  - Chiari malformations type I & II

- **Acquired hydrocephalus**
  - **Post infectious:** Most common cause of communicating hydrocephalus with a high incidence of this entity in the developing countries.
  - **Post hemorrhagic:** following subarachnoid or intra-ventricular hemorrhage
  - Secondary to intracranial masses
  - Following posterior fossa tumor surgery

DIAGNOSIS

The clinical symptoms and signs reflect increased intracranial pressure either in its acute or chronic form and may include irritability, vomiting, drowsiness, lethargy, poor neck control, bulging and widened anterior fontanelle, enlarged and engorged scalp veins, paralysis of upward gaze (sun setting sign), abducens nerve palsy, hyperactive reflexes, papilloedema and gait abnormality in the older child.

Diagnostic modalities are important for early diagnosis of hydrocephalus, permitting treatment to be instituted and ultimately for optimum neurologic function and good cosmetic result. The diagnostic modalities include:

1 **Ultrasound scan:**

This is the most readily available and essential investigative tool in the developing world despite its abuse and under-utilization. It can be employed in the following ways:

- **Abdomino-pelvic ultrasound scan of the mother:** this is done at various points during gestation and allows for prenatal diagnosis of hydrocephalus and allows for possible intra-uterine intervention not available in the developing countries which despite some recorded success remain a controversial issue even in the developed world. It however helps in planning the delivery of the baby. The inherent habit of home delivery and the role of the traditional birth attendants have limited the role of this invaluable diagnostic instrument.
- **Transfortanelle Ultrasound Scan:** this forms an important diagnostic tool in the developing world as it can readily assess ventricular size, be used to stratify patients for treatment: shunting/ETV and also forms an important tool in the follow up of patients.

2. **Cranial computerized Tomography scan:** This gives a better definition to the anatomy of the brain and the CSF pathways. Cost and availability still limits its effective use in the diagnosis of hydrocephalus though there's remarkable increase in the utilization of this modality in developing countries.
3. **Magnetic resonance imaging:** this is the hallmark of diagnosis with detailed anatomical description in three planes; axial, coronal and sagital. It is also able to demonstrate membranes, CSF flow voids as well as the surrounding vasculature. MRI machines are scantily few because they are capital intensive.

TREATMENT

The mainstay of treatment is surgical. There are however temporizing measures that can be instituted and may also serve as an adjunct to therapy. These include:

Diuretics

- carbonic anhydrase inhibitors (Acetazoloamide: 25-100mg/kg/day)
- loop diuretics: frusemide 1mg/ug/day

These reduce the rate of secretion of CSF from the choroid plexus.

Attention must however be paid to the electrolyte status of the patient as these can cause metabolic consequences. Mannitol is also used in acutely raised intracranial pressure in hydrocephalus.

SURGICAL INTERVENTIONS

EXTERNAL VENTRICULAR DRAINAGE (EVD): CSF is drained via a ventricular catheter connected to an external drainage bag. (A temporary solution in particular for patients with on going infections) If infection is absent, the main risk is contamination which can then delay treatment.

SERIAL VENTRICULAR TAPS: this is also a temporary measure to treat raised intracranial pressure.

SHUNTS: this is the most common method of treatment worldwide in which the ventricles are connected to other body cavities such as the peritoneum, atrium, pleural etc. The ventriculo-peritoneal shunt is particularly preferred in children as it enables the implantation of an important length of drainage catheter to allow for growth, and it predisposes to less severe infectious complications than the ventriculo-atrial or ventriculo-pleural (V-P) shunts.

The shunt comprises a proximal am distal catheter with a valve and reservoir inserted between the two. The available shunts in the developing countries are differential pressure valves i.e. they are pressure regulators which maintain a fixed pressure differential regardless of flow in contrast to flow regulators which maintain a contant flow irrespective of the pressure. A low, medium or high pressure alternative are available. The cost of these shunts partly account for the delay in the institution of treatment though there are inexpensive shunts manufactured in India (Chhabras). It is however admissible that these shunts have not been fund to perform less than the expensive western shunts. Adeloye also treated patients in Malawi with locally made shunts with results comparable to conventional shunts.

Although shunting is the standard method of treating hydrocephalus, it is fraught with complications which include infection, under or over drainage, mechanical failure etc. The North American Shunt Design Trial failure rate was more than 40% within 3 years of the shunt placement. The saying 'once a shunt always a shunt' still remains true today.

CHOROID PLEXUS ABLATION

This can be open or endoscopic but the former has essentially been discarded due to the mortality associated with it. Endoscopic coagulation of the choroid plexus does reduce CSF production but not

completely and may not provide an effective treatment solely. This is due to the existence of other sources of CSF such as ventricular ependymal lining and dural sleeves of spinal nerve roots. It can be combined with endoscopic third ventriculostomy.

ENDOSCOPIC THIRD VENTRICULOSTOMY

This has become the most common neuro-endoscopic procedure. Initially abandoned, after the first procedure by William J Mixter in February 6, 1923. It is an internal CSF diversionary procedure which involves creating an opening (a stoma) in the floor of the third ventricle, thus connecting the ventricle to the prepontine/interpenducular cisterns. It serves to drain the ventricles under physiologic control, re-establishing a uniform hydrostatic pressure regimen in the whole central nervous system and preventing the development of a pressure gradient on the vulnerable midline structures.

ETV reduces the risk of infection to a marginal level eliminating most of the complications of shunts and particularly conferring on the patient a shunt independent life. The success rate of this procedure has been quoted to be between 59 90% and is not limited to obstructive hydrocephalus secondary to aqueductal stenosis. Ben Warf in his work has quoted an almost 90% success rate in post infective hydrocephalus in Uganda. The selection criteria however remain unclear.

One must be aware that the decrease in ventricular size after an efficient third ventriculostomy is less significant than that after shunting. Evidence of flow through the ventriculostomy illustrated by phase contrast MRI and normalization of clinical symptoms are more important in evaluating the benefits of treatment than is the decrease in ventricle size.

There is an increasing preference for this procedure wherever it is available, as also evident on the service of the neurological surgery, University College Hospital, Ibadan, where almost all mothers choose it ahead of V-P shunt. Limitations in the developing world with respect to ETV will include the learning curve, limited resources for installation of equipment, maintenance, cost etc.

CONCLUSION

The challenge to the optimum management of hydrocephalus, in developing countries, still remains enormous. Efforts to salvage the situation are noted but there must be political will by the Governments in those countries in terms of education, manpower development, and provision of infrastructure to reduce the morbidity of this disease.

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CHILD ORAL HEALTH CARE – ROLE OF MOTHERS

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Summary

Mothers, as decision makers and best role models within the family, play vital roles in relation to health matters. Good oral health is important for child's growth, development and self esteem, and if started early will increase the opportunity for a healthy mouth as the child grows through childhood to adolescence and to adulthood.

Maintenance of good health and prevention of dental caries depend mainly on mothers as this lesion if left untreated has been known to lead to poor nutritional practices in children.

This paper discusses the role of mothers in preventing caries development.

Key words: Health care, Child, Dental caries, Mothers.

Introduction

Infant oral health is the foundation upon which preventive education and dental care must be built to enhance the opportunity for a life time free from preventable oral diseases.[1] Allied health professionals and parents must be involved as partners to achieve this goal. Parental influence on children oral habit is important if children must develop good habits. Mother's role in relation to health is very crucial as they make decisions within the family about life styles, food and health. Therefore preventive measures targeted at children should rightly be directed at mothers.

This article provides an overview of dental caries in children and the role of mothers as oral health care providers in its prevention.

Dental Caries

Dental caries also known as tooth decay is the commonest oral disease in children and also the main cause of tooth loss in children accounting for 64.4% of the causes of tooth loss in children in a previous clinic based Nigerian study.[2] The Centre for Disease Control and Prevention in America has reported that caries is the most prevalent of infectious diseases in their nation's children and more than 40% of children have caries by the time they reach kindergarten.[3] Dental caries a communicable disease, which causes destruction of teeth by acid – forming bacteria found in dental plaque.

Early childhood caries (ECC) can be a particularly virulent form of caries beginning soon after tooth eruption, developing on smooth surfaces, progressing rapidly and having a lasting detrimental impact on the dentition.[4,5]

Dental caries is a dynamic lesion with periods of demineralization alternating with periods of remineralization. Its development is dependent upon the interaction of a number of related factors which include host (tooth), bacteria in plaque (*Strepcoccus mutans*), dietary carbohydrate (sugars) and time.[4] These inter play with a variety of social, cultural and behavioural factors. Bacteria (*Streptococcus mutans*) in plaque ferment sugar in the dietary carbohydrate with a resultant acid formation on the tooth. Over time, in the absence of remineralization factors the acid causes dissolution of enamel. .The vertical colonization of *S mutans* from mother to infant has been well documented and the micro organism does not appear in the mouth of infants until later stages of primary tooth eruption.[6,7]

Dental caries in children is typically first observed clinically as a white spot lesion. If the tooth surface remains intact and non cavitated, then remineralization of the enamel is possible. If the subsurface demineralization of enamel is extensive, it eventually causes the collapse of the overlying tooth surface, resulting in a cavity

A severe form of ECC known as Rampant Caries has been documented.[8] It occurs when a child usually under 3 years of age is exposed to sugary feeds -liquids or solid for a continuous extended period of time. The practice of putting a baby to sleep with a bottle filled with milk or a child over 2years of age waking up in the night to feed many times are the major causes of this most difficult and challenging dental condition confronting the Pediatric dentist.[9] Salivary flow is diminished during sleep and since these sugar containing feeds cannot be eliminated from the oral cavity during this time, it pools around the tooth surfaces and cariogenic micro organisms which are opportunistic, exploit this conducive environment resulting in tooth decay. The longer the practice continues, the greater the damage to the primary dentition.

Dental caries in children especially in the under 3 years is better prevented than managed. If left untreated, dental

caries causes a lot of pain and discomfort to the child. A common complication of untreated caries is development of acute dentoalveolar abscess, a condition characterised by severe pain, swollen and tender jaw and inability of the child to feed. Caries in primary teeth can affect children's growth, result in significant pain and potentially life-threatening infection, and diminish overall quality of life.[8]

Preventive strategies and recommendations

Caries is a disease that is preventable. Due to the time of inoculation, steps to prevent caries ideally begins prenatally and continue with the mother and young child, beginning when the infant is approximately 6 months of age with the eruption of the first tooth.[10]

Since vertical transmission of *Streptococcus mutans* from mother to child has been established, prevention of caries prenatally in the mother begins with modification of the mother's oral hygiene, diet and the use of topical fluoride as these can have a significant impact on the child's caries rate.[11,12]

General anticipatory guidance for the mother includes oral health counselling during pregnancy which should include the following

Oral hygiene: Tooth brushing and flossing on a daily basis are important for the parent to dislodge and reduce bacterial plaque levels

Diet: Important components of dietary education for the parents include the caries potential of their diet, cariogenicity of certain foods and beverages, role of frequency of consumption of these substances and demineralization and remineralization process

Fluoride: Using a fluoridated tooth paste and where possible rinsing every night with an alcohol- free mouth rinse containing 0.05% sodium fluoride to help reduce plaque levels and help enamel remineralization

Caries removal: This involves routine professional dental care for the mother as this can help keep their oral health in optimal condition. Removal of active caries and subsequent restoration are important to minimize infecting the infant with the mother's oral flora

Delay of colonization: Education of mothers on sharing utensils (eg. Shared spoons, cleaning a dropped pacifier with their saliva),foods, and cups can help prevent early colonization of oral flora in their infants

Xylitol chewing gum: Recent evidence suggests that the use of xylitol chewing gum by mothers had a significant impact on decreasing the child's caries rate.[10]

General anticipatory guidance for children up to age 3 years includes the following

Diet: Restricting bottle/breast feeding to normal meal times and not allowing the infant to feed while sleeping after the eruption of the first primary teeth will prevent the development of ECC. The parents understanding of the cariogenicity of certain foods can help the infant and child eliminate or reduce their caries level

Oral hygiene: Cleansing the infant's teeth as soon as they erupt with either a washcloth or a soft tooth brush will help reduce bacterial colonization. The use of a chewing stick and or foam should be discouraged in children. The use of a tooth brush must have been established as soon as the child has multiple teeth erupted in the mouth. Tooth brushing must be done for children below 6 years by their mothers with a fluoridated tooth paste which should be used sparingly as infants and very young children have limited ability to expectorate. Pea size tooth paste on the brush is however recommended for all.[13] Fluorides are known to increase the resistance of teeth to decay and to acquire this maximum benefit, children are expected to spit out the paste after brushing but should avoid rinsing out with water.[14]

Establishment of a dental home: this involves making the child's first dental visit by his first birthday for a thorough oral examination and assessing the infants risk of developing dental disease especially dental caries

In conclusion, the main aim of every one responsible for the care of a child should be to promote his best physical and mental health. A mother should be alert to recognise, prevent or mitigate any condition that might mar or handicap the full potential, growth and development of the childs' body, mind and personality.

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TRANSFUSION REQUIREMEN IN SICKLE CELL DISEA

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ABSTRACT

Sickle Cell Disease has remained a scourge of the black race as individuals of Negroid descent are predominantly a Symptomatic treatment is the mainstay of management of this condition. Until recently, it was believed that routine tra of patients with sickle cell disease was necessary. However, the risks, ranging from iron overload to transmission of blood infections, far outweigh the benefits. Hence, there are now clear indications for transfusion in patients with sickle cell This review highlights the indications for transfusion in individuals with sickle cell disease.

Key words: Sickle cell disease, hemoglobin SS disease, anaemia, transfusion.

INTRODUCTION

Sickle cell disease is a common cause of morbidity and mortality among individuals of African ancestry. This is because of its high frequency amongst people of Negroid descent. Sickle cell anaemia is also prevalent in parts of Asia and the Indian sub-continent. It is also seen in parts of Europe and in areas where people from the above regions have migrated. However, the S gene exists in all the three major races of mankind black, white and oriental.[1] The term refers to a heterogeneous group of disorders caused by inheritance of a mutated â-globin gene which result in the replacement of glutamic acid by valine at position 6 of the â-globin chain. This abnormal gene may be inherited in a homozygous fashion or in a heterozygous combination with normal or other abnormal â-globins genes. The resulting haemoglobin (gene product) is less soluble and precipitation occurs in conditions of low oxygen tension. This results in sickling of the erythrocytes with resultant reduced red cell survival, micro vascular occlusion and ischaemic end organ damage.

Homozygous sickle cell disease individuals are those who usually exhibit the classical signs and symptoms of the disease. The different clinical entities in the spectrum of these disorders show varying degrees of severity. The most significant problem is anaemia which is multifactorial. However, the anaemia is well tolerated with some patients having haemoglobin concentrations as low as 6 g/dl. Most of these patients are stable with this chronic anaemia and may not require transfusion. However, there are several clinical situations in which the therapeutic use of transfusion is clearly defined.

Transfusion of red cells is life-saving and is a major component of the medical management of SCD. Transfusion therapy is used to prevent complications and also treat severe clinical conditions.

TRANSFUSION THERAPY IN SICKLE CELL DISEASE

The modern use of transfusion in clinical therapy has evolved from a simple procedure to a highly sophisticated and rapidly developing sub-discipline. It has come to occupy a crucial role not only in modern clinical me large but particularly in the management of sic disease. When used correctly, transfusion can be life and may slow down the progression of organ However when used inappropriately, transfusion more harm than good. Physicians and other profe involved in the care of sickle cell disease patients knowledgeable of the specific indicatio complications of transfusion therapy, types o products available as well as strategies to minimize of adverse events.

The indications for transfusion may be e (triggered by an acute need or medical interve chronic (usually for the long term suppression of sickle cells)[2]. Several methods of transfusion are and these include simple transfusion, partial e transfusion and erythro-cytapheresis. The meth depends on the specific indications. Broadly spea objectives of transfusion in sickle cell disease are firstly to correct the low oxygen carrying capacity severe anaemia and secondly, to improve micro perfusion by decreasing the proportion of sickle ce circulation

INDICATIONS FOR TRANSFUSION

1. EPISODIC INDICATIONS

Episodic transfusions are usually prompted b complications of the disease or a ne medical/surgical intervention.

Management of severe anaemia:

There are several situations in sickle cell patients w haematocrit may drop significantly below the base severe consequences. In these patients, simple tra should be used without taking any blood from the p

No universal parameters exist for transfusion of with sickle cell disease and management ha individualized. Some useful guidelines incl

obin values **less than 5g/dl**, (b) a twenty percent fall below patient's baseline, or (c) a drop in obin of **greater than 2g/dl** from the steady state.[5]

patients should be transfused if there is evidence siologic derangement such as heart failure, hypotension or marked fatigue.

cell disease, acute severe anaemia may commonly the following:

ic sequestration: When severe, this may result in emia and cardiovascular decompensation in to the anaemia. Patients require immediate on to prevent cardiovascular collapse.[8]

ion crisis, is however often less severe and initial transfusion should be avoided.

red cell aplasia: This is caused by Parvovirus B19 anaemia develops over a few days secondary to red cell survival without compensatory of new red cells.

anagement of sudden severe illness

syndrome, stroke, septicaemia and acute multi- are leading causes of death in sickle cell falling haematocrit usually accompanies these transfusion to improve tissue oxygenation and is indicated. When haemoglobin is low, many be treated with a simple red cell transfusion. In ases exchange transfusion/red cell pheresis is ded.

reparation for General Anaesthesia

ave compared perioperative complications atients with sickle cell disease undergoing major g. cholecystectomy).[9] It is recommended that all disease patients be transfused in advance to their anaemia to a haemoglobin concentration of ately 10g/dl and percentage Hb S to ately 60%. Although no standard guidelines have eloped with haemoglobin SC disease, the accepted practice is not to use preoperative in otherwise healthy/stable patients.[9]

ATIONS FOR CHRONIC TRANSFUSION

ransfusion therapy is indicated in several in which potential medical complications the risk of allo-immunization, infection and iron Transfusions are usually repeated at 3-4 week and the major goal of these programs is to the levels of haemoglobin S at 30-50% depending ific disorder. Simple transfusion can be used but ers recommend red cell pheresis / exchange s to reduce the risk of iron overload. The are the different clinical scenarios in which sfusion offers some benefit in sickle cell disease ment[3].

rimary stroke prevention

first application of chronic transfusion therapy is for potential complications of sickle cell disease. It is particularly useful in children with a high blood flow rate through the circle of Willis as measured by transcranial Doppler ultrasound[4].

- **Prevention of recurrence of stroke**

It is well documented that children with sickle cell anaemia who have suffered a vaso-occulusive stroke achieve a reduction in the recurrent stroke rate from 46-90% to well below 10% with chronic transfusion therapy [6]. This also minimizes the risk of progressive neurologic deterioration in children who have had a stroke. The current recommendation is to maintain the level of haemoglobin S at 30% or less for approximately 5 years[5]. There are currently studies underway in which the haemoglobin S is allowed to rise to 50% in patients with stable neurologic disease.

- **Pulmonary hypertensive and chronic lung disease**

Chronic transfusion therapy is well documented to be beneficial to patients with confirmed pulmonary hypertension and chronic lung disease. It has also been used to minimize sequelae in patients experiencing severe acute chest syndrome.

- **Vital organ failure**

Other vital organs such as the heart and the kidneys are progressively damaged in the natural course of sickle cell disease. The reduction of the circulating sickled erythrocytes is of tremendous benefit in slowing down the rate of vital organ damage and consequent failure.

- **Chronic debilitating pain**

Some sickle cell disease patients suffer unusually protracted and severe pain episodes. Although chronic transfusion may be used to alleviate this, its true value lies in being part of a multidisciplinary pain program rather than a 'stand-alone' modality.

3. OTHER INDICATIONS

These are conditions in which the efficacy of transfusion is unproven but may be considered under severe circumstances. They include the following:

- **Management of acute priapism**

Red cell transfusions in particular, exchange transfusions are indicated[10]. Patients are transfused as for a stroke protocol.

- **Preparation for infusion of contrast media**

Risk is due to red cell sickling in hypertonic contrast media. The use of new technology and non-ionic contrast media substantially lowers the risk associated with these studies.

- **Pregnancy**

Previously routine transfusion therapy was recommended for sickle cell patients due to poor pregnancy outcomes. The current recommendation is to confine transfusion therapy to women who suffer frequent complications during pregnancy.[5]

- **Management of silent cerebral infarct**

Exchange transfusion can be carried out when cerebral

infarct is detected by MRI.

- **Leg Ulcers**

Transfusion should be considered in recalcitrant or recurrent skin ulcers if conservative therapy fails.

4. NON-INDICATIONS

The following are considered to be inappropriate reasons for transfusions in sickle cell patients.

- Chronic (steady state) anaemia
- Uncomplicated acute painful crises
- Infection
- Minor surgery not requiring general anaesthesia
- Aseptic necrosis of the hip or shoulder
- Uncomplicated pregnancy

TYPES OF BLOOD PRODUCTS

Standard bank blood is appropriate for the sickle cell disease patient with the following provisions:

i The antigenic phenotype of the red cells should be determined in all patients with sickle cell disease older then 6 months of age. Ideally ABO, Rh, Kell, Duffy, Kidd, Lewis, Lutheran, P and MNS status should be determined and a permanent record maintained in the blood bank while a copy should be given to the patient or his/her family.

ii All blood used for sickle cell patients should be screened and confirmed to be haemogblobin S- negative.

iii Pre-storage leuco-depletion of red cells should be standard practice to reduce the incidence of febrile reaction, platelet refractoriness, infections and cytokine-induced complications. Washed red cells are indicated where there is a positive history of allergic reactions post transfusion while irradiated blood should be considered in potential candidates for bone marrow transplantation

iv. Autologous transfusion should be avoided in patients with sickle cell disease.

v. All patients with a prior history of transfusion should be screened for the presence of all antibodies which occur in some patients with a prior history of transfusion. Alloimmunisation is observed to be much less common in Africa where donor and recipient are often of the same race, in contrast to Europe and North America where donor and recipient are often racially different [2].

TRANSFUSION REGIMENS

As previously mentioned, 3 basic methods of transfusion may be employed in patients with sickle cell disease - simple transfusion, exchange transfusion and erythrocytapheresis - and these may be adapted as required to episodic or chronic transfusion protocols. Invariably, packed red cells are the blood components of choice except when marked volume expansion is needed [5,6].

Chronic simple transfusion - Once target Haemoglobin A levels of 60-70% is achieved, this may be maintained with simple transfusions at 2-4 week intervals. HbA levels must be monitored regularly with quantitative haemoglobin electrophoresis. Generally, pre-transfusion haematocrit 25-30% is adequate with a target post transfusion haematocrit of 36% or less (to forestall the risk hyperviscosity) [6].

Exchange Transfusion This type of transfusion reduces the concentration of sickle cells without increasing the haematocrit or whole blood viscosity. It may be done manually or by automation. The aim is to replace the sickle cells in the blood with normal red cells and to prevent further production of sickle cells in the short term. Blood containing HbS is removed from the patient and an equal amount of donor blood containing Hb A is transfused into the patient. The benefits include the following:

1. The volume of blood in the patient remains the same and the circulation is not dangerously overloaded.

2. The patient's Hb concentration rises because a volume of better haemoglobinized blood has been used to replace the same volume of less haemoglobinized blood.

3. Many sickle cells would have been replaced with Hb A cells thereby temporarily eliminating or preventing complications caused by sickled cells.

Rapid Partial Exchange - In this method, whole blood is removed from one arm at the same time that donor cells are transfused into the other arm. In adults, the procedure is performed in 500ml units and usually 6-8 units of blood are needed to optimally exchange an adult.

Exchange transfusions performed with whole blood are more efficient than those using packed cells.

A simple protocol is given below:-

i. Bleed one unit (500ml) of blood from the patient and infuse 500ml of saline.

ii. Bleed a second unit from the patient, infuse 2 units of blood

iii. Repeat step (i) and (ii) ; if the patient has a large red cell mass repeat once more.

Care must be taken to ensure that the final haemoglobin level does not exceed 10-12 g/dl to avoid hyper viscosity.

COMPLICATIONS OF TRANSFUSION

Transfusion complications may occur more frequently in sickle cell disease patients given their more frequent need for transfusion therapy than the general population.

These attendant complications include:

1. **Volume overload**- This can occur when too much volume is transfused too quickly with possible development of congestive heart failure and pulmonary oedema. Intravenous furosemide, partial removal of red cells, preserving fluid and slow rate of transfusion can help prevent this problem.

2. **Acute haemolytic transfusion reaction**

This follows major blood group mismatch and is rapidly fatal. The patient must be aggressively treated

maintain blood pressure and renal perfusion. These are best prevented by careful record keeping and sample identification to minimize clerical errors.

3. **Delayed haemolytic transfusion reaction**

This usually occurs following alloimmunization to red cell antigens in transfused patients with sickle cell disease. 30% or more of alloantibodies may disappear with time but the patient remains capable of mounting an anamnestic response to stimulation. [6]

4. **Transfusion-transmissible infections** - these include viral hepatitis B and C, Human Immunodeficiency Virus (HIV), parvovirus and rarely bacterial infections. Patient receiving transfusion therapy and especially those on a chronic transfusion protocol should be periodically screened and counselled.

5. **Febrile, non-haemolytic transfusion reactions**: These may be caused by antibodies developed to platelets and leucocyte antigens. This is best prevented by leuco-depletion measures.

6. **Iron overload**: This often occurs in sickle cell patients who require routine transfusions throughout their lives. However, the problem is often undetected and may not be treated. The most accurate test for iron overload is a liver biopsy. Chelation therapy is recommended when the iron is 7mg per dry weight or alternatively at cumulative transfusions of 120mls of packed red cells per kilogram body weight. Desferrioxamine at an initial dose of 25mg/kg/day given over 8-10 hours (subcutaneously) is quite safe but should be discontinued during an acute infection. Other iron chelators are the subjects of current studies.

CONCLUSION

While the benefits of transfusion are undoubted, the safest transfusion is the one which never occurred. Prudence must be the watchword in organizing and implementing effective transfusion practices. In this regard, the following measures are pertinent -

- Prevention of severe anaemia by adequate, continuous education and counselling of patients, routine folate supplementation, dietary advice and prevention/prompt treatment of infections.
- The precise need, quantity and duration of transfusion should be determined by appropriately qualified medical professionals
- Good record keeping
- Vaccination prophylaxis against transfusion transmissible infections (where available)
- Periodic reviews and audit of transfusion programmes and early detection of complications.

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PAEDIATRIC CENTRAL NERVOUS SYSTEM NEOPLASMS- CHALLENGES AND PROSPECTS

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Contrary to the classical teaching that central nervous system (CNS) neoplasms are uncommon in black children, these neoplasms are the fourth most common paediatric neoplasms in Ibadan. They have increased dramatically in frequency during the past five decades, largely due to expansion of neurological services. This paper reviews aetiopathogenetic and clinicopathological features of paediatric CNS neoplasms. There is evidence that brain tumour stem cells within germinal regions of the developing CNS contribute to these neoplasms. Childhood CNS neoplasms have a multifactorial origin, with a minority of cases being linked to heredofamilial disorders such as types 1 and 2 neurofibromatosis, tuberous sclerosis, Gorlin's syndrome, Turcot's syndrome and the Li-Fraumeni syndrome. Clinical presentation of paediatric CNS neoplasms varies, ***and may be non-specific sometimes,*** *depending on the location of the neoplasm, stage of development and age of the child. The prognosis also varies considerably, depending on morphological type and age of the child. The poor prognosis associated with intracranial neoplasms generally, is multifactorial. In addition, severe long-term effects of chemotherapy and irradiation on the paediatric CNS may occur. Childhood CNS tumours produce considerable socio-economic stresses on both the society at large and the immediate family of afflicted children, particularly in resource-poor settings such as Nigeria. They constitute a diagnostic challenge to the clinician, and a relatively unexplored, but extremely challenging field to the research physician. There is a persistent compelling need for further extensive studies in order to enhance our understanding of the inborn and environmental origins of these neoplasms.*

KEY WORDS- Central nervous system, brain, spinal cord, tumours, children, clinical challenges

*INTRODUCTION

Paediatric neoplasms constitute an important issue of public health concern, accounting for considerable morbidity and mortality worldwide. They comprise not one disease entity, but rather a spectrum of diverse and heterogeneous neoplasms that vary by type of histology, site of disease origin, race, sex, and age[1]. Childhood neoplasms provide basic health science and clinical researchers alike unique opportunities for elucidation of the mechanisms of tumourigenesis[2]. The topographical distribution, characteristic histological patterns and age distribution of certain childhood neoplasms suggest a probable genetic contribution to their aetiopathogenesis, which is attested to by the fact that approximately 10-15% of all paediatric neoplasms are hereditary or familial in nature[3, 4]. Mutations involving oncogenes, tumour suppressor genes, DNA mismatch repair genes, and nucleotide excision genes are important aetiopathogenetic factors of childhood neoplasms[5]. **They are also more common in children with hereditary neurocutaneous syndromes like neurofibromatosis and tuberous sclerosis.**

Central nervous system (CNS) neoplasms are the fourth most common paediatric neoplasms in Ibadan after lymphomas, retinoblastomas and nephroblastoma. In Caucasians, CNS neoplasms are the second most common paediatric neoplasms and the most common childhood solid tumours[6]. The relative frequency of paediatric CNS neoplasms has increased approximately six-fold in Ibadan, from 2.2% to 12.9%, during the past four to five decades[7]. Doubtless, a similar trend will also be reported from several other Nigerian centres, with the progressively increasing numbers of locally- and foreign-trained neurosurgery, neurology, neuroradiology, neuropathology and neuropaediatric specialist physicians and ancillary medical staff. Thirty-seven percent of all CNS neoplasms in Ibadan occur in children, with an overall male to female ratio of 1.3:1[8].

Being an extremely complex and specialized system, the CNS harbours a fascinating array of approximately 130 primary neoplasms[9]. The distinction between benign and malignant neoplasms is blurred in the brain, in that all CNS neoplasms may be eventually attended by fatal raised intracranial pressure (ICP), irrespective of degree of histological differentiation. **In this respect, many childhood CNS neoplasms often only come to clinical awareness after their initial non-specific symptomatology has evolved to more terminal ones like those of raised ICP.**

It is also noteworthy that the majority of primary cerebrospinal neoplasms arise from the resident stable cell population of glial epithelium. Neurones, being post-mitotic (permanent) cells do not commonly give rise to neoplasms. In this respect, medulloblastoma, which is the most frequent postnatal brain tumour, develops in the cerebellum, which is the main site of postnatal proliferation in the brain[6]. The ratio of cerebral to spinal cord neoplasms is approximately 10 cerebral neoplasms for every single case of spinal cord neoplasm. Another general aphorism is the fact that about 70% of all adult brain neoplasms are supratentorial, whereas 70% of all childhood neoplasms are infratentorial. However, it must be noted that in infancy, there is actually a predominance of supratentorial neoplasms, while in later childhood there is a predominance of infratentorial neoplasms.

AETIOPATHOGENESIS OF CHILDHOOD CNS NEOPLASMS

ere is significant experimental evidence that brain mours might originate from the transformation of differentiated precursor cells, which are found not only in germinal regions of the developing and early-postnatal CNS but also in areas of the mature brain, in which neurogenesis persists throughout adulthood. **In this respect, the subventricular zone is thought to be the most likely source of gliomas**[10]. Certain childhood CNS neoplasms, of which medulloblastoma is the archetype, apparently originate in the context of aberrant embryogenesis, which is reflected by the striking histological resemblance of this neoplasm to the foetal cerebellum[2]. Experimental mouse models have demonstrated that medulloblastoma cells possess gene products expressed by the foetal cerebellum at varying stages of development, as outlined later in this review.

tiology of Childhood CNS tumours:

The aetiology of the majority of childhood CNS neoplasms is not known. However, it is appreciated that they, like other neoplasms, have a multifactorial origin. A minority of paediatric CNS neoplasms are linked to familial disorders such as type 1 neurofibromatosis (pilocytic astrocytoma); type 2 neurofibromatosis (bilateral acoustic schwannoma); tuberous sclerosis (subependymal giant cell astrocytoma); Gorlin's (naevoid basal cell carcinoma syndrome (medulloblastoma); Turcot's syndrome (medulloblastoma, glioblastoma); and the Li-Fraumeni syndrome (astrocytomas, medulloblastoma and other tumours) (Table I)[9]. Another reported risk factor for childhood CNS neoplasms is male gender, in cases of medulloblastoma and ependymoma. The majority of other CNS neoplasms do not show any gender predisposition. Therapeutic ionising radiation exposure to the head in cases of tinea capitis and head and neck cancer increases the risk of developing brain neoplasms, especially **the meningiomas but this postradiation tumourigenesis is usually more prominent later in life**. Other studies suggest that childhood brain tumours are more prone to occur in the children of mothers that ingest cured meat during pregnancy and in those children with a family history of leukaemia, lymphoma, bone or brain tumours. The latter association might be explained on the basis of undetected Li-Fraumeni or other familial cancer syndromes in some cases. Finally, there are tenuous and other anecdotal associations of childhood brain tumours with electromagnetic field exposure, cellular phone usage, certain paternal occupations, pesticide exposure, head injury and a family history of either epilepsy or mental retardation[11].

SSIFICATION OF PAEDIATRIC CNS NEOPLASMS

re are many classification systems for categorising neoplasms, and none of them has emerged as the standard[11]. The most widely employed system is World Health Organization (WHO) classification of CNS outlined in Table II[9]. However, paediatric neoplasms are better classified for comparative epidemiological purposes using the International Classification for Childhood Cancer of 2000, which groups CNS neoplasms into broad diagnostic categories as follows[12]:

Ependymomas and choroid plexus tumours

Astrocytomas

Intracranial and intraspinal embryonal tumours

Other gliomas

IIIe- Other specified intracranial and intraspinal tumours

IIIf- Unspecified intracranial and intraspinal tumours

FEATURES OF SPECIFIC PAEDIATRIC CNS NEOPLASMS

a. Gliomas

About 70% of primary CNS neoplasms are of neuroglial origin. These are classified based on differentiation of the tumour cells into the 3 major cell lineages, namely astrocytic, ependymal and oligodendroglial.

i. Astrocytic neoplasms

Astrocytomas are the most common gliomas, accounting for about 39% of paediatric intracranial neoplasms[2]. The majority of paediatric astrocytomas originate in the cerebellum or pons, and a minority of cases are hemispheric. Optic nerve involvement has also been recorded[2,13]. Astrocytic neoplasms may be grouped in various differing ways:

1. Topographic

Supratentorial (cerebral hemisphere) astrocytomas are more common in adults, whereas infratentorial (cerebellar and brainstem) astrocytomas are more common in children. Cerebral hemisphere and brainstem astrocytomas have a worse prognosis than cerebellar astrocytomas of childhood; **the former usually because it is of higher grade, the latter for the obvious reason of its critical location, in addition to the fact that it is also frequently of the high grade type .** Childhood cerebellar astrocytomas are amenable to complete resection and prolonged survival of up to 40 years has been recorded in examples of this tumour.

2. Microscopic differentiation

Astrocytomas may exhibit fibrillary, protoplasmic, gemistocytic, pilocytic or pilomyxoid patterns of differentiation.

3. Prognostic

Several histological grading systems have been proposed, but that which is universally applied currently is the four-tiered system advocated by the World Health Organization[9]. According to this scheme, astrocytomas may be grade 1 (juvenile pilocytic), grade 2 (well-differentiated), grade 3 (anaplastic or malignant) or grade 4 (glioblastoma multiforme). These 4 groups are respectively associated with median survival times of up to 20 years, 6 years, 2 years and 6 months respectively. As a general rule, the majority of low-grade astrocytic neoplasms (pilocytic astrocytoma and subependymal giant cell astrocytoma) occur in children, while the majority of high-grade (anaplastic astrocytoma and glioblastoma multiforme) occur in adults[9].

ii. Oligodendrogliomas

Oligodendrogliomas are relatively uncommon in children, being primarily adult neoplasms[9]. They are characterized clinically by radiographically demonstrable calcification, and morphologically by nests of uniform cells with perinuclear haloes (fried egg appearance) interspersed by a chicken-wire mesh-work of capillaries. These neoplasms affect the frontal, temporal, parietal and occipital lobes in a ratio of approximately 3:2:2:1. The molecular signature of oligodendroglioma is characterized by deletions involving chromosomal segments 1p and 19q.

iii. Ependymal neoplasms

Ependymomas tend to arise from the lining of the fourth ventricle, more so in children, among whom these

Table I-Risk factors for childhood CNS neoplasms (modified from Gurney et al, 2001)

RISK FACTORS	COMMENTS
DEFINITE RISK FACTORS	
Sex	Medulloblastomas and ependymomas more common in males than females
Ionising radiation exposure to the hea	Three- to six-fold increase in risk
Neurofibromatosis, tuberous sclero Gorlin's syndrome, Turcot's syndror Li-Fraumeni syndrome	50- to 70-fold increase in risk
PROBABLE RISK FACTORS	
Maternal ingestion of cured meat dur pregnancy	Two-fold risk for child developing brain tumour
First degree relative with brain tumou	Four-fold risk
Family history of leukaemia, lymphor or bone tumour	Increased risk due to Li-Fraumeni syndrome
CONTROVERSIAL RISK FACTORS	
Electromagnetic field exposure	Inconsistent results between different studies
Father's occupation	Aircraft, agriculture, electronics, petroleum, painting, paper/pulp mill, printer, metal, ionising radiation, solvents, electromagnetic fields- also variable results
Pesticides	Limited anecdotal evidence
Head injury	Difficult to assess
Family history of epilepsy	Inconsistent data
Family history of mental retardation	Anecdotal data

neoplasms are almost invariably infratentorial. By contrast, in adults, supratentorial and infratentorial ependymomas have an equal frequency[9]. They are the most common spinal cord neoplasms. In the cauda equina, a peculiar variant, the myxopapillary ependymoma has been described.

iv. Mixed (oligoastrocytic) neoplasms

Oligoastrocytomas have a prognosis that is intermediate between that of oligodendroglioma and astrocytoma. These neoplasms predominate in middle aged adults, although childhood cases have been described[9].

b. Embryonal neoplasms and Primitive Neuroectodermal tumours (PNET).

The archetype of these tumour variants, also histologically referred to as the blue-cell tumours, is medulloblastoma.

Medulloblastomas are highly malignant predominantly childhood embryonal neoplasms arising from foetal external granular cells (classic medulloblastoma) or paraventricular neuroblasts (desmoplastic medulloblastoma). These neoplasms have a peak age at presentation of 7 years[9]. The Sonic hedgehog (Shh) signal-transduction pathway plays a role in the patterning, growth and survival of the CNS and other tissues. The Shh pathway ultimately activates the transcription factors Gli1 and Gli2. Patched, which inhibits the positive activation of GLI1, is mutated in Gorlin's syndrome (familial basal-cell carcinomas and medulloblastomas). The ShhGli pathway is also abnormally activated in sporadic medulloblastomas and Gli activation has been implicated in the development of gliomas. Inhibition of Gli function might therefore be a promising therapeutic target in these tumours[14].

c. Specialized neuroepithelial neoplasms

Choroid plexus papillomas are an interesting cause of hypersecretory communicating hydrocephalus. These neoplasms present in very young children, often under the age of 1 year. Their anatomical distribution is inversely related to that of ependymal neoplasms, with the vast majority of paediatric neoplasms being supratentorial, while fourth ventricle tumours are evenly distributed in all age groups[9].

d. Meningeal neoplasms

Meningiomas account for about one-fifth of intracranial neoplasms, being more common in adult females than males, which may in some way be related to the presence of steroid receptors in these neoplasms. The majority of cases occur in adults, while meningiomas associated with familial cancer syndromes tend to occur in younger children[9].

e. Nerve sheath neoplasms

Acoustic schwannomas may be sporadic or familial (type 2 neurofibromatosis), being bilateral in the latter instance. Paediatric cases are relatively uncommon[9].

f. Lymphoid neoplasms

Primary cerebral lymphomas mainly occur in children who have inherited immunodeficency syndromes. These neoplasms characteristically present by the age of 10 years[9]. The majority of cases are high-grade B cell non-Hodgkin lymphomas.

g. Vascular neoplasms

Haemangioblastomas are predominantly adult neoplasms, although familial cases (associated with von-Hippel Lindau disease) tend to occur at a significantly younger age[9]. Histologically, these are vascular neoplasms comprising lipid-filled stromal cells and proliferating capillaries. They usually occur in the cerebellar hemispheres, or rarely as cerebral hemisphere tumours. Apart from features of raised intracranial pressure, some patients may also present with secondary polycythaemia, due to elaboration of erythropoietin by the tumour cells[9].

h. Maldevelopmental neoplasms

Developmental neoplasms such as germ cell tumours (germinoma, teratoma), craniopharyngioma (Rathke pouch remnants) and vascular malformations may also occur. It should be noted that although foetal brain

umours are quite rare, these tumours are predominantly teratomas, reflecting the more primitive tem cells that are present in early brain tissue. Many eatures of these developmental neoplasms closely esemble the biology of the immature tissue in which ey arise[1].

Pituitary adenomas

hese neoplasms are common and potentially serious eoplasms that can cause mood disorders, sexual sfunction, infertility, obesity, visual disturbances, pertension, diabetes mellitus and accelerated heart isease. Although the majority of tumours are benign, a oportion may invade the surrounding structures such the sphenoid sinus, the cavernous sinus, and even the ain. Promoter hypermethylation of the human Ras sociation domain family 1A gene (RASSF1A), located 3p21.3, has been demonstrated to play a role in itary tumourigenesis[15].

Craniopharyngioma

Worldwide, this is the most common parasellar/suprasellar brain tumour in childhood [16]. This is also attested to by experience in this environment [13, 17, 18]. Craniopharyngiomas are histologically benign, but many times clinically malignant, neoplastic growth of the sellar/parasellar cell rests of the Rathke's pouch. The tumour, solid, cystic or mixed-consistency, usually has a ramifying pattern of growth in the parasellar region to involve the surrounding critical neurovascular structures, including the visual apparatus and the internal carotid artery. The usual challenge therefore is how to achieve total surgical resection of many such lesions without causing new neurological deficits. In most such occasions, all that is realistically achieved is subtotal tumour resection followed with adjuvant treatments, mainly radiotherapy.

Metastatic neoplasms

ally, a significant proportion of intracranial eoplasms are metastatic or secondary. In children in is environment, CNS involvement by Burkitt's mphoma is the most important secondary neoplasm. etastasis of cancer cells to the brain, as for other gans, occurs via a non-random sequential process, mprising invasion of surrounding tissue, entry into d survival in the bloodstream (intravasation), arrest d/or extravasation at the secondary site, and survival d proliferation. Recently, several metastasis ppressor genes, which can spontaneously suppress etastatic growth at any point in the metastatic cascade, ve been identified, such as NM23 and CD44. plication of advances in the understanding of the thogenesis of brain metastasis may lead to novel geted treatment models and a better prognosis for ients with brain metastatic disease[19].

CAL PRESENTATION

ally, the clinical diagnosis of childhood CNS eoplasms is highly confounded by many factors [20]. ildren less than 3 years often are not able to report eir health problems to their parents or care givers. It kes an acute sensitivity on the part of the parents detect subtle changes in the general behaviours of eir wards before they can begin to suspect that mething is the matter with them [21]. At other times, the ial complaints of a child with brain tumour may be so nspecific, like diarrhoea, nausea and vomiting, that ey are mistaken, even by the family physicians, for such ner conditions like gastroenteritis [22, 23]. Hence, late diagnosis is a continuing challenge in paediatric CNS tumours. This is even more so in resource-poor settings like ours here in Nigeria where there is that penchant by parents to either ignore, or seek alternative health solutions to, the initial complaints of their wards that might have aided in early diagnosis. This is to be viewed also in conjunction with the fact of the general gross inadequacy of the health systems to address the total health situations [17].

More Specific Presentation of Childhood CNS neoplasms

In general, the symptoms and signs described for children with intracranial tumours are headache, nausea and vomiting, ataxia, seizures, papilloedema, cognitive decline, head enlargement, visual deterioration, and other eye abnormalities [20]. For spinal tumours, presentation usually is that of back ache, gait incoordination, spinal deformity, sphicteric deficit and limb weakness [20]

However, the presentation of childhood CNS neoplasms could also be more specific to the location of the neoplasm, its stage of development and the age of the child[19,26].

Posterior fossa neoplasms, cerebellar or brainstem, present with ataxia, nystagmus, dysarthria, hypotonia, limb paresis, cranial neuropathy and early onset raised intracranial pressure, the last usually from obstructive hydrocephalus. Another unique presentation of posterior fossa mass lesions in children is neck stiffness from tonsillar herniation [24]. This may be mistaken for, and may necessitate prolonged treatment for, meningitis further militating against the timely diagnosis of the brain tumour. Some posterior fossa tumours may also be missed by computerised tomographic scanning, such that only after a cranial MRI study may a firm clinical suspicion of a posterior fossa tumour be dismissed.

Pineal region tumours present in children also with early onset of raised ICP from obstructive hydrocephalus. They also have characteristic eye signs like Parinaud's syndrome, that is, upgaze palsy, poor convergence and defects in accommodation.

Sellar/Parasellar masses present with visual symptoms and signs, features of raised ICP, and evidence of perturbations of the hypothalamo-pituitary endocrine functions. These include obesity, growth retardation or precocious puberty, and abnormalities of water and electrolyte homeostasis.

Cerebral hemisphere tumours present with hemiparesis, hemisensory loss, visual field abnormalities, seizures and declining intellectual performance. Mid line tumours present with visual loss, endocrinopathies, increased intracranial pressure, personality change and intellectual decline[16,26].

Diagnostic Management

Radiological investigations are an important part of the total care of CNS tumours. In the days before the contemporary era of computerised neuroimaging, plain skull X-ray films, cerebral angiography, ventriculography and pneumoencephalography were used in the evaluation of cranial mass lesions, whilst plain X-ray and spinal myelographies were utilized for spinal disease.

In the current era however, computerised tomography (CT) and magnetic resonance imaging (MRI), amongst sundry other computerised investigating studies, are the gold standard of radiologic work-up of CNS tumours.

They both, CT and MRI, have their strengths in the information derivable from each, and sometimes are complementary to each other. MRI is the radiological investigation of choice for posterior fossa mass lesions in any age group, more so in childhood, whilst CT is preferred in the acute situations, e.g. for the immediate post-operative evaluation of the surgical treatment. They each, or both of them taken together sometimes, help to establish the presence, the location and the local extent of a tumour in the neuraxis, and also to screen for possible extra-neural extension of the disease process. They are also utilized in the post-surgical excision care of the patients to screen for possible recurrence [22]

Tumour Markers

Some paediatric CNS tumours express tumour markers which can be recovered in the blood and or the cerebrospinal fluid, CSF. Examples of these are alpha fetoprotein, foetal alkaline phosphatase, and beta human chorionic gonadotropin expressed by germ cell tumours of the pineal region. These tumour markers can aid in the pre-histological diagnostic differentiation of the possible cell line of the mass lesion. They are also useful occasionally in the post treatment (surgical excision or otherwise) follow-up of individual cases for possible recurrence [22].

Treatment Paradigms for Paediatric CNS Tumours

The general aim of treatment usually is relief of the tumour burden and its attending neurologic deficits. This is carried out mainly by surgical excision or debulking followed by other adjuvant treatment modalities like radiotherapy, chemotherapy and others.

Surgery.

The goal of surgery is usually total excision/extirpation of the mass lesion and the tumour cells. This is one of the main good prognostic factors in brain tumour treatment [21] and is many times achieved in cases like cystic cerebellar astrocytomas, benign supratentorial ganglionic/neuronal cell tumours and occasionally some craniopharyngiomas. Some paediatric CNS tumours however can only be partially resected as for instance, supratentorial diffuse gliomas, or spinal cord intramedullary tumours. Efforts continue nonetheless to develop safe surgical corridors to such hitherto inaccessible mass lesions like pontine gliomas, especially the dorsally exophytic (into the 4th ventricle for instance) types, and tumours of the pineal region.

Table 2- Summary of major entities included in the 2007 World Health Organization classification of CNS neoplasms

TUMOUR CATEGORIES	EXAMPLES	WHO HISTOLOGICAL GRADE
1. Neuroepithelial tumours		
a. Astrocytic	Pilocytic astrocytoma Subependymal giant cell astrocytoma	Grade1
	Diffuse/Pilomyxoid/Xanthoastrocytoma	Grade 2
	Anaplastic astrocytoma	Grade 3
	Glioblastoma multiforme	Grade 4
b. Oligodendroglial	Oligodendroglioma Anaplastic oligodendroglioma	Grade 2 Grade 3
c. Ependymal	Myxopapillary ependymoma Subependymoma	Grade 1
	Ependymoma/anaplastic ependymoma	Grades 2/3
d. Oligoastrocytic	Oligoastrocytoma/anaplastic oligoastrocytoma	Grades 2/3
e. Choroid plexus	Choroid plexus papilloma Atypical choroid plexus papilloma Choroid plexus carcinoma	Grade 1 Grade 2 Grade 3
f. Neuronal	Gangliocytoma/ganglioglioma/others	Grade 1
	Anaplastic ganglioglioma	Grade 3
g. Pineal	Pineocytoma	Grade 1
	Pineoblastoma	Grade 4
h. Embryonal	Medulloblastoma	Grade 4
	Ependymoblastoma	Grade 4
	CNS primitive neuroectodermal tumour	Grade 4
	Atypical teratoid tumour	Grade 4
2. Meningeal	Meningioma (benign/atypical/malignant	Grades 1/2/3
	Haemangiopericytoma/anaplastic haemangiopericytoma	Grade 2/3
	Haemangioblastoma	Grade 1
3. Lymphoid	Primary CNS lymphoma	
4. Germ cell	Germinoma, Embryonal carcinoma, Y sac tumour, Choriocarcinoma, teratom Mixed germ cell tumour	
5. Sellar region	Craniopharyngioma/granular cell tum of neurohypophysis/pituicytoma	Grade 1

Table III- Clinical presentation of childhood brain neoplasms*

CEREBRAL HEMISPHERE TUMOURS (eg. Glioma, developmental cysts)	CEREBELLAR TUMOURS (eg. Medulloblastoma, ependymoma, astrocytoma)	MIDLINE TUMOURS (e.g. pituitary adenoma craniopharyngiomas, pineal region tumours)	BRAINSTEM TUMOURS (e.g. gliomas)
Hemiparesis Hemisensory loss Hemivisual loss Cognitive changes Seizures Raised ICP	Truncal ataxia Appendicular ataxia Scanning speech Hypotonia Pendular reflexes Nystagmus Early development of raised ICP	Visual loss Endocrinopathies (changing appetite, diabetes insipidus, stunted growth or precocious puberty) Changing personality Non-localising signs of raised ICP Some specific syndromes: Parinaud, Diencephalic etc	Cranial neuropathies Long tract signs Ataxia Raised ICP

* Modified from ref 26

er times, complementary or primary cerebrospinal (CSF) surgical diversion is carried out to relieve the athway obstruction and the complicating raised

herapy

on therapy is used to achieve one of several aims. is either as adjuvant therapy following surgical resection, partial or total, or as the primary nt in non-surgically resectable tumours. These diffuse pontine glioma, or certain radiosensitive rs like CNS lymphomas or germ cell tumours of eal region.

r, the potential and known complications of X- to the still developing nervous tissues of the ic age group limit the use of radiotherapy in od CNS tumours [22], generally for children less years of age but more so for those less than three. the complications of radiation exposures of the neuraxis in children include growth retardation, e decline and secondary neoplasms. For this radiotherapy is sometimes rather deferred in this up.

herapy

of CNS tumours, including in paediatric age are not chemosensitive. The exception here may CNS lymphoma and some germ cell tumours. r, studies have shown some benefits of herapy as a stop gap measure to prevent tumour ion in some children considered too young to adiotherapy for their brain tumour [25].

G REMARKS

three decades ago, it was widely believed that the e of gliomas was relatively low in black, as d to Caucasian, children[27, 28]. This statement will be constantly re-evaluated by workers in the paediatric neuro-oncology, particularly in the as medical services continue to expand in scope ability. It is very likely that the true magnitude of the problem of intracranial and other CNS neoplasms in Nigerian children is being underestimated by most studies due to the relative lack of neurological services in potential peripheral referring hospitals[2].

The poor prognosis associated with intracranial neoplasms generally, is due to several factors, namely, the inability to successfully completely debulk these neoplasms, the impermeability of the blood-brain barrier to chemotherapeutic agents, the low mitotic rates of many of these neoplasms and the toxicity associated with high doses of CNS radiotherapy[26].

The long-term effects of chemotherapy and irradiation on the paediatric CNS must be considered in survivors of paediatric brain tumours. The neuropathological features include a necrotizing leukoencephalopathy characterized by coagulation necrosis, fibrinoid vascular necrosis, demyelination and prominent swollen axons in the white matter. The late delayed effects of brain cancer therapy are irreversible[29]. Clinical manifestations include moderate to marked cognitive deficits with losses in IQ, learning disabilities, hormonal deficits from involvement of the hypothalamic-pituitary axis, growth retardation, and psychomotor retardation.[30]

Childhood cancer produces considerable socio-economic stresses on both the society at large and the immediate family of afflicted children[2], particularly in resource-poor settings such as Nigeria.[31] At least half of children may present with advanced disease, laboratory and radiological investigations and curative/palliative chemotherapy regimens may not be affordable to care givers, even if available, and the rate of default is very high[32,33].

However, improved information is required in order to allow empowerment of those with the ability to attract funding for the promotion of quality health care delivery services, and for the conduct of research into this grossly neglected field of paediatric neurosciences. There is therefore a persistent compelling need for further extensive studies in order to enhance our understanding of the inborn and environmental origins of childhood CNS neoplasms[2].

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CLIMATIC CHANGE: A TICKING TIME BOMB

By Ebekozien Osagie

troduction:

Climate refers to average weather condition anges from this average in a given place and usually 30 years), weather elements include haze, moist, sunshine, wind, humidity, dew, temperature.

in UK, forest fires in Indonesia, Lagos bar beach wing ... all attributed to extreme weather events, es in climate.

Climate change: a ticking time bomb; present rther changes in climate are feared to cause ms to mankind and environment. Climate change bal warming (as sometimes described) connotes se in the earth surface temperature; the world has en as warm as it is now for a millennium or more. ree warmest years on record have all occurred 998, 19 of the warmest 20 since 1980. Although erall rate of maximum temperature increase since is approximately 1°C per century, minimum erature have increased at twice that rate, and temperature are rising even faster near poles sea level (measured with tide gauge) has risen 20cm from melting ice at the poles.

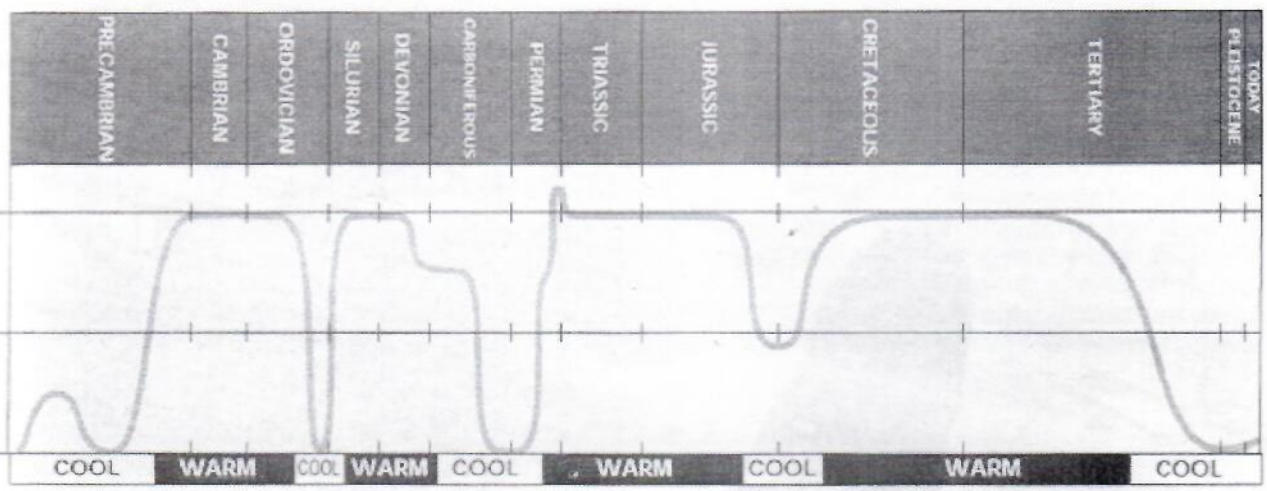
finition and Historical perspective

The earth's climate has never been constant, ions between cold and hot periods but the term change is "defined as change in climate which is ted directly or indirectly to human activities that composition of global atmosphere and which is to natural climate variability observed over a erable period of time". UNFCCC.

Studies have been done by archeologist to ne the past climate of the earth by mapping the ion of ancient coals, desert deposits, tropical salts deposits, glacial material as well as ion of plants and animals that are sensitive to such as alligators, palm trees and mangrove

dings are represented in the graph below.

showing different times on earth with different emperature.



Source: *http://www.climatehistory.com*

These climate alterations were due to natural causes and termed by the United Nations (UN) as "climate variability" as opposed to "climate change" mainly attributed to human activities.

3.0 Causes of Climate change

3.1 Natural: This is the reason for earth's climatic change before man's mastery of his environment in turn produced a negative effect on climate, vegetation, soils and water. Natural causes include emission of Nitrous Oxide (N_20) from ocean, decomposition of organic matter mashes (emission of methane) respiration of animals and plants (carbon dioxide release) these gases mentioned are called green house gases and their role in causing climate change explained below.

3.2 Man made

3.2.1 Green house gases

Gases like ozone, carbon dioxide, methane, nitrous oxide (N_20), chlorofluorocarbon (CFCs) are the major cause of climate change.

Heat from sun is trapped by these polluting gases in the atmosphere, gases trap heat by forming shield around earth, like a green house glass, effect known as green house effect. Human activities that increase these gases include burning fossil coal, oil, gas cutting down forest trees (supposed to trap carbon dioxide CO_2), agricultural activities, exhaust from motor vehicles, industrial burning activities (power plants), fertilizer use (Nitrous oxide release) refrigerator and air conditional use, aerosol propellant use (CFCs).

A study done by that Harvard medical school's centre for Health and the Global environment puts the current atmosphere concentration of carbon dioxide at 379 parts per million (PPM). It adds that the earth has not experienced CO_2 level above 280 PPM for at least 42,000 years. 0

3.2.2 Ozone Layer Depletion

Ozone (O_3) is important to the climatic system both through its absorption of solar radiation and as a green house gas through its absorption of infrared radiation from the sun. Following the publication of ozone depletion theory (1974) the role of Chlorofluorocarbon was better understood as a very important cause of our warming climate.

4.0 Implications and future expectations

Climate change, a ticking time bomb with widespread implications in different areas of human endeavor future expectations includes:

- Global mean temperature increase by between 1.4°C and 5.8°C. Northern hemisphere cover should decrease further, but the Antarctic ice sheet should increase.
  - *Sea level rise by between 9 and 88 cm*
  - Increase in extreme weather events.
  - These changes projected to last for many centuries.
  - Predictions made from emissions scenario using computer models.

4.1 Environmental Effect

- Warming varying by regions will be accompanied by changes in rainfall pattern leading to land slides, droughts, desertification, soil erosion, famine, floods, and heat waves.
  - Melt the ice caps in the cold region and cause thermal expansion of oceans, overflow of watering coastal region.
  - Rising global surface temperature could result in arched soils, shrink soils and increase chances of erosion and building subsidence.

4.2 Energy and Engineering

- Change in our energy source with new policies to curb this menace, resulting in development of new power source, solar power, geochemical, wind, and water power.
  - House building would have to change using green roof to reduce heat.
  - Reduction in the use of chlorofluorocarbon (CFC) containing aerosol propellant.
  - Redesigning and proper calculation in rail, bridge and road construction.

4.3 Life and Habit

- Extinction of Australian flying foxes due t extreme heat.
  - Proliferation of jelly fish and the need t protect fish stock.
  - Ocean acidification proposal would redu aquatic life.
  - Pines in California the dying off.

UN believes that there would be a northward sh 200km 300km for every 1°C

4.4 Economic Implications

In 2004, Munich Re, one of the biggest reinsuran company world wide, reported insurance loses, of $4 billion, associated mainly with extreme weather.

- Economic losses following disaster; change insurance and financial service.
  - Money for research and development of ne ways in curbing problem.
  - Diseases and medical conditions from th adverse weather would be another econom burden.
  - Scarce food resources, making available one more expensive.

4.5 Agriculture

- Farmers would have to adjust planting an harvesting dates.
  - Production of grain crops can be depressed rise in temperature from global warming, being particularly sensitive 1°C rise temperature above 32°C results in 3 reduction in yield.
  - Increase in genetic engineering to cope wi need
  - Proliferation of agriculture pest like aphi causing decrease in wood and woo production.
  - Loss of fertile coastal lands caused by rising sea level.

4.6 Health Implications.

"there is very, very big connection between th emergence of new disease and environmenta change" said Nick Nutall spokes person for th United Nation Environmental Program (UNEP

- Malnutrition would predispos individuals to lot of disease.

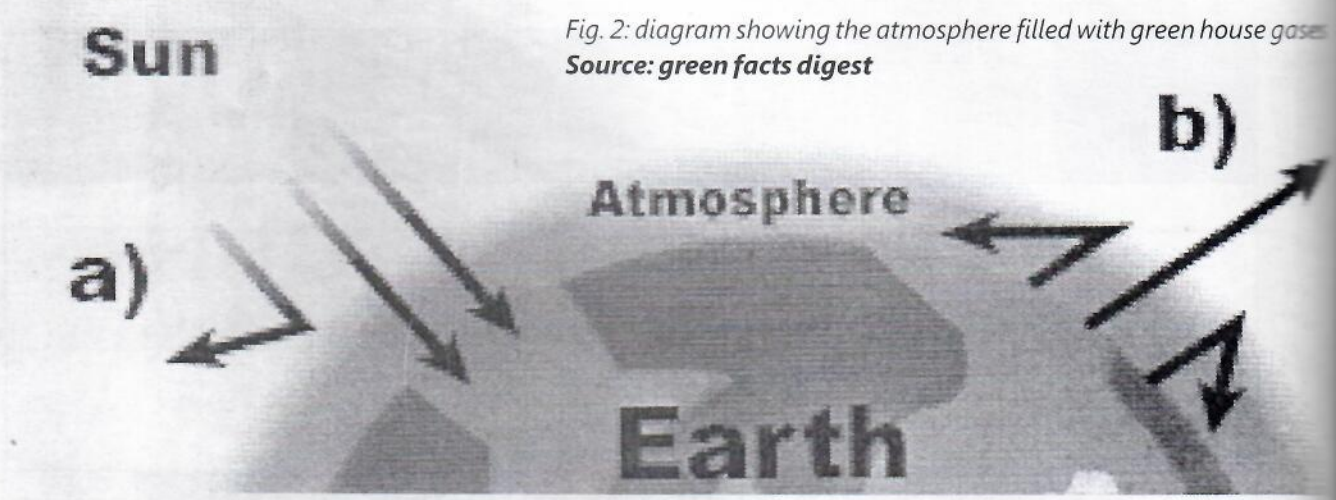


Fig. 2: diagram showing the atmosphere filled with green house gases
Source: green facts digest

- Death from disaster
- Chicago heat wave resulted in 700 deaths (John Hopkins University 2003)

- 25,000 people died in heat wave that hit Western Europe in 2003 (BBC News)
- Thousand have died and more would die from flood, wind slides, tornadoes and other disasters.
- Increase in new diseases and infection with existing ones, since 1976, WHO has noted emergence of 30 new diseases.
- Nipah virus infection from deforestation in Malaysia.
- Increase in malaria, study done in latin America showed that a 10% rise in deforestation resulted in 8% increase in malaria carrying mosquitoes, globally 10% of population that were not previously exposed are at risk of contracting malaria.
- Cerebrospinal meningitis likely to increase in the dry belt of Nigeria
- West Nile virus and Ross river virus from extreme drought
- Water borne disease like cholera, and parasitic infestation
- Malignant condition like basal cell carcinoma from UV radiation
- Respiratory disease and asthma increase because of high CO_2 level which favors pollen producing plants.
- Travel hazards from extreme weather condition

5.0 Africa and Climate Change

Africa, particularly Nigeria is not immune from the effect of climate change, unfortunately she is not as equipped as the developed world to combat this problem. Part of Africa's peculiarities include.

5.1 **Geographical Location:** Located in a coastal region, Coastland are more prone to floods and destruction from rising sea level, calculations are that 0.2m rise in sea level will inundate 3,400 Km^2 of coastland (especially the Niger Delta belt of Nigeria), (Onofeghera 1990). Location also makes her prone to drought and desertification.

5.2 **Poor Financial Sector:** With corruption, money trafficking Africa suffers a problem with wealth distribution if there is a catastrophe at Niger Delta the country would lose close to 9million naira.

5.3 **Power Production:** The supply of power in Africa is largely hydrological, climatic change is causing reduction in potential for power generation of kainji and Shiroro dam in Nigeria, Akosombo, and kpong hydropower reservoir in Ghana.

5.4 **Action by Nigeria on climate:** includes development of a climate change unit in the department of environmental assessment under the federal ministry of environment. Studies done at Nigerian institute for oceanography and marine researched centre for energy research and development Ile Ife.

6.0 PREVENTING DETONATION OF CLIMATE CHANGE BOMB

6.1 Global and government policies

6.1.1 Bonn conference, the Kyoto protocol

In 1977 the treaty setup to consider what can be done to reduce global warming. The treaty was established by United Nations Framework Convention on climate Change (UNFCCC) involving most world countries with exception of America, their role includes developing new methods to reduce CO_2 level, develop a standardize approach to managing the results of climate change

6.1.2 Montreal Protocol

Protocol on substance that deplete the ozone layer is a landmark international treaty that protects the stratospheric ozone layer by phasing out the production and consumption of about 100 man made chemicals, treaty was originally signed in 1987 and has been amended five times, fund implementing agencies include UNEP, UNDP, UNIDO and World Bank.

6.1.3 Other Policies

Different government have developed policies unique to their environment, prominent ones include.

Johannesburg summit, Delhi declaration, Rich V poor, USA policy, UK policy and Nigeria policy.

These policies cover industrial regulation, agriculture, power, engineering and health.

6.2 Industries and Individual

Industries must be aware of the changing climate and its effect, they should engage in only activities known to be "environmental friendly" from the type of fuel use to means of disposing waste product.

Individuals also have a role to play by using energy saving appliances, turning off TV, light when not in use, closing curtains to keep heat, favor more natural means of doing things, reduce food and water waste, proper disposal of waste amongst other would go a long way.

7.0 Conclusion

Latest evidence collated by international panel on climate change (IPCC) predicts that global temperature will rise between 1.5°C and 4°C of human activities double CO_2 (Carbon dioxide) level.

The 1st half of the 20th century scientist thought that climate, if it changes at all, evolves so slowly that the difference cannot be seen in human lifetime but that view has been proved wrong. Climate change is as real as life itself, so government, industries, non governmental organizations and individuals must stand to the fight of either preventing the blast or increasing the time before the blast of the climate change bomb.

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DOKITA NEWS

SSIONS

itor-in-Chief on behalf of the board hereby ratulates the following people on thier sion into DOKITA Editorial Board:

BER 2007
duwe Charles
utimi Busayo
onye Charles
emi Setemi
unuga Folusayo
nuga Abayomi

H 2008
deyemo Adewunmi
eyanju Timilehin
chukwu Onyeka
adipe Toyin

A Symposium

nd annual symposium of DOKITA was held on of June 2007 under the distinguished anship of Emeritus Professor E.O Akande ation Provost College of Medicine and s Professor of Obstetrics and Gynaecology, ity of Ibadan.

Infertility: Dread of the African Couple

emes: Infertility: The Nigerian Situation

Dr. C.O Chovwen
Senior Lecturer
Department of Psychology
University of Ibadan

Current Trends in the Management of Infertility

Dr. A.B Ajayi
Chief Executive Officer
NORDICA Fertility Center Lagos

The Role of the Family, Government and Non-Governmental Organizations in Infertility

Dr.O.A Roberts
Consultant Obstetrician and Gynaecologist
University College Hospital
Ibadan

PROFESSOR J.A.ADELEYE ESSAY COMPETITION:

The annual Professor J.A Adeleye Eassy Competition held from the 17th of September,2007 to the 2nd of March, 2008.

Topic: ***"The Climatic Change: A Ticking Time Bomb"***

Winners:

1st Prize-Ebekozien Osagie
(University of Ibadan)

1st Runner-up-Ogbodo Elisha
(University of Ibadan)

2nd Runner-up-Olaseinde Gbolahan
(Ladoke Akintola University of Technology)

ANNUAL GENERAL MEETING

This held on 17th February 2008.

Executives for the new Board year were elected,they are as follows:

Editor-in-Chief Miss Onwuka Nkechi
Board Secretary Mr.Akinbami Opeoluwa
Acting Production Manager Mr Oyenuga Abayomi
Distribution Manager Mr. Akinwunmi Femi
Financial Controller Miss Oderinde Motunrayo
Business Manager Miss Olokode Ayobami
Acting Publicity Editor Miss Olunuga Folusayo
News and Quiz Editor Mr. Ogungbemi Olawale

Compiled by the News and Quiz Editor,
DOKITA Editorial Board

POEM

INNOCENCE

Bright and vivid,
Innocence is clear,
Activated and livid,
His energy equals his peers.

Ignorance is bliss,
In my head their laughter ring,
That in a world like this,
The voice of innocence should still sing.

Arms splayed out in the sunshine,
Even malice seems pure,
Tears fall for so short a time,
That in their joy, of the gloom I'm unsure.

A long way to go it seems,
All you see is a face with a sheen,
That you'd give it all,
To have back your days.

Dedicated to the Child health Edition by Dewunmi.

UIMSA NEWS

1960

UNIVERSITY OF IBADAN MEDICAL STUDENTS' ASSOCIATION (UIMSA) NEWS FOR THE 2007/2008 EXECUTIVE YEAR

EXECUTIVE MEMBERS

Mr Adeluwoye Tiwalade	President
Miss Olakojo Olaotan	Vice President
Mr Okoror Collins	General Secretary
Mr Agbuduwe Charles	Assistant General Secretary
Miss Ekundayo Morenikeji	Treasurer
Miss Egbekun Ethel	Financial Secretary
Mr Raheem Kabiru	Public Relations Officer
Mr Ologbosere Nosakhare	Sports Secretary
Mr Oluranti Richard	Special Duties Officer (Clinicals)
Mr Odewole Odunayo	Special Duties Officer (Preclinicals)

SENATE OFFICIALS

Mr Omogiafo David	Chairman
Miss Azuka Chioma	Deputy Chairman
Mr Awosika Oloyede	Registrar
Mr Olasebikan Olubodun	Chief Whip

CONGRESS OFFICIALS

Mr Aiwonodagbon Benjamin	Chancellor
Mr Obogo Stephen	Deputy Chancellor
Miss Shittu	Scribe

The Executive council with the other arms of UIMSA was inaugurated on Monday 12th of November, 2007. In attendance were the foundation provost of the College of Medicine, Emeritus Professor E. Akande, Professor (Mrs) M.O. Onadeko (UIMSA staff adviser) and other eminent personalities. The motto for the Executive year is "Breaking New Grounds".

Association News

The first activity of the executive year was to revive the Faculty of Basic medical Sciences Liaison Office at the pre-clinical school and with the assistance of the Dean, Prof. O.O. Olaleye, the liaison office became effective on the 26th of November, 2007. 100 Level and Preclinical students can now go about their administrative activities there instead of coming to UCH.

The notice board welcoming students to the preclinical school was replaced by the Executive Council of the association with a brand new one which is a beauty to behold.

The Executive council with made a case and stood its grounds and with the assistance of the Provost, College of Medicine, was able to stop UIMSITES from paying other Student Associations' dues in UI..

Planning for the Year.

The Executive Council in its bid to ensure proper planning of activities of **UIMSITES** in the year, distributed year planners which were 1st of its kind in the history of our association.

100 Level Welcome programme

The programme came up on the 19th of January, 2008. This was the first phase of the programme which involved speakers like Dr Babalola of chemistry department, UI; Dr S.B. Olaleye (Department of Physiology, UI), Dr Owoeye (Department of Anatomy), Dr (Mrs) Moody (Snr. Academic Registrar, COM, UI). The students really benefited and got useful tips about keeping focused and staying strong in medical school. The second phase of the programme is the trip to Erin- Ijesha waterfalls.

ID card

The Executuve being concerned about about the welfare of ots members stood against the payment of N500 by the students before collection of their ID cards for the 2007/2008 session and also ensured that they got the ID cards without payment. For those who have problems concerning their ID cards, the executive are working directly with MIS in UI.

Hepatitis B vaccination

The executive council being sensitive to the plight of members carried out both the ***screening and vaccination*** for all preclinical students (200L and 300L) both MB, BS. BDS and physiotherapy students.

UIMSA 5-a-side

The revived annual UIMSA 5-a-side football competition took place between the 17th and 19th of April, 2008. The competition was an interesting one with the 2005 set winning the tournament. A side attraction was a novelty match between the MBBS ladies and Physiotherapy ladies.

Prof B.O. Osuntokun Quiz Competition

The 8th Edition of the competition took place between the 21st and the 25th of April, 2008. The competition was educative and informative with the MB, BS 2005 set emerging winners while the 1st and second runners-up were MBBS 2008 and 2006 respectively. A mini-party is to be organised for the winners and those who have been representing the association in quiz competitions nationally.

UIMSA 2008 World Health Day celebration

The first ever World Health Day celebration organised by the association took place on the 7th of April, 2008 with a public health campaign with the theme for the year being "Protecting health from climate change". The association organised an enlightenment campaign to Agbeni market, Ibadan where..... A courtesy visit to the Secretary to the Oyo State Govt. Chief Olakojo was made to show the association's support to government participation in qualitative health care delivery.

CMD Games 2008

The MB, BS football team won the football event for the second consecutive time beating its nursing counterpart 3-0. The team received the trophy at the dinner which was coulorful and interesting.

Buses for medical students

The executive council with the help of the college management was able to revive the transport scheme of its 2008 set from UI to UCH and back too UI after their lectures. The Executive Council with its diplomatic Prowess as being able to make the transportation scheme **free** for members of the 2008 set. Also buses are being arranged for clinical rotations and outside elective postings to Jericho, Eleta etc for Final year **UIMSITES** -The first of its kind! by an Executive Council.

Production of CBN'oscope Magazine

With the dedication of members of the Prelim press(100 Level), the Executive council assisted in ensuring the production of the first ever 100L magazine on behalf of the association. This has now added to the publications of the association and also carried 100 level **UIMSITES** along with the activities of the association.

Provision of hand gloves and soaps for dissection which has helped the 200 L members and their demonstrators a great deal

Intermedical Quiz competitions The association was represented in LAUTECH in Nov, 2007 and University of Ilorin in March 2008. The association was made proud by its representatives and honoured by the executive council.

Our own staff adviser and mother, Ass (Prof) Mrs M.O. Onadeko was on the 16th of May, 2008 made a full-fledged professor.

UIMSA Public Awareness Campaign 2008-06-07

This is the first of its kind and was organised in partnership with medriech pharmaceuticals with a campaign train to Bodija Market where a mobile clinic was set up and free consultations were made by members of the association to people who visited the clinic. This is a form of community development in going back what we've learnt in the hospital to the community.

WORLD BLOOD DONOR DAY

The Executive council, in collaboration with the Nigerian Red Cross Society organised a symposium to mark the 2008 World Health Day on the 14th of June, 2008. Professor Wuraola Shokunbi of the Department of Haematology, University College Hospital, Ibadan, was present at the occasion amongst other guests.

The president and financial secretary represented the association as part of the activities marking the day at the BCOS Live programme, *Saturday Special,* where they reached out to the public on the importance and advantages of blood donation. The vice-president led other members of the association to distribute hand bills made by the association to raise awareness within the halls of residence in the University of Ibadan and environs. This is the first of its kind by the association.

Upcoming Events

Awosika Memorial lecture 2008(October 2008)

Annual Health Week 2008 (October/Novermber 2008)

Provost cup/UIMSA sports fiesta (November 2008)

Unveiling of capital project for the executive year.

UIMSA PACKAGE 2008

This is something the Executive Council plans to use in saying a big thank you for the support it received from members during the tenure as it is going to be befitting for all medical students and will live long in the minds of members.

Also the Executive Council has made arrangement for all **UIMSITES** to have a presentable copy of the association's constitution and also the **Dokita** journal. Also the association's Magazine " the Ibadan Medscions" is to be published.

Mr. Okoror Collins

General Secretary

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Mr. Okoror Collins

General Secretary

COLLEGE NEWS

EVENTS

2006/2007 Faculty Lecture: Clinical Sciences-Challenges of the Back-Stage Actor in a Developing Economy by Dr. Olayinka.R.Eyelade MBBS, FWACS of the Department of Anaesthesia on behalf of the Faculty of Clinical Sciences, College of Medicine. The lecture took place on Monday, 20th August 2007 in Paul Hendrickse Lecture Thearte and was chaired by Professor A.O Omigbodun.

- Commissioning of the Extension Building of IAMRAT: The new extension of IAMRAT building was commissioned by Mr. Gamaliel .O. Onosode,OFR, The Chairman, Court of Governors, College of Medicine, and Pro-Chancellor and Chairman of Council, University of Ibadan, Ibadan. The event took place on Wednesday, September 5, 2007. Present at the event were members of the Court of Governors, some past provosts, deans and staff of the College of Medicine.
- 2007/2008 Inaugural Lecture: A Lecture titled "On the Trail of a Deadly Fuel" was delivered by Professor A. Sowunmi of the Department of Pharmacology and Therapeutics, on behalf of the Faculty of Basic Medical Sciences. The Vice-Chancellor, Professor Olufemi .A. Bamiro, FNSE, FAS, presided.
- The first ceremony of induction of "Achievers" and "Benefactors" into AIM Incorporation "Wall of Honour" was held on Wednesday, November 14, 2007 at Paul Hendrickse Lecture Theatre, University College Hospital, Ibadan. The plaque commemorating the ceremony was unveiled by the Chairman, Archives of Ibadan Medicine Inc., Professor Emeritus Oladipo .O. Akinkugbe, in the presence of Professor Adenike Grange, and His Royal Highness, Oba (Dr.) Samuel Odulana J.P, Odugade 1, The Olubadan of Ibadan Land.
- The new Physiotherapy Departmental Building was commissioned by Mr. Felix Ohiwerei, OFR, the former Pro-Chancellor and Chairman of Council, UNIversity of Ibadan, Ibadan. The event took plave on Wednesday, November 21, 2007.
- Foundation Laying Ceremony of New Animal House: The foundation of a new animal house was laid by the Provost, Professor A.O Omigbodun on Friday, November 23, 2007. The occasion was graced by eminent personalities and members of the 1982 Medical Class.
- University College Hospital 50th Anniversary: A week long (14th-20th November 2007) activities took place to commemorate the 50th Foundation Anniversary of the University College Hospital, Ibadan. The programme was used to raise funds for the hospital. Among dignitaries that graced the occasion were Honourable Minister of Health, Professor Adenike Grang, His Royal Highness, Oba (Dr.) Samuel Odulana J.P, Odugade 1, the Olubadan of Ibadan Land, the Pro-Chancellor of the University of Ibadan, MrGamaliel Onosode to mention a few. All the programmes were well attended by the staff of University College Hospital, College of Medicine and members of the public.
- To mark the 10-year Anniversary of the L'OREAL-UNESCO Awards for women in science (1996-2006), Professor Adeyinka G. Falusi of IAMRAT and Professor Emeritus Oladunni G. Taylor of the Department of Chemical Pathology were invited as part of the 52 laureates in celebrating a decade of commitment to the promotion of women in science. The L'OREAL-UNESCO initiative encourages the participation of women at all levels of science, and focuses on the role of women in the scientific research and developing regional fellowships for Africa.

BENEFACTORS

- Professor A. Olu Osoba, Former Professor and Head of Department of Medical Microbiology, College of Medicine. The donation of 650 volumes of Medical Journals and Books on Microbiology and Infectious Diseases to the Odekun Medical Library, College of Medicine on July 19, 2007.
- 1986 Alumni Group: The graduating class of 1986 of the College made a donation of N2,983,520.73 (two million, nine hundred and eighty-three thousand, five hundred and twenty naira and seventy-three kobo) only. The donation was meant for these projects:

a) Renovation of the Institute of Child Health
b) Improvement of the water supply and plumbing systems of the four departments in the Pre-Clinical School (Faculty of Basic Medical Sciences), University of Ibadan Main Campus.
c) Renovation of the Dean's office Faculty of Dentistry, College of Medicine.

- World Health Organization donated some textbooksnto the Professor Adetokunbo Lucas Public Health Library.
- The Chief Medical Director, University College Hospital, Ibadan on behalf of the hospital donated five units of

computers with accesories to Odeku Medical Library,College of Medicine University of Ibadan.

APPOINTMENTS

- July 17, 2007: Department of Anatomy-Professor Temitayo Sokunbi-Head for a period of fours years with effect from July 2, 2007.
- Dr. O. Owoeye, Department of Anatomy-Assistant Hall Warden Alexander Brown Hall for a Period of two tears with retrospective effect from October 2, 2007.
- Dr. E. A. Farombi-Acting Head of Department of Biochemistry, for a period of two years with effect from February 1, 2008.
- Dr. B.O.A.Adegoke-Acting Head, Department of Physiotherapy, for a period of two years with retrospective effect from August 1, 2007.
- Dr. C.O. Bekibele- Acting Head, Department of Opthalmology, for a period of eight months with retrospective effect from December 3, 2007.

LECTURES

- The 8th Archives of Medicine Incorporated "Honours Lecture": Professor Enitan Bababunmi, D.Sc., FRC Path, the Chairman GLOPAH TECH (UK) Ltd. and President, ENHICA Global Organization delivered lecture in memory of Professor Olumbe Bassir (1919-2001) on Wednesday, 5th December, 2007. The lecture was titled "How Safe is Our Food" and it was attended by dignitaries.
- Inaugural Lecture: The Dean, Faculty of Clinical Sciences, Professor O.M. Oluwatosin delivered an inaugural lecture on Thursday, 29th November, 2007 in the Faculty of Education Lecture Theatre, University of Ibadan. The topic of the lecture was "The Road to Success is always under Construction: A Re-Constructive Surgical Perspective". It was well attended by the Principal Officers the University/College.
- Faculty Lecture: Dr. Olukemi Kehinde Amodu of the Institute of Child Health delivered a Faculty Lecture titled "Old Adversary Modern Weapons" on Monday, December 3, 2007 at Paul Hendrickse Theatre. The lecture was chaired by Professor A.O. Akinkugbe, Provost College of Medicine. The lecture was well attended by members of the College and UCH communities.
- Department of Psychiatry Guest Lecture: Dr.A.Akinkunmi from the United Kingdom as part of his visit to the Department of Psychiatry, University College hospital, Ibadan delivered a lecture on Thursday, December 6, 2007 on "Forensic Psychiatry. The lecture was held at Paul Hendrickse Lecture Theatre and was well attended by members of Department and University College Hospital community.
- Late Professor Chris Ekundayo Famewo's 10th Annual Memorial Lecture: A memorial lecture titled "Medical Waste Management: Challenges in a Developing Economy" given by Dr. Olakunle Onakoya of Lagoon Hospital, Lagos, took place on Monday, January 7, 2008 at Paul Hendrickse Lecture Theatre in honour of Late Professor Chris Ekundayo Famewo, Pasr President, National Postgraduate Medical College of Nigeria, and former Don in the Department of College of Medicine. The chairman of the occasion was Professor Olufemi Bamiro, the Vice-Chancellor, University of Ibadan, represented by the Deputy Vice-Chacellor (Administrations) Professor Ayo Ogunkunle and was well attended by staff and students of the College of Medicine and members of the public.

ALUMNI MATTERS

- Year 2007 Alumni Day Programme: The Alumni Day Programme of the year 2007 took place on Monday, November 19, 2007. The event took the form of a distinguished lecture delivered by 1977 Set Alumnus, Vice Chairman/CEO, Premier Medicaid International HMO, Dr. Kayode Obembe under the chairmanship of the distinguished Senator of the Federal Republic of Nigeria, Senator (Prof.) Jubril Aminu. The lecture was titled "Community Health Insurance: The Panacea for Achieving the Millenium Development Goals in Nigeria". In attendance were notable personalities from the Nigerian Medical, Political, Education and Services circles. The event of this year's celebration took place at Paul Hendrckse Lecture Theatre in the College, within the UCH 50th Anniversary Week.
- Election of Officers: The New Executive Committee for ICOMAA was elected at the annual meeting of the association on Monday, November 19, 2007 as follows:

President
Dr.B.G.K. Ajayi
1st Vice Pesident
Professor Simbo D. Amanor-Boadu
2nd Vice President
Professor M.C. Asuzu
Secretary General
Dr. S.B. Olaleye
Asst. Secretary General
Mrs A.O. Fabowale
Financial Secretary
Dr. T. K. Hamzat
Publicity
Dr. M. Oyetunde
Treasurer
To be appointed by Executive Committee

- IMSG New Executive: Dr. A. Akinkunmi has succeeded Mr. Jimi Coker as the new President of Ibadan Medial Study Group (IMSG) UK and Dr. Wale Makanjuola as Vice President.
- ICOMAA-North America Branch: The Annual General Meeting of the ICOMAA, North America branch will be held on Friday, June 27, 2008 at Charlotte, NG, USA.

CROSSWORD PUZZLE

B	Q	I	O	U	Z	Y	O	N	W	I	Z
N	A	N	P	S	O	Z	U	A	O	T	F
N	I	D	Z	A	P	D	Z	H	B	O	Q
W	D	U	P	F	O	O	R	A	U	B	Y
O	N	C	T	B	Q	K	C	R	Q	A	Z
R	I	T	G	Z	P	I	D	A	A	Y	T
B	U	I	M	U	P	T	W	R	Z	T	H
A	M	O	F	O	V	A	N	E	O	I	A
O	B	N	H	S	T	Y	E	A	R	A	M
V	A	C	Q	W	Z	B	N	C	D	Q	E
Z	D	I	A	P	H	O	R	E	S	I	S
Q	S	O	T	Y	Z	W	C	F	M	Q	R
P	O	F	U	N	N	E	K	A	T	N	A

1. Provost of the College of Medicine, University of Ibadan.
2. Number of faculties in the College of Medicine, University of Ibadan.
3. The Capital of Zimbabwe.
4. Formal ceremony of acceptance to the medical profession.
5. Eyelid swelling
6. Excessive sweating
7. Acronym for the Paediatric clinic in UCH.
8. The name of the river that runs through London, UK.
9. The first professor of Medicine, University of Ibadan.
10. The world's largest democracy.
11. Journal of the University of Ibadan Medical students.
12. Nigeria's representative at Big Brother Africa II.

© Eromosele Charles Agbuduwe (2007)
400L, Medicine and Surgery.

DOKITA QUIZ

DOKITA **QUIZ FOR THE CHILD HEALTH EDITION**
SUBMITTED BY
EROMOSELE CHARLES AGBUDUWE

1. A patient complains of difficulty breathing through his nose and bony pain in his cheeks, near his nose. Physical examination and CT of the head revealed mass lesions involving the nose, pharynx, and sinuses. CT-guided biopsy demonstrates a non-keratinizing, squamous cell carcinoma. Which of the following disorders is associated with the same oncogenic virus that is the likely cause of this patient's cancer?

A. Adult T-cell leukemia
B. Burkitt's lymphoma
C. Cervical carcinoma
D. Hodgkin's lymphoma
E. Kaposi's sarcoma

2. A 73-year-old woman with a history of diabetes presents with left ear pain and drainage of pus from the ear canal. She has swelling and tenderness over the left mastoid bone. Which of the following microorganisms is the most likely causative agent?

A. Hemophilus influenzae
B. Klebsiella pneumoniae
C. Mucor sp.
D. Pseudomonas aeruginosa
E. Streptococcus pyogenes

3. A 42-year-old woman is pregnant and undergoes amniocentesis. The results of the amniocentesis are consistent with trisomy 21, but the woman wishes to carry the pregnancy to term. After birth, this child will be at increased risk for the development of which of the following disorders?

A. Acute lymphoblastic leukemia
B. Colon cancer
C. Tetralogy of Fallot
D. Glioblastoma multiforme
E. Hodgkin's disease

4. Which of the following neoplasms will not metastasize?

A. Squamous cell carcinoma of the bladder
B. Burkitt Lymphoma
C. Basal cell carcinoma on the nose
D. Mesothelioma
E. Lipoma

5. A PSA level is drawn from a 54-year-old man as part of a routine health evaluation. Which of the following embryonic structures gives rise to the organ being screened for carcinoma?

A. Genital tubercle
B. Processus vaginalis
C. Testis cords
D. Tunica albuginea
E. Urogenital sinus

6. A pregnant woman presents at 22 weeks gestation for a routine prenatal visit. Physical examination demonstrates ankle edema and new onset hypertension. Urinalysis reveals marked proteinuria. Development of which of the following would justify a diagnosis of eclampsia?

A. Diabetes mellitus
B. Hyperuricemia
C. Seizures
D. Systemic lupus erythematosus
E. Thrombocytopenia

7. A neonate has a 10-cm neck mass. She is diagnosed with a cystic hygroma of the neck. The physician is concerned that she may also have which of the following abnormalities?

A. Aneurysm of the ascending portion of the aortic arch
B. Aortic dissection
C. Coarctation of the aorta
D. Turner syndrome
E. Early severe aortic atherosclerosis

8. A 52-year-old male presents with epigastric pain that improves with meals. Endoscopy demonstrates a 2 cm ulcerated area located 3 cm distal to the pyloric junction. Basal acid output is within normal limits. Which of the following is most likely to have made the strongest contribution to the development of this disease?

A. Aspirin use
B. Chronic antacid use
C. Drinking alcohol
D. Helicobacter pylori infection
E. Smoking

9. A liver biopsy from a 54-year-old man shows many Mallory bodies. This finding is suggestive of which of the following diseases?

A. Alcohol abuse
B. Alpha1-antitrypsin deficiency
C. Primary biliary cirrhosis
D. Hepatitis B
E. Wilson's disease

10. Which of the following could cause macrocytic, non-megaloblastic anaemia?

A. Vitamin B12 deficiency
B. Chronic alcohol intake
C. Pregnancy
D. Aplastic anaemia
E. Gastrectomy

Answers

Adult T-cell leukemia (F)
Burkitt's lymphoma (T)
Cervical carcinoma (F)
Hodgkin's lymphoma (T)
Kaposi's sarcoma (F)

Hemophilus influenzae (F)
Klebsiella pneumoniae (F)
Mucor sp. (F)
Pseudomonas aeruginosa (T)
Streptococcus pyogenes (F)

Acute lymphoblastic leukemia (T)
Colon cancer (F)
Tetralogy of Fallot (T)
Glioblastoma multiforme (F)
Hodgkin's disease (F)

Squamous cell carcinoma of the bladder (F)
Burkitt Lymphoma (F)
Basal cell carcinoma on the nose (T)
Mesothelioma (F)
Lipoma (T)

Genital tubercle (F)
Processus vaginalis (F)
Testis cords (F)
Tunica albuginea (F)
Urogenital sinus (T)

Diabetes mellitus (F)
Hyperuricemia (F)
Seizures (T)
Systemic lupus erythematosus (F)
Thrombocytopenia (F)

7.
A. Aneurysm of the ascending portion of the aortic arch(F)
B. Aortic dissection (F)
C. Coarctation of the aorta (T)
D. Turner syndrome (T)
E. Early severe aortic atherosclerosis (F)

8.
A. Aspirin use (F)
B. Chronic antacid use (F)
C. Drinking alcohol (F)
D. Helicobacter pylori infection (T)
E. Smoking (F)

9
A. Alcohol abuse (T)
B. Alpha1-antitrypsin deficiency (F)
C. Primary biliary cirrhosis (T)
D. Hepatitis B (F)
E. Wilson's disease (T)

10.
A. Vitamin B12 deficiency (F)
B. Chronic alcohol intake (T)
C. Pregnancy (T)
D. Aplastic anaemia (T)
E. Gastrectomy (F)

STORIES

Dokita **Extras Story submitted by Mr. Onadipe for publication in the Child Health Edition of** Dokita

ONE LONG NIGHT

It was dark and the rain poured angrily.

The boy looked out of the shed; he couldn't see much, it was all blurred by the rain.

He looked at the sky; he couldn't make out any stars. He loved it when the sky had so many stars in it.

He was tired and hungry.

Drenched in rain water, he shivered violently and hugged his lean, small frame to keep warm.

The ground beside him bore the burden of the tray containing the fried yam he was supposed to be selling, it was ruined now. The tray, half filled with water. The yams were water logged. He felt his pockets and heaved a sigh of relief when he felt the small bundle ... the money was still in place. He was afraid that he had lost it in the commotion to find a place to hide from the rain.

His belly growled furiously.

He picked up one water logged piece of yam, wiped its surface with his shirt and examined it. It seemed good to eat ...

He looked around at the deserted market. It was all rain, water and mud. Most of the sheds were falling apart. It would have been fun playing in it. But the last thing he wanted to do right now was play in the rain. A nice warm bed was much more welcome.

The wind raved and ranted, he could almost here his father's voice in it threatening to kill him if he didn't come home immediately.

Rain poured into the shed. It was pointless remaining, but he couldn't leave.

He took off his shirt, squeezed hard and put back on.

The rain poured relentlessly.

The boy didn't seem worried about getting home, he was too far away from home anyway ... finding somewhere to spend the night usually wasn't a problem ... he was used to sleeping out every now and then.

He leaned against one of the poles holding up the roof of the shed ... his shivers suddenly got worse when a gust of wind blew.

He felt like crying ... he was so tired ... and so cold ... he'd have given anything for a cup of something warm ... a warm bed ...

He kept hoping the rain would stop soon.

He thought of home ... he shivered again... ***nah***... he didn't want to go home.

Suddenly anywhere was better than home. Home was sad. Home was a jobless drunkard father and a frustrated overworked mother, a pregnant 16 year old half sister, and 3 half brothers he hated in a dreary one room apartment.

Running away was so appealing ... but he wasn't going without his mother. The others could die for all he cared.

They didn't exist in his world, at least not anymore. He thought of what he would do when he finally grew up and started to make a lot of money. He would go far away with his mother, to a place where there are trees, flowers and pretty birds ... and every morning he would give a flower to his mother.

The splashing water woke him up. The rain had stopped. Someone was coming.

'Towards the shed?'

He couldn't tell. He was shivering hard, cold bursts of wind hitting hard against his lean frame. The footsteps in the water got closer. He crouched, hiding as silently as he could. His treacherous heart was beating so hard ... he was afraid it would betray him. He almost fainted from relief when sound passed and moved on.

He had to leave this place.

He emptied the contents of the tray into the gutter.

The streets were deserted.

It was pitch black... and quiet save for the wind ...

He looked left and right and started to walk briskly away from the shed.

"Hey!!!"

He froze

"You there!!! Come here!!!" the words were said in Yoruba. It was the voice of a man...harsh and terrifying.

He turned around. He couldn't see anybody.

He turned and started running ... he heard footsteps behind him quickly catching up with him.

He burst out onto the main road ... not looking left or right he continued running.

"***Crash!!!***"

The car smashed into him from the side ... knocked him over.

He screamed. The car didn't stop; it dragged him along the road a few meters and then it stopped.

It all happened so fast.

The boy laid on the muddy ground ... he spat out bloodied mud.

He tried to move, it was impossible. He couldn't feel his limbs. He couldn't breathe well. He felt faint.

He heard the doors to the car open and close.

He sighed. "*Someone to finally get him out of the mud.*" He thought with relief.

He heard two voices. Male and female.

"He's dead!" it was the female.

"We can't be too sure..."

"He's dead!" It was almost a shriek. "Let's get out of here!"

"We can't just leave him!" the man was almost shouting back.

The boy tried to talk; he couldn't find the strength to do so. He was choking on his own blood. There was mud in his mouth.

"Look! There's going to be a crowd soon, and then what? We'd be handed over to the police, or worse, burned! No way! Kola, please get into the car. Someone else will take care of this."

The man was hesitant. He looked closely at the figure lying on the road. '*It was just a kid*' he thought. '*What is a kid doing out here this late?*'

He heard the door to the car close as his wife entered.

'*Probably a ploy by robbers...*' he shivered. '*I better get the hell out of here.*'

The man got into the car, started it and drove off.

The boy couldn't see much, something was in his eyes; he couldn't tell if it was the mud or blood.

And then he heard foot steps again. He felt hands reach into his pockets roughly ... searching...

"No ... no ... no ... please ... it's for mother ... please I have to get all that money to my mother ...

The words didn't come out ...

He spat out blood and then he started coughing ... more blood.

The searching stopped ... he heard footsteps depart.

He was weak ... he was out of breath ... tears poured out

He felt so alone, so cold, so tired ... it was suddenly so quiet ... he could hear the wind as if in a shell placed over his ears ... then he heard music ... far away ... slow and mournful.

He couldn't think clearly anymore, he didn't care anymore, not about the mud, not about rain, not about the cold or the money ... he just wanted to sleep ...

It was supposed to be his birthday

Those were his last thoughts ... his birthday.

He was going to be ten ... today.

It was going to be special.

He was going to get a birthday gift for the first time ... from his mother.

"*...far away, to a place where there are trees, flowers and pretty birds...*"

He closed his eyes.

"*...where there are trees, flowers and pretty birds...*"

'*O God.*
I am so cold.
Please make me warm.
Please.
I would be a good boy.
Tell my mother I am fine, she shouldn't worry about me.
Tell her I would be home soon, with a lot of money.
And then, she wouldn't have to cry any more... ...'

Road traffic accidents, domestic violence, disease, malnutrition ... name it.

Our Children die every day.

Our world can be a better place, a safer place for the kids ... our kids.

Help make the world a better place. Help secure our future.

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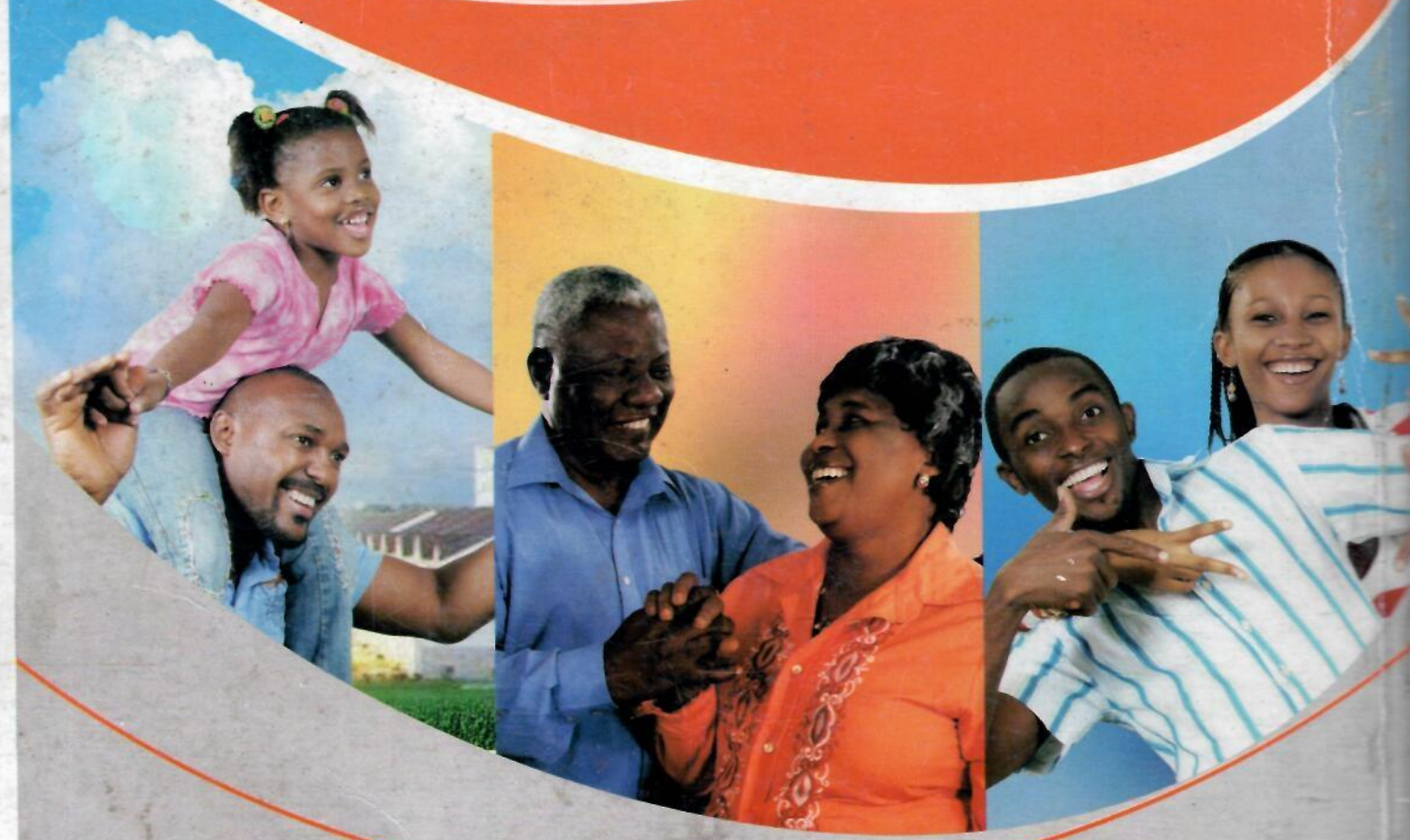

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