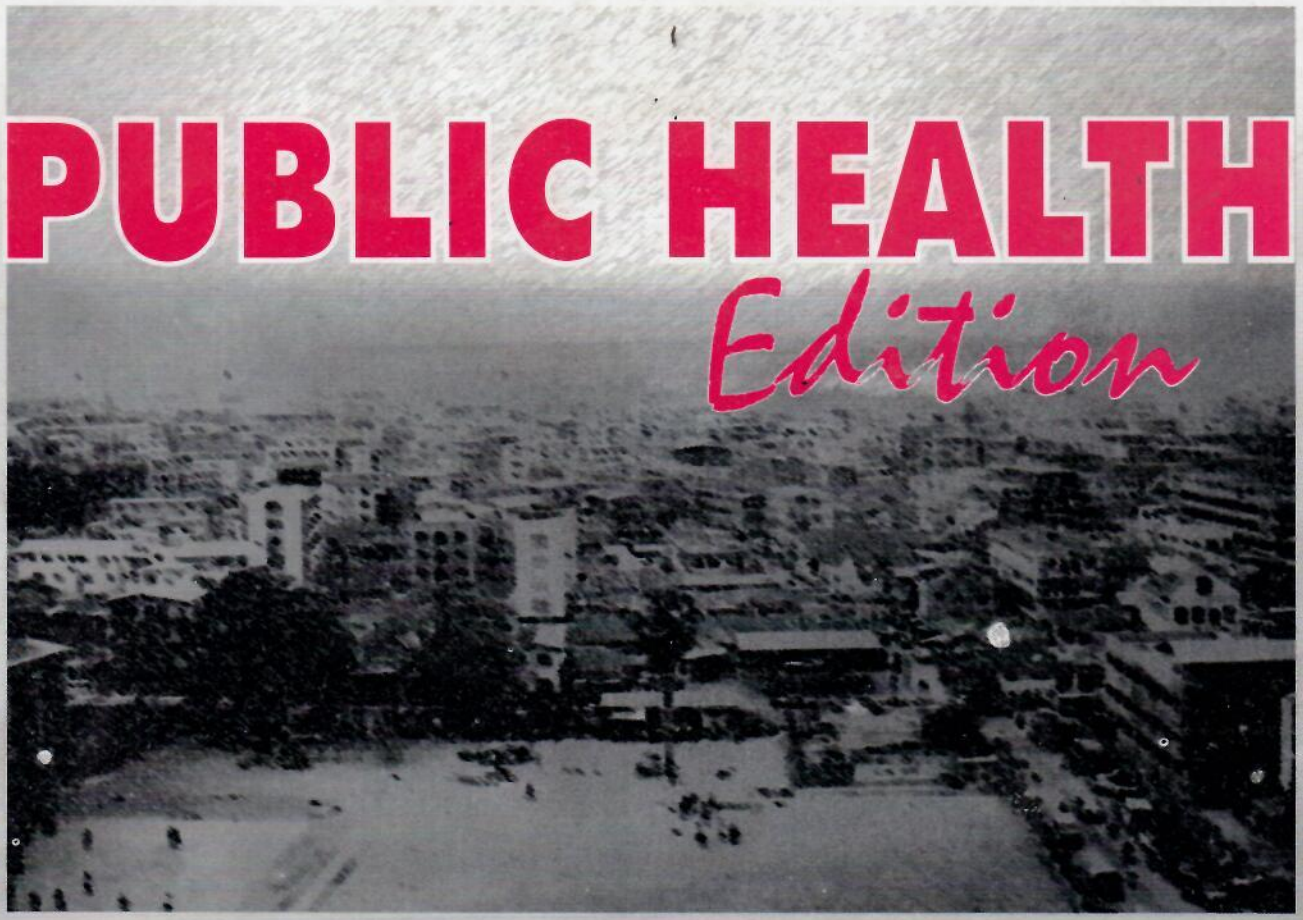
ISSN 00460508

DOKITA

1960

VOLUME 30 NUMBER 1

MARCH 2005



PUBLIC HEALTH Edition

Highlights

- Organization of Community Health Services in Nigeria
- Environment and the Emerging Health Risks
- Issues in Reproductive and Child Health in Nigeria
- Living Condition and Prevalence of Intestinal Parasites among Children in Ibadan Metropolis
- Disaster and Refugee Management in Nigeria
- Hypertension in Nigeria

JOURNAL OF THE UNIVERSITY OF IBADAN MEDICAL STUDENTS' ASSOCIATION

DOKITA

EMERGENCY MEDICINE EDITION

Volume 29 Number 1 May 2003

A MEDICAL JOURNAL PUBLISHED AT THE UNIVERSITY COLLEGE HOSPITAL, BY THE

DOKITA EDITORIAL BOARD

GENERAL INFORMATION

DOKITA is a WHO-recognized medical journal published by the medical students of the University College Hospital, Ibadan under the auspices of the **DOKITA** Editorial Board, which is an autonomous organization, composed of bona-fide medical students of the University of Ibadan. **DOKITA** provides a medium for publication of scientific papers written primarily by and for medical students and solicited manuscripts on specific subjects from experts.

Original articles, reviews, case reports and other articles on any subject of medical interest are invited. Manuscripts and other communications should be sent to the Editor-in-Chief, **DOKITA** Editorial Board, Alexander Brown Hall, University College Hospital, Ibadan. Articles are accepted with the understanding that they are offered to this journal only and that articles and reproductions can only be made by permission of the editorial board unless authors state before publication, that they reserve the right to themselves.

Reprints can be purchased at reduced prices if authors would indicate their requirements- a minimum of fifty reprints at the same time of submission of the manuscripts.

MANUSCRIPTS REQUIREMENTS

The requirements for submission of articles to **DOKITA** is in accordance with the "Uniform requirements for manuscripts submitted to biomedical journals" (the Vancouver style) as revised and published by the International Committee of Medical and Journal Editors in the British Medical Journal (BMJ 1991: 302:338-41)

All papers should be submitted in duplicate original type script (no carbon copies). Double spacing with ample margins is desired throughout the text except for quotations.

The manuscripts should have the following components each of which should begin on a new page in the following sequence:

1. Title page with the full title of the paper and the author's full names and highest academic degrees and departmental institutional affiliations.
2. Running title of no more than 40 characters (letters and spaces included) with the name and address of the author responsible for the correspondence about the manuscript.
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
6. Reference
7. Tables
8. Legends for illustrations

REFERENCES

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).

(a) For standard journal articles: author's name(s) and initials, title of article, name of journal, year of publication, volume in Arabic numerals (with number n 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.

(b) For books: author's name and initials, full title, edition, place of publication, publishers and year of publication.
e.g. Adetuyibi A. Companion to Clinical Medicine in the Tropics. London: Macmillan. 1980.
If the author(s) is/are (an) editor(s), this should be indicated after the name(s) and initials.

TABLES AND ILLUSTRATIONS

Tables should be numbered consecutively in Roman numerals in the order of their first citation in the text and should each have a brief title. they should be referred to in the text but set out on double spaced sheet separate from the text. Explanatory notes should be placed in the footnote. not in the heading. Do not use internal horizontal and vertical rules. Ensure that each table is cited in the text

Illustrations should be numbered consecutively in Arabic numerals and should be submitted in duplicates. They should be good quality unmounted glossy photographs and positive prints, not x-rays or lanternslides. Drawings should be black Indian ink in white paper and in a finished state suitable for reproduction and legible enough for suitable reduction; authors should carefully examine recent issues to ascertain the width of columns. Coloured illustrations will be published out only at the expense of the author(s).

Legend for illustration should be typed double-spaced on a separate page with Arabic numerals corresponding to the illustrations. A key explaining any symbols used in the illustrations must be included in the legend. Credit must be given for any previously published illustration.

SUBMISSION

Manuscripts should be submitted in duplicate in a heavy paper envelope, accompanied by a covering letter signed by all co-authors. addressed to:

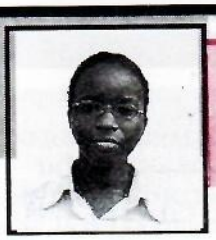
THE EDITOR-IN-CHIEF,
DOKITA EDITORIAL BOARD,
Alexander Brown Hall,
University College Hospital,
Ibadan.

Manuscripts may be accompanied by a $3^1/_2$ diskettes of the text if available(Preferably Microsoft Word). Not more than 5 Pages please

The article can be sent by e- mail to: dokita@37.com

THE EDITORIAL BOARD

CONTENT



EDITORIAL

It is not the Critic who counts...
The credit belongs to the man who is actually in the arena, whose face is marred by dust and sweat and blood, who strives valiantly, who errs and comes up short again and again because there is no effort without error or shortcoming, but who knows the great enthusiasms, the great devotions, who spends himself for a worthy cause; who, at least he fails while daring greatly, so that his place will never be with those cold and timid souls who knew neither victory nor defeat.

Theodore Roosevelt, Paris, 1910.

The Emergency Medicine Edition is finally out. The Editorial Board has over the past year gone through its fair share of experiences (we prefer to call them challenges).
The New board year started with the organisation of the 2nd Edition of the Prof. O.O Akinkugbe Biennial Quiz Competition. It was quite an experience and was indeed a meeting of the best minds in Medical Schools across the country. It is hoped that subsequent Editions will be held in other medical school to increase participation by a larger number of medical schools and increase diversity.

One major factor that has kept the board on its toes is the issue of funds. We have in recent times experienced a 'donor fatigue' in response to our appeal for funds from individuals and corporate bodies. This response has been attributed to the dwindling fortunes in every aspect of our economy. To counteract this unfortunate syndrome, the board has come up with the **DOKITA** Editorial board endowment fund – a scheme intended to raise Two Million Naira (from a thousand units of Two thousand naira). It is hoped that the interest generated from the deposit of this sum in a reputable bank will be sufficient to run the board and put an end to our letters soliciting for funds.

The Words " **Emergency Medicine**" bring to mind a scene of a patient being rushed in a fully equipped ambulance to the nearest hospital with all the necessary medical equipment and of course medical personnel available to save lives. This scenario is sadly not the case in Nigeria as can be attested to by anyone who has come upon an accident site on any of our expressways or who has rushed a relative to the hospital only to be told that there are no facilities available to manage the patient. We hope and pray for better times.

The Emergency Medicine Edition is a cornucopia of articles written by experts on various emergencies in their fields. Such articles are Compartment Syndrome, Acute glaucoma, Sickle Cell Crises, Diarrhoeal diseases, etc. The Student papers also reflect common emergency presentations seen in the emergency room. The winning essay for the annual Prof J.A Adeleye Essay Competition and the Best Igbo-Ora. Project are as usual included in this journal. On the lighter side, the DOKITA Extras Section includes various views on life as a house officer and as a Casualty officer and poems by medical students. As usual excellence in every sphere is emphasized.

As usual the Editorial Board in its tradition of excellence has set out to produce a journal of the highest standards. We have faced different challenges but they have only made us more determined to continue the work started forty-three years ago. Like our predecessors we shall pass on our torch still brightly burning.

The Editorial Board would certainly not have achieved so much this year on its own.
We give thanks to Our God and Father who has given us life and the strength and grace to give our best even when we did not think we were capable of doing so.
We would also like to thank Prof. O.O Akinkugbe for the fatherly advice, support and help he gives us whenever we ask for it. We are indeed grateful.
To Prof A.O Omigbodun and all our Editorial Consultants, thank you for giving us your time and helping us out whenever we needed it. We look forward to many more years together.
To Prof Jaiye Thomas, we appreciate your contributions to the board over the years and we are indebted to you for your role in lifting the board to the heights we have attained. Thank you very much.
I would also like to appreciate Prof A.O Oyegunle who wrote the foreword to this year's journal.
Finally, to the team who worked hard to make this possible, the members of the **DOKITA** Editorial Board, you have spent yourselves for a worthy cause, well done.

Olaoluwatomi Lamikanra
Editor- in Chief
May 2003

FOREWORD

EMERGENCY MEDICINE has different meanings to different categories of medical practitioners and specialists.

To the "surgeon" it may mean acute abdomen, ruptured viscus, fractured long bone, fractured skull, chest trauma, compartment syndrome, severe accidental or traumatic injury; to the "obstetrician and gynaecologist", it could be ruptured ectopic pregnancy, ruptured uterus or foetal distress. The "physician" would be concerned about bronchial asthma, diabetic coma, myocardial infarction, etc. An anesthetist would consider a patient with severe tetanus, full stomach and accidental or suicidal poisoning as serious emergency.

The common denominator in all types of emergency medicine is the need for initial active resuscitation and the maintenance of **Airway, Breathing and Circulation;** followed by specific attention to the aetiology of the emergency and the utilization of the management skills required.

The effort of the Editorial Board in putting the articles together for this package is commendable and would stimulate further thoughts on this important and involving topic.

PROFESSOR A. O. OYEGUNLE
Department of Anaesthesia,
Obafemi Awolowo College of Health Sciences, Sagamu,
Olabisi Onabanjo University,
Ogun State.

SICKLE CELL CRISES

AKEN'OVA, Y.

Professor Yetunde Aken'Ova is a of Professor Haematology at the College of Medicine, University of Ibadan and a Consultant Heamatologist to the University College Hospital, Ibadan

INTRODUCTION

The sickle cell disease (SCD) is an inherited haemoglobin disorder arising from the inheritance of the S gene alone or where the S gene exists in combination with another variant haemoglobin. It is the most common inherited disorder world wide by virtue of the survival advantage the heterozygous carriers of the S gene have, especially in *Plasmodium falciparum* malaria endemic areas.

Sickle cell disease is one disease that has a worldwide distribution. Its existence is well known especially amongst the blacks. It is also associated with malarial endemic areas. It has been well studied at the molecular level yet no cure has been found. However, knowledge of the pathophysiology is incomplete.

The sickle cell disorder includes SS, SC, SG, Sß thal, SD, SE. In fact about 400 other variants have been described. Although patients with sickle cell disease are found in many continents, about 89% of the 231,000 HbS born annually are born in Africa. Some West African indigenes with SC, or homozygous CC may be carriers with a normal haematological profile and a normal life expectancy. They do not have symptoms like SCD except for a rare haematuria caused by papillary necrosis.

Sickle cell crisis is used to describe the recurrent paroxysm of illness in sickle cell anaemia. The hallmark of sickle cell anaemia is the recurrent and painful vascular occlusive crisis interspersed with periods of stable steady state with no symptoms or signs other than that due to chronic haemolytic anaemia. The chief burden of the disease lies in Africa where the minimal care for patients is lacking.

PATHOPHYSIOLOGY

The S heaemoglobin has the ability to form polymers in the deoxy state. The polymers can cause cellular injury responsible for the clinical manifestations of Sickle Cell Disease. The vaso-occlusive features of the sickle cell disease are unique. By occluding small blood vessels and sometimes, large ones, sickle cells cause vascular injury. The severity differs from patient to patient. The complexity of the process of vaso-occlusion provides many possibilities for therapeutic intervention.

Vaso-occlusion is initiated and sustained by interactions among sickle cells, endothelial cells and constituents of plasma.

Vaso-occlusion is responsible for most of the severe complications of sickle cell disease and this can occur wherever blood flows. However, general principle of management include psychological stability, good nutrition, daily folic acid, antimalarial prophylaxis and healthy life style.

Neonatal screening can identify infants with sickle cell disease and introduce the parents to comprehensive care programmes. Early in life usually when the risk of infection is highest, counselling parents as regards importance of immunization, antimalarial prophyxlaxis, detection of a rapidly enlarging spleen in children, increasing pallor and prompt institution of therapy can be life saving.

CRISES IN SICKLE-CELL DISEASE

VASO-OCCLUSIVE CRISIS

A pathophysiologic feature of Sickle Cell disease is the episodic occurrence of vaso-occlusive events that precipitate in painful episodes. Vaso-occlusion of small and large vessels in sickle cell disease accounts for much of its morbidity and mortality.

A major contributor to vaso-occlusion in sickle cell is the increased tendency of sickle cell to adhere to vascular endothelium. This adherence of sickle cells to vascular endothelium will impede blood flow and thereby increase capillary transit time and increased cell adherence can initiate and propagate vaso-occlusion[1]. Factors that can activate endothelial cells and thereby enhance endothelial adhesivity of sickle red cells and trigger vaso-occlusive episodes are: Tissue necrotic factor (TNF), Interferon alpha, Interleukin 1ß, Vascular endothelial growth factor (VEGF), thrombin, histamine, the effects of hypoxia and reperfusion. The extent of sickle cell adhesitivity correlates with vaso-occlusive severity. Pain in sickle cell can be acute, chronic

intermittent, recurrent or persistent.

Sickle cell pain is the result of vaso-occlusion that leads to local hypoxia, ischaemia and tissue damage. The tissue damage mediators can activate or sensitize afferent nerve fibres and posterior horn cells of the spinal cord by different mechanisms that affect pain perception. This could be biochemical, neurological or electrochemical events.

Precipitating or predisposing factors include cold weather, infection, metabolic acidosis, menstruation, pregnancy and post partum, physical and emotional stress.

Vaso-occlusive crises are unpredictable in location and timing. They are repetitive or intermittent. Pain can be acute or chronic. Vaso occlusive painful crisis is associated with acute pain whereas those of leg ulcers, vascular necrosis of humeral or femoral head and bone infarcts are chronic.

Acute pain is associated with anxiety, fear, helplessness, sleep deprivation and fear of death.

Patients with sickle cell disease who suffer from both acute and chronic pain syndromes are seriously disadvantaged.

Acute pain in sickle cell disease is encountered in bone infarction, acute chest syndrome, hepatic crisis, bowel infarction and necrosis, priapism, cholecystitis, hand-foot syndrome, hepatic and splenic sequestration.

Management of vaso occlussive crises

Patients benefit from either a single drug or combination of various drugs including opioid analgesics, NSAIDs and adjuvant drugs.

Physicians could combine such drugs that would alleviate the patient's pain. Excessive dosage of some of the drugs can cause

(1) Hepatic necrosis and even death.

(2) Addiction should be guarded against in the patients.

Routes of drugs can be:

Oral e.g NSAID, Opioids

Controlled-release e.g Indomethacin

Parenteral - I.V, I M, subcutaneuos e.g. Opioids

Rectal - Indomethacin

Patient's pain should be classified into mild, moderate or severe and appropriate analgesics should be given. Dosage can be adjusted with improvement in pain symptoms.

ACUTE SPLENIC/HEPATIC SEQUESTRATION.

Patients with Sickle cell anaemia whose spleens have not undergone multiple infarction and fibrosis (auto-splenectomy) can experience sudden intrasplenic pooling of large amounts of blood. This can also occur in the liver or any organ. In children this can begin to occur after 6 months of age. Splenic sequestration occurs more commonly in children who still have a spleen. Hepatic sequestration occurs more in the adults because by adulthood most SCA patients would have undergone auto-splenectomy. This crisis is usually associated with viral infection.

- The spleen or liver become massively enlarged; filling the abdomen and can extend into the pelvis.
- The haemoglobin level also drops to very low levels, precipitating hypovolaemia, shock and even death.

Acute sequestration can also occur in the mesenteric vessel, or in the pulmonary tissue.

CLINICAL SIGNS

Sudden weaknesses, pallor, breathlessness, dizzy spells, abdominal fullness. Minor episodes can resolve spontaneously. Sequestration crisis can occur in older patients with SC, Sß thal.

Treatment of sequestration crisis

- Prompt correction of hypovolaemia with plasma expanders
- Give whole blood transfusion
- The sequestration crisis may re-occur however, the role of splenectomy is doubtful.

APLASTIC CRISES

This can occur after parvo-virus type ß 19 infection. It can also occur in siblings. Patients can present with increased fatigue and dyspnoea at rest or mild exertion or heart failure with reticulocytopaenia. The Hb may fall to 2.0 – 5mg per dl.

Aplastic crises may occur in mini epidemics and terminates spontaneously after 5 to 10 days. During the convalescent phase, hyper haemolysis may be assumed because of severe anaemia and high reticulocytosis. Treatment is symptomatic. Transfusion with packed cells should be given promptly. The Haemoglobin of the blood being transfused should be A:

HYPER-HAEMOLYTIC CRISIS

The recurrent episodes of anaemia resulting from haemolysis in sickle cell disease is referred to as haemolytic crisis. This is accompanied with jaundice, increased serum bilirubin and urobilinogen in the urine. However, other episodes when there is an exaggeration of the haemolysis is referred to as hyperhaemolytic crisis.

The episode is characterised by a drop in the level of haemoglobin while the reticulocyte count increases during the active phase of the hyperhaemolysis. There is also a rise in the level of total bilirubin and serum lactate dehydrogenase (LDH). The level of conjugated bilirubin is high. Hyperhaemolytic crises is a term used to describe a life-threatening situation with severe anaemia following mas-

sive destruction of red blood cells and the patient will have reticulocytosis. This can be triggered by Malaria, G6PD deficiency or any infection and may occur with or independent of painful crisis.

Patient should be watched closely. If the haemoglobin drop is acute, packed red cell transfusion should be given.

ACUTE CHEST SYNDROME

This is thought to be as a result of an increase in the haematocrit. Acute chest syndrome may encompass pneumonia, fat embolism, and thromboembolism. However, a major cause of acute chest syndrome is altered circulation of the blood through the pulmonary circuit leading to altered circulation, altered ventilation, perfusion and hypoxia. It is potentially fatal disease. The clinical signs include pain, dyspnoea, hypoxia and fever[4]. Pain is usually pleuritic and the diaphragm may be involved thereby causing abdominal pain. The signs may vary from mild to moderate to severe and can be life-threatening. The syndrome may be caused by pulmonary oedema, rib or sternal infarction, pneumonia or pulmonary infarcts due to in-situ sickling, pulmonary fat embolism or pulmonary embolism[5,6].

It is a common cause of death and hospitalization and is closely associated with painful crisis in adults.

Predisposing factors include high steady state haematocrit, low foetal haemogblobin and high steady state leucocytosis.

Investigations should include

- Chest X-ray
- Sputum and Blood Examination for microscopy, culture and sensitivity.
- Monitoring of arterial blood gases and haemoglobin level.
- Ventilation and perfusion scans
- Sputum and Bronchial washing analysis

Management Includes

- Broad spectrum antibiotics
- Hyperbaric oxygen – given intranasally
- Exchange transfusion is helpful in severe cases
- Anticoagulation has been helpful
- Caution should be exercised in giving opioids to hypoxic patients.
- Respiratory rate should be monitored

It is mandatory to establish baseline blood gases and pulmonary function tests in all patients. These would help in evaluating patients with acute onset of pulmonary signs and symptoms. Usually the haemoglobin level decreases on days 5 to 6 and maximum on days 6 to 7.

Other serious clinical conditions of serious concern include:

PRIAPISM

This is a painful penile erection. The pathophysiology is not well understood. However therapeutic approaches are controversial while medical or surgical approaches fail most times.

It is often thought that acidosis resulting from dehydration and hypoventilation during sleep may be a precipitating cause.

Bicorporal priapism involving both corpora-cavernosa is often seen in children. It is characterised by short repetitive reversible painful episodes referred to as stuttering priapism. The condition may reverse after a few hours. The prognosis is good for stuttering priapism.[6]

Tricorporal Priapism involves both corpora-cavernosa and the corpus spongiosum. This occurs more in the adult patients. The erection can last up to several days or weeks and this may lead to complete or partial impotence. The prognosis is worse when there is surgical intervention. Some other workers have concluded that priapism in adult males is a marker of severe disease.[8,9,10]

Management of Priapism includes

- Potent analgesia
- Sedatives
- Prevention of precipitating factors
- Rehydration
- Catheterization of urinary bladder
- Exchange transfusion has been helpful
- Hypertransfusion regime
- Stilbestrol[10]

If the above should fail, then surgery might have to be undertaken.

PELVIC CRISES

Some authors have described pelvic crisis. This is due to vaso-occlusive crises of some organs in the pelvis.

Management is:

- Analgesia
- Sedation
- Hydration

STROKE

This is a neurological deficit of sudden onset with focal rather than neurological dysfunction. Symptoms may last more than 24 hours or could result in death. This could result from thrombi, embolism, haemorrhage or could be iatrogenic.

Transient ischaemic attack can occur in the sickle cell anaemia patient.[10,11]

Investigations should include, computerized tomography(CT), magnetic resonance imaging (MRI).

Patients benefit from exchange transfusion.[11] long-term

hypertransfusion regime, hydroxyurea therapy and anticoagulation.

The risk of discontinuation of hypertransfusion is that about 50% of the patients will re-stroke even more than nine years after hyper transfusion is discontinued.[12]

CONCLUSION

Crises are important pathophysiologic features of sickle cell disease. They comprise of vaso-occlusive crises (a hallmark of the painful episodes), hyperhaemolytic crises, aplastic crises and acute hepatic and or splenic sequestration crises. These are chronic haemolytic state of the disease. Crises are life-threatening events and if not managed promptly can result in high morbidity and are responsible for the high mortality in these groups of patients.

Other crises of concern are acute chest syndrome(which is the second most common cause of death in SCA), priapism and stroke.

REFERENCES

1. Sickle Cell Pain – Samin K. Ballas , MD. IASP Press. Seattle. Sickle Cell Pain Syndromes Part II Chaper 3; pp – 43-50
2. Haematology 2000 – The American Society of Haematology Education Program Book
New views of sickle cell disease pathologysiology and treatment.
3. Wendell F. Rosse Mohandas Narla Lawrenece D. Petz, and Martin H. Steainberg. Page 2-17.
Sickle cell pain – Samin K. Ballas. IASP Press 1998.
4. Acute Painful Episodes, Chapter 4; pp 51-89
5. Athanason NA, Hatton C Mc Gee JOD, weatherall DJ. Vascular occlusion and infarction in sickle cell crisis and sickle chest syndrome. J. Clin pathol 1985; 38:659-664.
6. Barrett-Connor E. Pneumonia and Pulmonary infarction in sickle cell anaemia JAMA 1973); 224: 997-1000
7. Vickinsky E willians R, Das M. Pulmonary fat embolism; a destruct cause of severe acute chest syndrome in sickle cell anaemia. Blood 1994; 84:107-3112
8. Powers Dr, Johnson CS. Priapism. In sickle cell disease Hematoloncol Clin North Am 1996; 10: 1353-1372.
9. Emond Am, Holman R, Hayes RJ-Priapism and impotence in homozygous sickle cell disease. Arch Intern med 1980; 140:1434-1437.
10. Serjeant GR Stilbestrol and stuttering priapism in homozygous sickle cell disease. Lancet 1985; pp: 1274-1276
11. Asher S.W. Multiple Cranial nerve Neuropathies trigeminal neuralgia and vascular headaches in sickle cell disease a possible common mechanism. Neurology 1980; 30: 210-211.
12. Sydenstricker V.P further observations on sickle cell anaemia JAMA 1924 b; 83:12-15.

PREVENTION OF LIFE-THREATENING BLOOD TRANSFUSION REACTIO

ABJAH, U. M. A.

Dr. Abjah, FWACP, at the time of writing this article was a Senior Registrar, in the Department of Haematology, Univer College Hospital, Ibadan.

INTRODUCTION:

Blood transfusion in clinical practice is a life saving procedure. Blood and blood products are administered in order to improve the oxygen uptake and to arrest bleeding in patients with anaemia and bleeding disorders respectively. This procedure may be punctuated by adverse reactions, which may be life threatening.

Various transfusion reactions have been described in the literature, which can be classified broadly as haemolytic and non-haemolytic. These reactions may be immediate or delayed. An immediate blood transfusion reaction is defined as symptoms and signs that occurred within 24 hours of blood and blood products transfusion.

Blood transfusion reactions are under-reported, but published data indicate an incidence ranging from 1 in 1,000 to 1 in 20,000 transfusions. Successes attained in transfusion medicine are greatly influenced by careful donor selection. The essence is to protect both the donor and recipient from any ill effects. However, with the few numbers of recruited voluntary, non-renumerated donors: commercial, paid and walk-in donors have unfortunately assumed a greater significance in our environment.

AETIOLOGY

Causes of blood transfusion reactions can be broadly classified under:

(a) Immune mediated
(b) Anaphylactic (allergic) responses
(c) Use of mismatched donor blood and
(d) Transfusion of damaged blood during heat treatment or long storage.

Specific causes of Blood Transfusion reactions include

- Clerical errors (and not serological errors) which are the most common causes of incompatible transfusion reactions.
- Mislabeling of samples of blood particularly when dealing with a large number of patients.
- In emergency situations, errors in cross-matching could occur, particularly if a full crossmatch is not done.
- Multiply transfused patients – where clinically nificant antibodies may be stimulated in an ana tic response by a recent transfusion, and such not be detected unless fresh samples are taken.
- The concept of Blood group 'O' as a universal nor could be dangerous if the donor has h titre(haemolysins) anti – A and/or anti-B and transfused to recipient groups A, B or AB.

HAEMOLYTIC TRANSFUSION REACTIONS

There is increased destruction of red cells as a res interaction between the transfused red cells with the cipient blood group antibodies. This is usually caused ABO incompatibility,Rh (D) incompatibility and o antibodies e.g Lewis, kell, kidd etc.

Haemolytic antibodies are generally Ig M or rarel G and are complement binding. The binding of such tibodies to antigen on the red cell surface activates the complement cascade, and this leads to severe intra cular haemolysis with a fatality rate of up to 10%. intravascular haemolytic event will cause liberation of and C5a anaphylatoxins during complement activa which in turn causes smooth muscle contraction, pla aggregation, increased capillary permeability and rele of vasoactive amines and hydrolases from mast cell granulocytes respectively.

Thromboplastin-like substances are also liberated wh will activate the coagulation cascade and lead to Disse nated Intravascular Coagulation. The bleeding diath and increased haemolysis (involving both donor and cipient RBCs) will further exacerbate the problem.

NON-HEMOLYTIC TRANSFUSION REACTIO

Febrile reactions, due frequently to sensitization to wh cell antigens and rarely to platelet antigens, with urtic are the most common type of immunological reactio blood transfusion. Of recent, it has been demonstra that, antibodies may be directed against HLA antigen against granulocytes and platelet specific antigens, co with inflammatory cytokines that are released from taminated leukocytes. Storage time of platelet, red and contaminating WBC in platelet concentrate

strong relationship to the development of NHTR

TRANSFUSION RELATED ACUTE LUNG INJURY (TRALI)

Acute lung injury following transfusion is the second most serious blood transfusion reaction and the FDA has classified it as the 3rd most common cause of transfusion associated mortality.

Its pathophysiology remains unclear, but it is believed to be caused by leucoagglutinins or by other complement activating antibodies (comprising HLA antibodies, granulocytic (lymphocytotoxic) antibodies and biologically active mediators in stored blood. These antibodies mediate granulocyte aggregation and activation, resulting in microvascular pulmonary injury.

Characteristic pathologic changes include intra-alveolar oedema, hyaline membrane formation, alveolar cell hypertrophy and scanty interstitial inflammation.

CLINICAL FEATURES

The signs and symptoms of blood transfusion reactions vary. In a severe haemolytic reaction, typically within less than an hour of start of transfusion, the patient complains of heat in the vein, throbbing in the head, flushing of the face, chest tightness, nausea and lumbar pain. These symptoms are usually accompanied by tachycardia, profound hypotension and cardiovascular collapse, followed by rigors and pyrexia .

In heavily sedated or anaesthetized patient, these initial symptoms may be modified or masked.

Allergic reactions include hives, urticaria, pruritus, erythema, bronchospasm and hypotension.

In severe allergic reactions (anaphylaxis), the patient will have a fever, red or inflamed skin rash, and itching. This may progress to shock and collapse. Other signs may include difficulty in breathing, cough, cyanosis and shivering due to loss of body heat.

In TRALI, it is characterized by dyspnoea, hypotension, cyanosis, cough, fever and lung oedema after an hour or so of commencing transfusion. These symptoms may develop between the 4th and 6th hours of transfusion, may be unrecognized because of overlap of symptoms.

DIAGNOSIS

Reactions to blood transfusion are diagnosed by the presence of symptoms and signs during or shortly or long after transfusion.

- Complete blood count-will indicate blood abnormalities such as further drop in haematocrit or extremely low haemoglobin.
- Urinalysis will reveal haemoglobin in the urine.
- Blood cultures may reveal the offending organism if infection is responsible
- Chest x-ray will reveal bilateral pulmonary infiltrates without vascular congestion and normal cardiac silhouette (in case of TRALI)
- Bilirubinaemia and haemoglobinaemia are diagnostic of haemolytic reaction.

EMERGENCY CARE OF THE PATIENT WITH BLOOD TRANSFUSION REACTION

- The nurse is expected to notify the physician in-charge at once.
- Stop the transfusion
- Frequent evaluation of the patient i.e : vital signs (temperature, pulse rate, blood pressure, respiratory rate and urinary output)

If Haemolytic reaction is suspected:

- Intravenous fluids are administered to maintain blood pressure and kidney function.
- Hydrocortisone.
- Identification of the patient and units transfused should be checked against the appropriate documentation.
- Blood samples must be taken for the following investigation:
- Regrouping
- Re-crossmatch: major cross match, minor cross match
- Direct Antiglobulin test
- Pre-transfusion samples should be tested in parallel.
- Bacteriological test e.g. culture
- Urinalysis.

If reaction is non-haemolytic:

- The transfusion rate is slowed down and an antipyretic is administered.

PREVENTIVE MEASURES

- Donor's blood and recipient blood should be cross matched prior to the transfusion to ensure compatibility (major cross match.).
- Blood group 'O' donors must be screened for the presence of high titre haemolysins and labeled accordingly, hence, it should not be used for blood groups A, B and AB patients.
- Caution should be taken during handling of samples to avoid mislabeling. Antibody screening/Antibody identification should be done in donor blood and recipient blood, particularly in multiply transfused patients (minor crossmatch) .

REFERENCES

1. Njoku O. S. Modern Blood Transfusion practice. The Nigerian Family Practice. 1993: 3 (1) 61-68.

2. Nel. T.J. Haemolytic ,Transfusion Reachioins XVth meeting of the International Society of Heamatology. African and European division. 1999. 36-37

3. Filed S. P. Non Heamolytic Transfusion reactions. XVth meeting of the International Society of Heamatology. African and European division. 1999. 35-36

4. Politis C. Advance in Blood Transfusion Medicine 16th Congress of the International society of Haematology. European –.African division 2002 . 66-72

5. Blumberg N. Bove J.R Un-Cross-matched blood for Emergency Transfusion. JAMA. 1978. 240 (19). 2057 – 2059.

6. Jeter E.K., Spivey M.A. Non infectious complication of blood transfusion. Transfusion medicine II. Haematology / oncology clinic of North America. 1995 9.1. 187 – 204

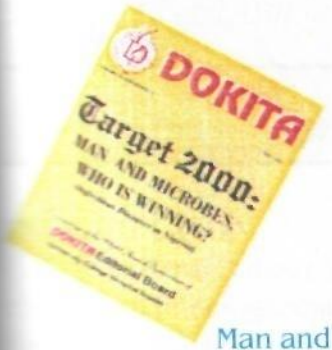
7. Colovic MC, Colovic R, Masirevic V, Barisic G. acute lung inju related to blood transfusion Acta Chirurgica Lugoslavica 200 47(3):87 – 90.

8. Lenahan SE, Domen RE, Silliman CC, Kingsley CP, Romano F Transfusion – related acute lung injury secondary to biological active mediators. Arch of path and laboratory medicine 2001. 125(523-6.

9. Rizk A, Gorson K.C, Kenney L, Weinstein R. Transfusion relat acute lung injury after the infusion of IVIG. Transfusion. 2001 (2). 264 – 8.

10. Reddy V, Goverder S, smart E. The laboratory investigation Transfusion reactions. XVth meetings of the international soci of Haematology. African and European division 1999. 38 – 39

OTHER DOKITA PUBLICATIONS AVAILABLE ON SALE



Man and Microbes: Who's winning?
DOKITA Vol. 26 supplement 1

Heart and Lung Edition.
DOKITA Vol. 26 no 1

The Dancing Quill.
A Collection of Selected Speeches and Quotes of
Prof. (Emeritus) O. O. Akinkugbe.

40th Anniversary Edition.
Dokita Vol. 27 no 1

Reproductive Health Edition
Dokita Vol. 27 no 1

SUBSCRIPTION INFORMATION

DOKITA, journal of the University of Ibadan Medical Students" Association

ORDER FORM:

Please enter my subscription to **DOKITA**

I enclose.....

Name of subscriber.....

Institution.....

•Student •Health care professional (Tic option)

Organization..... Title.....

Address.....

City.....

State/Provindence.....

Country.....

Postal code/zip code.....

E-mail Address.....

Fax Number.....

Please include N100 or $5 for National and International postage respectively.

Cheques should be made out to the address below. Please all cheques should be crossed.

Subscription Rate (Circle all that apply)	Student	Professional
• USA & CANADA	$8	$12
• UNITED KINGDOME.	E6	E9
• Other European Countries	Dollar (U.S.)	Equivalent
• Within Africa	N500	N750
• Within Nigeria	N300	N500

Subscription Request should be addressed to:

The Distribution Manager
DOKITA, Editorial Board,
Alexander Brown Hall,
University College Hospital,
Ibadan,
Nigeria.

MANAGEMENT OF ACUTE RENAL FAILURE

**LAMIKANRA, O. K. **ADESOKAN, D. I. *AJIBOYE, O. B. *AKINOLA, I. T.

*At the time of writing, ** were 2nd year Clinical students and * were 1st year Clinical students at the College of Medicine, University of Ibadan, Ibadan.*

INTRODUCTION

Acute Renal Failure (ARF) may be described as the clinical conditions associated with rapid loss of renal function and steadily increasing serum creatinine with azotemia. The situation may occur with or without oliguria (<400mL/day). It often signifies acute suppression of renal function and urine flow falling within 24 hours to less than 400 ml. ARF can be caused by a variety of conditions including organic vascular obstruction, severe glomerular disease, acute tubulointerstitial nephritis, massive infection (pyelonephritis), DIC, urinary obstruction by tumors, prostatic hypertrophy or blood clots and acute tubular necrosis (ATN).[2]

EPIDEMIOLOGY.

The distinction between community- and hospital-acquired ARF is important for the differential diagnoses, treatment, and eventual outcome of patients with ARF. The annual incidence of community acquired ARF is approximately 100 cases per 1 million population and it is diagnosed in only 1% of hospital admissions at presentation. On the other hand, hospital-acquired ARF occurs in as many as 4% of hospital admissions and 20% of critical care admissions.

This increased incidence of hospital-acquired ARF is multifactorial; It may be related to an aging population with increased risks of ARF, the high prevalence of nephrotoxic exposures possible in a hospital setting, and increasing severity of illness.

Because most cases of community-acquired ARF are secondary to volume depletion, as many as 90% of cases are estimated to have a potentially reversible cause.[7] Hospital-acquired ARF often occurs in an ICU setting and is commonly the end result of multiorgan failure. This dichotomy in the aetiology of ARF explains the increased mortality rate, dialysis requirements, and rates of progression to end-stage renal failure seen in hospital-acquired ARF compared with community-acquired ARF.

It may be related to an aging population with increased risks of ARF, the high prevalence of nephrotoxic exposures possible in a hospital setting, and increasing severity of illness. Because most cases of community-acquired ARF are secondary to volume depletion, as many as 90% of cases are estimated to have a potentially reversible cause.[7] Hospital-acquired ARF often occurs in an ICU setting and is commonly the end result of multiorgan failure. This dichotomy in the aetiology of ARF explains the increased mortality rate, dialysis requirements, and rates of progression to end-stage renal failure seen in hospital-acquired ARF compared with community-acquired ARF.

Mortality rates for ARF have changed little since the advent of dialysis at 50%. This curious statistic simply reflects the changing demographics of ARF from community- to hospital-acquired settings. Currently, the mortality rate for hospital-acquired ARF is reported to be as high as 70% and is directly correlated to the severity of the patient's other disease processes[7]. The mortality rate among patients presenting to the Emergency Department with prerenal ARF may be as low as 7%. With the advent of dialysis, the most common causes of death associated with ARF are sepsis, cardiac failure, and pulmonary failure.[7] Interestingly, patients who are older than 80 years with ARF have mortality rates similar to younger adult patients. Paediatric patients with ARF represent a different set of aetiologies and have mortality rates averaging 25%.[7]

ARF is not a benign disease. In a recent study, a 31% mortality rate was noted in patients with ARF not requiring dialysis, compared with a mortality rate of only 8% in matched patients without ARF. Even after adjusting for co-morbidity, the odd ratios for dying of ARF was 4.9 compared to patients without ARF. Mortality rates are generally lower for nonoliguric (>400 mL/day) ARF than for oliguric (<400 mL/day) ARF, reflecting the fact that nonoliguric ARF is usually caused by drug-induced nephrotoxicity and interstitial nephritis, which have few other systemic complications. Males and females are affected equally and the patient's age has significant implications for the differential diagnoses of ARF.

Between January 1990 and December 1998 a total

of 1,716 cases of ARF was treated at Sindh Institute of Urology and Transplantation (SIUT) Dow Medical College, Karachi-Pakistan. (Criteria of defining ARF was a rise in Creatinine of >2mg% and normal size kidneys on ultrasound in a person without any previous systemic illness). Of these,

746 43.4% were due to medical causes;
485 28.2% were due to surgical causes;
298 17.3% were due to obstetrical causes;
169 9.8% miscellaneous and unknown;
35 4.58% snake bite.

From November 1995 through June 1996, acute anuric renal failure was diagnosed in Haiti in 86 children, most (85%) of whom were aged less than or equal to 5 years. An accompanying study entitled, "Epidemic of Pediatric Deaths from ARF caused by Diethylene Glycol (DEG) Poisoning," by doctors from the US National Center for Infectious Disease and others, followed the efforts to uncover the source of the epidemic of acute renal failure. This unusual cause of childhood death was found in 32 children admitted to the University General Hospital in Port-au-Prince from November 1995 to May 1996. The study reviewed the grim medical record of DEG contamination. DEG is a toxic chemical found in, among other things, anti-freeze. It was the source of epidemics of acute renal failure in Argentina, Bangladesh, Spain, Nigeria and South Africa.[3]

AETIOLOGY.

A study comparing the experiences of the incidence and aetiology of acute renal failure in pregnancy (ARF-P) from the same institution revealed 42 patients with a diagnosis of ARF-P during a 3year period from 1990 to 1992. The main contributor to obstetric-related causes was PE: E.[4] In another study, Olabanji J.K et al found 11.6% of 474 patients with burns in a ten-year review of burns cases at the Obafemi Awolowo University Teaching Hospitals' Complex (OAUTHC). However, in general the causes of acute renal failure can be subdivided into 3 groups.

- Prerenal as a result of inadequate renal perfusion
- Renal
- Post renal due to urinary tract obstruction.

Prerenal causes:

*Hypovolemia from any cause including blood loss (hemorrhage) secondary to trauma.

*Causes of acute circulatory failure.

Renal causes:

* Acute glomerulonephritis.
* Acute tubular necrosis from ischaemia, toxins, haemoglobinuria, myoglobinuria, radio-contrast agents.
* Heavy metal or barbiturate poisoning.
* Collagen disease e.g. systemic lupus erythematosus, polyarteritis nodosa.
* Renal papillary necrosis.
* Malignant hypertension
* Renal arterial or venous obstruction.
* DIC with renal cortical necrosis.
* Intrarenal precipitation due to hypocalcaemia, urates, myeloma protein)

Post-renal causes:

Acute urinary tract obstruction from:

- Prostatic enlargement
- Calculi
- Bladder, pelvic or retroperitoneal tumors
- Retroperitoneal fibrosis
- Ligation of the ureters during surgery within the pelvis e.g. Total abdominal hysterectomy or some other

TABLE 2: MAJOR CAUSES OF ACUTE RENAL FAILURE

PRERENAL

Fluid and electrolyte depletion,
Haemorrhage
Septicaemia
Cardiac Failure
Liver Failure
Heatstroke (Myoglobinuria + fluid/electrolyte depletion)
Burns (fluid/electrolyte depletion + myoglobinuria and haemoglobinuria)

POSTRENAL

Prostatism
Bladder, pelvic, or retroperitoneal tumors
Calculi

RENAL

Acute tubular injury (ischaemia, toxins, radiocontrast agents, haemoglobinuria, myoglobinuria).
Acute glomerulonephritis
Disseminated Intravascular Coagulopathy with cortical necrosis
Arterial or venous obstructio
Acute tubulointerstitial nephritis (drug reaction, pyelonephritis, papillary necrosis)
Intrarenal precipitations (hypercalcaemia, urates, myeloma protein)

forms of tubal surgery.

PATHOGENESIS OF ATN.

The critical events are:

- Tubular injury.
- Persistent and severe disturbance in blood flow.

Tubular injury:

Due to the sensitivity of tubular epithelial cells to ischaemia and their vulnerability to toxins, these cells are particularly prone to damage from any cause employing this method of pathogenesis. The epithelial cells possess a vast electrically charged surface for a tubular reabsorption, active transport systems for ions and organic acids and the capability for effective concentration. Ischaemia may lead to structural and functional changes including celiular swelling, loss of brush borders, bleb formation, loss of polarity, cell detachment, necrosis and apoptosis.

The biochemical basis of this structural and functional changes include:

- Deletion of ATP
- Accumulation of intracellular calcium.
- Activation of proteases resulting in cytoskeletal rearrangement.
- Phospholipases-damage of membranes.
- Generation of reactive oxygen species.
- Activation of apoptotic cell death.

ARF from acute tubular injury may have 3 typical phases: prodromal, oliguric, and diuretic/recovery. The prodromal phase varies in duration depending on causative factors, such as the amount of toxin ingested or the duration and severity of hypotension. During the oliguric phase, urine output typically varies between 50 and 400mL/day. However, many patients are never oliguric and have a lower mortality, morbidity, and need for dialysis. Serum Creatinine typically increases by more than 1 to 2 mg/dL/day and the urea nitrogen by 10 to 20mg/dL/day. However, serum urea nitrogen levels may be misleading as an early index of renal function because elevated values frequently are associated with increased protein catabolism due to surgery, trauma, sepsis, transfusion reactions and gastrointestinal or internal bleeding.

In the diuretic phase, urine output gradually returns to normal levels; however, serum creatinine and urea levels may not fall until several days later. Tubular dysfunction may persist and is manifested by Na^+ wasting, polyuria that is unresponsive to vasopressin, hyperchloremia and metabolic acidosis.[1]

DIAGNOSIS

The history of a patient with suspected renal disease should include review of:

- Recent clinical events.
- Inventory of all the patients' prescription and non-prescription medications.
- In outpatient and hospitalized patients, information includes the blood pressure, pulse rate, alterations in daily weights, daily fluid intake and water output.

The essentials of diagnosis include:

- A sudden increase in blood urea nitrogen/serum creatinine
- A progressive daily rise in serum creatinine.
- Associated oliguria.
- Symptoms and signs depend on the cause.

INVESTIGATIONS.

1.Urinalysis.

1. Normal urine sediment is seen in the prerenal azotemia and the urine microscopy in such cases is often unremarkable.
2. The urine may contain crystals, pus and blood, as seen in the obstructive disease.
3. When the source of the ARF is renal tubular cells, cellular casts and proteinuria are present.
4. White cell casts, tubular cells/casts, eosinophiluria and isosthenuria suggest tubulointerstitial nephritis.
5. Urine culture is desirable to detect infections, which may cause or complicate acute renal failure.
6. Specific gravity is about 1.015 where ARF is caused by severe hypovolemia.
7. Intrinsic renal disease may give an s.g value of 1.010.
8. Urea concentration-prerenal-330mmol/L (1980mg/dl) -Intrinsic renal disease-<100mmol/L (600mg/dl)

2.Electrolytes and Urea- Serum Na^+, K^+, Ca^{2+}, BUN, creatinine, uric acid and CK.

3. Imaging studies of the kidneys by Ultrasonography or CT Normal or enlarged sized kidneys favours reversibility whereas small size suggests chronic renal insufficiency.

4. ASO and complement titres, antinuclear antibodies.

Characteristic Laboratory findings in ARF are those of progressive azotemia, acidosis and hyperkalaemia. A modest daily rise in serum Creatinine (1 to 2 mg\dL) and urea nitrogen (10 to 15mg?dL) usually occurs. A rise of the serum Creatinine > 2mg/dL/day suggests that over-production is occurring from rhabdomyolysis.

Acidosis is ordinarily moderate, with plasma CO^2 content between 15 and 20 mmol/L. Serum K^+ concentration increases slowly. However, when trauma, sepsis, surgery, or steroids markedly accelerate catabolism, or urea generation is accelerated by amino acid infusions, the serum

urea nitrogen may rise at an excessive rate of 30 to 100 mg/dL/day and the serum K^+ by 1 to 2 mmol/L/day. Hyponatraemia usually is moderate (serum Na^+ 125 to 135 mmol/L) and is related to fluid retention. The haematologic picture is that of a normochromic normocytic anaemia of moderate severity. Haematocrit usually ranges from 25 to 30%.[1]

TREATMENT

The goals of treatment of acute renal failure include identifying and treating any reversible causes of the kidney failure (e.g. use of nephritic medications, obstructive uropathy, volume depletion e.t.c). Additionally, treatment focuses on preventing the excess accumulation of fluids and wastes, while allowing the kidneys to heal. The kidneys may gradually resume function. Hospitalization is required for treatment and monitoring.

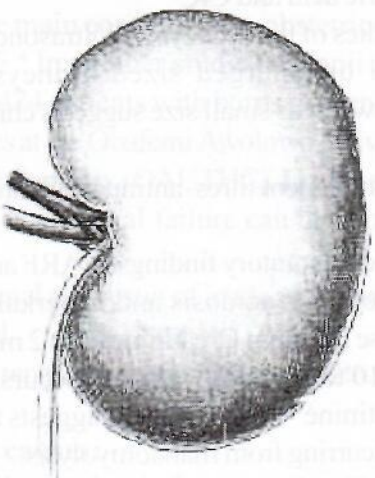
Fluid intake may be severely restricted to an amount equal to the volume of urine produced plus the previous day's output. Specific dietary modifications to reduce build-up of toxins normally handled by the kidneys include following a diet plan that is high in carbohydrates, low in protein, salt and potassium intake.

Antibiotics may be used to treat or prevent infection. Diuretics may be tried in an attempt to increase the excretion of fluid from the kidney in the early stage.

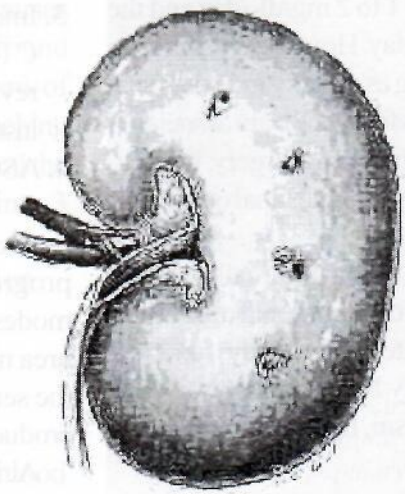
A major priority in treatment is to control dangerous hyperkalemia. A variety of different medications may be utilized for this including IV calcium ions, glucose/insulin

TABLE 3:Therapeutic Approach to Hyperkalemia[6]

Modality	Dosage	Onset	Duration	Mechanism
Ca-gluconate	10-20ml (10%)	<5min	30-60min	Increase threshold potential
Insulin + Glucose	10unit in 50 ml	30-60min	2-4hrs	ICF shift
NaHCO3	44-50meq	30-60min	1-2hrs	ICF shift
Albuterol	20mg	30min	2-4hrs	ICF shift
Kayexalate	Enema: 50g	1-4hr Oral: 20g q4-6hr	Few hrs	Removal of potassium



Pale,Swollen Kidney

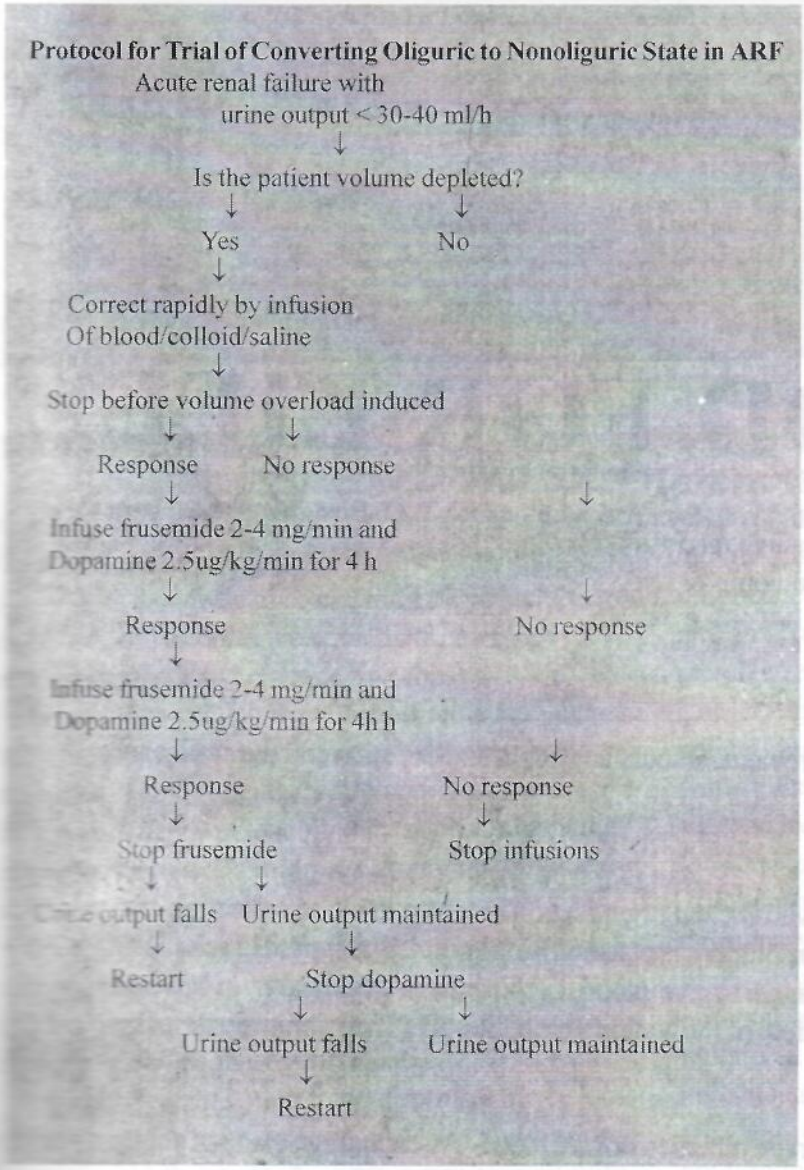


Acute renal failure

Diagnostic plan
history
physical appearance
abdominal palpation
urinalysis
blood work
abdominal x-rays
kidney biopsy

Therapeutic plan
Fluid therapy
Diuretics
phosphate binders
sodium bicarbonate
drugs to control stomach
peritoneal dialysis

Dietary plan
a diet with controlled and appropriate
levels of protein, phosphorus, sodium nad calories



and oral or rectal administration of potassium exchange

In ARF, the role of dialysis is to prevent morbidity associated with complications of ARF and provides temporary support until the renal insufficiency resolves. The decision to initiate dialysis therapy is based on the patient's clinical conditions rather than particular numerical values of BUN or serum creatinine concentration. Uraemic symptoms that can be resolved only with dialysis are considered absolute indications for dialysis. Problems, such as volume overload, hyperkalemia and acidosis, are considered relative indications for dialysis; that is, dialysis should be instituted when conservative management has

Dialysis may be used to remove excess waste and fluids. This often makes the person feel better and may make the condition easier to control. Dialysis may not be necessary for everyone, but is frequently lifesaving, particularly if serum potassium is dangerously high. Decreased mental status, seizures, increased potassium levels, total lack of urine production, fluid overload and uncontrolled accumulation of nitrogen waste products (serum Creatinine > 10mg/dl and blood urea nitrogen > 120mg/dl) are common indications for dialysis.

In ARF, the role of dialysis is to prevent morbidity associated with complications of ARF and provides temporary support until the renal insufficiency resolves. The decision to initiate dialysis therapy is based on the patient's clinical conditions rather than particular numerical values of BUN or serum creatinine concentration. Uraemic symptoms that can be resolved only with dialysis are considered absolute indications for dialysis. Problems, such as volume overload, hyperkalemia and acidosis, are considered relative indications for dialysis; that is, dialysis should be instituted when conservative management has failed.[6]

CONCLUSION

Acute renal failure that requires a high index of suspicion on the part of the physician using the physical signs which depend on the cause of the Acute Renal Failure (ARF) and the results of the chemical investigations. The management of ARF requires aggressive treatment, i.e correction of fluid and electrolyte imbalance and monitoring of vital signs, and symptoms. Patients with significant ARF need to be transferred to a center with facilities for haemodialysis.

ACKNOWLEDGEMENT

We would like to appreciate the Supervisory support given to us during the presentation of this article by Dr. B.L Salako, Consultant Nephrologist, University College Hospital, Ibadan.

REFERENCES

1.Robert Berkow, M.D., Editor-in-Chief, Andrew J. Fletcher, Mark H. Beers, M.D The Merck Manual of Diagnosis and Therapy 16th Edition.

2.Ramzi S. Cotran, Vinay Kumar, Tucker Collins. Pathologic Basis of Disease, Sixth Edition.

3. http://www.wsws.org.

4.Randeree IG, Czarnocki A, Moodley J, Seedat YK, Naiker IP. "Acute renal failure in pregnancy" Renal Failure 17(2): 147-53, 1995 Mar. MEDLINE Abstract.

5. Olabanji JK, Oginni FO, Bankole JO, Olasinde AA. A ten-year review of burn cases seen in a Nigerian teaching hospital. J Burns & Surg Wound Care [serial online] 2003, 2(1): 1. Available from: URL: http://www.journalofburns.com.

6 http:// WWW.ch.org.tW/EBM/eBm/0105/Acute%20 Renal%20

Failure.doc

7 http:// www.emedicine.com/emerg/topic500.htm
Richard Sinert D.O. Peter R. Peacock Jr. MD. "Acute Renal Failure"
e medicine.com, Inc.

MEDICAL EMERGENCIES IN DENTISTRY

ADEKUGBE, O. P.

At the time of writng, Mr. Olubunmi Adekugbe was a second year Dental Surgery Student at the College of Medicine, University of Ibadan.

INTRODUCTION

Now more than ever before, when one considers the revolutionary advances that are occurring in medicine and the fact that a rapidly growing segment of the population consists of the medically compromised patients, geriatric patients, the importance of medical emergency and its relationship to dental practice becomes clear.

It may be accepted that given a sufficient number of dental patients, medical emergency will occur in every dental office. The frequency of such problems will vary with the type of dental practice and the patient population, but emergency may occur in any age group and in all socio-economic status.

The dental surgeon, for the benefit of patients and as a matter of his or her own self interest is obliged to re-evaluate continually the knowledge of prevention, recognition and treatment of emergency.

Physicians on the other hand also have the task of ensuring that they are well informed of the various emergencies that can arise from co management with dental practitioners.

CASE REPORT

A 22year old woman presented with three impacted mandibular for extraction. She had experienced several episodes of pericoronitis. Her medical history was negative. Physical examination revealed a well developed, physically fit, athletic young woman. There was evidence of gingival erythema and swelling in the right third molar region, but the remaining oral examination was normal. There was no evidence of respiratory or cardiac pathology. Laboratory studies, consisting of urinalysis, serum electrolytes and complete blood count, were well within normal limits.

She was admitted for outpatient surgery and taken to the operating room for extraction of three impacted mandibular. General anesthesia was induced with propofol 200mg, and the patients was paralyzed with 100mg of succinylcholine. The patient on recovery from anesthesia began to experience respiratory difficulty characterized by profound inspiratory effort with no air exchange, suprasternal and intercostals retraction, and occasional crowing. She became mildly cyanotic and her SaO_2 dropped to 60%. A diagnosis of larygnospasm was made and she was managed with 100% oxyger and positive pressure ventilation, followed by 20 mg of succinylcholine. The laryngospasm resolved in less than 1 minute and the patients regained spontaneous respirations. Consultation with a pulmonologist was requested and a chest radiograph was obtained that showed subtle infiltrates in the right lower lobe and ground glass radiolucencies of both lung fields. The pulmonary consultant's diagnosis was noncardiogenic pulmonary edema secondary to laryngospasm and acute upper airway obstruction. She was admitted for close observation on the regular medical-surgical unit. She was monitored with continuous pulse oximetry and maintained her SaO_2 at 90% to 95% with supplemental oxygen, but the values decreased to 80% to 85% while breathing room air. Gradually through the night the coughing episodes became less frequent and the dyspnoea lessened. By the following morning she was able to maintain good oxygen saturation without supplemental oxygen and the dyspnoea had ceased. A follow-up chest radiograph showed that the previously noted infiltrate had cleared. She was discharged without further sequlae.

DISCUSSION

As in the case just reported a potentially life threatening condition was observed in an apparently healthy dental patient undergoing surgical disimpaction. Prompt diagnosis and collaborative treatment were invaluable in management of this case. It is to be noted that the emergencies to be discussed below can be seen in healthy, and the increased medical risk individuals.

FAINTING

Fainting (vasovagal syncope) is the most common cause of sudden loss of consciousness with up to 2% of

patients fainting before or during dental treatment.

Predisposing factors

These include pain, anxiety, fatigue, relative hyperthermia, hunger and hot humid atmosphere. Some patients have a tendency to faint readily in response to particular stimuli such as injections and sights of blood. Characteristic signs and symptoms are feelings of dizziness and nausea, pale, cold and clammy skin, a low, thin thready pulse which rebounds to become rapid and loss of consciousness with collapse, if unsupported.

A fainting attack may mimic far more serious conditions most of which can be excluded by a familiarity with the patients past medical history. These include strokes, corticosteroid insufficiency, drug interaction, epileptic attacks, heart block, hypoglycemia and myocardial infarctions.

Prevention

- Avoid predisposing factors
- Treat patients in supine position unless specifically contraindicated (e.g. heart failure, pulmonary oedema).

Management

- Lower the head to the level of/or below the heart. This is best achieved by laying the patient flat.
- Loosen clothing (in the presence of a witness)
- Monitor pulse and if recovery does not occur rapidly, then reconsider the diagnosis.
- Determine precipitants and avoid in future

ACUTE CHEST PAIN

This in dental practice is usually the result of ischemia of the myocardium. The principal differential diagnosis is between angina and myocardial infarction. Both exhibit severe retrosternal pain described as heavy, crushing or band like. It is classically preceded by effort, emotion or excitement and may radiate to the arm, neck, jaw and occasionally to the back or abdomen. Angina is rapidly relieved by rest and glyceryl trinitrate (0.5mg) given sublingually, which patients with angina carry with them. Failure of these methods to relieve pain with a history of hypertension, and co-existing breathlessness, nausea, vomiting and loss of consciousness with a weak or irregular pulse suggest myocardial infarction.

Management

Ensure patients is placed in a supported upright position as the supine position increase pulmonary edema and hence breathlessness

In Dental Practice

- Call for help
- Administer analgesia: Give nitrous oxide and oxygen mixture or intravenous morphine (10mg at 2 mg/min).
- Don't panic. Be prepared, should cardiac arrest supervene, give aspirin 75 – 150mg Par Oral

In hospital

- Nurse upright. Give oxygen
- Establish IV assess and give opioid analgesic available (2.5 - 10mg of diamorphine is most useful)
- Give aspirin.

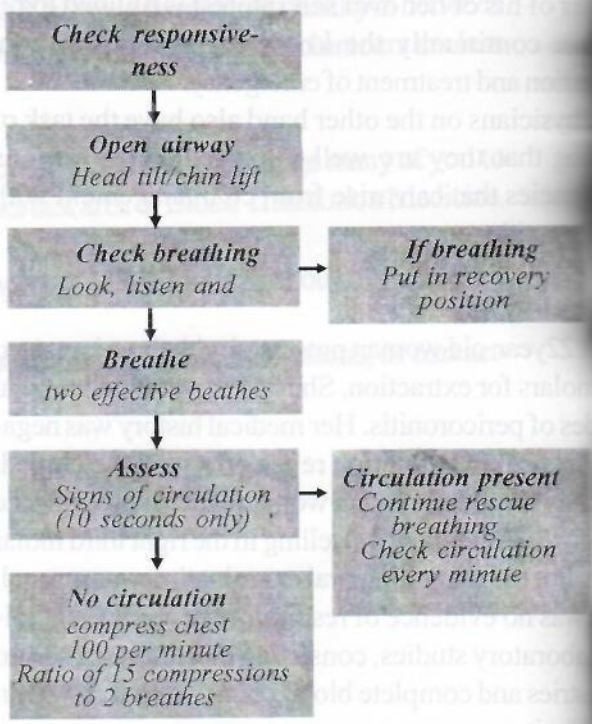
CARDIAC ARREST

This describes the sudden and complete loss of cardi function. There is no pulse, the patient loses consciousne and respiration ceases almost immediately. Death inevitable unless effective treatment is given prompt Cardiac arrest may be due to ventricular fibrillatio ventricular tachycardia, asystole or electromechanic dissociation. 90% of deaths from cardiac arrest outsi the hospital are due to ventricular fibrillation. T commonest underlying cause is ischaemic heart disea but other causes, especially in younger people may be ac asthma, anesthesia, drug overdose, electrolyte imbalan electrocution, and immersion hypothermia \or inappropri medication may increase susceptibility to ventricu fibrillation.

Management

- Ensure immediate expert help is called for.

Figure 1



Algorithm for adult basic life support

- After convulsion ceases, turn into "recovery" position (semi-prone)
- Ensure airway is clear.
- Do not insert anything in mouth.
- If convulsions continue for more than 5 minutes or recur without patient on regaining consciousness, summon urgent medical attention
- Give oxygen to offset cerebral hypoxia
- Give intravenous anticonvulsant (e.g. diazepam 10mg) ONLY if convulsions are continuous and repeated.
- Transfer to intensive care unit, monitoring neurological condition, blood pressure, respiration and blood gases.

HYPOGLYCEMIA

Hypoglycemia is a biochemical abnormality, arbitrarily defined as blood glucose concentration below 2.2 mmol/l. This is seen as acute and dangerous requiring urguent attention. Precipitation factors such as missed meals, excess insulin or increased calorific need due to exercise or stress. Most diabetic patients have no difficulty in recognizing hypoglycemic symptom. Recognition of this state is essential and an acute collapsed diabetic should be assumed hypoglycemic until proven otherwise.

Diagnosis

Disorientation, irritability, increasing drowsiness, excitability or aggression in a known diabetic suggests hypoglycemia.

Treatment

If conscious, give glucose orally in any available form. If unconscious, protect airway, place in recovery position, establish IV assess and give up to 50ml of 20 – 50% dextrose. If available 1 mg of glucagon IM may be given. Ensure help is requested.

ACUTE ASTHMA

Asthma is defined as a chronic inflammatory disorder of the airways characterized by reversible airflow obstruction, causing coughing, wheeze, chest tightness and shortness of breath. Attacks can be precipitated in the dental office by specific allergen such as drugs and other stimuli such as anxiety, infection, cold and exercise. Characteristically, the patients complains of tight chest and shortness of breath. Examination will reveal breathlessness, with wide spread expiratory wheezing. The accessory muscles of respiration may be used to support breathing. If patients are unable to talk, you are dealing with a potentially fatal episode.

Management

Use patients own anti-asthmatic drugs such as salbutamol inhaler ideally, this should be administered, in the form of a nebulizer using 24% oxygen and nebulized salbutamol.

Steroids should be administered either as oral prednisolone, if patient carries these with them or as IV hydrocortisone up to 200 mg IV.

Management in Dental Practice

- Keep patients upright
- Administer salbutamol by inhaler
- Give Oxygen
- Give Steroids

Allow patients go home if responsive, but if in doubt send to accident and emergency department.

Management in Hospital

- Nurse patients upright
- Give nebulized salbutamol 2.5 – 5 mg (with oxygen) up to two hourly
- Give nebulized ipratropium 500 micrograms (with oxygen)
- Establish IV access and give up to 200mg hydrocortisone IV or prednisolone 40 mg per oral
- Monitor peak expiratory flow, arterial blood gases and pulse oximetry
- Obtain chest X-ray to exclude infection pneumothorax.

PROLONGED HEMORRAGE

This can be very alarming and mind – boggling to the dentist and the patients but major vessel damage can conveniently ruled out in the routine treatment in the dental office. However cases of mortality have been recorded in dental offices with prolonged bleeding being the cause of shock.

Causes

* Bleeding disorders - Hemophilia
* Coagulation defects
* Platelet disorder
* Von Willebrand disease
* Anticoagulant therapy
* Others

Bleeding disorders

Hemophilia:

Hemophilia refers to a number of congenital factor deficiencies that result in bleeding diathesis. The most common deficiency are Hemophilia A (factor VIII deficiency) Hemophilia B (factor IX deficiency) and notable is the Hemophilia C (factor XI deficiency) all have absence of or decrease in, or deficient function of implicated factors. Hemophilia is found worldwide, incidence, in Nigeria is estimated at 2.5 per 100,000. Usually presents in childhood as haemathrosis. Following trauma, bleeding appears to stop, but intractable

ACUTE GLAUCOMA

UBAH J.N., ASHAYE A.O.

Dr (Mrs) Ashaye is a Consultant Ophthalmologist at the University College Hospital, Ibadan and a Senior lecturer in the Department of Ophthalmology, College of Medicine, University of Ibadan.
Dr Ubah, at the time of writing this article, was a Senior Registrar at the University College Hospital, Ibadan.

INTRODUCTION

Glaucoma comprises a group of diseases characterized by glaucomatous optic neuropathy, peculiar field changes with raised intraocular pressure as a risk factor. It is a cause of irreversible blindness.

The acute glaucomas are usually accompanied by high-rise in intraocular pressure and are faster at damaging the optic nerve when compared to the chronic glaucoma. They could be primary or secondary. The primary acute glaucomas are of the angle closure variety; with some anatomical and physiological predispositions while the secondary type have some pathological ocular predispositions.

They are usually dramatic in presentation and need intervention to safeguard the optic nerve, which can readily get damaged by the sudden rise in intraocular pressure (IOP).

Primary Angle Closure Glaucoma.

Primary angle-closure glaucoma (PACG) is a condition in which aqueous outflow is obstructed solely as a result closure of the angle by the peripheral iris. Population surveys show clear evidence of racial difference in the prevalence of primary angle closure glaucoma. It is rare before the age of 40years and 2 to 4 times more common in women. It is also familial.[1,2]

Ocular Characteristics

There are some anatomical predispositions, which are:

1. Shallow anterior chamber
2. Relative anterior location of the iris-lens diaphragm
3. Hypermetropia
4. Small corneal diameter
5. Short axial length of the eyeball

In predisposed eyes, it occurs when the dilator muscle of the iris contracts, increasing the apposition between the iris and the anteriorly located lens. This enhances some degree of physiological pupil block; the peripheral iris becomes more flaccid, pressure builds up in the posterior chamber causing a forward bulge of the peripheral iris referred to as iris bombe. The angle eventually becomes obstructed by the peripheral iris leading to rise in intraocular pressure (IOP)[3,4].

The precipitating factors are illumination, emotional stress, and dilating drops in eyes that have occludable angles.

An occludable angle is one in which three quarters or more of the trabecular meshwork is occluded by peripheral iris when viewed with the gonioscope

The process of angle closure occurs in stages, though not strictly from one stage to the other in an orderly sequence. The stages are:

1. Primary angle-closure suspect: the angle is occludable but no other abnormality is present in the eye.
2. Latent angle-closure glaucoma: the angle is occludable with peripheral anterior synechiae or a positive provocation test with normal IOP, optic disc and visual field.
3. Manifest glaucoma: which could be intermittent or chronic.

Intermittent

Intermittent PACG attacks may be precipitated by physiological mydriasis, such as watching TV in a dark room, or physiological shallowing of the anterior chamber when a person assumes a prone or semi prone position to sew or read. Emotional stress may occasionally be a precipitating factor. Without treatment some eyes develop an acute attack whereas others pass straight to the chronic angle–closure phase.

The condition is diagnosed on the basis of intermittent symptoms such as pain and haloes. The patient presents with transient blurring of vision associated with haloes around lights resulting from corneal epithelial oedema. There may be an ache or frontal headache. Examination during an attack shows corneal epithelial oedema. In some cases the pupil may be semidilated but the globe is congested. Eyes look normal in between attacks.

Acute Congestive Angle-Closure Glaucoma

There is a sudden persistent, symptomatic rise in IOP

conjunctival injection and corneal oedema. It is characterized by pain, redness and blurred vision. The pain is typically a severe, deep ache, which follows the trigeminal distribution and may be associated with nausea, vomiting, bradycardia and profuse sweating.

There is ciliary flush caused by injection of the limbal and conjunctival blood vessels. The blurred vision is also typically marked and may be due to stretching of the corneal lamellae initially and later to oedema of the cornea, as well as a direct effect of the IOP on the optic nerve. The IOP is markedly elevated, in the range of 40 to 60mmHg.

The anterior chamber is shallow with peripheral endothelial contact. Pupil is semi dilated, vertically oval and non-reacting to light and accommodating. The pupillary change is thought to result from paralysis of the sphincter which apparently is due to a reduction in the circulation induced by the elevated IOP.

Subsequent examination after corneal oedema has subsided shows aqueous flare and cells, oedematous and hyperaemic optic nerve head. Gonioscopy shows completely closed angle of drainage. The fellow eye usually has a shallow anterior chamber and a narrow angle.

In the post congestive state, the IOP may be normal, subnormal or raised. If the IOP was reduced rapidly, there will be folds in descemet's membrane. Fine pigment granules may be present on the corneal endothelium and iris transillumination. There is also stromal iris atrophy. Pupil is fixed and semi dilated from posterior synechiae and sphincter atrophy. Small grey white, anterior capsular or sub capsular lens opacities (Glaukomflecken) may be seen in the pupillary zone.

Chronic Angle-Closure Glaucoma

The features are similar to primary open angle glaucoma with progressive loss of vision, high IOP, pale, cupped disc and field loss. Unless routine Gonioscopy is carried out on all glaucomatous eyes, the diagnosis will be missed.

Treatment is both laser iridotomy and medical therapy. Filtration surgery may be required.

Differential diagnosis

The conditions that could mimic acute angle closure include:

Intumescent cataract: a swollen hyper mature cataract.

Subluxated displaced lens: The affected eye may show symptoms similar to those of the acute angle closure glaucoma but the fellow eye does not show narrow angle.

Neovascular glaucoma, especially when associated with iridocyclitis, inflammation, haemorrhage and rubeosis.

Secondary Acute Glaucoma

In this category, there is pathology in the eyes causing the glaucoma. Common examples are:

a) **Red cell glaucoma**. Here there is a sudden rise IOP, secondary to pupil block by blood clot, or blockages of the trabeculae meshwork by red blood cells. Occurs frequently after blunt eye injuries with hyphaema.

b) **Traumatic uveitis.** There is increased inflammation, which causes trabecular meshwork blockage and subsequent rise in IOP.

c) **Anterior lens dislocation** as following trauma or in persons with weak zonules e.g. Marfan's syndrome.

Management of Acute Glaucoma

The acute glaucoma are emergency cases that need prompt and urgent intervention. The patients are admitted immediately and started on treatment to prevent optic nerve damage. Prevention of attack is also a vital aspect of the management.

Preventive Measures

A patient suspected of having narrow anterior chamber angles should be treated prophylactically to prevent an attack. It is difficult to tell which eye with narrow angles will have an attack. Because of this, some surgeons utilize provocative test to determine such eyes. These tests are:

Mydriatic Provocative test:

A short acting mydriatic like 0.5% tropicamide is instilled and a rise of 8mmHg in IOP or more is considered to be a positive test. There should be gonioscopic confirmation of angle closure however.

Dark Room Provocative test:

Mydriasis is induced by placing the in a dark room for 60-90 mins. The patient should remain awake during this period to avoid the miosis of sleep. Pressure rise of 8mmHg or more and gonioscopic confirmation of angle is necessary.

Prone Provocative test:

Patient is placed in prone position for 60minutes, and a pressure rise of 8mmHg or more is taken as positive. The mechanism for this may be forward shifting of iris-lens diaphragm.

Treatment of acute primary angle closure glaucoma

The recommended prophylactic treatment is YAG-Laser iridotomy. Intermittent attack is treated during attacks with intensive miotic therapy (2% Pilocarpine every 5minutes). The fellow eye is treated prophylactically with 1% Pilocarpine, four times daily. Bilateral laser iridotomies should be performed as soon as possible.

Acute attacks: This is treated with 2% Pilocarpine drop frequently, Timolol 0.5% twice daily and intravenous acetazolamide 500g stat, then orally up to 1gm daily in divided doses. Hyper osmotic agents such as intravenous mannitol and oral glycerol are usually reserved for cases which fail to respond after several hours of Pilocarpine and acetazolamide. Analgesics, antiemetic and anti inflammatory drugs may be necessary. The fellow eye is treated prophylactically with 1% Pilocarpine drops four times daily until a prophylactic iridotomy is done. Subsequently, the affected eye is treated with laser iridotomy or surgical iridatomy but if more than half of the angle is permanently blocked, filtration surgery is performed.

Secondary Acute Glaucoma: The treatment of red cell glaucoma could be conservative with anti glaucoma anti-inflammatory drugs, or surgical depending on the clinical findings and IOP levels. Traumatic glaucoma secondary to uveitis is managed conservatively with anti inflammatory anti glaucoma drugs.

The definitive treatment for lens dislocation is urgent ract extraction.):37-39.

Conclusion

Acute glaucomas present as emergency and s be treated as such. They are not as common as the c glaucomas which are equally devastating but insid onset.

REFERENCES

1. Kanski J.J. Clinical Ophthalmology. Butterworth Heinemann
2. Shields M. Bruce: Textbook of Glaucoma. Williams and W pgs.
3. Foster P.J. Primary Angle Closure Glaucoma: Community E 1996. 9(18): 22-24
4. Foster P.J. Advances in the understanding of Primary Angle C cause of Glaucomatous Optic Neuropathy. Community E 2001 14(39

COMPARTMENT SYNDROME - AN ORTHOPAEDIC EMERGENCY.

OMOLOLU, B

Mr. Bade Omololu, FRCS, is a Consultant Orthopaedic Surgeon to the University College Hospital, Ibadan and a Senior Lecturer in the Department of Surgery, College of Medicine, University of Ibadan.

Compartment syndrome is a common orthopaedic emergency that may lead to devastating consequences such as amputation or even death if complicated by infection. Diagnosis is usually made by careful clinical observation for the typical signs of undue pain, swelling and tightness in the compartment concerned. Pain on passive stretching of the muscle in a given compartment may be the earliest clinical indicator. Once diagnosed, emergency fasciotomy is needed to avoid permanent neurologic sequelae. The surgeon must have a high index of suspicion for compartment syndrome for all patients.

Key words: compartment syndrome, emergency.

INTRODUCTON

Compartment syndrome (C.S.) is a common surgical emergency and is usually seen in the arm, leg and abdomen.[1] Other areas can potentially be involved for example the shoulder, post dislocation, in the thigh, post intramuscular injection, the ankle after a fracture/dislocation and even the eye after a blow out fracture[2]. It has also been postulated that the pelvis may occasionally be involved in compartment –like syndrome.[3] The word compartment comes from the anatomical compartments as derived by anatomical divisions; for example there being four in the leg (anterior tibial, superficial posterior, deep posterior, and peroneal). Whilst usually acute it is also seen as a chronic problem in athletes whereby the deep peroneal compartment is involved.[4]

Despite attempts to document the pathophysiology of compartment syndrome, the clinical recognition of this is frequently difficult. If left untreated compartment syndrome not only results in the loss of nerve and muscle function but also may lead to infection, myoglobinuria, renal failure and even amputation.[5]

It is thought to be due to circulation and function being compromised due to an increase in pressure within a closed space due to increase in the intra-compartmental pressure or a decrease in the size of the compartment [6]. Impaired circulation causes impairment of venous outflow and occlusion of arterial input and decreased tissue perfusion [7]. The underlying pathogenesis is thought to relate to acidosis within the compartment [8]. It can also be caused by revascularisation of the compartment[9]. Essentially the tissue becomes ischaemic and usually presents with the usual symptoms of an ischaemic limb (pain on movement and at rest, pallor, and paraesthesia, pulseless and cold).

Any age group may be affected; there are reports of a CS in neonates (for example after a distal tibial physeal injury) [11] and one of a toddler who had just simply been hanging his leg off a bed [12]. It has also been suggested that not only are women affected more commonly but that they tend to do less well post-surgery [13].

Recommended management comprises early recognition to prevent irreversible ischaemia., open fasciotomy over the affected compartment and delayed skin closure. Early diagnosis may be assisted not only by clinical acumen but also by measuring the intra-compartment pressure[13]. The role of the synthetic cAMP phosphodiesterase inhibitor, cilostazol, which is said to enhance smooth muscle cell vasodilation and may be even cyclo-oxygenase inhibitors, remain uncertain [14] .Where CS is a chronic problem treatment by endoscopic decompression has also been reported[15].

A Medline search unveiled many causes of CS (Table) the commonest being traumatic[15] (especially after fractures and arterial injury) and post operative [10],[16] interestingly including arthroscopy and knee arthroplasty[17] In the abdomen CS has not only been reported after abdominal surgery but also with severe burns and excessive fluid resuscitation[18].

THE CLINICAL PROBLEM

Despite an increased sensitivity of clinicians to the diagnosis of CS, few criteria are available to serve as guidelines for making the diagnosis. The subjective criteria include pain, sensory changes, but the sole objective criterion is the measurement of intracompartmental pressures. However, even the definition of abnormal tissue

Table: Causes of Compartment Syndrome

- Alcohol [1]
- Injection of narcotics [3]
- Sepsis [4]
- Exercise (acute, chronic and acute on chronic) [5]
- Non-traumatic muscular rupture [18]
- Post –partum in a neonate [19]
- Malignant hyperthermia [8]
- Aggressive fluid replacement [20]
- Post –operative use of local anaesthetic [21]
- Epidural analgesia [22]
- Bleeding post venepuncture in a patient on warfarin [22]
- After a ruptured Bakers Cyst
- After removal of a tattoo using laser treatment [23,24]

pressure is difficult as anatomical compartments are not homogenous and an equilibrium of pressure cannot be expected.[18]

Heckman et al[8] recommended the measurement of pressure at multiple sites.

For the clinician the fundamental problem is the inability to identify the pressure at which the nerve becomes ischaemic. There is no reliable objective method to determine when a fasciotomy is required; and despite the development of various techniques for the measurement of intracompartmental pressure , it is not appropriate to rely on this measurement only; the diagnosis of CS is made from a constellation of clinical findings.

The clinician should consider several key points when evaluating a patient for the development of CS:

- Intracompartmental pressures are not a measure of muscle and nerve ischaemia;
- The development of muscle ischaemia depends on the magnitude and duration of the elevated pressure and
- The tolerance of muscle to Ischaemia may vary among patients because of associated conditions such as shock, compensatory hypertension, or altered tone of the resistance vessels.

Probably because of the variable factors mentioned above,different critical values have been identified by various authors [3, 7, 11, 5]. Some authors have tried to identify an absolute tissue pressure above which the risk of tissue necrosis is great enough that a fasciotomy should be performed. This value has been determined to be 30-40 mm of mercury (30-40mmHg) .

Others have suggested that the critical value must reflect a decrease in tissue perfusion which occurs when the intracompartmental pressure approaches the perfusion pressure as reflected by some measurement of the systemic blood pressure. This critical difference or differe pressure (DP) has been suggested to be 30-40 Hg.[11,12,15,23,24]

DIAGNOSIS

The mechanism of injury is the first indication t patient may be at risk for a CS. According to Tsch and Gotzen [25],the more severe the initial soft tissue i the greater the probability that soft tissue complica including CS will develop.

Because the development of a compartment syndr is unpredictable, close observation is required until the swelling begins to subside.

Severe or increasing pain, tightness in the leg and sen changes are frequently the first symptoms.

A careful physical examination is necessary and s include testing of muscle strength in the leg and f well as sensory testing of the superficial and deep per nerves and the tibial nerve. Because nerve tissue i sensitive to ischaemia, sensory changes frequently h the onset of decreased time perfusion.

OPERATIVE TECHNIQUE FOR FASCIOTO

Performing an adequate fasciotomy requires an inc that approximate the proximal-distal length o compartment to be decompressed. A long fasciot required for reliable decompression of an a compartment syndrome [5]. Regardless of the appr used, all compartments of the involved areas of the most likely the limbs must be thoroughly decompr This is necessary at the time of the initial fasci because after one compartment has been rel hyperemia may precipitate increased pressures in a compartments.

In most instances, the two-incision technique a better exposure of the four compartments in the l release of the soleus from the fibula is not required.

A lateral incision is made over the inter-muscular s between the anterior and lateral compartments to these two compartments.

The medial incision is made two centimeters fr medial crest of the tibia shaft. After the superficial p compartment has been released, the deep p compartment is exposed by retraction of the su compartment posteriorly.

The interval between the superficial and d compartments is best identified in the distal one-t the leg where the gastrocnemius-soleus unit b tendinous. The deep posterior compartment sh released throughout its entire length.

After the fasciotomy a bulky compression dressing and a splint are applied. The foot should be placed in slight dorsiflexion to prevent an equinus contracture. The incision for the fasciotomy usually can be closed after three to five days. When two incisions have been made it is not possible to close both, delayed primary closure of the medial wound should be performed.

On the lateral side, where there is good muscle coverage over the bone, the wound may be closed by one of several methods involving the use of split-thickness skins grafts, relaxing incisions or skin-stretching devices. Regardless of the method, excessive skin tension must be avoided with closure.

OUTCOME OF COMPARTMENT SYNDROME

The most important determinant of a poor outcome from acute CS after injury is delay in diagnosis [1, 5]. The complications are usually disabling and include infection, contracture and amputation. When the diagnosis of a CS is made early and a fasciotomy is performed promptly, most patients have few sequelae.

Rorabak amd Macnab [26] documented that patients who had a release within six hours of the diagnosis had a full recovery whereas those who had a release after six hours (meantime to operation, eighteen hours) had sequelae.

In a study of malpractice costs associated with a missed diagnosis of CS in eight patients[27], the average indemnity was nearly $280,000.00.

The sequelae included amputation and complete loss of function of the lower or upper extremity. The costs were high because CS affects the young productive working force of a community.

CONCLUSION

A CS of a limb may lead to devastating consequences such as amputation. In patients who are conscious, sensory changes usually occur before motor changes. Pain on passive stretching of the muscle in a given compartment may be the earliest clinical indicator

Intracompartmental pressure is the sole objective measurement and constitutes an indirect measurement of muscle and nerve ischaemia.

Once a CS has been diagnosed, emergency fasciotomy is needed to avoid permanent neurologic sequelae.

A delay of more than six hours in the diagnosis or improperly performed fasciotomy usually leads to permanent weakness. The surgeon must have a high index of suspicion for compartment syndrome for all patients.

REFERENCES

1—Blick, S.S; Brumback, R.J;Poka, A; Burgess, A.R; and Ebraheim,N.A. Compartment syndrome in open tibial fractures. J.Bone and Joint Surg., 1986 68-A: 1348-1353.

2—Chapman,M.W:Fractures of the tibial and fibular shafts.In surgery musculoskeletal system,edited by C.McC.Evarts. vol.3, pp.8:5-8:62.New York,churchill Livingstone,1983.

3—Gaspard, D. J., and Kohl, R.D., Jr.: Compartmental syndromes in which the skin is the limiting boundary.Clin. Orthop., 1975 133:65-68

4— Hamza K. N., Dunkerly, G. E, and Murray, C.M.: Fractures of the tibia. A report on fifty patients treated by intramedullary nailing. J. Bone and Joint Surg., 1971 53-B(4):696-700.

5—Hargens, A.R.; Romine, J.S.; Sipe,J. C.; Evans, K.L.; Mubarak, S.J.; and Akeson, W.H.: peripheral nerve-conduction block by high muscle-compartment pressure.J.Bone and Joint Surg., 1979 61-A: 192-200.

6—Heckman, M.M.; Whitesides,T.E. Jr, Grewe, S.R and Rooks,M.D. Compartment pressure in association with closed tibial fractures.The relationship between tissue pressure, compartment, and the distance from the site of the fracture.J. Bone and Joint Surg., 1994 76-A: 1285-1292.

7—Heckman M.M.; Whitesides,T.E.;Jr.; Grewe,S.R.; Judd, R.L.;Miller, M.; and Lawrence J.H.,III:Histologic determination of the ischemic threshold of muscle in the canine compartment syndrome model.J.Orthop. Trauma, 1993 7:199-210.

8—Heppenstall, R. B.,Scott,, R.; Sapega, A.; Park, Y.S.; and Chance, B.: A comparative study of the tolerance of skeletal muscle to ischemia. Tourniquet application compared with acute compartment syndrome.J.Bone and Joint Surg. 1986,68-A:820-828.

9—Hepenstall,R.P.;Sapega,A.A.;Scott,R.;Shenton,D.;Park,Y.S.;Maris,J.; and Chance B.: The compartment syndrome.An experimental and clinical study of muscular energy metabolism using phosphorus nuclear magnetic resonance spectroscopy.Clinorthop. 1988,226:138-155.

10—Ho, Y.K., and Lau, P.Y.:Compartment syndrome after intramedullary interlocking nailing of a tibial fracture.Injury. 1991 22:490-491.

11—Kelly,R.P., and Whitesides,T.E., Jr.: Transfibular route for fasciotomy of the leg (abstract).J. Bone and Joint Surg. 1967, 49-A:1022-1023.

12—Koval,K.J.; Clapper,M.F.; Brumback, R.J.;Ellison,P.S.,Jr.;Poka, A.;Bathon, G.H.; and Burgess,A.R.:Complications of reamed intramedullary nailing of the tibia.J.Orthop.Trauma, 1991 5:184-189.

13—McQueen,M.M., and Court-Brown,C.M.: Compartment monitoring in tibial fractures:the pressure threshold for decompression.J.Bone and Joint Surg. 1996, 78-B(1):99-104.

14—McQueen, M.M.;Christie, J.; and Court-Brown, C.M.: Compartment pressures after itramedullary nailing of the tibi.J.Bone and Joint Surg. 1990,72-B(3):395-397.

15—McQueen, M.M.; Christie,J.; and Court-Brown,C.M.: Acute compartment syndrome in tibial diaphyseal fractures.J.Bone and Joint Surg. 1996,78-B(1):95-98.

16—Masten,F.A.,III:Compartment syndromes.Part A. Pathophysiology of compartment syndromes.in Insructional course Lectures,The American Academy of orthopaedic Surgeons. Vol. 38,pp. 463-466.park Ridge,illinois, The American Academy of Orthopaedic Surgeons.1989.

17—Masten,F.A.,III; Winquist,R.A.; and Krugmire,R.B.Jr.: Diagnosis and management of compartmental syndromes.J.Bone and Joint Surg. 1980, 62-A:286-291.

18—Matsen,F.A.,III;Mayo,K.A.;Krugmire,R.B., Jr.; Sheridan, G. W.;and Kraft,G.H.:A model compartmental syndrome in man with particular reference to the qualification of nerve function.J.Bone and Joint Surg. 1977,59-A:648-653.

19—Mawhinney, I.N., Maginn,P.; and McCoy,G.F.: Tibial compartment syndromes after tibial nailing.J.Orthop. Trauma, 1994 8:212-214.

20—Moed, B.R., and Strom,D.E.: compartment syndrome after closed intramedullary nailing of the tibia: a canine model and report of two cases.J.Orthop.Trauma, 1991 5:71-77.

21—Mubarak,S.J.;Owen, C.A.;Hargens,A.R.;Garetto,L.P.;and Akeson;W.H.; Acute compartment syndromes:diagnosis and treatment with the aid of the wick catheter.J.Bone and Joint Surg. 1978,60-A:1091-1095.

22—Shakespeare,D.T., and Henderson,N.J.:Compartmental pressure changes during calcaneal traction in tibial fractures.J. Bone and Joint Surg. 1982, 64-B(4):498-499.

23—Soejima, O., Ogata,K.;Ishinishi,T.;Fukahori,Y., and Miyauchi,R. Anatomic considerations of the peroneal nerve for division of the fibula during high tibial osteotomy.Orthop.Rev. 1994,23:244-247.
24—Stitgen,S.H., Cairns,E.R.;Ebraheim,N.A., Neimann,J.M., and Jackson,W.T., Anatomic considerations of pin placement in the proximal tibia and its relationship to the peroneal nerve. Clin. Orthop. 1992,278:134-137.
25—Tscherne,H., and Gotzen,L. Fractures with Soft Tissue Injuries.New York,Springer,1984.
26—Rorabeck,C.H., and Macnab,I. The pathophysiology of the anterior tibial compartmental syndrome.Clin.Orthop. 1975, 113:52-57.
27—Templeman, D., Schmidit, R.D., and Varecka,T.F., The economic costs of missed compartment syndromes. Orthop. Trans., 17:989,1993-1994.

UPDATE ON THE MANAGEMENT OF ACUTE URINARY RETENTION

*IFESANYA, O., *OGEBE, O.S., *SALAMI, A. O., *ABIKOYE E. O., **OLAPADE – OLAOPA, E. O.

*The Authors * are residents in the Department of Surgery, ** is a consultant Urologist to the University College Hospital and a Senior Lecturer in the Department of Surgery, College of Medicine, University of Ibadan.*

INTRODUCTION

Urinary retention (acute or chronic) is a condition in which urine cannot be voluntarily expelled from the bladder. Acute urinary retention (AUR) is an unpleasant experience characterized by sudden onset of painful inability to urinate, which usually requires immediate intervention. It usually occurs within a volume range of 500 – 1,500 mls. There may or may not be a history suggestive of previous obstructive uropathy.

In contrast, chronic urinary retention occurs over a period of time (months rather than days). It is usually associated with features of longstanding bladder outflow obstruction and overflow incontinence. Characteristically, the patient is not in pain, but there may be recurrent episodes of acute retention superimposed. Residual urine volume is usually more than 1,500mls.

EPIDEMIOLOGY

Acute retention of urine is common in men, (a study from the Western world revealed an incidence rate of 4.5 per thousand per year in men 45 years and above and the incidence increases with age [2]. It is rare in females (incidence of 0.07 per 100 women per year [3]) in whom neurological disorders (e.g. multiple sclerosis) is the commonest cause.

Risk factors associated with this condition include:

- Low peak urinary flow rates (less than 15ml per sec.) [1,4]
- Elevated serum prostate specific antigen (PSA) [5]
- Large prostate gland [5]
- Moderate to severe urinary obstructive symptoms based on the International Prostatic Symptom Score [2a]
- Use of drugs with adrenergic or anticholinergic side effects (e.g. scopolamine), which may cause excessive bladder relaxation[2].

AETIOLOGY (see Table 1)

CLINICAL FEATURES

Symptoms:

The patient with AUR complains of painful inability to void urine of a short duration of few minutes or hours. Further questioning may reveal urinary obstructive symptoms (such as hesitancy, straining, poor urinary stream or terminal dribbling) or irritative bladder symptoms (nocturia, urgency and frequency), which are commoner in BPH and carcinoma of the prostate. Haematuria and passage of tissue fragments or stones could suggest carcinoma of the bladder or urolithiasis respectively. There may be a history of trauma to the penis, perineum or pelvis in a patient with urethral rupture in which cases urethral bleeding may coexist.

Other symptoms to look out for include:

1. Low back pain and pathological fractures, which would suggest metastatic cancer of the prostate or bladder. Back pain, chronic cough and haemoptysis and weight loss associated with the urinary symptoms in spinal tuberculosis.
2. Polydipsia, polyphagia and polyuria in diabetic cystopathy [18], other diabetic complications should also be excluded.
3. A recent operation under general anaesthesia; suprapubic or perineal operations.
4. Dysuria, urethral discharge, painful ejaculation and constitutional symptoms could suggest an acute urethritis or prostatitis.

Complication due to urinary retention should be ruled out in the history; these include :

1. Fever, chills and rigors of urinary tract infection.
2. A patient with longstanding urinary obstruction presenting with acute-on-chronic retention may have uraemia which is characterized by anorexia, nausea vomiting, hiccups and weakness. Hydronephrosis may produce a dragging loin pain.

Signs:

On examination, the patient is typically restless, pacing up and down the room, sweaty and in acute painful

distress. There may be fever if the condition is already complicated by urinary tract infection. The finding of uraemia fetor, anaemia and altered sensorium may suggest uraemia complicating a background chronic retention.

There is a tender suprapublic mass, which is dull to percussion (a very painful manouever that should be done gently). Aspiration of the mass yield urine. The kidneys may be ballotable due to hydronephrosis in acute-on-chronic retention. There is a positive pelvic stress test if the condition is due to pelvic fracture with urethral rupture. The diagnosis of meatal stenosis is made by examining the glans penis. In penile shaft fracture, there is a tender, swollen penis, which may be deformed. A tender perineal swelling close to the midline may point to a rupture of the bulbar urethra. Palpable induration along the urethra may be found in urethral stricture or urethral calculus (when it is tender). Periurethral abscess and urethrocutaneous fistulae (*"watering can perineum"*) may be found especially in utter obstruction.

Digital rectal examination may reveal an enlarged prostate, which is smooth and firm with preservation of the medial groove and lateral sulci in BPH. In carcinoma the prostate, the gland is nodular, and the medial groo and lateral sulci obliterated. In acute prostatitis, whic uncommon, there is exquisite tenderness of the pros and the patient might not allow a digital rectal examinat Examination of the systems would help to confirm ot

TABLE 1: Causes of Acute Retention

A. Mechanical

1 ***Urethral Conditions***

a. *Meatal stenosis*

b. *Traumatic rupture*
- *Pelvic fracture, falling astride, perineal kicks, penile fracture*

c. *Strictures*
- *Younger (sexually active) males*
- *Could be post-traumatic*

d. *Acute urethritis*[7]

e. *Urethral foreign bodies e.g. stones,* [7,8,9]
Posterior urethral valves, Congenital fibro-epithelial polyp of the prostatic urethra [10,11]

2. ***Diseases of the Prostate***

a. *Benign prostatic hyperplasia (BPH)*
-The commonest cause
-Affects most men over 40 years however only 25% are symptomatic [3]

b. *Carcinoma of the prostate*
- *A disease of ageing*
- *Co-exist with urethral stricture in 2% of cases* [12]
- *Tumor metastases to prostate* [13]
- *Leukaemic infiltration*

c. *Acute Prostatitis*

3. ***Diseases of the Urinary Bladder***

a. *Carcinoma of the bladder*

b. *Foreign bodies e.g. calculi* [7,8]

c. *Ureterocele* [14]

d. *Haematuria and clot retention* [15,16]

B. Neurogenic

1. *Spinal Cord injury, spina bifida*
2. *Tuberculosis of the spine (Pott's disease)*
3. *Cauda equina and conus medullaris lesion* [17]
4. *Diabetic cystopathy* [18]
5. *Tabes dorsalis*
6. *Multiple sclerosis*
7. *Bladder neck dysynergia*
8. *External urethral sphincter spasm (Isaac's syndrome)*
9. *Anticholinergic drugs, chloroquine induced neuromyopathy* [20].

C) Other Causes

(1) Post Operative urine retention
- *Following general anesthesia*
- *Following perineal operations e.g. Haemorrhoidectomy, fistulectomy and fissurectomy*[21]

(2) ***In females***

Psychogenic – a diagnosis of exclusion [3,22]
Pelvic masses e.g. gravid uterus ectopic pregnancy uterine fibroids [5,23,24]
Urethral mucosal prolapse[25]
Urethral leiomyoma [26]

D) Rare causes

Perineal diseases e.g. anogenital herpes[27,28] *and ischiorectal abscess*[29]
Renal hydatid disease with hydatiduria.

causes of urine retention. Neurological examinat reveal a gibbus in cases of spinal cord injury or col vertebra following metastatic tumour deposits and nal tuberculosis (in which case, the diagnosis is str ened by the presence of chest signs). There may be ciated sensorimotor deficits in the limbs.

PRELIMINARY TREATMENT

This is aimed at relieving the patient's discomfo venting complications of urinary retention and trea underlying cause.

a. Analgesics should be given to relieve both visc well as somatic pain as soon as the diagn been made and prior to preparation for ca ization. This would make the patient m

fortable and co-operative during catheterization. Hyoscine (Buscopan) usually is given for the former and pentazocine for the latter.

It is important to relieve the patient's discomfort by catheterization and emptying the bladder. Urethral catheterization can be diagnostic as the catheter may be held up at the site of a stricture or an impacted urethral stone. It should be done under antibiotic cover (80 – 250 mg of gentamicin stat after excluding allergies) to avoid acute onset urosepsis, which occurs in 2 – 5% of patients and may be associated with significant mortality if not adequately treated. If catheterization is not possible or is contraindicated (as in partial urethral rupture for fear of converting it to a compete rupture), a suprapubic cystostomy (SPC) should be carried out. In situations where an SPC cannot be done due to lack of expertise or equipment, repeated suprapubic aspiration may be done before the patient is referred. This is of particular importance in pediatric patients with AUR (usually secondary to posterior urethral valves). The quantity of urine evacuated is measured and a sample sent for microbiology.

If the residual volume of urine drained at once exceed 1.5 litres the patient should be kept under observation and fluid and electrolyte replacement with normal saline infusion be commenced. This is because these patients commonly experience a post obstructive diuresis with increased glomerular permeability and reversible renal tubular damage. This is commonly associated with proteinuria as evidenced by increased excretion of albumin and alpha$_1$ – macroglobulin by the kidney, which resolves within 6 months [31].

Apart from the prophylactic antibiotics given, a course of broad spectrum antibiotic should be prescribed in patients with infected urinary tract as well as those liable to infection (e.g. urethral rupture and when the catheter is to be retained).

The patient is then fully investigated and definitive management of the cause of acute urinary retention instituted.

INVESTIGATIONS

These should be done include:

1. Haematological

   A full blood count and erythrocyte sedimentation rate (ESR) may show anemia in uremia or severe haemorrhage, neutrophilia in urinary tract infection or relative lymphocytosis in spinal tuberculosis. ESR is non-specific and is elevated in carcinoma of the prostate, spinal tuberculosis, other chronic diseases, malignancies and multiple sclerosis. It can also be used to monitor progress.

2. Biochemical

   a. Baseline serum urea, creatinine, electrolytes (E/U, creat) with calcium, phosphate and alkaline phosphates – hyponatraemia may occur in patients with diuresis, 12–24hours following relief of urinary retention. Disseminated prostate cancer may cause hypercalcaemia and elevated alkaline phosphatase levels.

   b. Urinalysis may reveal glycosuria (in diabetes mellitus), crystalluria (in urolithiasis) proteinuria (in renal parenchymal disease and urinary infection confirmed by urine culture); microscopic haematuria is non specific.

3. Radiological

   a. Ultrasound of renal tract, bladder and prostate – define the anatomy of the urinary tract and the prostate; hydronephrosis due to back pressure effect on the upper urinary tract, stones, tumours and bladder pathologies are also detected.

   b. Plain KUB X-ray – 80% of urine stones are radio-opaque and may be detected. Pelvic fractures are visualized. Spinal X-ray is useful in Pott's disease of the spine as well as spinal injury.

   c. Intravenous urography (IVU) – No longer commonly done. It may however give crude information about renal function, as a minimum of 25% function is necessary for excretion.

   d. Retrograde urethrography (RUG) is indicated when urethral rupture, stones and strictures are suspected.

   e. Micturating cystourethrography (MCU) - confirm posterior urethral valves and identifies the proximal extent of the urethral stricture.

4. Urethrocystoscopy

   Indicated in non-prostatic causes of urinary retention and may be therapeutic as well as investigative e.g. endoscopic realignment of urethral rupture can be done during this procedure; similarly, polyps of the urethra and bladder as well as posterior urethral valves can be ex-

cised[11].

5. Others:
   a. Urine flowmetry – assesses severity of prostatic obstruction and responses to treatment. This is the simplest urodynamic study that can be performed on all patients with bladder outflow obstruction. It consists of measurement of the peak flow rate in millilitres per second and the total voided volume.
   b. Urodynamics – evaluates detrusor physiology and is commonly diagnostic in neurogenic and obstructive urinary retention. It entails the recording of pressure/volume changes within the bladder during filling and pressure/flow relationship during voiding.
   c. Tumour marker assay – the prostate – specific antigen (PSA) is a serine protease produced by the prostate epithelium with the function of liquefying the gel, which surround spermatozoa to enable them to become fully mobile. It is usually elevated in prostate cancer. Unfortunately, many centres in the tropic and the third world still depend on prostate-specific acid phosphatase activity.

COMPLICATIONS OF ACUTE URINARY RETENTION AND ITS TREATMENT

1. **Septic complication** – Gram-negative septicemic shock may occur and this is associated with a high mortality rate. It may also complicate catheterization carried out in an infected system especially without an intravenous antibiotic cover. Other septic complications are cystitis and pyelonephritis.
2. **Post – obstructive diuresis** may follow relief of urine retention. This is characterized by salt and water depletion resulting in hyponatremia, which may be significant enough to cause cerebral edema. Shock may also result from intravascular volume depletion. Iatrogenic polydipsia may follow over – enthusiastic compliance with physician's instruction to drink plenty of (hypotonic) fluid during rehydration of patients[32].
3. Other complications of urethral catheterization include urethral rupture, encrustation and stone formation at catheter tip, traumatic urethral bleeding, false passage and oedematous bladder mucosa due to irritation by catheter balloon.

CONCLUSION

Acute retention of urine is a condition that requires urgent intervention in order to relieve the discomfort as well as forestall the complications notably urinary tract infection and septicaemia which are quite formidable. There are premonitory features, hence, high-risk subjects may be identified and treated. Worthy of note is the dearth of local epidemiological data on this subject.

REFERENCES

1. Boyle P. Some remarks on the epidemiology of acute urinary retention. Arch Ital Urol, Androl 1998 ; 70 (2) 77 – 8
2. Meigs J.B., Barry M.J. Giovanucci E, Rimm E, B. Stampfer M. Kawachi I. Incidence rates and risk factors for acute urinary retention: the health professionals follow-up study. J Urol 1999 ; 162 (2) 376 – 82
3. Van der Linden E.F. Venema P.L. Acute urinary retention in women. Neth Tijds voor Genees 1998; 142 (28) 1602
4. Jacobsen S. J. Jacobsen. J,. Girman CJ. Roberts R. O. Rhodes T. Guess H.A. Lieber M.M. Natural history of prostatism: risk factors for acute urinary retention J. Urol 1997; 158 (2) 418 – 7
5. Roehrborn C.G. McConnell J.D. Lieber M, Kaplan S. Gittelman M. Malek G. H. Castellanos R, Coffield S, Saltzman B. Rudlinger M. Cook T. J, Waldstreicher J. Serum prostatic specific antigen concentration is a powerful predictor of acute urinary retaniona and need for surgery in men with clinical benign prostatic hyperplasia. PLESS study group. Urol 1999 ; 53 (3) 473 – 80
6. Barry M.J. Fowler F.J. Jr. o' Leary M.P. Brukewitz R.C. Bishop H.L. Mebust W. k. Cockett A.T.K. and the Measurement Committee of the American Urological Association. The American Urological Association symptom index for benign prostate hyperplasia J. Urol 1992; 148. 1549
7. Larkin GL. Weber J.E. Giant urethral calculi: a rare cause of urinary retention. J. Emerg Med. 1996; 14(6) 707
8. Bedii Salman A. Urethral calculi in children. J Paed Surg 1996 (10): 1379-82
9. Garcia Riestra V, Vareal Salgado M, Fernandez Garcia L. Foreign bodies. Apropos 2 cases (Spanish) Actas UROL 199 ; 52 (1) 74 - 6
10. Mattei F.M. Giovannelli V, Del Vecchio M.T. C Compagnoni firbroepithelial polyp of prostatic urethra in a adult. Ital Urol, Androl 1998; 70 (4): 173 – 5
11. Amrani A, el Quessar A, Belkacem R, Outarahout O. Benosman Ammar H. Polyps of the posterior urethra in children. Apropos of a case (French). Ann Uril 1997 31 (4) 225
12. Yeboah E. D Retention of urine. In Badoe E.A. Archampong E.Q. Jaja M.O.A (Eds) Principles and Practice of Surgery including pathology in the Tropics. Assemblies of Literature Centre. Ghana. 1994 790 – 792
13. Benekli M, Buyukasik Y, Haznedaroglu I.C. Savas M.C. Ozsan O. I. Chronic lymphocytic leukaemia presenting with urinary retention due to leukaemic infiltration of the prostate. Ann Haemat 1996 ; 73 (3) 143 – 4
14. Sekine H, Kojima S, Mine M, Yokokawa M. Intravesical prolapse cele presenting bladder outlet obstruction in an adult. Internat J. Urol (1): 74 – 6
15. Yeboah E. D et al. The causes and management of haematuria in Accra. Ghana MED. J. 1975 ; 14 : 299
16. Osegbe D. N. Amaku E. O. Haematuria in Nigeria: A prospective study. J. Trop Med. Hyg. 1984 : 27 :115
17. Fujisawa H, Igarashi S, Koyama T. Acute cauda equina syndrome secondary to lumber disc herniation mimicking conus medullaris syndrome – case report Neurol Med Chir (Tokyo) 1988, 38 (7): 429 – 31
18. Olapade – Olaopa E. O., Morley R. N. Carter C. J. Wessely H. Diabetic cystopathy presenting as primary acute urinary retention in a previously undiagnosed young male patients. J. Diabetes & Complic 1997; 11(6) 290
19. Tiguert R, Lewis R.A. Gheiler E.L. Tefilli M.V. Godoroja M. report : acute urinary retention secondary to sacral drome. Neurol & Urodynam 1999 ; 18 (2) 135

20. Dhote R, Lestang P, Zuber M, Gheradi R, Christoforov B. A cause of acute urinary retention; chloroquine – induced neuromyopathy (letter). Revue Du Rhumatisme, English Edition. 1996; 63 (1) 69.
21. Laitsikii N.A Aivazia I. A., Al-Shukhri S. Kh Grobachev .G.· The treatment of acute reflex urinary retention after operations in the ears of the rectal sphincter (Russian). Vest Khirugu Imen Grek 1996; 155 (6): 80
22. Espejo E, Cozar J.M. Tallada M. Psychogenic urinary retention. Diagnostic therapeutic approach (Spanish). Arch Espan Urtol 1997. 50 (6) : 603 – 7
23. Melilli G.A. Di Gesu G, Loizzi V, Vimercati A, Cormio G. Acute urinary retention in uterine myoma: description of a case (Italian). Arch Ital Urol, Andorl 1998; 70 (40): 163 – 4.
24. Abi Aad S.A Opsomer R. Obstructive retention in a young female: case report act Urol Belg 1996; 64 (4): 19 – 21.
25. Kisanga R.E., Abound M.M. Urethral mucosa prolapse in young girls. Cent. Afric J. Med. 1996; 42 (1) 31 - 3
26. Leung Y. L. Lee F, Tam P.A. Leiomyoma of female urethra causing acute urinary retention and acute renal failure. J Urol 1997; 158 (5): 1911 – 2
27. Yamanishi T, Yasuda K, Sakakibara R, Hattori T. Uchiyama T. Minamide M, Ito H. Urinary retention due to herpes virus infections. Neurol & Urodyn 1998; 17 (6): 613 - 9
28. Ginsberg P.C. Harkaway R.C. Elisco A. J. 3rd, Rosenthal B.D. Rare presentation of acute urinary retention secondary to herpes zoster. J Amer Osteopath Assoc 1998; 98 (9): 508 – 9
29. Lennon G.M. Desmond A.D. An unusual case of ischiorectal abscess presenting as acute urinary retention. Irish J. Med Sc. 1997 ; 166 91) 26 –7
30. Benghanem Gharbi M. Hachim K. Ramdani B. Zaid D. Acute urine retention Another presentation of a hydatid cyst of the kidney (French) J Urol 1997; 103 (1-2) 44 – 5
31. Mustonen S, Ala- Houshala I, Tammela T.L. Proteinuria and renal function during and after acute urinary retention. J Urol 1999; 161 (6): 1781 – 5
32. Olapade – Olaopa E. O. Morley R. N. Ahiaku E. K.N. Bramble F. J. Iatrogenic polydipsia: a rare cause of water intoxication in urology. Brit. J. Urol 1997; 79 488.

MANAGEMENT OF SEVERE BURN INJURY

OGUNDOKUN, A. O, ASHAYE, A. O,* ADETOSOYE, F

*At the time of writing, ** were second year and * first year Clinical Students at the College of Medicine, University of Ibadan.*

INTRODUCTION

Burns are coagulative lesions involving the surface layers of the skin usually caused by heat. Other causes include chemical agents and radiation. It may be classified based on the depth of tissue damage into partial thickness and full thickness varieties.

Burn is a universal occurrence and the home has been found to be the commonest site of injury with particular reference to the kitchen; use of hot water, boiling oil e.t.c. Higher incidence of burns has been reported in urban areas than rural areas, most of the affected populations are adults of working age group.

CLINICAL SIGNIFICANCE

Clinical significance of burns depend on:

- Age of patient; more fatal at the extremes of life
- Depth of burns; given the same total body surface, full thickness burns is more debilitating
- Percentage of body surface involved; >30% of body surface involvement is severe.
- Possible presence of inhalational injuries from inhalation of hot or toxic fumes.
- Promptness of therapy and prevention of wound infection and control of infection if wound is already infected.
- Partial thickness burn > 15% in a child is severe, partial thickness burn >30% in an adult is severe
- Full thickness burn >7.5% in a child is severe, full thickness burn > 15% in an adult is severe

EPIDEMIOLOGY

The epidemiology of burns has been observed to vary with mode of living and modification of the environment.

In Ibadan, Nigeria, most patients affected are above 15 years, most burns occur in December/ January, least incidence is between April and July. The home was the site of injury in most cases (66%), accidents in the street e.g. pedestrian road traffic accidents (30%) and work related burn injury (4%)[2]

In Malaga, Spain, most cases of burn injury occur in urban environment (89.5%). Most of these accidents occurred at home (65.8%) especially in the kitchen involving hot liquids. Incidence was higher in women (33.0%) against 21.1% in males. Most burns occurred on the hand

In Tehran, Iran, most of the patients involved were male (63%), the highest incidence recorded in age group 1-25. The most common cause of burns being kerosene in children however, the most common cause was boiling water. It was also noted that burn injury occurred more in illiterate people with men surviving more than women.

In the developed countries of the world, the incidence of burn injury was found to be due more to activity and way of living. In the developing countries, the way of life is a major factor in burn injury cases. Most people are of low socio-economic status, living in overcrowded flats lacking proper hygiene and using naked light source (candles and kerosene stoves without any protective measures.

AETIOLOGY

The causes of burns include

- Dry heat - naked flames
- Moist heat – hot liquids
- Chemicals – caustic soda, concentrated acids that can be accidentally swallowed or spilled
- Electricity – depending on resistance of tissues, extent of injury is higher in bone and skin
- Parasuicidal – burn injury inflicted on oneself in suicidal attempts

LOCAL RESPONSE TO BURNS

Skin

- There is disruption of basement membrane
- Blistering
- Vascular changes

3 zones are seen

a. Coagulation – cells in the area affected are damaged and undergoing necrosis
b. Stasis - peripheral to the zone of coagulation undergo heat injury and may become necrotic survive
c. Hyperemia - cells have been affected but recover normal conditions, there is vasoconstriction follo

by vasodilatation.

SYSTEMIC RESPONSE TO BURNS

Release of Mediators

- Cytokines; Interleukin (IL 1,2,3), γ- interferon. These may lead to:
  - Increase vascular permeability
  - Increase catabolism of muscle
  - Increase destruction of red blood cells
  - Initiation of wound healing
  - Fever
- Oxygen radicals; super-oxide, hydrogen peroxide. These cause
  - Alteration of vascular permeability
  - Red blood cell haemolysis
  - Disruption of the interstitial matrix
- Arachidonic acid metabolites (via cyclo-oxygenase pathway) prostaglandins which may cause
  - Vasosdilatation (via the lipooxygenase pathway)
- Leukotrienes C_4, D_4, causes
  - Vasoconstriction

Molecular response to trauma results in the release of:

- Catecholamines
- Aldosterone
- Antidiuretic hormone
- Histamine
- Thyroxine

The release of these mediators of trauma and burns lead to systemic effects, which include:

Cardiovascular system

- There is vascular damage and increased blood cell destruction.
- There is increased vascular porosity.
- Catecholamines cause peripheral and splanchnic constriction thereby decreasing blood supply to the gut.
- There is oedema and hypovolemia due to large extent involvement.
- Cardiac muscle damage from fluid over load, mediators, drugs and sepsis.

Renal

- Renal failure due to hypovolemia.
- Reduced glomerular filtration rate.
- Reduced parenchymatous flow.
- Precipitation of myoglobin and hemoglobin in tubules

Gastrointestinal Tract

- Curling's ulcer due to decreased splanchnic blood flow.
- Reflex paralytic ileus
- Gastric dilatation
- Gut barrier disruption and bacterial translocation due to splanchnic vasoconstriction

Upper Respiratory Tract

- No direct injury to airway except from steam
- Upper airway edema from components of smoke and chemicals e.g. noxious gases

Lower Respiratory Tract

- Restricted chest expansion from eschars may cause atelectasis.
- Interstitial damage and pulmonary edema.
- Carbon monoxide and cyanide poisoning.

MANAGEMENT OF ACUTE BURN INJURY

- ABC of resuscitation and first aid
- Fluid administration
- Parkland's formula – 1st 24 hours from onset of trauma, give 4mls/kg/% burnt body surface area. Half of the fluid total is given in 8 hours and the other half over the next 16 hours. This is limited to burns ≤50% of body surface.
- Hartmann's solution (Ringer's lactate) is preferred. Hypertonic saline is given to CVS patients but with a different formula.
- History – To note the site of injury, nature of injury (chemical, heat etc), whether injury was in a confined or open space.

Examination and evaluation of burns

- Initial investigations
  - Packed cell volume
  - E & U ± Cr (electrolyte, urea and Creatinine)
  - Chest radiography
  - Ventilation / perfusion ratio
  - Blood gas analysis
  - Parenteral analgesia + anti-tetanus toxoid. antibiotic cover (not at onset)
  - Antihistamines
  - Anti H_2 receptor blockers
  - Anti-coagulants to prevent DVT (deep venous thrombosis)
  - Intubation – should be performed early if/when there is inhalational injury

CLINICAL EVALUATION

- **Extent and depth of burns should be estimated**
  - ***Wallace's rule of nine***

Head and neck	-	9%
Each upper limb	-	9%
Anterior trunk	-	18%
Posterior trunk	-	18%
Each lower limb	-	9%
Perineum	-	1%

- *Lund and Browder chart in children below 12 years*

- **Depth – full and partial thickness burns**

Partial thickness burn – shows blisters, which sometimes burst. Surface of wound is pink and there is excruciating pain in the area, texture of skin is soft. It is divided into superficial and deep dermal burns.

Full thickness burns - No blisters, surface looks brown or white, charred or blackish. There is less pain, insensitivity to needle prick, areas of thrombosed vessels.

MANAGEMENT OF SUB-ACUTE PHASE

- Injury to red blood cells leading to anemia, blood transfusion is done
- Early wound cover, tangential or fascial excision if wound does not take skin graft. Tangential excision is contraindicated if burn is >10% due to excessive bleeding.
- Delayed wound cover on granulation tissue after escharotomy. However, there might be infection at this time.
- Treatment of fever due to wound infection, urinary tract infection (due to urethral catheterization, thrombophlebitis (I.V line), airway (chest infection).
- Physiotherapy
- Nutrition

INHALATIONAL INJURY

Inhalational injury occur in persons trapped in closed burning spaces e.g. cars, buildings and may be due to direct effect of the heat on the mouth, nose and upper airway or inhalation of toxic components in smoke.

Unlike shock, pulmonary manifestations may not develop for 24 hours, but this does not exclude the presence of injury. Inhalational injury is one of the determinants of mortality in major burn patients. Mortality ranges between 19% and 84%.[3]

Pathophysiology

Chemical injury from products of combustion, ammonia, nitrogen dioxide, sulphur dioxide and chlorine

- Irritation by this injury and damage to the mucosa.
- Oedema and airway obstruction.
- Increased airway resistance, reduced compliance and hypoxemia in immediate post-burn period.

Most mortality results from inhalation of toxic products and not from burn injury. Carbon monoxide combines with haemoglobin impairing oxygen availability. Cyanide paralyses the mitochondrial respiration by binding reversibly with cytochrome oxidase to stop phosphorylation causing hypoxia and acidosis.

Diagnosis

History of burns in a closed space, stridor, hoarseness circumhumoral burns and tachypnea occurring a few hours after a burn.

Management

- Life support measures, patent airway by intubation.
- Fibre-optic bronchoscopy.
- Blood Oximetry.
- Head of bed is elevated to 30° to decrease airway oedema and reduce pressure from abdominal contents which limit diaphragmatic excursion.
- If circumferential, full thickness burn of the thorax present, there is need for escharotomy.
- Therapeutic coughing and chest physiotherapy.
- Oxygen and oxygen tent.
- Fluid therapy to avoid hypovolemic shock, renal failure and early death in post- burn period.
- Inhalation injury increases fluid requirement of patients with burn by 40% – 70% in first 24 hours.
- Broad-spectrum antibiotics for prophylaxis before transfer to intensive care unit.

COMPLICATIONS OF BURNS

The complications of burns can be grouped into acute and chronic complications

Acute complications

- Shock
- Inhalational injury
- Infection
- Hypovolemia
- Acute renal failure
- Heart failure in the elderly
- Septicaemia
- Deep Venous thrombosis
- Hypoproteinemia
- Compartment syndrome

Chronic complications

- Chronic infections
- Ulcers
- Dyschronia
- Hyperpigmentation
- Hypertrophic scars
- Contractures, reduced joint mobility
- Marjolin's ulcers in burn scars

CONCLUSION

Despite the high incidence of mortality in patients with severe burns, adequate first aid management can increase the chances of survival of these patients.

PREVENTION

- Avoiding use of naked flames in and around the home and work places. Careful handling of hot water and boiling oil e.t.c. in the home.
- Proper labeling of chemical agents to prevent accidental; ingestion or spillage
- Provision of fire exits in high rise buildings, fire extinguishers in work places and well aerated work environment to prevent closed space injury
- Education on need for fire extinguishers in cars and buses to combat fire incidents
- Education on proper handling and trans port of patients from accident site
- Oxygen face mask or nasal catheter must be given to patients on transfer to the hospital
- Early monitoring of blood gases
- Early skin coverage to ensure reduction of septic complications and prevent contractures.

ACKNOWLEDGEMENT

Dr. O. M Oluwatosin, Consultant Plastic Surgeon, University College Hospital, Ibadan for his support and contributions in the writing of this article.

REFERENCES

1. Kumar V., Cotran R. S, Robins S. L. Basic Pathology 1997, 6th Edition 239.
2. O. M Fasika, Changing Pattern of Burn Epidemiology and Compliance Factor in Management at Ibadan. The Nigerian Postgraduate Medical Journal June 1997; 4
3. I. O Adigun, O. M. Oluwatosin, S. D Amanor-Boadu & O. A. Oluwole Inhalation Injury in Burns Patients in Ibadan. The Nigerian Journal of Surgical Research June 2001; 3
4. Fernandez – Morales E et al. Epidemiology of Burns in Malaga, Spain Medline. 23(4): 323-32 June 1997
5. Soltani K. et al. Epidemiology and Mortality of Burns in Tehran, Iran Medline. 23(4): 325-8 June 1998

EVALUATION AND MANAGEMENT OF PERIPHERAL VASCULAR INJURIES

SALAMI, M

Dr. Salami is a registrar in the Department of Surgery, University College Hospital, Ibadan and a former member of the Editorial Board of **DOKITA**

INTRODUCTION

Patients with peripheral vascular injuries present daily in emergency departments and trauma centers worldwide.These injuries may require immediate intervention to prevent loss of life or limb. Some may present with only subtle or occult symptoms or signs. Not surprisingly therefore, the presence of vascular injury goes unrecognized or is recognized late with resultant high incidence of complication and limb loss after delayed repair[1]. The aim of this paper is to highlight the fundamentals of peripheral vascular trauma and provide a current approach to the diagnosis and management.

AETIOLOGY

In Nigeria as in most developing countries, road traffic accidents remain the commonest cause of trauma in the civilian population[2,3]. In recent times, penetrating injuries especially from gunshots have featured more prominently as aetiological factor among patients[4]. In a combined retrospective and prospective study[5] of Gunshot Injuries (GSI) that presented to the National Orthopaedics Hospital Lagos, it was noted that gunshot injuries has almost become an epidemic unlike before when they used to be a sporadic event in Nigeria. The review showed a significant rise in trend ($P<0.05$) of the cases of GSI when the period of retrospective review was compared with that of prospective review. Vascular injuries occurred in 9% of the study population. Arterial and venous structures are most commonly injured by penetrating trauma with a much higher incidence in gunshot wounds than for stab injury. Blunt trauma also carries a significant injury rate, and iatrogenic vascular injuries are increasing with radiological and minimal access procedures becoming more commonplace.

PATHOPHYSIOLOGY

In the upper extremity, the axilla, medial/anterior upper arm and antecubital fossa particularly are considered high-risk areas due to the superficial location of the axillary and brachial arteries in these regions. Whereas in the lower extremity, the inguinal region, medial thigh, and popliteal fossa particularly are considered high-risk locations.

The injured artery may be contused, punctured, lacerated or partially divided; the intima may be dam or the vessel may be completely divided with separ of the ends.

These injuries result in hemorrhage, spa occlusion, thrombosis, dissection or the developm false aneurysms and arteriovenous fistulae. In a repo the management of 8 Nigerian patients with perip aneurysms, trauma was the main aetiological factor i these patients[16].

Both sharp arterial transections and crus tearing injuries cause arterial spasm; this combined the hypotension caused by blood loss and rapid pla deposition, quickly leads to reduction in, or cessati bleeding. Bleeding may be prolonged if there is a l tear in the artery or the laceration is held open. Whe blood pressure recovers muscle spasm may wear of platelet thrombi are expelled resulting in a reacti hemorrhage. A secondary hemorrhage can occur if infe erodes the arterial wall; this is usually between 7 a days after the initial injury.

Concealed bleeding and reactionary ede following injury may cause a rise in the intracompartm pressure leading to compartment syndrome.

Vascular injury has two main consequenc haemorrhage and ischemia. Ischaemia result from an a interruption of flow of blood to the limb. Oxygen supp inadequate to meet demand and anaerobic metab takes over, producing lactic acidosis and activating cel and humoural inflammatory pathways. If the arterial su is not re- established in time, cell death occurs.

Skeletal muscle can be rendered ischaemic f 6 hours and still recover function. Peripheral nerve more sensitive to ischaemia, and prolonged neurol deficits may result from relatively short period of t ischaemia. The sudden release of inflammatory medi lactic acid, potassium and other intracellular materi the circulation can cause profound myocardial depr generalized vasodilatation and initiate a syst inflammatory response.

The pathological changes in arterial ischae reversible in early stages and recover if treated vigo If they are allowed to progress, it leads to isch

sis and contracture.

AGNOSIS

The diagnosis of an arterial injury may be pletely clear-cut or equivocal. Hence, a high index of picion is a great aid in the preoperative diagnosis of rial injury. In some instances diagnosis will be made operative exploration of the vessel in question. The al presentation forms the bedrock of diagnosis with tional clarifying help from investigations.

NICAL FEATURES

STORY

Important historical information includes hanism of injury because this is an early pointer to the sibility of vascular involvement.

This is buttressed by the high incidence of rating trauma (63% - 82%) as aetiological factor in reported series.[1,7-12] The exact time of the injury is portant because the limits of warm ischaemic time essitate repair of arterial injury within six hours before versible nerve and muscle damage occurs. The umstances surrounding the injury (which could be of nic importance), patient's occupation, avocation and isure; co-morbid medical conditions (which include existent vascular disease, diabetes, AIDS and use of munosuppressive medication) and presence of ptoms of ischaemia such as pain, numbness or ness and paraesthesia suggesting peripheral nerve

SICAL EXAMINATION

The diagnosis of significant vascular injury rest entirely on the physical examination. Vascular injuries to be detectable solely by clinical examination in a ficant number of patients with a true positive rate of

Examination begins with palpation of the pulses the injury and comparison with the corresponding the uninjured limb. Detection of a pulse deficit is ely unreliable indicator of arterial injury however, quires further investigation rather than immediate

Specific signs are categorised into hard and soft differences in incidence of vascular damages.

Hard signs of vascular injury [12]

- Pulsatile bleeding
- Expanding or large haematoma
- Absent or reduced distal pulses
- Coolness of the extremity
- Lifelessness of the limb distal to the wound
- Pale limb
- Uncontrollable bleeding with direct pressure
- Presence of bruit or thrill

Soft signs of vascular injury [13].

- Presence of small non- expanding haematoma
- Peripheral nerve deficit
- Diminished pulse
- Injury In the proximity of major vessels (Proximate injury)
- A history of bleeding

Over 90% of patients manifesting hard signs have arterial injury while up to 35% of patients with soft signs have arterial injury[14].

In penetrating trauma to the extremities occult vascular injuries is found in 9% to 11.1% of cases[9, 15]. There are other asymptomatic patients who have sustained high-risk injuries; these include knee dislocation or severely displaced long bone fractures or dislocations.

In a retrospective study[16] of 52 patients at the Jos University Teaching Hospital (JUTH), the 3 most common presentations were active bleeding in 81%, pulse deficit in 65% and hypervolemic shock in 54%. Fixed skin staining was stated to be a late and omnious sign indicating unsalvageable limb. Furthermore, frank gangrene of the extremity was a relatively common finding occurring in 16% of cases due to initial intervention by traditional bonesetters in their environment.

INVESTIGATIONS

In the presence of massive arterial haemorrhage, there is no place for arteriography or other investigations. However, blood sample should be sent for grouping and cross matching before the patients is taken to theatre.

NON- INVASIVE TESTS

1. **Laboratory studies:** do not help diagnose injury but may assist with management:
   - Full blood count, electrolytes, urea and Creatinine
   - Prothrombin time and activated partial thromboplastin time
   - Serial hemoglobin measurements.
2. **Pulse Oximetry**: a reduction in oximeter readings form one limb, as compared to another is suggestive of vascular injury.
3. **Doppler Ultrasound:** a limb with a pulse deficit should be examined by means of a hand – held Doppler unit that amplifies sounds. A change in the quality of the pulse from the normal triphasic sound to biphasic or monophasic suggests a partial arterial

occlusion.

4. **Ankle / Brachial Index (ABI) or Arterial Pressure index (API);** The systolic arterial pressure in the injured limb is compared to the equivalent pressure in the uninjured extremity (API) or the systolic measured at the ankle is divided by the systolic pressure in the arm (ABI). A ratio less than 0.9 to 1.0 is considered abnormal and is ground for further investigation.

5. **Duplex Ultrasound:** The combination of Doppler with B -mode is called Duplex ultrasound. Greater information can be obtained regarding both venous flow (low-pitched, near continuous sound) and arterial flow (high – pitched, triphasic sound) in addition to directly visualizing vessels. Duplex can detect intimal tears, thrombosis, false aneurysms and arteriovenous fistulae. Doppler pressure measurements and Duplex scans have reported sensitivity of 83 to 95% and specificity of 97 to 100%[17,18]

6. **Plain X-ray** of injured extremity will help in determining the presence of fractured bones and foreign bodies. Certain fractures (e.g. supracondylar, humeral or femoral fractures) have a higher incidence of vascular injuries and recognition of these types of injuries alerts the clinician to the risk of vascular injury.

INVASIVE STUDIES

1. **Angiography**: This is now the gold standard for investigating arterial injury. It could be done in the angiography suite (for haemodynamically stable patients) or in the operating room. It is an expensive, invasive procedure that requires the mobilization of a specialized team to perform.

In most traumatic injury settings, angiography is best performed in the operating room. Indication for angiography include cases of diagnostic uncertainly, injuries peripheral to the axillary and common femoral arteries, presence of significant abnormalities on Doppler or Duplex scan and after repair to identify any unsuspected technical errors [19]. In the presence of hard signs arteriography will demonstrate arterial injuries in 90 – 98% of cases[12] while arteriography for " proximate injury" is positive in 3%[20], 4.6% [21], 13%[13]and 16%[22] of cases.

2. **Digital subtraction angiography**

This is an excellent alternative to the above. The advantages of seeing the vessels without background interference from bone together with the low volume of contrast that required for intra – arterial injection has established digital subtraction angiography as technique of choice.

MANAGEMENT

Airway control and respiratory assessment take priority over management of the circulation. The priorities of vascular injury are arrest of haemorrhage and restoration of normal circulation.

Care of the patients begin at the scene of the injury. prehospital care include stabilization of the extremity in anatomic position and control of bleeding by direct pressure to the area or proximally. Tourniquets are to be avoided unless there is no other means to control bleeding. The its application must be carefully monitored as the risk distal ischaemia and metabolic derangement following release is considerable. Tourniquet-related irreversible ischaemia is a known primary cause for amputation of injured arm [23].

FLUID RESUSCITATION

Two large bone venous accesses are necessary for giving warmed fluid rapidly in two phases: before and after haemorrhage control. Prior to haemorrhage control minimal fluid should be administered. Raising the blood pressure will increase haemorrhage from the vessel injury and dislodge any clot that has already formed.

Once haemorrhage control is achieved, then a phase of aggressive volume resuscitation to restore circulating blood volume. Warmed fluids – crystalloids, blood or clotting factors as necessary are administered to correct acidosis, hypothermia and coagulopathy, and restore perfusion rapidly to shut – down organ systems.

SURGERY

The use of manual injection arteriography has been demonstrated to have a sensitivity of 95.5% and specificity of 97.7%[13] and is especially relevant in developing countries where angiographic suites are not available or functional [4].

The basic principle of vascular repair is to obtain proximal and distal control of the relevant vessels before investigating the site of injury. Next debridement of devitalized tissue and definition of the wound edges is done and an assessment of inflow and outflow is made. If inadequate a balloon (Fogarty) catheter is passed proximally and distally to extract any thrombus. Heparinized saline is then instilled proximally and distally to further anticoagulate the vessels. The methods of vascular repair vary depending on the type, extent and location

...ury. Arterial repair may be by simple suture, lateral ...ntinuous suture, patch repair end-to-end anastomosis ...terposition grafting.

Vein graft replacement may be done using ...henous vein or the basilic vein. Prosthetic material ...Dacron) or polytetrafluoro ethylene (PTFE) can be used.

Also of importance is repair of major venous injury, ...gh ligation is considered acceptable when repair is ...t feasible[24,25].

In patients with fracture associated with vascular ...ma restoration of blood flow should be the priority ...s when intervention is early and signs of ischaemia ...minimal[8,24,26].

Need for fasciotomy is dictated by the degree of ...perative ischaemia and may precede the vascular ...r[8]. In cases of combined arterial and venous injury ...se of fasciotomy has merit [27].

The presence of soft signs of vascular injury is an ...tion for repeated clinical evaluation of the patients. ...se of ABI/API and ultrasound (Doppler or Duplex) ...paramount importance. Abnormal ultrasound findings ...erioration in pressure indices on repeat examination ...prompt surgical exploration [14].

...AGE CONTROL SURGERY

The principle of damage control surgery is ...cable to vascular trauma. The basic damage control ...ques are ligation and shunting [28]. These are very ...vessels that cannot be ligated in the extremities at ...risk to life and limb. Where there is a significant ...limb loss intraluminal shunts may be employed to ...rily restore flow.

...-OPERATIVE CARE AND COMPLICA-...

Post-operatively, frequent monitoring and vascu-...cks (e.g. pulse presence, quality capillary refill), ...continue for the first 24 – 48 hours. Anticoagula-...antiplatelet agents are important but are contrain-...in the presence of multiple injuries especially with ...ement of brain, spinal cord and eye [1,8,29]. Adequate ...n and urine output must be ensured.

Common complications include thrombosis of the ...owing of the vessel with primary repair and kink-...graft. Breakdown of an arterial repair can occur ...wound infection.

...COME

The site of injury, the associated injuries, and the ...erval prior to intervention determines the outcome ...ment. The two poorest prognostic indices of vas-cular trauma are increased ischaemic limb tissue time(ILT) and massive haemorrhage[16] .Patients with substantial soft tissue and skeletal injury accompanied by denervation usually end up with primary amputation[29, 30]. Though every attempt should made for salvage in lower limb injuries, the need for stratification according to damage is important for realistic prognosis and avoidance of futile efforts at reconstruction[30].

There is increased morbidity due to delay in diagnosis and management, particularly in orthoarterial trauma[1,6]. Fasciotomy significantly affects outcome; its omission or delay was responsible for 5 of 11 amputations in a series[25].

In the Jos series[16], the definitive surgical treatment of vascular trauma was accomplished in 49 patients. 46 arteries and 36 veins were surgically managed in the 82 vessels involved. There was a hospital mortality of 15%. Wound infection was recorded in 25% and limb disability in the form of paraesthesia and instability in 70%. Chronic venous insufficiency in 4% of the total number of patients was recorded 2 years following repair of vascular injuries. Also, knowledge of the anatomy of the brachial artery is crucial when indications for repair and the anticipated result are considered. Amputations rates vary considerably for injuries above and below the profunda brachial artery. During the World War II experience[31], the amputation rate was 56% when ligation occurred above this level and only 26% when ligation was below the origin of this major muscular collateral artery. The amputation rate was reduced to less than 5% in the Vietnam experience[32] due to prompt routine repair, and in some civilian series amputation has not been reported after repair[33].

CONCLUSION

A high index of suspicion and prompt management are the keys to management of peripheral vascular trauma. Improved emergency medical and surgical services faster transport time and improved surgical techniques will surely help in limiting morbidity and mortality. The need for an increased awareness of the possibility of major vascular injuries in patients with extremity trauma cannot be over-emphasized. This will lead to elimination of delay in diagnosis and ultimately improved outcome.

REFERENCES:

1. Adebo O. A. and Osinowo, O. (1986) Management of Peripheral Arterial Injuries at Ibadan. Vasc. Surg. 2. 55-60
2. Kale, O.A. and Aina K.A. (1976). Patterns of injuries in 455 people killed in road traffic accident. W. Afr. J. Surg 1 (3) 171 – 173
3. Jaja, M. O.A. (1976). The changing pattern of injuries in Africa W. Afr. J. Surg 1 (3) 162 – 166.
4. Adebo O. A. (1996) Limb Salvage in Peripheral Vascular

Trauma W. Afr. J. Med. 15 (3) 139 – 142.
5. Yinusa, W. and Ogirima, M.O. (2000): Extremity gunshot injuries in civilian practice. The National Orthopaedics Hospital, Igbobi experience, W. Afr. J. Med 19(4), 312-316.
6. Walker, G.F. (1963) Peripheral aneurysms in Nigerians. W. Afr. Med J. 12, 116 -122.
7. Anyanwu, C. H. Umeh, B.U.O.and Swarup, A.S. (1982); Experience with civilian vascular injuries in Eastern Nigeria. Angiology 33 (2), 90 – 92.
8. Keeley, S.B. Snyder, W.G. 3d, and Weigelt J.A. (1983) Arterial injuries below the knee; Fifty-one patients with 82 injuries J. Trauma 23 (4), 285 – 292.
9. Pasch, A.R. Bishara, R.A.; Lim. L.T; Meyer H.P. Schuller J.H. and Flanigan, D. P. (1986): Optimal limb salvage in penetrating civilian vascular trauma. J. Vasc. Surg. 3(2), 189 – 195.
10. Even, N. Ozgen, G, Ener. B.K. Solak H. AND Furtun, K, (1991): peripheral vascular injuries in children J. Paed. Sugr. 26(10), 1164 – 1168.
11. Andreev A., Kavrakov T., Karokolev J. and Penkov, (1992); Management of acute arterial trauma of the upper extremity.
12. Ordog. G.J., Balasubramanium, S, Wasserberger, J. Kram H, Bishop M, and Shoemaker W. Extremity Gunshot wounds (1994); Part one – Identification and treatment of patients at High risk of vascular injury J. Trauma 36 (3) 358- 368.
13. Itani K.M.F. Burch J.M.Spjut – Patrinely. V., Richardson, R, Martin, R.R. and Mattox, K.L. (1992) Emergency center Arteriography J. Trauma 32 (3); 302 – 306.
14. Edward Newton; (2000): Assessment and management of peripheral vascular injury in the ED. E Med Home. Com.
15. Shayne, P.H. Sloan E. P. Rydman. R and Barrett, J.A. (1994): A case – control study of risk factors that predict femoral arterial injury in penetration thigh trauma. Ann. Emer. Med. 24 (4), 678 – 684.
16. Igun G.O., Nwadairo A.Z., Sule V.M., Ramyil and Dakun N.K. (2001): Surgical experience with the management of vascular injuries. W. Afr. J. Med 20(2) 102-106.
17. Johansen, K. Lynch. K., Paun. M and Copass M, (1991) Non – invasive vascular tests reliably exclude occult arterial trauma in injured extremities J. Trauma 31 (4); 515 – 519.
18. Anderson R. J. Hobson R. W2d., Lee, B.C. Manno. J. Swan K.G. Padberg. F. T. Jamil, Z, Cambria R.A. and Breitbart. G.B. (1990) Reduce dependency on arteriography for penetrating extremity trauma: Influence of wound location and non-invasive vascular studies J. Trauma 30 (9), 1059 – 1063.
19. Robert W. Hobson II and Norman M. Rich (1994): Vascular injuries of the Extremities in Vascular surgery. Principles and practice 2nd Ed; 975 – 986.
20. Smyth, S. H. Pond G. D. Johnson P.L. Tauch R.F. McIntyre K.E.(1991): Proximity injuries; correlation of result of extremity arteriography J. vasc. And Intervention. Radiology. 2 (4): 451-454.
21. Trooskin S.Z. Sclafani S. Winfield J. Duncan A. Scalea Vieux E., Ateweh N, and Gertler J. (1993) The management of vascular injuries of the extremity associated with civilian firearms. Surg. Gynaecol. Obstet. 176 (4): 353 – 354.
22. Geuder J.W. Hobson R.W. 2d, Padberg. F. T. Lynch J. Lee B.C. and Jamil Z. (1985): The role of contrast angiography in suspected arterial injuries of the extremities. Am. Surg. 51 (2); 89 – 93.
23. Charles A. Hunt and John R. Kingsley, (2000) Vascular injuries of the upper extremity. South Med. J. 93(5): 464 – 468.
24. Menzolan, J. O. Logerfo. F.W. Doyle J.E. Hirsch E.F. Nowak M. Sequeira J.C. and Weitzman A.F. (1982): Management of Vascular injuries to the leg. Am. J. Surg. 144, 2[illegible]
25. Corgile J.S. Hunt, J.L. and Purdue G. F. (1992): Acute injuries of the femoral Artery and vein J. Trauma. 32(3): 364 – 370.
26. Hardy J.D. Faju S., Neely W.A. and Berry D.W. Aortic and other Arterial injuries. Ann. Surg. 181 (5): 649 – 653.
27. Padberg F.T. Hobson R.W. Fasciotomy in acute ischemia Semin. Vasc. Surg. 5: 52. 1992.
28. Karim Brohi (2002): Vascular Trauma Basics in Trauma Org. 7.3.
29. Drost, T.F. Rosemorgy. A.S. Proctor, D., and Kuman (1989); Outcome of treatment of combined orthopaedic and arterial trauma to the lower extremity J. Trauma. 29: 1331 – 1334.
30. Whitman G. R. McCroskey B.L. Moore, E.E. Pearce and Moore F.A. (1987): Traumatic popliteal and [illegible] tion vascular injuries determination of functional [illegible] vage. Am. J. Surg. 154 (6): 681 – 684.
31. DeBakey M.E., Simeone F.A. (1946): Battle injuries of teries in World War II. An analysis of 2471 [illegible] Surg123, 534.
32. Rich N.M, Baugh J.H., Hughes C.W. (1970): Acute injuries in Vietnam: 1000 cases. J. Trauma. 10:359.
33. Peacock J.B., Proctor H.J., (1977): Factors [illegible] tremity function following vascular injury. J Trauma. 17: 534.

DIARRHOEAL DISEASES IN CHILDHOOD

AKINBAMI, F. O.

Dr Felix O. Akinbami is a consultant Paediatrician at the University College Hospital, Ibadan and a Senior lecturer in Paediatric Gastroenterology, Department of Paediatrics, College of Medicine, University of Ibadan.

INTRODUCTION

Diarrhoeal disease remains a major cause of morbidity and mortality in children especially in the less developed countries of the world. It is estimated that in a year approximately one billion episodes of diarrhoea occur among children less than five years of age in Africa, Asia and Latin America, with more than 4 million deaths. Studies in Nigeria have also shown diarrhoea as the commonest cause of death among hospitalized children less than 5 years of age. Acute diarrhoea kills by causing dehydration, which leads to hypovolaemia and metabolic acidosis. Many children suffer repeated episodes of diarrhoea which lead to malnutrition as the result of anorexia, inadequate calorie and protein intake, and increased catabolism from infection. With the advent of Oral rehydration therapy (ORT), the mortality picture has changed in many parts of the world. ORT is effective in the prevention and treatment of dehydration caused by diarrhoea and has the potential to allow substantial reduction in the morbidity and mortality of acute diarrhoea in children.

DEFINITIONS

Diarrhoea simply means a change in the consistency of the stools to being abnormally loose or fluid and increase in the frequency of stools more than is normal for the age of the child. There is a wide range of normal stool habits in children which make more precise definition difficult, for example, Nigerian pre-school children open their bowels once every other day to 5 times per day [4]. However the passage of 3 or more loose stools in a day during infancy is generally considered abnormal.

Acute Diarrhoea (Gastroenteritis) :This is of sudden onset, and usually self-limiting with majority subsiding within a few days. It is the most common type of diarrhoea.

Persistent Diarrhoea: this term applies to diarrhoea that begins acutely but is of unusually prolonged duration (at least 14 days). In developing countries up to 13-20% of acute diarrhoea in children progress to persistent diarrhoea.

Intractable and Protracted diarrhoea are terms applied to diarrhoeal episodes, which are chronic (more than 4 weeks) for which no cause can be found and which do not respond to specific or non-specific forms of treatment.

Toddlers Diarrhoea means recurrent episodes of mild to moderate diarrhoea of variable duration in toddlers, for which no cause can be established and which are not associated with constitutional symptoms or nutritional impairment.

Dysentery: This is diarrhoea with visible blood in the stool. The most important cause is *Shigella spp*; other causes include *Campylobacter jejuni, enteroinvasive E. coli and Salmonella.*

EPIDEMIOLOGY

Acute diarrhoea occurs frequently in children between the ages of 6 months and 3 years. Diarrhoea in infants below 6 months is usually associated with early introduction of infant formula feeds, which are readily contaminated. Many factors predispose children to diarrhoeal disease including poor personal and environmental hygiene, poverty, lack of clean water, contaminated food supplies, overcrowding especially in the urban slums, illiteracy, ignorance and malnutrition. Several studies in developing countries have shown that the increase in diarrhoeal diseases is related to the decline in breast feeding, poor weaning and the increasing trend towards bottle-feeding.

SEASONALITY

In some parts of the world, there is a seasonal variation in the prevalence of diarrhoea. In temperate climates, bacterial diarrhoea tends to occur more commonly during the warm season, whereas viral diarrhoeas, particularly due to Rotavirus, peak during the winter. In tropical countries, including Nigeria, viral diarrhoeas, tend to occur throughout the year, with an increase during the drier, cooler months, while bacterial diarrhoeas tend to occur

more during the warmer, rainy season.

AETIOLOGIC FACTORS

Diarrhoea is in most cases caused by 3 major groups of microorganisms - viruses, bacteria and protozoa or parasites (Table 1). All over the world, viruses especially Rotavirus have been identified as the major causes of acutediarrhoea, in childhood. Studies in Nigeria also found viruses as the major cause of diarrhoea in up to 60% of cases and bacterial organisms accounting for only 10-20%.

Table 1 Diarrhoea causing pathogens

Viruses
Rotavirus
Norwalk and Norwalk-like agents
Coronae-like virus
Adenovirus

Bacteria
Escherichia coli
Shigellae
Salmonella (non typhoid)
Campylobacter jejuni
Vibrio cholerae
Yersinia enterocolitica
Clostridium perfringens and difficile

Parasites
Cryptosporidium
Giardia lamblia
Entamoeba histolytica

PATHOPHYSIOLOGY

In the gastrointestinal tract, there is a normal homeostasis maintained by an interaction between absorption and secretion of fluids and electrolytes. Diarrhoea ensues as a consequence of derangement of the normal homeostasis. There are several mechanisms, which may be operative in acute diarrhoea:

(i) **Toxin production** (e.g. *Vibro cholerae, Enterotoxigenic E. Coli*). Bacterial pathogens after ingestion proliferate and elaborate enterotoxins within the intestinal lumen, which act on a morphologically intact mucosa. These enterotoxins stimulate receptors at the mucosal surface and induce the production of excess cyclic adenosine monophosphate (cAMP) by stimulating the action of adenyl cyclase. This inhibits influx of sodium chloride and water into the villous cells and also induces secretion of NaCl and water by the crypt cells. The net result of these two changes is the secretion of water and electrolyte. The glucose-stimulated sodium absorption of the gut is however not affected.

(ii) **Mucosal adherence with local cytopathic fect** (e.g. *Enteroadherent E. Coli or Enteropathog E. Coli*). These organisms adhere tightly to the muc and cause effacement of the microvilli without invading crovilli.

(iii) **Mucosal invasion** (e.g. *Shigella Enteroinvasive E. Coli.*) These organisms invade destroy mucosal epithelial cells resulting in shedding cells with formation of micro-ulcers and an overlying bl exudate. These changes occur mostly in the colon terminal ileum. Rotavirus replicate within the villous thelium and cause patchy mucosal damage. There is associated loss of disaccharidase enzymes.

(iv) **Osmotic diarrhoea** – increase in the osmo of the intestinal luminal content e.g. ingestion of os cally active substance such as lactose by children wit tase deficiency.

(v) **Alteration in intestinal motility.**

(vi) **Inhibition of the active transport in th lon.** Two types of congenital defects involving (i) H^+ and (ii) Cl^- - HCO_3^- exchange mechanisms have reported. These congenital transport defects are ver and they give rise to very watery diarrhoea presen birth.

CLINICAL FEATURES

Apart from the diarrhoea that the child will p with there are some other relevant features which m helpful:

- Prodrome from the diarrhoea illness shou one suspect viral agents.
- Vomiting is a common complaint and ofte cedes the diarrhoea by up to 48 hours.
- Fever is more frequent with invasive orga but is of no diagnostic value as it occurs both in v bacterial diarrhoea and may also be due to dehy It should be assessed in its own right. The fever m result in febrile convulsions.
- Abdominal pain, blood and mucus in st gest invasive organisms.
- Ask about urine output. No urine passed eral hours is an important sign of severe dehydra

MANAGEMENT OF DIARRHOEA

The basic objectives of treatment of diarrhoea (a) prevention of dehydration, (b) correction of d tion (c) maintenance or improvement of nutriti treatment of the aetiological agents.

Prevention of dehydration

When a child passes one diarrhoeal stool, it s assumed that dehydration would set in, even if the appears well initially. Therefore the first line of

Table 2 : Comparison of stool electrolyte composition in diarrhoea due to differential organisms and the WHO recommended ORS

	Electrolyte (mmol/L)					
Aetiology	Na^+	K^+	Cl^-	HCO_3^-	(citrate)	Osmolality (mOsmol)
Cholera	88	30	86	23		300
Rotavirus	37	38	22	6		300
ETEC	53	37	24	18		300
ORS	*90*	*20*	*80*	*30(10)*		*300 (+ 110mmol glucose)*

is prevention of dehydration, which can be achieved should start at home by the mother or the caretaker. mothers or caretakers are advised to (i) give extra orally, (ii) prepare a standard salt sugar solution (SSS) 2) and give the child slowly with a cup and tea- (iii) continue breastfeeding, (iv) take the child to ital if the diarrhoea persists or if the child is becoming

dration

Risk Factors

Children are at higher risk of developing dehydration use of their larger body surface area/body weight ratio, ger body content of water/unit body weight and higher al metabolic rate. Studies have shown that factors such age below 12 months, vomiting (>2/day) and severe utrition are high risk factors for dehydration in a child diarrhoea and should be looked for.

rrection of Dehydration

is is simple both in concept and execution, more so with advent of ORS. It involves the following steps:

Weigh the child

Assess for presence/severity of dehydration based on (a) recent weight loss (if possible) OR (b) clinical signs of dehydration

Calculate and correct the initial loss (deficit)

Provide maintenance and replace on going losses

Rehydration (deficit correction) can be achieved her orally or intravenously. For most children, except se severely dehydrated and who cannot drink, the oral te is used by giving oral rehydration salt solution.

Oral Rehydration Therapy (ORT)

During the last three decades, oral rehydration therapy has had unparalled success in the treatment of diarrhoea. The most widely used solution is the World Health Organisation oral rehydration solution (Table 3). ORT olves the mother and provides an opportunity for health ucation that has an educational effect on the community.

The use of ORS is based on 2 scientific facts:

(i) Solution containing both Na^+ and glucose will maximize water absorption in the small intestine by using the electrogenic Na^+ pump.

(ii) More important, the sodium/glucose co-transport mechanism and other absorptive mechanisms of the gut are maintained during acute diarrhoea, even in the face of considerable intestinal damage.

Amount of ORS required

This depends on the degree of dehydration

- For some (mild) dehydration, 75 ml/kg of ORS over 4 hours
- Severe dehydration (>10%), 100-150ml/kg as ORS over 4-6 hours
- If the child vomits, ORS is given in small frequent volumes
- The child should be reassessed after 4 hours and more frequently in severe cases

If the child is well hydrated, he is discharged home. Before discharge, the mother is advised on the child's need for extra fluid, increased feeding and also to give ORS, 10ml/kg for every watery stool passed.

ORT is inappropriate for

- Initial treatment of severe dehydration with signs of shock
- Patients with paralytic ileus or marked abdominal distention
- Patients unable to drink – ORS solution can however be given to such patients through Nasogastric Tube, if IV access cannot be obtained.

ORT will not be successful in

- Patients with very rapid stool loss (≥15ml/kg/hr)
- Patients with severe, repeated vomiting
- Patients with glucose malabsorption (rare)

Intravenous Therapy (IVT)

This is mainly used for initial treatment of severe (life threatening) dehydration, to rapidly restore blood volume and correct shock and sometimes in situations mentioned above.

In *severe dehydration with shock*, 30ml/kg of normal saline (or ringer's lactate) is given rapidly over 30-60 minutes and can be repeated until organ perfusion is restored. If the child can drink, continue rehydration orally with ORS.

In case the child is still *not able to drink*, after boluses above, rehydration is continued intravenously using 0.45% Dextrose (or 0.18% saline in 4.3% dextrose based on serum sodium values) thus:

- give 70 ml/kg in 5 hours (for infants), or in 2 ½ hours (for the older child)

OR calculate the deficit and maintenance and give:

- ½ deficit and 1/3 maintenance in 8 hours and the remaining deficit and maintenance over the next 16 hours.

* Add KCl to intravenous fluid (10mmol/500ml bag) as soon as urine is passed.

Role of Drugs in the Treatment of Acute Diarrhoea

The goal of drug therapy is to decrease stool water and electrolyte losses, thereby limiting the morbidity resulting from dehydration. In addition, the drug should obviate the need for IV therapy, must be safe and effective, compatible with ORT and cheap. It should not affect normal gut function. To date, several drugs have been tried in the treatment of acute diarrhoea, but none has met the requirements enumerated above. In addition, most episodes of acute diarrhoea in children are caused by non-bacteria agents and are self-limiting. Thus no anti-diarrhoeal, anti-emetic or anti-secretory agent is of any proven practical value and some are also dangerous.

Antimicrobials are **only** indicated in shigella infection and systemic salmonellosis or salmonella enteritis in the very young, in the immunocompromised and in those who are systemically ill.

ORT and Nutrition

One of the main arguements for not feeding during acute diarrhoea is based on the idea that intestinal absorptive function is compromised during acute diarrheoa and that feeding may worsen the diarrhoea. However, studies have clearly shown that the digestive and absorptive functions of the gut remain near normal during an episode of diarrhoea. Fasting reduces intestine enzyme activity, secretion of gastric acid and results in flattening of the intestinal villi. In addition many researchers have demonstrated that in spite of continued feeding, both stool volume and duration of diarrhoea were reduced in children with acute diarrhoea compared to a control group who were kept fasted. Therefore in order to avoid nutritional injury, t emphasis during ORT, is on continued feeding (includi breast feeding). If the child is on infant formula, it may necessary to temporarily reduce the amount of the m Non-lactose containing formula can be used, as an alt native for a few days before normal feeding is re-est lished. When diarrheoa has subsided, extra feeding sho be encouraged.

PREVENTION AND CONTROL OF DIA RHOEAL DISEASE

Health education is a very important component i programme of control of diarrhoeal disease in childr During ORT mothers are educated on personal and e ronmental hygiene, the causes of diarrhoea in children, h to recognise simple signs of dehydration and how to p pare the standard SSS at home. Other effective prev tive measures include promotion of breast feeding, mea immunization, supplementary feeding, improvemen water supply and sanitation facilities.

COMPLICATIONS OF ACUTE DIARRHOE DISEASE

Oliguria:

- This indicates either continuing dehydrati acute renal failure
- Rehydrate fast with 20-30ml/kg NaCl or pl over 30 minutes
- Above may be repeated and then continue a vere dehydration
- If urine is inadequate at four hours, give fur 1ml/kg.
- If urine is still inadequate treat as Renal fail

Table 3- Formula for Salt Sugar Solution

1 level (3ml) teaspoon of salt - 2g (60 mmol/l Na)
10 level (3ml) teaspoon or 5 cubes of sugar - 20g (80 mmol/l sucrose)
1 beer bottle (or 2 soft drink bottles) of clean water - 600mls

Hypokalaemia:

- Presents as hypotension, abdominal disten bradycardia during dehydration
- Malnourished children particularly at risk
- DO NOT start correction unless the child i ing urine.
- DO NOT give potassium as a bolus. It sho added to the IV fluid.
- IV fluids should NOT contain more tha 60mmol/lit of potassium. Ideally correction sh be under ECG monitor.

...osis:

- Give 1ml/kg $NaHCO_3$ (diluted) IV slowly over 15-20 minutes or use formula – (0.3 x wt x Base deficit) ml and correct over 8 hour period.

...mulsion:

- Could be due to fever or fluid/electrolyte imbalance (especially hypernatraemia)
- Exclude Meningitis
- Treatment – Paraldehyde or Diazepam

...mary Oedema:

- Due to simple overload
- commoner with acidosis
- treatment – IV frusemide

...e intolerance

- Persistent lactose intolerance (lasting more than 10 days) may occur
- lactose free diet indicated

...MARY

1. Acute infectious diarrhoea is a major killer of children in the developing world
2. It is also an important cause of malnutrition
3. Rehydration therapy especially ORT, is the cornerstone of treatment of acute diarrhoea
4. During rehydration therapy, feeding should be continued
5. Antisecretory and antidiarrhoeal agents have no place in the treatment of acute diarrhoea
6. Antimicrobials should be used prudently
7. Promotion of breast-feeding, immunization against the preventable killer diseases and health education on personal and environmental hygiene are important control measures.

REFERENCES

1. Cutting W.A.M., Omer R.I., McLean S.D. A worldwide survey on the treatment of diarrhoeal disease by oral rehydration. Annals Trop Ped 1981; 1: 199-208
2. Snyder J.D and Merson M.H. The magnitude of the global problem of acute diarrhoeal disease: a review of acute surveillance data. Bulletin of the World Health Organization 1982; 60: 605-613
3. Okeahialam T.C. Gastroenteritis in children. The Nigerian Med Pract. (Suppl) 1983; 3: 11-14
4. Akinbami F.O., Erinoso O., Akinwolere. Defaecation pattern and intestinal transit time in children. Afr J Med Sci. 1995; 24: 337-341
5. Maiya P.P., Pereira S.M., Matham M. et al. Aetiology of acute gastroenteritis in infancy and early childhood in Southern India. Arch Dis Chidh 1977; 52: 482-485
6. Rhode J.E., Northrup R.S. Taking science where the diarrhoea is in CIBA Symposium 42, Acute diarrhoea in Childhood. New York, Elsevier Excerpts Medica, 1976
7. Molla A.M., Rahman M, Sacker S.A., Sack D.A. and Molla A. Stool electrolyte content and purging rates of diarrhoea caused by rotavirus, enterotoxigenic E. coli and V. cholerae in children. J. Pediatr 1981; 98: 835-838
8. Editorial. Oral Rehydration. The time has come. Lancet 1983; 2:259
9. Grange A.O., Okeahialam T.C., Seriki et al. Standardization of home made salt sugar solution for the treatment of acute diarrhoeal disease of childhood in Nigeria. Nig J. Paed. 1985; 2: 246-249
10. Okeahialam T.C. and Grange A.O. Oral rehydration therapy. An overview. 1998

END ORGAN CHANGES IN HYPERTENSIO A TRIPLE TRIAD

*ASANGANSI I, OGUNSUA A

* At the time of wrting, the writers were 1st year clinical students of the College of Medicine, University of Ibadan

INTRODUCTION

Hypertension is one of the world's major scourges. It has for long been recognised globally as the most prevalent cardiovascular disease and is a known risk factor in the development of stroke, coronary artery disease, congestive heart failure, and renal insufficiency.

Stephen Hales first measured blood pressure in 1773. He described the importance of blood volume in blood pressure regulation. The suffix ***-tension*** is related to the word ***tone***, which was first described in relation to peripheral arterioles by Lower in 1669 and subsequently by Senac in 1783. With the likes of Bright, Johnson, Gull, Sutton, Bernard, Edouard, and many others, the 19th century was a busy period of research that exposed more facts about this disease.[1] However, it was the observations of Janeway and Walhard that led to the recognition of target organ damage in hypertension and consequently the branding of hypertension as 'the silent killer'.

TABLE 1 - CLASSIFICATION OF SEVERITY OF HYPERTENSION

CATEGORY	*SYSTOLIC*	*DIASTOLIC*
Optimal	*<120*	*<80*
Normal	*<130*	*<85*
High Normal	*130-139*	*85-89*
Grade 1 hypertension (mild)	*140-159*	*90-99*
Subgroup borderline	*140-149*	*90-94*
Grade 2 hypertension (moderate)	*160-179*	*100-109*
Grade 3 hypertension (severe)	*?180*	*?110*
Isolated systolic hypertension	*?140*	*<90*
Subgroup borderline	*140-149*	*<90*

DIAGNOSTIC CRITERIA

According to the World Health Organization (1999), hypertension is defined as a systolic blood pressure of 140mmHg or greater and/or a diastolic blood pres 90mmHg or greater in subjects who are not taki hypertensives[2]. The table below shows the classification of blood pressure levels (in mmHg) o over 18.

When the systolic and diastolic blood pressure of a patient fall into different categories, the higher should apply. Classification is based on the averag or more readings taken at each of two or more vis initial screening.[3]

TARGET ORGAN DAMAGE (TOD)

After a long period, hypertension tends to cau damage to the heart, the aorta and small arter kidneys, the retina and the central nervous syste process, as described by Sharma et al[1], begins co with **pre-hypertension** in persons aged 10-30 ye **early hypertension** in persons aged 20-40 years (increased peripheral resistance is promine **established hypertension** in persons aged 30-5 and finally to **target organ damage** in perso years and beyond. Although, this is the usual s variation in age of onset and rapidity of progressio in individual patients, probably determined in part b influences, including underlying genetic f environmental factors, early diagnosis and treatm drug compliance.

GENERAL PATHOPHYSIOLOGY OF TA ORGAN DAMAGE

Hypertension is a vascular disease and mu pathogenesis involves structural changes in resista vessels leading to the classical picture of arterios It causes small vessel arteriosclerosis kn arteriolosclerosis and large vessel arteriosclero as atherosclerosis. The third classical pat arteriosclerosis, Monckeberg medial calcific s though found in old age, has not been li hypertension.[5]

...iolosclerosis (small vessel arteriosclerosis)

...mechanical effects of high blood pressure on the ...vessels cause arteriosclerosis. The two patterns of ...sclerosis include:

- Hyaline arteriolosclerosis and
- Hyperplastic arteriolosclerosis

...e arteriolosclerosis

...the smallest arteries and arteriles, hypertension ...se a glassy, homogenous, pink hyaline thickening ...blood vessel walls known as hyaline ...sclerosis. Benign arteriolosclerosis is a term that ...the change that occurs in mild chronic hypertension. ...four mechanisms- changes in pulsatile flow, ...lial cell damage, smooth muscle growth, and ...remodelling- contribute to the development of ... arteriolosclerosis. Progression of the ...sclerotic process leads to leakage of plasma ...nts and increased extracellular matrix production ...muscle cells. This form of arteriolosclerosis is ...evident in renal parenchyma, where it causes ...ischaemia and symmetrical shrinking of the ...referred to as benign nephrosclerosis.[5]

...plastic arteriolosclerosis

...plastic arteriolosclerosis is generally related to ...or very severe elevations of blood pressure ...fore characteristic of but not limited to malignant ... (malignant hypertension is defined as diastolic ... > 140mmHg normally with features of ...a, fibrinoid necrosis and other target organ ... Under light microscopy, it is visible as an ***onion*** ...centric, laminated, thickening of the walls of ... with progressive narrowing of the lumina. ...microscopy reveals that these laminations consist ...muscle cells and thickened and reduplicated ...membrane. When cell necrosis is combined with ...of plasma proteins in the vessel wall, it is termed ***necrosis***. ***Necrotizing arteriolitis*** is a term ...to the accompaniment of hyperplastic changes ...necrosis. Arterioles in all sites of the body ...to hyperplastic arteriolosclerosis but certain ...to be particularly susceptible such as the gall ...peripancreatic/intestinal arterioles, and the ...where it is called ***malignant nephrosclerosis***).

...sclerosis (Large vessel arteriosclerosis)

...is large- and medium-sized vessel ...sis, characterized by the formation of intimal ...ques that have a central grumous core rich in lipid. Atherosclerotic lesions develop because of two critical processes:

(1) Proliferation of intimal smooth muscle cells and
(2) Accumulation of lipid.

As the lesions form, there is infiltration by macrophages, fibroblasts, and lymphocytes. The endothelium breaks down and small capillaries may penetrate the vessel wall. There is vascularisation of the plaque with endothelialised channels called ***vasa plaquorum***. The lesions expand, coalesce, and may eventually cover the entire surface producing the final clinical result, occlusion of the artery.

However, in addition to hypertension, other factors have been associated with atherosclerosis and they facilitate the formation and spread of the lesions. These factors include:

Non-modifiable factors - aging, male gender, family history, and genetic abnormalities

Potentially modifiable factors - other components of the metabolic syndrome X (diabetes, some forms of obesity, hyperlipidaemia) and cigarette smoking.

Lesser/non quantitated factors - some forms of obesity, physical inactivity, stress (type A personality), homocysteinaemia, postmenopausal oestrogen deficiency, alcohol, lipoprotein (a), hardened (trans) unsaturated fat intake, and ***Chlamydia pneumoniae.***

Common sites of clinically significant atherosclerosis in order of frequency include 1.abdominal aorta and iliac arteries, 2.proximal coronary arteries, 3.femoral and popliteal arteries and thoracic aorta, 4.internal carotid arteries and 5. The vertebrobasilar system.

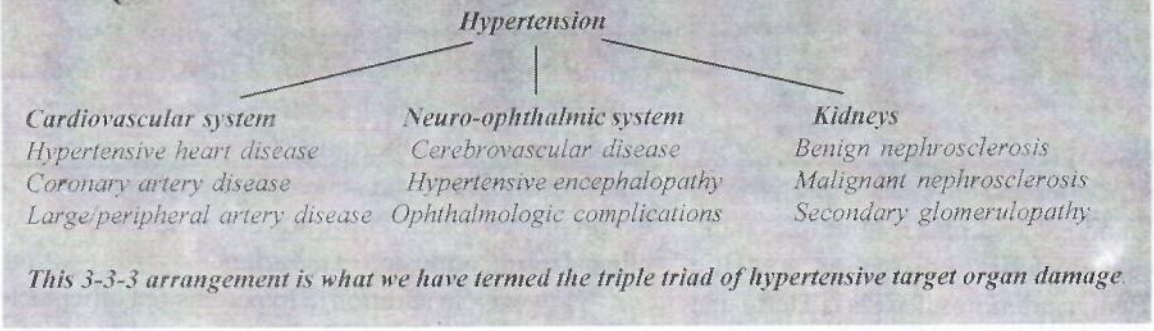
STUDIES ON TARGET ORGAN DAMAGE IN HYPERTENSIVES

In his paper published in 1955, Perera reported his findings on 500 men with untreated hypertension, which he followed until their death. Their mean age was 32 years and mean survival was 20 years. He found out that the presence of end organ damage greatly reduced life expectancy. The presence of left ventricular hypertrophy and congestive heart failure reduced the mean survival to eight and four years respectively. The occurrence of other complications such as coronary artery disease (CAD), cerebrovascular disease (stroke), encephalopathy, accelerated hypertension, and azotaemia had a significantly negative impact on observed mortality[6]. Numerous other studies have supported his findings and it Is now generally accepted that chronic elevation of blood pressure involves the cardiovascular system, the renal system, the eyes, and the central nervous system.

The possible complications of hypertension areshown

shown in the schematic diagram below: shown in the schema below:

FIGURE 1: TARGET ORGAN DAMAGE IN HYPERTENSION



This 3-3-3 arrangement is what we have termed the triple triad of hypertensive target organ damage.

The presence of target organ damage must be searched for in patients with longstanding hypertension. This is because when such changes are found along with an elevated blood pressure, an aggressive reduction of blood pressure is required especially if the target organ changes are acute, the condition is then termed a hypertensive emergency. Malignant (accelerated) hypertension is a hypertensive emergency. Malignant hypertension is said to occur when blood pressure rapidly rises with diastolic pressure greater than 140 mmHg usually with characteristic findings of fibrinoid necrosis histologically and papilloedema clinically. There is a risk of cerebral oedema and haemorrhage.

There are marked changes in the retinal vessels and these are diagnostic of malignant hypertension. The separation of malignant hypertension from accelerated hypertension is based on the presence of ***flame-shaped haemorrhages*** and exudates (stage 3 Keith-Wagener-Barker retinopathy) in accelerated hypertension and papilloedema (stage 4 retinopathy) in malignant hypertension.

However, there is evidence that the survival rate of those with papilloedema (malignant hypertension) or without papilloedema (accelerated hypertension) is so similar that there is little reason to separate the two.

CARDIOVASCULAR COMPLICATIONS

Hypertensive heart disease

Uncontrolled and prolonged hypertension can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes can lead to development of left ventricular hypertrophy (LVH), coronary artery disease (CAD), various conduction system abnormalities and systolic and diastolic dysfunction of the myocardium, which clinically present as angina, myocardial infarction, cardiac arrhythmias (especially atrial fibrillation) and congestive heart failure (CHF). Hypertensive heart disease is generally applied to heart diseases such as left ventricular hypertrophy (LVH), coronary artery disease (CAD), cardiac arrhythmias, and congestive heart failure by direct and indirect effects of elevated blood pressure. Acute elevation of blood pressure can also lead to diseases traditionally associated with chronic hypertension.

Left ventricular hypertrophy (LVH) is an adaptive response of the myocytes to pressure overload. Most of people with hypertension have left ventricular hypertrophy. Pressure stress on the ventricular wall leads to the production of new myofilaments within the myocytes. There is an increase in the number of mitochondria, ribosomes and nuclear enlargement occurs. New myocytes are not produced but there may be production of new sarcomeres alongside existing ones. At the molecular level the alpha-1 adrenergic receptors activate intracellular transduction proteins and RNA transcription factors. Angiotensin II acting on angiotensin I receptors in myocytes also leads to the growth of interstitium and collagen components. There is an association between left ventricular hypertrophy in hypertension and the presence of ACE genotype, which is an ACE gene deletion polymorphism.

In compensated hypertensive heart disease the heart size may reach 500g (normal 250 - 300g in females and 300 - 350g in males) and the thickness of the ventricular wall may exceed 2 cm (normal 1.3 - 1.5cm). Hypertrophy plays a protective role but later it leads to dilatation and ultimately systolic dysfunction. LVH is a powerful predictive marker in hypertension and its pattern is important. The alterations in diastolic and systolic dysfunction may lead to a progressive pump failure or congestive heart failure. About 91% of people who develop CHF have pre-existing hypertension.

Coronary artery disease (CAD)

The development of CAD in hypertension is multifactorial. Hypertension accelerates

...iosclerosis and causes increased coronary vessel ...istance. However, angina in the absence of epicardial ...nary artery disease (syndrome X) could occur in ...ertension for two major reasons, which include:

1. Compromise of coronary blood flow in diastole secondary to increased left ventricular wall tension and transmural pressure.
2. Dysfunctional microvasculature beyond the epicardial coronary arteries making the coronaries unable to compensate for increased metabolic and oxygen demands.

...ypertension is also associated with decreased nitric ...ide, which further promotes the development of ...eriosclerosis and fibrofatty plaque formation.

...rrhythmias

...rrhythmias common in hypertensive patients include ...l fibrillation, premature ventricular contractions, and ...tricular tachycardia. The atrial fibrillation in hypertension ...uld be paroxysmal, chronic recurrent or chronic ...sistent. The risk of sudden death is increased. Theories ...plain these arrhythmias include altered cellular structure ...metabolism, lack of homogeneity of the myocardium, ...r perfusion of conducting tissue, myocardial fibrosis, ...ctuation in after-load.

...rge vessel disease

...neurysms of the thoracic or abdominal aorta (fusiform, ...ular, cylindrical, or even dissecting), renal, mesenteric, ...iac arteries, and atherothrombotic obstruction of the ...minal aorta or its branches have been documented in ...ferent studies of end organ damage in hypertensive ...jects. Aneurysms greater than 6cm rupture within 10 ...rs. Severe hypertension can cause aortic root dilatation ...ding to significant aortic insufficiency.

...ripheral vascular disease

...hronic hypertension predisposes to peripheral vascular ...ease, which manifest as intermittent claudication or the ...re ominous gangrene of the extremities. Generally, ...ertension doubles the risk of peripheral vascular ...ease. Other factors- prominent among them is diabetes- ...also involved.

...EURO-OPHTHALMOLOGIC ...MPLICATIONS

...EREBROVASCULAR DISEASE

...The brain needs a constant supply of large amounts of ...gen and nutrients via the blood because of its incredibly ...gh rate of metabolism. To maintain the flow of blood, ...regulatory mechanisms that function by changing the ...ure and luminal diameter of brain resistance vessels (in ...nse to changes in perfusion) are in place. However, arteriosclerosis, the hardening of vessels, impairs this autoregulation. Therefore, hypertension, which predisposes the aorta, the vertebral and carotid arteries to arteriosclerosis and causes arteriosclerosis and lipohyalinosis in small diameter penetrating end arteries, impairs this autoregulation.

Ischaemic cerebrovascular disease

Tissue ischaemia (ischaemic strokes) occurs when perfusion of the brain or part of the brain falls below a critical level. In hypertensives, 75-80% of strokes are ischaemic, either embolic or thrombotic. A good number of ischaemic strokes occur in the morning.

Transient ischaemic attacks (TIAs) in hypertensives are usually due to temporarily inadequate blood supply from embolisation of atherosclerotic plaques.

Lacunar stokes represent 20% of the ischaemic stokes. They occur when the penetrating branches of the circle of Willis, vertebral artery or basilar artery become occluded secondary to arteriosclerosis. The important clinico-pathologic outcome of these arteriolar lesions is the development of single, or multiple, small cavitary infarcts – lacunes or lacunar state (etat lacunaire) also called lacunar softening. These lake-like spaces (not more than 15mm in diameter) consist of lost tissue and surrounding reactionary gliosis. Depending on where they are located, lacunar softening may be either symptomatic or asymptomatic. Of all stroke types, lacunar strokes have the best prognosis. However, minute silent infarcts occur commonly in the elderly and may lead to dementia.

Watershed infarcts also known as border zone infarcts also occur (usually due to general hypoperfusion).

Hemorrhagic cerebrovascular disease

Intracranial haemorrhage may be intracerebral, subarachnoid, or mixed and a vast majority of intracranial bleeds are secondary to hypertension (most commonly malignant or accelerated). Cerebral haemorrhages that occur without trauma are referred to as *'spontaneous'*. Hypertension, however, is the most common and important cause of spontaneous haemorrhage in blacks (amyloid angiopathy in Caucasians).

Hypertensive intracerebral haemorrhage occurs at preferential sites, which are in order of frequency, the basal ganglia - thalamus (65%), the pons (15%) and the cerebellum (8%). When cerebral arteriolar walls are weakened by lipohyalinosis, small fusiform aneurysms called Charcot-Bouchard aneurysms are formed. These microaneurysms could rupture causing damage to adjacent brain tissue. The number of these aneurysms increases with age and duration of hypertension. Subarachnoid haemorrhages develop from rupture of berry aneurysms.

Hypertensive encephalopathy

Oppenheimer and Fishberg, in 1928, first introduced this term to describe the encephalopathic changes associated with the accelerated phase of hypertension. It is a term used to describe the clinico-pathological syndrome of fibrinoid necrosis of intracranial arterioles and small arteries, petechial haemorrhages and symptoms including headaches, confusion, nausea, vomiting, convulsions, visual disturbances, and sometimes coma.

The onset of symptoms usually occurs over 24 - 48 hours with neurological progression over 24 - 48 hours. Papilloedema is normally seen. Post mortem examination usually reveals cerebral oedema, with or without transtentorial or tonsillar herniation along with petechiae and fibrinoid necrosis in grey and white mater.

Progression of this condition could lead to a clinical syndrome characterized by dementia, gait abnormalities (usually a shuffling gait with small steps called marche a petits pas), and pseudobulbar signs, often with focal neurological deficits. This syndrome is called vascular (multi-infarct) dementia and is caused by multifocal vascular disease consisting of:

1. Cerebral atherosclerosis
2. Thrombosis or embolisation from the carotids or from the heart and
3. Cerebral arteriolosclerosis.

There may be confusion clinically with Parkinson's disease; this has been called ***'atherosclerotic parkinsonism'*** in the past.

Binswanger disease refers to a preferential sub-cortical white mater affectation seen on CT as low-attenuation areas in hypertensives with encephalopathy, TIAs, and stroke like episodes.

OPHTHALMIC VASCULOPATHY

Systemic hypertension causes hyaline arteriolosclerosis. Leakage of plasma components across the endothelium and increased extracellular matrix production by smooth muscle cells causing hyaline thickening of the vessel walls and narrowing of the lumen.

Further changes manifest in the retina, choroid, and optic nerve through mechanical changes in the retina and choroid and possibly release of local mediators.

Three entities caused by hypertension include:

- Hypertensive retinopathy
- Hypertensive choroidopathy and
- Hypertensive optic neuropathy.

Hypertensive retinopathy

The first account of retinal changes in hypertension was by Liebreich in 1859, shortly after the invention of the direct ophthalmoscope by Helmolz.

In 1939, Keith Wagener and Barker devised the m widely used (though not universally accepted) classificat of hypertensive retinopathy:

TABLE 2 KEITH-WAGENER-BARKER CLASSIFICATION

Grade I	*Benign hypertension. Mild narrowing/sclerosis retinal arterioles. Copper wiring or silver wiring. No symptoms; good general health*
Grade II	*Marked sclerosis, arteriovenous nicking (Gunn sign), and venous compression. Increased venous tortuosity and exaggeration of the arterial light reflex.*
Grade III	*Mild angiospastic retinopathy. Retinal oedema cotton wool spots and haemorrhages. Blood pressure is very high and sustained. Symptomatic. Prognosis is poor.*
Grade IV	*Malignant hypertension - cardiac and renal functions may be impaired. Papilloedema (disc oedema). Prognosis poor.*

The basis for these changes is that hypertension le to endothelial necrosis, which invites the disruption of t blood-retina barrier. The changes are classified into tw

1. Vaso-occlusive retinal changes (copperwiring silverwiring, A-V nipping and ischaemia visible cotton wool spots).
2. Extravascular retinal lesions (retinal micro-aneury and haemorrhage, retinal and macular oedema and li deposition)

Hypertensive Choroidopathy

Seen more commonly in young patients with acu hypertension, it involves arteriolar constriction leading changes in the choroid layer.

The three characteristic fundal changes seen are:

- Choroidal vascular occlusion (acute or chronic
- Retinal pigment epithelial lesions (persiste choroidal ischaemia results in degenerative lesions know as Elschnig's spots)
- Serous detachment of the neurosensory retina

Hypertensive optic neuropathy

There is optic nerve oedema (stage IV retinopathy Patients present with haemorrhages at the optic di margin, blurring of disc margins, congestion of retinal ve macular exudates, and florid disc oedema.

Some ocular diseases are related to but not caus by systemic hypertension. They include:

- Retinal vascular occlusion,
- Ocular ischaemic syndrome
- Carotico-cavernous fistula
- Cranial nerve palsies
- Spontaneous subconjunctival haemorrhage

- Diabetic retinopathy
- Expulsive choroidal haemorrhage

THE KIDNEY (HYPERTENSIVE NEPHROPATHY)

The relationship between hypertension and kidney disease is an interesting one for, either of them can lead to the other. Target organ damage to the kidneys by hypertension is generally referred to as hypertensive nephropathy, though this is more of a chemical entity. Three pathologic patterns can be seen in hypertensive nephropathy:

- Benign nephrosclerosis
- Malignant nephrosclerosis
- Secondary glomerular disease

Benign nephrosclerosis

This is the term used to describe renal arteriolosclerosis. It occurs more in blacks than in whites and has been found in some non-hypertensive patients (especially diabetic patients). Its pathogenesis has been discussed previously. Grossly, the kidneys have a fine, even granularity that resembles grain leather.

Histologically, there is narrowing of the lumina of arterioles caused by thickening and hyalinization of the walls (hyaline arteriolosclerosis). In addition, fibroblast hyperplasia resulting from increased myofibroblastic tissue in the intima, with consequent narrowing of the lumen has been described.

Uncomplicated benign nephrosclerosis causes anemia or renal insufficiency and sometimes, mild proteinuria. Three groups of hypertensives with benign nephrosclerosis are at risk of developing renal failure:

- Blacks
- Severely hypertensive patients and
- Patients with secondary underlying disease (e.g. diabetes)

Malignant Nephrosclerosis

This pattern occurs in malignant or accelerated phase hypertension. It is often superimposed on benign nephrosclerosis and is an uncommon cause of death from uremia. It predominantly affects younger individuals, is more in men and in blacks. Its characteristic features, already discussed, include fibrinoid necrosis, hyperplastic arteriolosclerosis and a background markedly elevated plasma renin.

Hypertensive Glomerulopathy

According to Cotran et al, a variety of glomerular alterations could occur consequent to vascular narrowing and patch ischaemic atrophy. These include

1) Collapse of glomerular basement membranes
2) Deposition of collagen within the Bowman space
3) Periglomerular fibrosis and
4) Total sclerosis of glomeruli (glomerulosclerosis).

Hypertensive glomerulosclerosis has also been attributed to direct transmission of raised blood pressure to the glomeruli.

SUMMARY

Hypertension has for long been globally recognised as the most prevalent cardiovascular disease and is an acknowledged potent risk factor in the development of target organ damage[8]. This involves a triad of cardiovascular, neuro-ophthalmic, and renal involvements, each of which interestingly involves its own triad of complications (check diagram). This triple triad (the triad of triads or the 3-3-3 arrangement), as we have called it, are the end organ/target organ changes seen in hypertension and have been dubbed by some, 'the hypertensive diseases'.

REFERENCES

1. Sharma S, Kortas C. Hypertension. eMedicine Journal, 2002 Jan; 3(1) http://www.emedicine.com/med/topic1106.html
2. Roberts WC. Cardiovascular consequences of systemic hypertension: a morphologic survey In Cardiology 1. Hypertension. Butterworth's Scientific. 1982 78-79, 92.
3. World Health Organization. 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines Subcommittee. J Hypertension 1999. 17(2) 151-83
4. Kaplan NM. Systemic hypertension: Mechanisms and diagnosis. In: Braunwald E. Heart disease. Saunders 1992. 822-844
5. George OT. Target organ complications of Hypertension. Archives of Ibadan Medicine 1999. 1(1) 13; 13-16
6. Perera GA. Hypertensive vascular disease: Description and Natural History. J Chronic Disease 1955; 1: 33-42.
7. Kumar P, Clark M. Clinical Medicine. Saunders. 1998, 739, 1050-1051
8. Rubin E, Farber JL. Pathology. J.B. Lippincott. 1994, 851-853. 1397-1401.
9. Cotran RS, Kumar V, Collins T. Robbins Pathological Basis of Disease. Saunders. 1999, 498-515, 981-984, 1313-1314.
10. Akinkugbe O.O. Current epidemiology of hypertension in Nigeria. Archives of Ibadan Medicine 1999. 1(1). 3-5

ACKNOWLEDGMENT

We are grateful to Dr. Aje of the Cardiology Unit, Department of Medicine, and Prof. E.E.U. Akang of the Department of Pathology, University College Hospital, Ibadan, for helping us review the manuscript.

70 MILESTONES: THE MAKING OF AN AFRICAN COLOSSUS

17th of July 1933...
Nature spread its morning dews
To tend the greenness on the velvet land
The tropic sunrise broke...
Pangs of labour pain, sweat as viscous balls
Body doing rhythmic dance to the tunes played by pain
A mighty push: every muscle contracting in obedience
Cries of a baby, a sigh of relief, tears of joy...
Drum beats, Dancing feet
Hearty chuckles Bellowing laughter
As cups of wine graced the lips of men
OLADIPO, OLUJIMI...

Dark iron's skin, kinky black hair
Calloused soles, blistered palms: result of hard work
Typical of African-
Hunched from heavy load of culture
And spoken limitations of tradition
Crippled he may have appeared but
His mind was intact

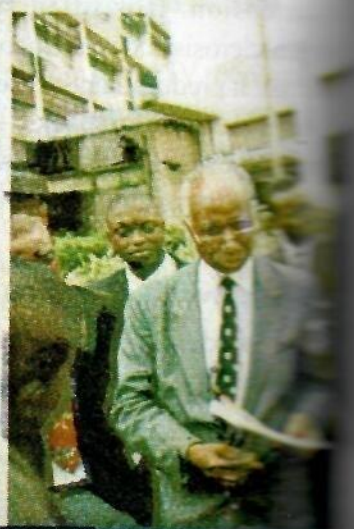
Shedding the chains of cultural and racial prejudice
He conquered the academic battle
From primary through tertiary levels
The picture: Clad in the white man's suit
Hair cut with a side parting
A tie of great semblance to a spade
A portmanteau in hand
Ox coloured shoes with black heels
Stone to brilliance
He set out-Far beyond the seas
Not just to acquire the ACCENT
Through London, Moscow & Montreal
Boston, Washington & Cleave land
New Orleans, Atlanta & Kingston

He went; leaving them staring at the black prowess
Awed by excellence and amassed wisdom
While he added more feathers to his cap

Today he is a medical and literary colossus
Effortlessly bridging the gap between the two professional worlds
To the literary world He is
A synthesizer of ideas
A smooth word smith
His speeches are a gourmet's delight
For a good literary buffet
Dr. 'Mellifluous'-master of the spoken word
To the medical world He is
The master of medicine
A teacher of teachers
an antidote to the silent killer-HYPERTENSION
An Emeritus Professor

To us his children, he is;
A father, A mentor, & A record to beat
One who must remain proud of us

Sir, we say it's been 70years of fulfillment
CONGRATULATIONS, WELL DONE, EKU ISE, NDEWO
SANU DA AIKI, FELICITATIONS!
BON-ANNIVERSAIRE!!!



This Poem was written by Ikeaguoha Ogadinma (Miss) and Bekwelem Wobo, both members of the Editorial Board of DOKITA

CAUSES AND TREND OF MORTALITY IN IBARAPA

AWOJOBI O. A, OLALEYE O. A.

Dr. O. A Awojobi is the Consultant Surgeon at the Awojobi Clinic, Eruwa, Oyo State. Mr. Olaleye was a 2nd year Clinical student of the College of Medicine, University of Ibadan at the time of writing this article.

INTRODUCTION

The Faculty of Medicine, University of Ibadan, Ibadan, Nigeria and the government of Western Nigeria set the pace in primary health care delivery 15years ahead of the World Health Organization's Alma Ata Declaration on the same subject when, in 1963, they established the Ibarapa Community Health Project.[1] The programme is based at the Rural Health Centre (now a General Hospital) in Igboora, the largest of the seven towns in rural Ibarapa district.

The District Hospital, Eruwa was opened in 1970 and secondary level health care thus became available. Senior Registrars and Registrars from the Departments of Surgery and Obstetrics and Gynaecology of the University College Hospital, Ibadan provided surgical services on a regular basis. This was boosted when a Consultant Surgeon took up appointment in the hospital in 1983.[2,3]

The Alma Ata report on primary health care emphasized evaluation by those providing the service, those using them and those responsible for managerial and technical control at various levels.[4] One of the methods of evaluation is the study of the causes and trends of mortality in the community.

Ayeni and Oduntan[5] reported on infant mortality rates and trend in Igboora for the period of 1965 to 1975. The report assessed the effects of the provision of primary health care on the standard of health of the people.

This retrospective study reports on the causes and trend of mortality at Awojobi Clinic, Eruwa, the major secondary level hospital in the district established in October 1986 and offers suggestions for the improvement in the health status of the Ibarapa community.

MATERIALS AND METHODS

The case notes of all patients who died at Awojobi Clinic, Eruwa between January 1st 1987 and December 31st 2001 were retrieved. Cases of maternal and postoperative mortality were excluded from analysis. Data extracted included the sex, age and the major cause of death. Autopsy was not performed on any of the patients.

The data were analyzed in three 5year periods to detect any change in mortality trend and the effects of health education conducted by members of staff of the clinic in the community during the period.

RESULTS

In the 15year period, 14, 354 (Fourteen thousand three hundred and fifty four) patients (excluding maternal and surgical patients) were admitted and 1, 336 (One thousand, three hundred and thirty three) deaths were recorded giving a mortality rate of 9.3%.

Table 1 shows the quinquennial distribution of admission and mortality. Mortality was twice as common in children (14.9%) as in adults (7.1%). There was no change in overall mortality trend in both children and adults.

Table 2 shows the age/sex distribution in the patients that died. In the paediatric age group, the under-fives constituted 40.2% and the infants were 30.2%. The male to female ratio was 1.3:1. In the adult population the 60-69year age group (18.8%) was the most vulnerable. The male to female ratio was 2.2:1

Table 3 indicates the major causes of death in children. Respiratory tract infection, anaemia (usually due to severe malaria and poor nutrition), gastro-enteritis, septicaemia, meningitis and neonatal tetanus were the major diseases; all accounting for 67.1% of all cases. Anaemia and meningitis showed an increasing trend over the years while neonatal tetanus dropped sharply in the third quinquennium after an increase in the second. Deaths from snakebites are peculiar to this environment as poisonous snakebites are common.

The causes and trend in mortality in adult patients are shown in Table 4. Infective diseases alone accounted for 56.3% of deaths. Tuberculosis and typhoid fever were the most common infections. Hypertensive cerebrovascular accident was the main cardiovascular cause of mortality while cancer was responsible for 3.9% of cases. There were 11 cases of primary liver cell carcinoma and three cases of prostate cancer.

SSION

etrospective hospital-based study has shown that e and preventable diseases are the major causes of in the paediatric and adult population of Ibarapa of Oyo State, Nigeria. This picture is similar to

TABLE 1: ADMISSION AND MORTALITY AT AWOJOBI CLINIC ERUWA

Period	CHILDREN			ADULT		
	Total admission	No of deaths	% mortality	Total admission	No of deaths	% mortality
7 - 1991	1 875	282	15.0	3 522	231	6.6
2 - 1996	1 259	176	13.9	3 567	260	7.3
7 - 2001	838	137	16.3	3 293	250	7.6
TOTAL	3972	595	45.2	10 382	741	12.5

eports from both urban and rural communities.[6-10] Children under five years of age are the most able. This prodigious waste of life is compensated a corresponding rise in birth rate as shown by rising ation figures and the high twinning rate of 40/1000 ths for which this district is noted.[11]

ough the introduction of the National programme unization over a decade ago has virtually eradicated and tetanus and reduced the effects of measles in cy and childhood, neonatal tetanus is still common se many babies are still born at home where sanitary ions are less than satisfactory. This situation results from inadequate and expensive transportation of ant women to the maternity centers for delivery. It been our policy to encourage mothers to bring their born babies to the clinic soon after delivery at home ssive immunization using the equine antitetanus serum. may have been responsible for the drop in the incidence eonatal tetanus as a cause of death in the third quennium under review. Routine active immunization adults presenting in all health institutions and during ission to colleges and tertiary institutions will reduce cidence of tetanus.

Malaria is a serious public health problem of the rural ace, causing a high mortality in preschool children the attendant anaemia and febrile convulsion. Febrile ulsion in children is still being treated with herbal drugs aining tobacco. Prevention of malaria infection in the areas will be very difficult as the use of the mosquito plain or insecticide-impregnated) is not common and rally inappropriate. Early and vigorous treatment, with the addition of haematinics to the regimen, remains the practical solution, coupled with improved environmental sanitation.

In the adult population, infections (especially pulmonary tuberculosis) still constitute the most common cause of mortality. This has been the situation in other reports in the past when there was no HIV/AIDS epidemic. It is only recently that cases of the pandemic are presenting in the rural environment and this is setting a stage for a disaster in a population that is permanently under-nourished. The only hope lies in a massive intervention through persistent health education by governments at all levels and nongovernmental organizations. Improvement in the provision of potable water, adequate transportation and general nutritional status of the rural populace will eventually reduce mortality from infectious and communicable diseases.

TABLE 2: AGE/SEX DISTRIBUTION

Age	Male	Female	%
Children			
0 – 1 month	51	38	15
< 1year	97	83	30.2
< 5years	136	103	40.2
< 12years	54	33	14.6
TOTAL	**338**	**257**	**100**
Adults			
(years)			
13 – 19	53	25	10.5
20 – 29	100	25	16.9
30 – 39	65	28	12.6
40 – 49	57	43	13.5
50 – 59	70	42	15.1
60 – 69	100	39	18.8
70 – 79	54	30	11.3
80 – 89	10	-	1.3
TOTAL	**509**	**232**	**100**

Hypertension is common in the rural population.[12] Due to low level of health awareness and poverty, it often presents with the complication of cerebrovascular accident and the attendant high morbidity and mortality. The cost of antihypertensive drugs should be substantially subsidized for better treatment compliance.

TABLE 3: CAUSES OF DEATH IN CHILDREN

Causes	Number (%) 1987 - 1991	1992 - 1996	1997 - 2001	Overall %
Respiratory tract infection	46 (16.3)	27 (15.3)	17 (12.4)	15.1
Anaemia	37 (13.1)	34 (19.3)	38 (27.7)	18.3
Gastroenteritis	36 (12.8)	10 (5.7)	10 (7.3)	9.4
Septicaemia	33 (11.7)	21 (11.9)	13 (9.5)	11.3
Protein energy malnutrition	22 (7.8)	2 (1.1)	3 (2.2)	4.5
Febrile convulsion	19 (6.1)	6 (3.4)	1 (0.7)	4.4
Meningitis	16 (5.7)	11 (6.3)	15 (10.9)	7.1
Neonatal tetanus	13 (4.6)	18 (10.2)	4 (2.9)	5.9
Infective hepatitis	8	4	6	3.0
Typhoid fever	8	3	3	2.4
Measles	7	9	9	4.2
Poisoning	5	6	2	2.2
Haemoglobin S + S	5	4	2	1.8
Tetanus	4	2	–	1.0
Pulmonary tuberculosis	4	1	1	1.0
Snakebite	2	1	1	0.7
Others	17	17	12	7.7
TOTAL	**282**	**176**	**137**	**100**

TABLE 4 CAUSES OF DEATH IN ADULTS

Cause	1987 – 1991	1992 – 1996	1997 – 2001	Total (%)
(Infective)	137 (59.3)	146 (56.2)	134 (53.6)	417 (56.3)
Cardiovascular	28 (12.1)	40 (15.4)	41 (16.4)	109 (14.7)
Trauma	6 (2.6)	11 (4.2)	15 (6.0)	32 (4.3)
Gastrointestinal **(non-infective)**	10 (4.3)	8 (3.1)	13 (5.2)	31 (4.2)
Cancer	8 (3.5)	6 (2.3)	15 (6.0)	29 (3.9)
Haematologic	8 (3.5)	9 (3.5)	3 (1.2)	20 (2.7)
Diabetes	4 (1.7)	5 (1.9)	8 (3.2)	17 (2.3)
Renal	1 (0.4)	3 (1.2)	3 (1.2)	7 (0.9)
Respiratory **(non-infective)**	4 (1.7)	1 (0.4)	1 (0.4)	6 (0.8)
Others	25 (10.8)	31 (11.9)	17 (6.8)	73 (9.9)
TOTAL	**231**	**260**	**250**	**741**

In no other disease is prevention not only cheaper but better than cure than in snakebite in rural Ibarapa district.[13] Majority of the bites are poisonous and mortality, due to severe haemorrhage from all orifices and internally, is high. Most of the populace are peasant farmers and bites have occurred near and in the homes as well as on the farms. Health education and wearing of boots when working on the farms should provide the solution to the problem.

The incidence of cancer, and as a cause of death in this community is low.[14] As in other communities it will come into prominence as the communicable diseases are br under control. However, it is imperative that the cu habits, particularly the eating habit, that appear to le the life expectation of black Africans who survive m age should not be lost or given up for Western Eur dietary habits, if degenerative diseases, in parti coronary heart disease, are to be minimized.[6]

REFERENCES

1. Oyediran A B O O and Brieger W R 25 years of The

Community Health Programme, 1989. Printed by African Press Limited.
2. Awojobi O A Surgery in Nigerian rural health care delivery – the Ibarapa experience Nig Med Pract 1987; 13: 49 – 51.
3. Awojobi O A Principles of rural surgical practice Dokita 1998; 25: 1 – 2.
4. Primary Health Care. Report of the International Conference on Primary Health Care, Alma Ata, USSR, 6 – 12 September, 1978.
5. Ayeni O and Oduntan S O Infant mortality rates and trends in a Nigerian rural population. J Trop Pediatr 1980; 26: 7 – 10.
6. Junaid T A Mortality in middle-aged Nigerians. An autopsy study. Trop geogr Med 1979; 31: 389 – 394.
7. Abdulrahaman M B Why our children die: A study of mortality pattern in an emergency paediatric unit in Kaduna, Nigeria. Nig Med Pract 1983; 5: 157 – 162.
8. Ezenwa A. A study of mortality in rural population in Bendel State, Nigeria. Nig Med Pract 1985; 9: 17 – 19.
9. Ogunmekan G. O. Analysis of medical admissions to Adeoyo State Hospital, Ibadan, 1969. Nig Med J 1973; 3: 5 – 12.
10. Ogun S A, Adelowo O O, Familoni, O B et al Pattern and outcome of medical admissions at the Ogun State University Teaching Hospital, Sagamu – a three-year review. W Afr J M 2000; 19: 304 – 308.
11. Nylander PPS The frequency of twinning in a rural community in Western Nigeria. Ann Hum Genet London 1969; 33: 41 – 4.
12. Akinkugbe O. O and Ojo A. O. The systemic blood pressure in a rural Nigerian population. Trop Geogr Med 1968; 20: 347 – 56.
13. Abara O E, Ayandiran D O and Adeloye A. Snakebites in Igbo-ora, Ibarapa district, Western State, Nigeria. Nig Med J 1977; 7: 107 – 111.
14. Awojobi O. A. Malignant tumors in rural Western Nigeria. *Workshop on the State of the Art in Oncology in Ibadan and Ife*. National Headquarters of Cancer Registries in Nigeria, UCH, Ibadan. Solanke TF and Adebamowo C (editors) 1996: 13-16.

DRUG USE AND ABUSE AMONGST SENIOR SECONDARY SCHOOL STUDENTS IN IGBO - ORA

ACHAKA A.M, AJAYI A.O, ALUKO B.F (MISS), AJIBADE A.T, ADEGOKE D.A, ABDUL M.M (MISS), AGBEDEYI G.O (MISS) AKHETUAMEN P.O, AJAKAIYE O.A (MISS), AJUMOBI O.O (CAPTAIN), ABDULMALIK J.O, AGORO K.O, ABIAHU J.A

This submission is the project report of SUBGROUP 1 of GROUP D 98 and it was nominated as the best Igbo Ora project for the set.

ABBREVIATIONS

NDLEA	*National Drug Law Enforcement Agency*
CNS	*Central Nervous System*
WHO	*World Health Organization*
USA	*United States of America*
UK	*United Kingdom*
NIDA	*National Institute on Drug Abuse*

INTRODUCTION

The widespread use of psychoactive substances has become a subject of public concern worldwide. It has grown into a social menace that threatens the fabric of man's existence.

Most of the drugs that are abused are psychoactive substances capable of stimulating or inhibiting the central nervous systems in such a way that the mood, cognitive process and behaviour of the individual involved are involuntarily impaired.

Drug abuse is the maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by one or more of the following occurring within a 12-month period[1].

1. Recurrent substance use resulting in the failure to fulfil major roles; obligations at work, school or in the home.
2. Recurrent substance use in situations in which it is physically hazardous; for example driving automobile when impaired by substance use..
3. Recurrent substance related legal problems.
4. Continued substance use despite recurrent social and interpersonal problems.

Drug dependence is based on 3 or more of the following criteria within the previous year[2].

1. A strong desire or compulsion to take the drug.
2. Subjective awareness of impairment in one's capacity to control the use of the drug.
3. Substance use to relieve withdrawal symptoms
4. Withdrawal state
5. Evidence of tolerance
6. A narrowing of the personal repertoire of patter drug use
7. Progressive neglect of alternative ways of pleasu
8. Persisting with drug use despite clear evidence overtly harmful consequences.

STATEMENT OF THE PROBLEM AND JUSTIFICATION OF THE STUDY.

There is an upsurge in the use of psychoactive substances in Nigeria. Thus, her status has changed from mere transit point in the international drug market to nation with an increasing number of drug users and abusers. This upsurge has been characterised by an increase in the crime rate, mental disorders and cult activities in both the higher institutions of learning, and the secondary schools.

The adolescent period (between the ages of 10 and 19 years) may be described as a transition from childhood to adulthood. It is often characterised by psychological and emotional turmoil. Adolescents often wish to experiment with anything and are strongly influenced by peer group pressure.

Most studies from both the developed and the developing worlds clearly identify the fact that a vast majority of drug users start in their adolescent years[3,4,5,6,7].

The resultant loss to the nation on account of these problems afflicting her youth cannot be quantified in material terms. Examples include the issues of "Area boys" in such states as Lagos state that do not have meaningful jobs and have thus caused a surge in the crime rates in such

...eas. Such area boys and girls' also lose the privilege of ...aining an education, as their main purpose in life is to ...ain money to purchase the various drugs and other psy...active substances that they cannot live without. These ...e just a few examples and based on such facts we found ...ecessary to carry out this study, to see how deleterious ...g use and abuse amongst adolescents is. The study ...aims at providing useful information on the prevailing ...ation amongst young people in secondary schools.

...DY OBJECTIVES

...ERAL OBJECTIVES

To determine the point prevalence, pattern and the ...rs influencing drug use and abuse amongst secondary ...l students with the aim of obtaining useful informa... for social monitoring and the planning of preventive ...egies.

...IFIC OBJECTIVES

To determine the prevalence and type(s) of drug(s) used ...ng secondary school students.

To identify the pattern of initiation into drug use and ...se of drugs

To identify socio-demographic and other factors that ...associated with the use and abuse of drugs.

...ERATURE REVIEW

A psychoactive substance is defined as a substance ...e of stimulating/inhibiting the CNS in such a way ...e mood, cognitive process (especially the judge... and thinking) and behaviour are involuntarily im...

Hazardous use of a drug implies that it carries a risk of ...damage to physical or mental health, while harmful ...s that the use is already causing harm to the body. ...rrence of both hazardous and harmful use to... called DRUG ABUSE [9].

...MIOLOGY

Generally, in the USA, the National Institute of Drug ...has reported that in 1991, 37% of the population ...illicit substances in the past year, and 6% in the ...before the survey[1].

Alcohol abuse and dependence is by far the most com... substance related disorder in the US[10].

National Institute on Drug Abuse reported that for ...about 73% of the US population aged 12 and older ...oked cigarettes in their lifetime, 32% in the past ...and 27% in the past month[4]. They also reported that ...% of the population has used marijuana at least once in their lifetime, 9.5% in the past year and 4.8% in the past month and that adults aged 26-34 were the most likely group[4].

Studies conducted in the UK have also reported that alcohol abuse was very common in the adolescent age group[9].

In Scotland, alcohol consumption was highest among men who were unmarried, separated or divorced. A report find that 3% of the population, mostly single men who were in their late teens or twenties were responsible for 30% of all alcohol consumption in Scotland [5,12].

Wilson confirmed these findings in studies carried out in England and Wales[13]. Little however is known concerning the prevalence of other psychoactive substances in the UK[9]. Rates are high among adolescents, particularly around school leaving age. The prevalence of psychoactive substance in Britain among people under 20 yrs which rose steeply in the mid and late 1980s may have declined in the 1970s, but has again increased in the 1980s[9]. Solvent abuse prevalence in the UK is uncertain but occurs mainly in boys aged 8-19, with a peak in those aged 13–15[14]. Adhesives- containing solvent and acetone are coming the most frequently used[6].

In Asia, a study in Taiwan with 4,358 adolescent students aged 16 - 18 yrs old estimated the prevalence of alcohol drinking as 70-71%, tobacco smoking 56%, illicit drug use 6% [7].

A similar trend of drug use and abuse in adolescents and young adults has been observed in Nigeria. Alcohol, tobacco, cannabis, amphetamine, heroine, cocaine, caffeine, and kola nuts are included [15,16].

In a cross sectional study of drinking behaviour and social character among 2,079 senior secondary school students in Abeokuta and Ibadan, prevalence rates of 51.5% and 56% were reported for Abeokuta and Ibadan respectively. The age for commencing alcohol use was as low as 10-11 yrs.[4]

Adelekan et al in Ilorin found among Nigerian undergraduate students a prevalence rate of 77% for alcohol, stimulants 69.28% and cigarettes 37.4%. The use of most substances started in primary school.[15]

Solvent (glue and petrol) abuse by youths has been recently reported in different parts of the country.[17] The abuse of solvent was observed to be common among young persons who are school dropouts now working as mechanics, shoe repairers and petrol attendants, and bus conductors[1].

Studies done in USA by NIDA showed that males were significantly more likely than females to have smoked cigarettes in the past month; 58% compared with 44%. These differences were greater in adults > 26yrs. Ciga-

rette smoking was also significantly more common in males than females[4]. Marijuana smoking was found to be twice as common in males than females[4].

In contrast to this, studies in Columbia as a sample population, aged > 12 years showed that conditional prevalence of dependence on alcohol, marijuana and cocaine were significantly higher among females than males, except for cocaine[18].

In the UK, male: female ratio of alcoholics, formerly 5:1 has changed with alcohol problems being significantly higher among female than males, except for cocaine[18].

In Taiwan, a study done by the school of nursing, Kaohsing medical college in 4,358 adolescent students aged 16 - 18yrs showed a higher incidence of drug abuse in male students: - alcohol: (boys & girls) 75.1% : 51.4%, tobacco 61.8% : 30.2%, illicit drug use 6.6% : 5.6%[7].

There was a similar trend in Nigerian studies, a study on senior secondary school students reported a male: female ratio of 1.1:1 (Ibadan), 1.2:1 (Abeokuta)[3]. In 1970, Elegbeleye and Pearse showed that amongst 1,026 male and 947 female school students, there was an incidence of 17.5% male and 2.7% female smokers[20]. A study by Adelekan in 1992 in Ilorin on undergraduate students showed that males were significantly more likely to use alcohol and cigarettes than females, who tended to use stimulants more[15]. A study done by Obot (1990) with 1,271 Nigerian adults showed that males smoked more than females[21].

Studies in Tennessee, USA on 217 adolescents aged 12 - 19yrs showed that as attendance of religious services increased, alcohol and other drug abuse decreased[22]. Studies done in Ilorin, Nigeria undergraduate students showed a correlation between lack of religious devotion and the use of alcohol, cigarette, and cannabis[22]. There was also a correlation between belonging to the Christian religion and reduced use of these substances[16, 22]. According to the Surgeon General's report in US (1979), if an older sibling and both parents smoked, then the child was 4 times more likely to smoke[23].

Other researchers also reported similar findings that children followed their parents drinking behavior[9,10,11].

A study done in Quebec, Canada, on about 1,000 children showed that paternal alcoholism is an important factor in the development of substance abuse problems in adolescents. Parental supervision was found to have protective influence, reducing the risk of substance abuse in children of alcoholic fathers.

A study in Hong Kong showed that adolescents perception of parenting styles, family functioning and parent adolescent conflict were significantly related to psychological well-being, academic performance and problem behaviours i.e. drug abuse[25].

In Nigeria, studies by National Drug Law Enforcement Agency (1993) on students in secondary school hospital admissions and people arrested for drug trafficking in a population survey showed a profile of youth, most male school dropouts, with no parental control. Many were destitutes in big cities, referred to as 'Area boys'[4].

Others took up menial jobs in market places, motor parks and other public places[3]. Odejide and Sanda (19[illegible] also highlighted in their study the effect of parental deprivation on children, such as deaths, divorces, separation, discard, finding a strong association between these and drug use/abuse[26].

A study done in Canada showed that the risk of alcoholism decreased with personality traits such as low thrill seeking behaviour and a propensity for inhibition[24].

A study in USA in the New York School of Medicine showed that physical abuse added significantly to their factors in accounting for major depressions, conduct disorder and drug abuse.

Studies done in Taiwan showed that risk factors for drug use included behavioural problems, non - negative attitude towards patient's substance use and peer influence[7].

In 1982, Nevadomsky in Nigeria reported that some behaviour endemic among adolescents such as experimentation, rebellion and desire for independence are associated with drug use[27]. Akindele (1976) also identified peer pressure as a factor in Nigerian adolescents[28].

Societal factors such as poverty, drug availability, unemployment, and frustration caused by tension between improved levels of education and shrinking employment opportunities has also been identified[3,4].

According to Akindele (1976), Western culture has made drug use prevalence high among the high socio-economic class[28]. Abiodun (1991) also reported that drug abuse (cocaine, heroin) was very common in big cities.

Studies on the effects of the media (exposure to advertisements promoting the use of psychoactive substances) on drug use, and observed an increased consumption with the use of television adverts which portray alcohol and cigarettes as being linked with social and financial success, with messages such as ***'DISCOVER GOLD'*** and ***'SYMBOL OF EXCELLENCE'***. Notable stars in the sports and music world are used to advertise these social drugs[17, 26, 30].

A study in the USA showed that whites are more likely to be dependent on NICOTINE and blacks on COCAINE, than other races.

MATERIALS AND METHOD

BACKGROUND INFORMATION ON STUDY AREA

Igbo-Ora town, the study area, is located in the rain forest region in the outskirts of Oyo state in the Southwestern part of Nigeria. It is the larger of 2 towns in the present Ibarapa Central Local Government Area- the other being Idere. The study area is located about 100km from Ibadan.

Formally, it was under the Ifeloju Local Government Area, which comprised 6 other towns viz., Ayete, Idere, Ilupa, Igangan, Lanlate and Eruwa with its headquarters at Igbo – Ora.

Igbo–Ora has an estimated population of 70,000 people. The predominant occupations are subsistence farming and petty-trading. The Yorubas constitute the bulk of the native inhabitants and are of low socio- economic status. Islam is the predominant religion - though there are some Christians and Traditionalists.

Igbo–Ora has 17 public primary schools and 7 secondary schools. The main means of transportation is motorcycle and the main source of water is by dug out wells - whether public or privately owned.

The town is divided into 6 blocks, which are further subdivided into 62 enumeration areas; each containing varying number of family compounds. Apart from this system there is also a primary health care numbering system used by the local government based on a number of political wards.

STUDY DESIGN.

This descriptive study was cross sectional in design to determine the prevalence of drug use among senior secondary school students in Igbo-Ora town. Variables of interest include socio-demographic correlates of drug users, pattern of use-whether single or multiple, family background and history, presence of drug abuse and or dependence.

The target population was senior secondary school students in Igbo-ora. A sample frame was drawn up. It was made up of all the secondary schools in Igbo-ora town:

1. Igbo-Ora High School. *
2. Lasogba Grammar School.
3. Islamic High School.
4. Methodist Grammar School. *
5. Ogboja Grammar School. *
6. Answar-ud-deen Grammar School.
7. Lajorun Grammar School.

Two schools were then selected through simple random sampling using the ballot method - they are asterixed above.

Using the formula $n = Z^2pq/d^2$ by Kish and Leslie [31] (1965), the sample size was estimated.

N = minimum sample size

Z = 1. 96 (2 S.D) at 95% confidence level or

Q = 5%

P = 0.5

Q = 1- p = 1 – 0.5 = 0.5

D = precision expected at 95% confidence limit (0.05%)

The calculated value of the sample size was 384

INSTRUMENT OF DATA COLLECITON

Based on the established objectives, an appropriate questionnaire was developed structuring each question strictly around our specific aims and objectives. These questions were then translated into the local language (Yoruba) with the assistance of a Health worker at the Records Department of Igbo-Ora Comprehensive Hospital.

There were 5 sections to the questionnaire

Section I – Demographic data

Section II – Drug use

Section III – Drug dependence

Section IV – General health questionnaire

Section V - Drug abuse.

The questionnaire was pretested at Lajorin Grammar School by 3 members of the group who administered a total of 10 questionnaires (this school was not a part of our study population). A teacher in the school selected the ten students 5 males and 5 females for interview through the stratified random sampling method.

VALIDITY AND RELIABILITY

The questionnaire was effectively tested on senior secondary school students in Lajorin Grammar School. Back translation of particular terms from local language was carried out to avoid ambiguity and to enhance uniformity in translation. In addition to this the confidentiality of information given was stressed and respondents were encouraged to be as truthful as possible.

Questionnaires were then administered to all the senior students of each school present at the time of the interview. At Methodist Grammar School, a total of 122 students were interviewed - 45 in senior secondary 1(SS1) and 77 in SS2 classes. Igbo-Ora High School - a total of 144 students were interviewed; 58 students in SS1 and 86 students in SS2. 128 students were interviewed at Ogboja Grammar School- 55 being in SS1 and 73 in SS2. In all, 394 students were interviewed. No students in SS3 of any of the schools were interviewed because these students were writing the West African Examination Council

(WAEC) Senior School Certificate Examination (SSCE) and were thus unavailable.

METHODS OF DATA PRESENTATION

1. Tables and charts were used for the descriptive aspect of he study.
2. Pictoral representation - pie and bar charts were used

METHODS OF DATA ANALYSIS

Chi - square test of significance on proportion was used to determine the significant association between the independent variables.

LIMITATIONS OF THE STUDY

The study project was an interesting one but we encountered the following limitations viz:

1. Difficulty in administering questionnaires due to problems encounter in communicating adequately with the respondents. This was overcome by using in addition, a standardized questionnaire translated into the local language.
2. Reluctance on the part of respondents in divulging information concerning details of drug use, family and home environment. Moreover, they were reassured of the confidentiality of the divulged information.
3. Lack of adequate means of ascertaining the validity and truthfulness of responses given by the respondents.
4. Timing of the study limited it to students in SS1 and SS2 classes, as the SS3 students were unavailable.

Despite these limitations, we were able to obtain substantial and relevant information.

RESULTS

DISTRIBUTION BY DEMOGRAPHIC VARIABLES

In this study, a total of 394 senior secondary students from 3 different schools were interviewed. Out of these, 11 (2.8%) were aged 10-14 years, 352 (89.3%) aged 15 - 19 years and they constituted the majority while 31 (7.9%) belonged to he 20 - 24 years age group (Table 1). The mean age was 17.1±1.6 years.

Two hundred and seventeen students (55.1%) were males while 177 (44.9%) were females. As regards religion, majority of the students 231 (58.6%) were Muslims and 163 (41.4%) were Christians. Out of the 394, 206 (52.3%) belonged to the polygamous setting, 188 (47.7%) were from monogamous setting.

Amongst the respondents, 277 (70.3%) were brought up by married parents while 117 (29.7%) grew up in dysfunctional families (parents are separated /divorced). 176 (44.7%) were engaged in menial occupation such as hawking etc as additional sources of income while the remaining 218 (55.3%) were not.

TABLE 1. DISTRIBUTION OF RESPONDENTS BY AGE

AGE (YEARS)	NUMBER	%
10-14	11	2.8
15-19	352	89.3
20-24	31	7.9
Total	394	100.0

The point prevalence of drugs used was 69.3%. Out of these drugs, alabukun was the most ever used (64.5%) and currently used (12.1%). Its prevalence was 44.7% (Table 2).

Hashi was the least used ever and at the time of the study there were no current users in the preceding 1-week. There were more users of cocaine than Hashi both ever before and at the time of the study. There were equal numbers of students who had ever used alcohol and Valium in their lifetime. Summarily, in those who have ever taken to any of the drugs; users of alabukun were> kola nut> coffee> alcohol which was the same as users of Valium> Evostick> petrol>Librium> Cigarette> snuff> Cocaine> Hashi (Table 2)

In the week preceding the time of the study, alabukun was the predominantly used drug. The incidence rate was 8.4%. The users of coffee and Valium were the same in number; likewise those of petrol and Evostick as well as users of snuff and Librium. In summary, incidence were: users of Alabukun were> Alcohol> kola nut> coffee> Valium> petrol> Evostick> Cigarette > snuff >Librium> cocaine. (Table 2)

Out of 394 respondents, 123 (5.1%) used only a single drug while 150 were multiple users. Amongst the latter, 14 had used any 2 of the social or recreational drugs, 2 had used any of the stimulants while few (7) had used any 2 of the so-called hard drugs (Table 3).

The prevalence of drug abuse and drug dependence was 4.1% (16 students) and 3.6% (14 students) respectively.

Most of the respondents were introduced to the use of drugs by their friends 49 (12.4%), closely followed by a family member 42 (10.7%). A large number took to use of drugs without prior introduction by anybody (Table 4). As regards drug use and associated factor; significantly respondents in 20-24 years age group were more likely to use drug than those in the 10-14 and 15-19 years age group ($x^2 = 6.08, p<0.05$) Table 5.

There was a significant relationship between the use of drugs and gender of respondents. Males were more likely to use drugs than females with a ratio of

TABLE 2. DRUG USE AMONG RESPONDENTS

DRUGS	EVER USED	CURRENTLY USED	NO	%TOTAL RESPONDNETS	%DRUG USERS	NO	%TOTALRESPONDENTS	%DRUG USERS
CIGARETTE	12	3.0	4.4	4	1.0	1.5		
HASHI	2	0.5	0.7	0	0	0		
ALCOHOL	74	18.7	27.1	30	7.6	11		
KOLANUT	100	25.4	36.6	28	7.1	10.3		
VALIUUM	74	18.7	27.1	23	5.8	8.4		
ALABUKUN	176	44.7	64.5	33	8.4	12.1		
PETROL	16	4.1	5.7	8	2.0	2.9		
COFFEE	83	21.0	30.4	23	5.8	8.4		
SNUFF	10	2.5	3.7	2	0.5	0.7		
EVOSTICK	22	5.5	8.0	8	2.0	2.9		
COCAINE	4	1.1	1.4	1	0.3	0.4		
LIBRIUM	15	3.8	5.5	5	0.5	0.7		

7.11,p<0.01) Table 6. In addition there was a highly tically significant relationship between peer influence rug use (x^2 = 26.22,p<0.0000001) as well as be-

TABLE 3. RESPONDENTS AND MULTIPLE USES OF DRUGS

GROUP	DRUGS	NUMBER
SOCIAL DRUGS)	Cigarette, alcohol, snuff	14
STIMULANTS)	Valium, Alabukun, coffee, Librium, kolanut	122
HARD DRUGS)	Hashi, petrol, evostick, cocaine	7

care giver influence and drug use 3.47,p<0.000001) Table 7 and 8 respectively. Sig- tly respondents who were engaged in after school re more likely to use drug than those who were = 5.37,p<0.05)

wever, the type of family (x^2=2.88,p>0.05), level uality (x^2=0.01,p>0.5) and marital status of par- =0.01,p>0.5) had no significant effect on the use gs.

USSION

his study showed a point nce of 69.3% signifying a high lence of drug use amongst scents in senior secondary schools -Ora. This is in keeping with the nducted by Odejide et al in 1997 secondary school students in Ibadan and Abeokuta 51.6% and 51.5% were respectivley reported[3] and een demonstrated in this study that the most widely use drug is alabukun, followed by kolanut. Moreover among current users, alabukun was still the most widely used, followed by alcohol. This is in contact with a similar study among the same set of students, which showed cannabis to be the most widely used drug[20].

The present study showed that drug use and abuse is commoner in adolescents aged 20-24 years than 10-14 and 15-19 years age group. Similar findings have been reported; in which rates are high among adolescents particularly around the school leaving age[9].

The study has revealed a higher preponderance of drug use in males than in female (1:5:1). Previous studies have supported this[3,7,15,17,19,21]. It is evident from this study that no relationship exists between drug use and being brought up in dysfunctional families where parents were either separated, divorced or (one / both) dead. It has showed that there is no significant affectation of adolescent drug behaviour whether either parents or someonelse raises

TABLE 4. INTRODUCTION TO DRUGS

Introduced to drugs by	Number of respondents	%
Nobody	164	41.6
Father	37	9.4
Mother	35	8.9
Family member	42	10.7
Friend	49	12.4
Medical/Nursing personnel	15	3.8
More than 1 person	52	13.2
TOTAL	**394**	**100.0**

TABLE 5. DRUG USE AND AGE

AGE (YRS)	DRUG USERS (%)		NON - DRUG USERS (%)		
10-14	5	(45.5)	6	(54.5)	11
15-19	242	(68.8)	110	(31.2)	352
20-24	26	(83.9)	5	(16.1)	31
Total	273		121		394

TABLE 6. DRUG USE AND SEX

SEX	DRUG USERS %	NON - DRUG USERS %	
MALES	163 (75.1%)	54 (24.9%)	217
FEMALES	110 (62.1%)	67 (37.9%)	177
TOTAL	273	121	394

her. All these are however in contrast to similar studies.[25,26] This is probably due to absence of nuclear bond in most families in Igbo-Ora since majority of respondents were from polygamous family. They probably get along well with life in the absence of the two parents.

This study demonstrated a significant relationship between drug use and those who are employed outside the home as motor park attendants, hawkers, farmers, mechanics etc. Thus, it is similar to results obtained in studies carried out by other researchers [3,15,27,32].

Moreover, this study has shown that peer pressure significantly affects drug use in adolescents.

This study went further to establish a relationship between drug use and poor role modeling, similar observation abound[9,10,11,21,23,27].

However, the association between drug use and problem with law enforcement agencies like the police could not be demonstrated. The possible reasons are as follows. It is not an absolute criterion but one of the criteria for drug use. Those who abuse drugs amongst the respondents probably have a way of keeping it under control.

There was no significant association between involvement in spiritual activities and drug use in adolescents in this study, which is in contrast to some previous studies[16,22].

In this study, more adolescents took to simple than multiple drug use which is in contrast to a previous study[3].

TABLE 7. DRUG USE AND PEER INFLUENCE

RESPONDENTS CLOSE FRIEND	USE DRUGS %	DONT USE DURGS %	
USE DRUG	100 (88.5%)	13 (11.5%)	113
DON'T USE DRUG	173 (61.6%)	108 (38.4%)	281
TOTAL	273	121	

This is probably due to their non- adventurous nature and financial constraint.

CONCLUSIONS AND RECOMMENDATIONS

This study has demonstrated that there is a widespread use of drugs amongst senior secondary school students in Igbo-Ora. It is worthy of note that there are actually cases of d abuse and dependences. These are by far uncommon as they account for 4.1% (16 respond and 3.6% (14 students) respectively. The risk fac for drug use in this group of respondents are age (years), male sex, peer group influence, poor modelling, and engagement in menial jobs. Thus significance of parental influence, peer group pres etc on drug use in adolescent cannot be over- emphasi This influence has untoward effects on their health, psy personality and future.

Moreover, in order to save our youth, who are bedrock of any nation, the aforementioned risk fac have to be taken into cognisnace in establishing a d awareness and rehabilitation programme.

Based on the findings of our study, we recomm the following:

1. Education of the society at large on the use abuse of drugs. This has a pivotal influence on both parents and these adolescents. Once the parents are with the essential knowledge, they can take up responsibility of keeping their children drug free.

2. Parents should help their children develop that s esteem which is unshakable by any form of peer pres Every adolescent should see himself/herself as a pers be reckoned with in the society.

3. Vocational intervention for the parentall deprived should be considered by the government to g them both the emotional and financial support. This sho not be that tasking as to warrant the use of psychoac drugs.

4. Parents should closely supervise their child (especially those of adolescent age group), know t group of friends and keep them busy after school h e.g. by sending them to extramural cla 'devil finds work for an idle hand say adage'.

5. Existing laws guiding sale distribution of drugs should be f executed; as such we commend the e of NDLEA

6. Finally, medical students sh

TABLE 8. INFLUENCE OF CARE GIVER ON DRUG USE

RESPONDENTS CARE GIVER	USE DRUGS %	DONT USE %	
USE DRUGS	131 (83.4%)	26 (16.6%)	157
DON'T USE	142 (59.9%)	95 (40.1%)	237
TOTAL	273	121	394

engage themselves in this type of project that focuses on vital issues not only for academic purposes but also as their contribution to the society. They should take part in more health talks as the upcoming adolescents see them as role models.

ACKNOWLEDGEMENT

Special thanks to Almighty God for the grace with which we commenced the project and brought it to completion; gratitude to our supervisor Dr(Mrs) T.O Lawoyin for her invaluable advice and penchant thoroughness. Dr Osungbade for proof-reading the project and Dr Sangowawa (miss) for her ever willingness to assist.

Finally, I appreciate my group members who worked conscientiously to make the project a reality.

Ajumobi Olufemi,
Subgroup Captain

TABLE 9. DRUG USE AND ENGAGEMENT IN AFTER SCHOOL/ ODD JOB

ODD JOB	DRUG USERS %	NON-DRUG USERS %	
ENGAGED	133 (75.6%)	43 (24.4%)	176
NON- ENGAGED	140 (64.2%)	78 (35.8%)	218
TOTAL	273	121	394

TABLE 10. DRUG USE AND LEVEL OF SPIRITUALITY

LEVEL OF SPIRITUALITY	USE DRUGS %	DON'T USE %	
Passive	6 (68.7%)	21 (31.3%)	67
Active	227 (69.4%)	100 (30.6%)	327
TOTAL	273	121	394

TABLE 11. FAMILY TYPE AND DRUG USE RESPONDENTS

FAMILY TYPE	USE DRUGS %	DONT USE DRUG %	
MONOGAMY	22 (6.9%)	66 (35.1%)	188
POLYGAMY	151 (73.3%)	55 (26.7%)	206
TOTAL	273	121	394

TABLE 12. MARITAL STATUS OF PARENTS AND DRUG USE IN RESPONDENTS

	USE DRUG %	DON'T USE DRUG %	
MARRIED PARENTS	191 (69.0)	86 (31.0)	277
SEPARATED/ DIVORCED PARTNETS	82 (70.1)	35 (29.9)	117
TOTAL	273	121	394

REFERENCES

1. Diagnostic statistical Manual DSMIV4th edition. American Psychiatric Association 199; 103-143
2. World Health Organization ICD IO Chapter V. Mental behaviour and developmental disorder: clinical description and diagnostic guidelines, Geneva. WHO 1993
3. Odejide AO. Adolescents and young adult substance use problem in Nigeria. A paper presented at the centre for health services, training, research and development, Ibadan. 1997.
4. Lasebikan VO. Dissertation on pattern of drug use among commercial drivers in selected motor part in Ibadan 2001:112
5. Dight SE. Scottish drinking habit: a survey of Scottish drinking habits and attitudes towards alcohol; office of population censes and survey, HMSO London 1976.
6. Sourindrin I, Baird JA. Management of solvent abuse: a Glasgow community approach. British Journal of addiction 1984; 79: 227 – 32
7. Yang MS, Yang MJ, Liu YH, KO YC. Prevalence ad related risk factors of illicit and licit substance use by adolescent students in southern Taiwan. Public Health 1998; 112 (5): 347 - 52.
8. Odejide AO. Drug use and abuse: Facts, consequences and remedies. Paper presented at the development policy centre Ibadan 1998.
9. Gelder M, Gath D, Mayor R. Alcohol and other psychoactive substance abuse Oxford textbook of Psychiatry, 2nd edition. 1998 Chapter 13 pp 521. OUP.
10. Kaplan H, Sadock B. Psychoactive substance use disorders. Synopsis of Psychiatry 7th edition. 1994 page 383 – Lippincolt, William & William
11. Hawker A. Adolescents and Alcohol 1978. Edsal London
12. Edward G, chandler J, Hemman C. Drinking in a London suburb. Quarterly Journal of studies on Alcoholism 1972 (Supl°) 6: 69 – 128
13. Wilson P. Survey of drinking in England and Wales. Office of population census and survey. HMSO London 1980.
14. Sourindrin I. Solvent abuse. British medical Journal 1985; 290: 94 –5.
15. Adelekan ML, Abiodun OA, Obayan AO, Oni G, Ogunremi OO. Prevalence and Pattern of substance use amongst undergraduates in Nigeria University. Drug and Alcohol dependence, 1992; 29: 255-261
16. Adelekan ML. The epidemiology and social contents of amphetamine and psychostimulant use in Nigeria. Paper presents for WHO (PSA) meeting, Geneva 1996.
17. Odejide AO, Ohaeri JU. Drug related admission in 28 mental health institutions in Nigeria in 1989. Commissioned by the federal ministry of health. Drug and alcohol dependence 1993; 31: 101-109
18. Kandel D, Chenm K, Warner LA, Kessler RC, Grant B. Prevalence and demographic correlates of symptoms of last year dependence on alcohol, marijuana and cocaine in US population. Drug and Alcohol Dependence 1997; 44 (1): 11-29.
19. Shaw S. The causes of increasing drinking problem among women. in women and alcohol 19980. Tav Stock – London
20. Elegbeleye OO and Pearse D. British Journal of preventive and social medicine 1976; 30: 60-70
21. Obot IS. The use of tobacco products among Nigerian adults. A general population survey in Drug alcohol dependence 1990; 25 (2): 203-8
22. Ndom RJ, Adelekan ML. Psychosocial correlates of substance use among undergraduates in Ilorin University, Nigeria. East African Medical

Journal 1996; 73 (8): 5441 – 7

23. Surgeon General's Report United States 1979 cited in status of adolescents and young adults in Nigeria by Odejide AO 1997. A paper presented at the centre for health services, training and development centre, Ibadan.

24. Vitaro F, Tremblay RE, Zoccolillo M, Alcoholic father, adolescent drug abuse and protective Factors. Canadian Journal of psychiatry 1999; 44 (9): 901 – 8

25. Shek DT. Family environment and adolescent psychological well being, school adjustment and problem behaviour: a pioneer study in a Chinese content Journal of Genetic Psychology 1997; 158 (1): 113- 28

26. Odejide AO, Sanda AO. Observation on drug abuse in western Nigeria. African Journal of Psychiatry 1976 5 (1&2:21-29

27. Nevadomsky J. Self reported drug use among secondary school students in two rapidly developing Nigeria towns. Bulletin on Narcotics 1982; 34:21 – 32

28. Akindele MB. Student and drugs. Ghana Medical Journal 19 3:184-187.

29. Abiodun OA. Knowledge and views on drug abuse of prim health care workers in Nigeria. Drug alcohol dependence 1991; 28 (2): 1 82.

30. Ebie JC, Pela OA. Substance in Nigeria: review of epidemiolog studies in Ebie JC and Tougue EJ edition handbook of the Nigerian Tra course on drug dependence ICAA, Benin city 1981.

31. Kish and Leslie. Survey sampling. John Willey and Sons. N 1965

32. Obot IS. Drinking behaviour and attitudes in Nigeria: A gen population in the middle belt. Research Monographs Jos; Centre Development studies, University of Jos.

MOLECULAR BIOLOGY RESEARCH:

A Student Perspective and Experience.

DANIA, Simpa

At the time of writing, the Author was a Second year clinical student at the College of Medicine, University of Ibadan.

"I wish to work miracles..." - Leonardo da Vinci

INTRODUCTION

Molecular biology has changed the landscape of the medical world greatly. It has given us a better understanding of the aetiology, pathophysiology, diagnosis and management of disease conditions. This rapidly developing field without doubt is the key to the future of medicine.

Molecular biology offers boundless unexplored opportunities for the curious questioning mind; the potentials for a rewarding research in molecular biology are simply irresistible. Since I have a passion for research, my elective posting therefore offered me a great opportunity to begin to build a foundation in this field.

I was priviledged to join Dr. Fiemu Nwariaku's (Assistant Professor, Surgery, UT Southwestern Medical School, Dallas, USA) research team. I spent about five weeks at the laboratory, which was located at the Veteran Affairs Hospital, Dallas, Texas.

BRIEF OVERVIEW OF RESEARCH WORK

The research was to determine the mechanism of Tumor Necrosis Factor-alpha(TNF-α) induced endothelial permeability with central hypothesis of a mitogen activated protein kinase (MAPK) dependent reduction of VE- cadherin. Its clinical application is related to formation of exudative oedema especially in disease conditions such as adult respiratory syndrome, systemic inflammatory response syndrome.

The method of research was divided into three main parts:

- Permeability study - this determined the effect of TNF-α on endothelial permeability as well as the time frame of this effect.
- Intracellular signaling pathway - this formed the central theme of the research trying to elucidate the pathway using various protein inhibitors as well as markers.
- VE- cadherin study - this was to describe the cytological location of the VE-cadherin in relation to the effect of Tnf-α.

RESEARCH EXPERIENCE

During my stay, despite being short I was able to fulfill to a large extent my goals, which are outlined below.

An opportunity to learn some fundamental concepts of molecular biology:

Fundamental concepts in molecular biology I learnt about were:

- The mechanism of cellular signaling.
- Cell to cell adhesion.
- Microvascular permeability.

Acquiring the basic practical skill required in molecular biology:

The skills or techniques I did my best to acquire were: protein assay, immunoprecipitation, Western blot, PCR, Southern blot, gene cloning, immunoflourescent staining, and permeability study using transwell.

Understanding research methodology and modalities:

I now understand that:

- The research begins with a clinical problem and the quest to understand the underlying mechanism.
- Research is funded by grants which are competitive.
- You must be up to date with the current knowledge base in regards to your area of interest before embarking on your work and during the course of it.
- Articles are published as new; interesting and relevant results are made during the research process.
- Creative thinking and critical analysis are required in the interpretation of results and development of new research method.

- The importance of collaborative work within and out of the team.
- Keeping of the journals detailing daily laboratory procedures and results is very important and key to the research.

This trip was not without its challenges within and outside the laboratory. Learning to work without having to fill a log book to show to someone at the end of my stay as well as determining my learning process was one, but in retrospect, I believe that I handled it well. The volume of information to be sourced and assimilated from various journals was another. I was happy when I got an article that proved to be a very significant one. Developing good interpersonal relationships was important to me during this trip. I indeed made some friends and they helped in making my stay a rewarding one.

PERSONAL REFLECTIONS

One issue that struck me was the exposure a lot of the medical students had concerning research especially in molecular biology unlike the case here, where molecular biology is just a mystery understood by a few and experienced only in the pages of textbooks. Students are given stipends to spend their breaks undertaking one form of research or the other. Here, even our teachers find it so difficult to carry any and when they do, students are relegated to the background. It became increasingly glaring how terrible our "research culture" was. The desire to question or investigate is either lost or suppressed in our young minds. More baffling is the fact that a research experience is a plus for any postgraduate form of study. Is there any way this can be resolved?

THE POSSIBILITY OF ESTABLISHING A MOLECULAR BIOLOGY RESEARCH LABORATORY IN U.C.H., IBADAN.

Whatsoever can be conceived in the mind can be achieved. It is indeed possible to undertake this Herculean task of setting up a laboratory. Without doubt the benefits of having such a laboratory cannot be overemphasized. These benefits include:

1) Stimulating interest in medical research amongst students and lecturers alike.
2) Making the teaching of molecular biology less esoteric.
3) The research experience would be of advantage especially for postgraduate medical training.
4) Providing funds for further development of the College of Medicine, University of Ibadan.
5) Make medicine a lot more interesting.
6) Facilitate greater interaction between College of Medicine, University of Ibadan and other medical schools abroad.

While the availability of facilities may seem beyond reach, we must accept that all limitations have solutions no matter how far fetched they may look at first. I would like to suggest the following concerning some likely shortcomings:

- Constant electricity via the use of solar energy.
- Provision of adequate waste disposal system.
- Procurement of materials
  - Appeal to manufacturers to supply various kits.
  - Role of international/national research funding bodies.
- Availability of literature – collaboration with other libraries outside, with access to their collections via the Internet.

The main determining factor is the willingness of the institution to establish such a laboratory; I strongly believe that with our collective efforts, we can make this a reality.

APPRECIATION

I am grateful to the following people who have immensely contributed to this wonderful experience: Dr. Fiemu Nwariaku, Mr. E.O. Olapade-Olaopa (FRCS), Dr. George Sarosi, Dr. Lance Terada, Dr. Rhonda Souza, Dr. Odukogbe, Drs. Ojeshina, Zhuo Zhu, Liu, Christie Lopez-Guzman, Lawrence Fan and Mrs. Julie Landrith.

CHAOS IN CASUALTY

OCHULOR, K

Dr. kenneth Ochulor is a Registrar in the Department of Medicine, University College Hospital, Ibadan.

On a certain day not too long ago, I was through with work and headed home. Not quite tired out, but somewhat roughened up. One of those days when no truly bad thing quite happens: hidden hands of frustration merely push you back and forth, dangle you briefly on the brink of exasperation, and return you back all intact. No harm done or humour gained. You know how it is when you're in one of these funny states: not exactly where the pull is, but tugging at the leash all the same. On this innocent day, with my nerve not quite well tuned, I somewhat forgot where exactly I was headed, took the wrong turn, and found myself in the Casualty. I paced unsuspectingly into that unspeakable red corridor by 5.00p.m, and didn't get out till the early hours of the next morning. And had enough trouble in one night to last me for months....

I had no business at A&E that day, but I suppose my cup was already full and merely waiting to spill. It's time they drag me down there once in a while, when I'm the Medical Registrar 'on take'. But I have always been careful not to take. Not when I can avoid it. Why should I? I'm learning how to be satisfied with the little I have. And though the cynical may call this clinical detachment of mine a dubious virtue, nobody is any the worse for it. What's more, I have generally made it my business not to get entangled in other people's emergencies. Because, to tell the truth, in such circumstances, I'm usually more of a nuisance than a help. For I have a record. Once, while a student, in the heat and clamour of it all, I was instructed to get a tourniquet for a line. Carried away by the mood of emergency, I grabbed the nearest thing I could find: a black white piece of tubing. With hindsight, I suspected it must have been a discarded cable from an old suctioning machine. There was so much agitation that I couldn't make sense out of so many orders barked out at the same time. But with such medley about me, I was forced to take the initiative. I eagerly strung the thing firmly around the only part of the patient that was free. And that was the neck! Almost strangled..., but let's not speak about the humanity...

Since then, I think I've learnt a few of the fine stuff in the books: the ABC of acute care and all that. But still when it comes to the real thing, with those powerful casualty chaps sweating it out in fever of resuscitation. I find myself too weak for that kind of exertion, and never manage to rise above the patent absurdities of the situation. For it is clear that the poor victims of the daily casualty experiment will do better dying at home. Why go through all that hassle just to have your death certificate signed in a hospital? No man –to say nothing of a woman –of modest strength will survive that ordeal, what with so many tubes thrust in, and as many robust hands grinding the last breath out of you. Those who make any effort to cry out are immediately gagged with an oro-tracheal airway, so that they can only manage occasional grunts, staring pathetically with their terrified eyes before the final event. I sympathise with them. And since I do not wish to have some kind but senseless friend surrender me to such terrors, I have put it into writing that I should be left in peace at home whenever my own crisis begins. At least I'd be able then, in between fits, to savour a fine novel or two, before the end...

Now don't misunderstand me. I love, venerate and esteem the Practice. Casualty is after all a wonderful place for those that need it. And our boys there are performing near miracles with the cutting edge of old technology. Just recently, some friend of mine, in spite of our hopes, slipped through the cold hands of death, thanks to his haven gotten to A&E early enough. It was a triumph of medical diligence. I don't remember what it was in the first place that took him there. But they worked him thoroughly, and literally beat the demons out of him overnight. He's now one of the candidates for an ongoing study on multiple rib fracture post resuscitation. But he's not complaining. For one thing, he is grateful to have escaped with enough of his lungs to permit an occasional puff of cigarettes, the old rascal! Which is not such a bad bargain, when you weigh it objectively.

So I got to the casualty, strolled casually up to the reception, launched into a favourite pastime, in the act of beguiling the matron on for that afternoon with one of my contrived nonsense, when the commotion began. A number of people were shouting outside, the nurses too began shouting, the matron took off to find out what was going on, and before you knew it, some men were rushing in three horribly mutilated bodies, with blood splashing here and there. Of course I wasn't going to get involved. I simply stepped aside when the first tide of chaos swept past. The first fellow they were carrying had his left hand hanging by a thin strand. Looked already dead, from what I could see. The second had multiple cuts all over the body, and it didn't look too good a case either. A similar instrument must have been applied with fatal intent on the head of the third, who was bleeding profusely, and letting out muffled whimpers of pain, half gone. I overheard that they were victims of student warfare at the Polytechnic. Happily, I had no wish to know anything further about it. I was slowly wrapping up my ward coat and getting ready to steal away, when the matron came back. Began pleading that I stay. Hands were needed. The second of the casualty officers for that afternoon hadn't turned up. So what? That was no concern of mine...

But in the end I stayed. No sterile gowns available, as usual. I first began gingerly, suturing with delicate moves, and hoping soon to be relieved. It was no use though. In the thick of the thing, I had to abandon both the pretext and the hope. Suture from every conceivable angle, and in the end soaked up my ward coat in blood. The third chap looked like he would make it. But when my colleague began that inane procedure on the second victim, who was already at entry, I gently eased off. Only to run into some wild looking fellows who wanted to take pictures of the bodies, and were threatening to take them away to some other place. This being a crazy country, I was careful not to inquire whether they were the police or not, and simply pointed to where the traumatized remains were.

I had hardly settled down in the consulting room when another rumble began. Came out into the corridor to see a woman flanked by two other women and accompanied by a man. It was quite a spectacle. The woman in the middle was dancing in a most strange way. She would contort her face and body into such queer postures, sway this way and that, and then suddenly leap into the air. Back down, and then after a few limps forward, repeat the amazing sequence. Meanwhile the man, who must have been the husband, was fanning her frantically, meanwhile the two other women with her were both shouting at the same time, making cutting gestures with their hands intervals. The nurse who was supposed to conduct the kept a safe distance, merely pointing the man towards And in this way the bizarre procession made its way in direction...

For about three minutes after getting into consulting room, the shouting and gesturing and fanning and dancing continued: a thorough madhouse. It turned out that the two women were mouthing incantations Pentecostal way, to exorcise the other woman of whatever devil had taken hold of her that evening. And since appeared the mayhem wouldn't stop if I didn't intervene I was forced in the end to shout them down, devil and All that seeming hysteria would have been a matter for psychiatrists. But the real case was even simpler. The poor lady was in a mute agony from a scorpion sting. She had seen the wicked bad thing scurrying away into a dark corner after the assault, and was only able to tell her husband about it before the excruciating pains took over.

Meanwhile these two 'sisters' who were visiting seeing her soundless but disturbing contortions, were convinced it was a case of demonic possession, and immediately launched their own line of attack. I had little trouble calming everyone down and administering analgesic to the pain-crazed woman, before the nurse dragged me off to some other case.

And so the night wore on. I was hardly through with one thing when I was rushed off to another. You'd be amazed at the variety of animals, human and otherwise, that people live with in this town, and what happens when they quarrel. Or the peculiar game playing that accompanies illness. Here the sick usually wait and waste away at home only coming to the hospital at the very last moment, at night! And often accompanied by a riot of people, usually in a shabby bus, most of who would begin to sneak away beginning with the eldest-once you start to talk about money...

But what broke my back that day was the very last case; a literal last straw! I was already fretting, as it began to seem the guy for the night shift also wasn't going to show up. And just as I was deciding how to extricate myself from all this madness, they now rushed in a guy who was convulsing all over and foaming at the mouth. His hands were tied. And you could tell someone had been trying to force something into his mouth, as it was all bruised and bloody. The fellow smelt like he had been in the belly of some fish for weeks. The whole of A&E was heavy

with the odour, and the facemasks were of no use. As the resuscitation room was full, they moved him to one of the cubicles, near a patient with multiple fractures from an accident. Much to my surprise, this man immediately dragged himself up, balanced on his one remaining good one, and hobbled away to safety some miles away. The smell was that bad! So I braced myself for the task. With the nurses holding him down, and all of us dodging his arm swings and savage kicks, managed to set up an infusion line. We sedated him heavily, and when he was calmed, went out to take history from those who brought him in. But I saw no one, nor any cars packed outside. No one could tell. We spent quite some time looking stupidly at each other, confounded. And by the time we went back to the cubicle, the patient was not to be seen either.

I imagine you do not believe in collective hallucinations. Neither do I. And yet somehow the whole thing began to look somewhat eerie. How could he have vanished? And what of those who had brought him? By now it was long past midnight, and finally at my wits end, I stalked out of the casualty, rubbing my sleepy eyes a couple of times and feeling quite rattled and dull...

But in the morning, they told me what had happened. The tall zealous porter, appearing suddenly while I was away, and asking no questions, had briskly whisked off the sedated patient, drip line and all. Down to the mortuary. And that was that...! So you'd better watch next time you find yourself on the couch at casualty.

By the way, just last week, they rushed one of my consultants down to casualty. You can imagine what happened: all hell broke loose. But then that's another story...

PATIENT-DOCTOR RELATIONSHIP: MEDICO-LEGAL ISSUES

OSHIN, BAYO

Mr. Bayo Oshin is a 2nd year clinical student in the College of Medicine, University College Hospital, Ibadan

"Although a Doctor may know much about a person's illness, if he or she knows little about the person, little or no healing will take place"

Hippocrates

INTRODUCTION

The Doctor-patient relationship is the foundation of modern medical ethics. It is the touchstone for professional conduct and a very vital issue to medical practitioners. The Doctor-patient relationship is changing, and so is medical ethics.

The importance of the medico-legal aspect of the medical practice in Nigeria cannot be overemphasized. It is all about the duties of the doctor in partnership with the law and how the doctor discharges these duties in practice and in court. The doctor-patient relationship represents the fundamental expectation of how doctors and patients are supposed to behave towards one another. The traditional doctor-patient relationship is vital to the survival of the physician, and to the physical survival of the patient.

Legal or forensic medicine has attracted considerable media attention particularly in the context of the insanity defence in criminal trials. In addition, it is clear that in the late 20th and early 21st century, medical practice and research have raised crucial ethical and legal issues, surrounding, for example, abortion, transplantation, euthanasia, cloning, etc.

Fundamentals of the doctor patient relationship

The doctor-patient relationship is basically the reciprocal trust, cooperation and understanding between doctors and their patients. A successful medical practice requires a relationship of trust between doctor and the patient. The purpose of medical practice is to relieve the suffering of the patient. The word 'patient' is derived from the Latin word *patiens*, meaning sufferance or forbearance[1].in order to achieve this purpose, it is important to make a diagnosis, know how to approach treatment and design an appropriate scheme of management for each patient. It is therefore essential to understand people as much as possible, whatever their social, religious, ethnic, or cultural background. The key to this and the beginning of the doctor patient relationship is communication.

Communication

Communication is a two way process between two or more people. Communication between the doctor and the patient can be verbal or non verbal. Effective use of non-verbal communication can help develop an atmosphere of trust. It starts as soon as the patient walks into the consulting room. Body language, eye contact, facial expression all help to enhance the interview with the patient. The patient's appearance, attitude and demeanour all give indications to the nature of the problem. The doctor should welcome his patient with a smile, introduce himself and offer a greeting. Frequent eye contact should be maintained during the consultation.

Listening and talking are important components of verbal communication. Doctors are encouraged to do more of active listening and allow patients to tell their story. This helps the doctor to fully understand the patient's condition although the physician should know when and how to prevent the patient from rambling on. Language difficulties may also pose a problem to effective verbal communication.

Comportment

The doctor should not allow his emotions to show. This however does not mean that he should not empathize with his patient. Patients who are allowed to express themselves as freely as possible are often able to describe their symptoms better and aid the doctor in reaching a conclusion.

While it is true that the doctor's role is always to establish a relationship with the patient, this cannot always be successfully accomplished. The patient could be ill because of anger, stress, anxiety, confusion or grief.

Trust and Understanding

The doctor-patient relationship should be based on mutual trust and understanding. Trust has been shown to improve the patient's compliance. The doctor must encourage his patients to describe the particular

at worries them most, as this helps to rid the patient's ind of any suspicion about the doctor's intentions. Pa- ents should also be open with their physicians, as those o are discovered to be withholding information betray e doctor's trust and confidence in them.

Understanding a patient's concerns and worries with- t offering false reassurances can help to comfort the tients. The patient in turn should understand the doctor- s long, stressful working hours, continually growing pa- nt list and limited working conditions.

cial, Cultural and Religious Factors

The social status, cultural background and religious liefs of both doctor and patient go a long way in termining their relationship. Well-to-do patients often l pompous and do not tell the truth or comply with ctor's instructions while on the other hand, poor patients unable to follow the treatment plan due to poverty.

Beliefs of religious sects such as the refusal of blood nsfusion by Jehovah's witnesses and the abstinence of slims from pigs and pig products affect the doctor's ity to totally care for them. The doctor must know how ndle these peculiar situations and proffer alternatives.

The doctor should not allow these factors to affect sponsibility to the patient's welfare keeping in mind ath he swore. (See No. 8 of Appendix A)

torical Overview of Medical Ethics

As far back as the fifth or sixth century B.C., the er of modern medicine, Hippocrates recognized the d for a code of conduct for practitioners of the art of ing and laid down a statement of code of medical ethics wn as the Hippocratic oath[2]. The oath recognized that sician's duty was to his patient and also, the special e of the doctor-patient relationship.

The oath, dedicated to the welfare of the sick patient, ndergone certain modifications to bring it in line with ctice and language of modern medicine. This resulted Geneva Declaration created by the General Assembly World Medical Association in 1948[3].

l System in Nigeria

The law of Nigeria may be divided into two viz: *inal law*, concerned with offences against the State ligations imposed on citizens by law and *Civil law*, ed with disputes between citizens. The state or ividual injured by the wrongdoing may institute crimi- civil proceedings against a doctor[4].

The courts that handle matters of the law in Nigeria Customary, Magistrate and High (State and Federal) Appeals from these cases go to the higher courts – the Court of Appeal and the Supreme Court.

It is important to know that two codes of criminal acts apply in Nigeria. The *criminal code* applies in the southern states of the country while the *penal code* applies in the northern states[5].

Regulatory Body of Medical Practice in Nigeria

The establishment of the Nigerian Medical and Dental Council in 1963, was by the enactment of the first Medical Act of Nigeria known as Act No.9. The purpose of the Act was to regulate the practice of the Medical and Dental professions in Nigeria[6].

ISSUES OF MEDICOLEGAL INTEREST IN NIGERIA

Abortion

Legally, abortion is defined as the termination of pregnancy before 28th week of gestation[7]. Abortion may be spontaneous or induced. An abortion, which has been illegally induced, is a criminal abortion.

Abortion is governed by the laws found in Sections 228-230 of the criminal code of the Laws of the Federation of Nigeria 1958[8]. For the offence to be committed the abortion must be performed 'unlawfully' but the law does not define what 'unlawfully' means. In other words, when is it lawful to perform abortion? Mr. Justice Macnaghten in the case of Rex versus Alex Bourne in England in 1938 stated that 'it is unlawful except for the purpose of preserving the life of the mother'[9].

In Nigeria, majority of those who procure abortions are young unmarried girls. They resort to quacks because they are afraid of informing their parents and also are unable to pay the doctor's bill. Methods of procuring abortion may be invasive or non-invasive. Non-invasive methods are ingestion of drugs or alcohol while the invasive methods are either by injections, insertions or instruments.

Our society today has become increasingly permissive in sexual matters. Premarital sex and adolescent sexuality are facts of life in today's Nigeria. The use of modern contraceptive methods only reduces, not eliminate, unwanted pregnancies. The enactment of an updated, relevant and enforceable abortion law would go a long way in reducing the incidence of illegal abortions and save the human suffering and wastage resulting from them.

Medical Negligence

Once a doctor undertakes to treat a patient, whether or not there is an agreement between them, a duty of care[10] arises. However, a doctor who refuses to respond to a distress call or refuses to treat a patient who is not his is

not to be considered negligent. A breach in the duty[11] occurs when a doctor fails to come up to the standard of skill and care required of him. Examples of conduct that constitutes breach of duty are: failure to admit a patient who requires hospitalization, failure to cross-match transfused blood and leaving a surgical instrument in the body of patient after an operation.

An accusation of negligence often implies that the doctor-patient relationship has broken down. The patient must suffer some form of damage (not necessarily physical) as a result of the doctor's negligence before he can succeed in an action against the doctor. It is not sufficient that the doctor was negligent but it must be shown that the harm was caused by the doctor's negligence. The burden of proving this is on the patient who is the plaintiff in this case.

However, in certain circumstances, the plaintiff's burden of proof is made easier by the application of *res loquitur*- Latin for the fact speaks for itself[12]. It applies only in the absence of an explanation. For example, a patient who enters hospital for treatment of Dupuytren's contracture of two fingers ends up, after treatment, with four stiff fingers and a hand that was useless[13].

Appearance in Court

A doctor may be called upon as a witness by the court to give evidence in a case. This evidence may be his testimony in a civil or criminal suit or a medical report on an autopsy in a murder case, for example. The doctor must therefore learn about court technicalities, how to conduct himself in court and give evidence in an acceptable manner.

Evidence, in legal terms, is made of facts into which the court inquires and the legal means of attempting to prove these facts[14]. In court, the doctor is both a witness of fact and an expert witness. Before giving evidence, the doctor swears an oath to tell the truth, the whole truth and nothing but the truth according to his religious belief.

It is true there are delays in hearing cases in Nigerian courts but many judges allow experts to give evidence early to release them for other commitments. A doctor must of necessity understand and comply with the court proceedings and as much as possible give their evidence truthfully and accurately. A doctor is guilty of perjury when he willfully gives false testimony after taking an oath[15].

Consent

Consent to medical examination or treatment may be expressed or implied. Express consent is given where the patient orally or in writing expresses agreement to the treatment. The holding out of a hand for an injection or lying down on a couch for an examination implies that the patient has given his consent to the procedures. There are three requirements for consent to be legal[16]:

1. The patient must be mentally and legally competent to give consent.

2. The patient must have been sufficiently well informed to be able to give consent.

3. Consent must have been given voluntarily, and not under duress.

Consent is valid only if it is an informed consent. This requires that the doctor should inform the patient adequately of the ailment, proposed treatment and possible risks involved to enable the patients understand and make an intelligent decision.

In Nigeria, necessary details are not often communicated to patients and as a result, informed consent is not always obtained. This is probably due to poor educational background compelling patients' reliance on the doctor to make the decisions concerning treatment. Lawsuits against doctors for unauthorized treatment are rare but the situation is changing. Doctors should therefore protect themselves against possible legal action by providing sufficient information for the patient to make a decision.

Confidentiality

All aspects of the medical consultation should be confidential. This allows the patient the freedom to express himself in the knowledge that his 'secrets' will be kept by the doctor. This principle of confidentiality applies to medical records too. Hospital records should only be available to members of staff involved in management.

These records are only available with the doctor's permission and in certain instances such as an overriding duty to the larger society. This principle of confidentiality of medical information was recognized by Hippocrates and is incorporated into the modern Declaration of Geneva (see No. 5 of Appendix A)

Last Word

Medicolegal issues are gradually gaining prominence and it is essential for both patient and doctor to be aware of them so that they don't run foul of the law and his oath. Pertinent issues today not mentioned include HIV/AIDS, death and autopsy, child abuse and clinical research. Several issues yet to gain relevance in Nigeria but with time will are organ donation and transplantation, surrogate motherhood and cloning.

Doctors need to be aware of these issues and of the legal requirements and implications to prevent unwarranted lawsuits. The general public must also be educated in these matters to aid them in making the right decisions when the need arises.

The dearth of knowledge on aspects of medicolegal practice has stemmed from the absence and/or the poor teaching of these subjects in medical schools in Nigeria, the lack of relevant specialists and the absence of comprehensive subject material. There is an urgent need to teach these subjects so as to improve on the efficiency and image of the medical profession.

NOTES

1. Umerah, B.C., ed. Medical Practice and the Law in Nigeria. Nigeria: Longman Nigeria Ltd., 1989.
2. Ibid.
3. Ibid.
4. Ibid.
5. Ibid.
6. Ibid.
7. Ibid.
8. Ibid.
9. Ibid.
10. Ibid.
11. Ibid.
12. Ibid.
13. Ibid.
14. Ibid.
15. Ibid.
16. Swash, M., ed. Hutchinson's Clinical Methods. Edinburgh: W.B. Saunders, 2002.

REFERENCES.

1. Umerah, B.C., ed. Medical Practice and the Law in Nigeria. Nigeria: Longman Nigeria Ltd., 1989.
2. Swash, M., ed. Hutchinson's Clinical Methods. Edinburgh: W.B. Saunders, 2002.

APPENDIX A

The Declaration of Geneva on admittance to the medical profession:

1. I will solemnly pledge myself to consecrate my life to the service of humanity.
2. I will give my teachers the respect and gratitude, which is their due.
3. I will practice my profession with conscience and dignity.
4. The health of my patients will be my first consideration.
5. I will respect secrets that have been confided in me, even after the patient has died.
6. I will maintain by all the means in my power the honour and noble traditions of the medical profession.
7. My colleagues will be my brothers.
8. I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient.
9. I will maintain the utmost respect for human life from the time of conception. Even under threat I will not use my medical knowledge contrary to the laws of humanity.
10. I make these promises solemnly, freely and upon my honour.

EDITOR'S NOTE

Mr. Bayo Oshin's piece was the first prize winning essay in the DOKITA Editorial Board organized Annual Prof. J. A. Adeleye Essay Competition

Other participants are:
Mr. Oni Ebenezer
Mr. Abiola Niyi
Miss Opurum Adaeze
Mr Olagbenro Adeola

Acknowledgement

We would like to appreciate the efforts of Dr. A. Malomo and Dr(Mrs) O. Omigbodun in assessing the entries for the DOKITA Annual Prof. J. A. Adeleye Essay competition.
Thank you very much

DOKITA NEWS

APPOINTMENTS

The new Provost of the College, Professor Isaac Folorunsho Adewole of the Department of Obstetric and Gynaecology assumed duty on Thursday, 1 August 2002. Professor Adewole is the seventh elected Provost of the college.

Also on the same day, Professor F.A.A. Adeniyi of the Chemical Pathology Department and Professor Olaitan A. Soyannwo of the Department of Anaesthesia assumed duty as Dean of Faculties of Basic Medical Sciences and of Clinical Sciences respectively. Also, Professor J.D. Adeniyi of Health Promotion and Education department assumed duty as the first elected Dean of the Faculty of Public Health.

Provost usually serve a four-year term while Deans serve a two-year but are allowed by the Statute of the College to re-contest again immediately after their first term.

The University Council has approved the appointment of Professor Kikelomo Osinusi M.B.B.S. {IB] FNMC {PAED}, FWACP {PAED} as the Deputy Provost of the College of Medicine for two years {2002-2004}, having won the election conducted on 08 November 2002.

BENEFACTIONS:

The College acknowledges with thanks the following donations.

(i) Professor B.O. Onadeko, Department of Medicine:
-Donated the sum of two hundred and fifty dollars ($250) to the Department of Medicine being the first installment towards the upliftment of teaching facilities.

(ii) Professor Ajovi B. Scott-Emuakpor, USA:
-Donated two Resuscitation Dummies to The Department of Surgery for the training of Medical Students and Residents on the basic aspects of resuscitation.

(iii) Mr. Akinbola A. Adeniyi, U.K:
-Donated 60 issues of journals (Virology News 1997-2001; BJU International September 1998- February 2002) to the Department of Surgery.

(iv) Dr. Fiemu E. Nwariaku, USA:
-Donated Gift subscription to Selected Readings in General Surgery (the issue Surgical Infections and Antibiotics, Part 3 of 3) to the Department of Surgery.

(v) Association of physiotherapy students sponsored the renovation of their lecture theatre.

(vi) Dr. Devell R. Young, MD from USA:
-Donated Textbooks of Surgery by Beauchamp Evers Mattox (16th Edition) and Textbook of Medicine by Goldman Bennett (21st Edition) to the Colleges.

OBITUARY

With a Heavy heart, the Board announces the Death of a foremost Professor of Paediatrics, Professor Olikoye Ransome – Kuti. Professor Ransome – Kuti was once the Federal Minister of Health, Federal Republic of Nigeria and has Chaired 2 DOKITA Symposia. He died on Monday, 2nd June 2003, aged 75years.

The Board also announces the death of one of our colleagues, Miss Busola Adeyemo, a 400l Medical student at the time of death.

GRANTS

(i) Grants Information on the Fogarty International Centre (FIC) is available at http:/www.nih.gov/fic/grantsinformation/Grant.html

(ii) Bill and Melinda Gates Foundation has established the William H. Feoge Fellowships in Global Health to honour the career and an achievement of one of the world's leading figures in public Health. Supported by a $5million Endowment. The new Fellowship Programme will be housed in the Rollins School of Public Health at Emory University. For complete information, visit http:/ www.gatesfoundation.org

USA FULLBRIGHT PROGRAMME

Information on the Fullbright Academic Exchange Programme is available at the Corporate Affairs Unit.

DOKITA SYMPOSIUM

The 37th annual symposium of **DOKITA** held on the 9th of July 2002 under the distinguished chairmanship of Professor Ayo Binite. The theme of the symposium was: **A BEAUTIFUL MIND: RE-EXAMINING SOCIAL ISSUES IN MENTAL HEALTH**

The speakers at the event were: Dr. Olaosebikan, Dr. C.O. Mume, Dr. O. Aina, and Dr. O. Olley.

NATIONAL QUIZ COMPETION

The **DOKITA** Editorial Board biennial Professor O. O. Akinkugbe National Quiz Competition was probably the event of the year for the Board. This event took place between the 12th and 14th of November, 2002 with Nine Medical schools across the country in participation. The schools were:

University of Ibadan;
University of Maiduguri;
Ahmadu Bello University, Zaria;
University of Nigeria, Nsuka;
Obafemi Awolowo University, Ile-Ife;
University of Lagos;
Ladoke Akintola University, Oshogbo;
University of Benin; and
University of Port-Harcourt.

The preliminaries produced the finalists: University of Ibadan and University of Benin. University of Ibadan won the first prize with the University of Benin and University of Lagos becoming the 1st and 2nd runners-up respectively.

DOKITA WEBSITE

In the last year, the Board was able to successfully set up her website. The domain name is www.geocities.com/dokitaboard/dokita

POEMS

LIVE THE DREAM

If you will not dream
Smiling at the thought of the unseen
Then how will others scream?
Rejoicing at the sight of a new being

If you will not dare
Laughing at the dangers of the unknown
Then how will others share?
Delighting in the discoveries of great renown

If you will not leap
Scaling over the fence of common reason
Then how will others flip?
Transforming at the reach of a new season

If you will not cry
Wailing over the passive multitudes
Then how will they ever sigh?
Observing their needless servitude

But if you will,
Then, they too, will!

OLOWOLAIYE MIRIFO
2000 Set Group B

A DIFFERENT BREED

The same hands have pierced perfect ears;
have held an open heart together
The same hands have touched life;
have signed death ticket
The same hands have warred with life;
have courted death to win life
The same hands have milked life
The same eyes have watched it drain
through the same fingers

KEMI LAWANI
200 LEVEL MBBS

WHY NOT NOW

Why not now?
I'll do it soon
Well before noon
Why not now?
I'll do it then
Please don't ask when
Why not now?
I'll do it just yet
Just at sunset
Why not now?
I'll do it at night
When all is right
Why not now?
I'll do it now
Cos "now" is "how"
No other time but now
Soon you'll say wow!
Its good I did it now
So, why not now?

OLOWOLAIYEMO MIRIFO
2000 Set Group B

GENETIC CODE

Sixty-four horns of a clustering veil
Twisted to meet a seer's tale
Forth their wings bud and sail
To soar the glories of a hallowed dream
Round merries of a masquerade's thread
Joyous dances of an unveiling wand
Swung across the marker's bier
To swell an eternal seed
That grows not far from mortality's fair

FADEYI ABIMBOLA AKINTUNDE
400 LEVEL MBBS

AS A PAEDIATRICIAN

I looked up to watch.
them ushered across the floor
I was moved with emotions
for the tiniest, of the little ones.

I looked into those eyes
I see innocence raw
sanctity and sanity
devoid of any law.

I'm moved when they cry
the only language they understand
for want, pain or fear.
a "shriek" followed by tears

I want to handle your cares
I want to bear your pains
I want to see you smile
relieved from your pains
I want to watch you crawl
and gradually make it into a run
I want to you a chance
to taste the sweet and.....
..... maybe the bitter.....
..... of life.'

My gifted hands are masterinbg skills
driven by a desire to see you smile
not just profession, but compassion
giving to tomorrow a future...
.....a hope.

I want to watch you crawl
I want to watch you run
I want to give you a chance
to taste the sweet....
..... and the bitter.....
.....of life

ADEWUMI BABAJIDE
Group B2000

TEN WORDS HATED IN HOUSE JOB

BELLA, S

Dr. Sesan Bella was a House Officer at the University College Hospital as at the time of writing.

As my House job days draw to a close, I reminisce on the peculiar joys and sorrows that the entire experience has brought my way. What I find is that in the everyday work of house job, happiness comes when patient management is tending towards a certain direction, whereas certain other developments tend to put me firmly down in the dumps. Here is a short compilation of ten words (or phrases), which, whenever they fell from the lips of my senior colleagues on a ward round, always made my face fall and my heart weighed down with the prospect of impending stress.

ADMIT

Call me indolent if you wish – after all, isn't admitting a patient the first step towards beginning work of any kind or intensity? Is hating to admit not the same thing as hating to work at all? You'd probably have a point there, but the truth is we all love it when a patient is seen and can be dismissed as not needing medical attention or can be promptly referred to another, more appropriate unit for admission. Or, better still, can be given treatment he can receive in the peace, comfort and nosocomial infection-free environment of his own home.

ENSURE

This ubiquitous word falls easily from the boss's lips and usually translates into " Do the Absolutely Impossible or I get Absolutely Mad at you!!" It is a powerful, broad spectrum command that can mean anything from ensuring a computer error credits your patient's bank account with a million naira so that he pays for a CT scan, to ensuring haematology technologist on call develops a personality disorder that makes him agree to run a clotting profile for your patient on a Sunday.

REPEAT

It's very painful when you've just finished congratulating yourself on finally getting a patient to do a Full Blood Count, only to be asked to repeat it a day after differential WBC count is eventually released. The worst part is the patient knows he is repeating the exact same test he paid through the nose for, and it's up to you to convince him why, without going into a 2 hour lecture on Clinical Biochemistry.

RETRIEVE

If only they knew what it takes to retrieve histopathology reports, radiographs, WBC differentials, blood culture and several other investigation results. If a house officer is to be a good retriever (no offence intended, though a breed of dog goes by that name), he or she has got to be a sleek sycophant of laboratory staffs throughout the hospital. A good sense of direction is also needed to worm oneself into the furthest nooks of the labs where sunlight does not enter.

CONTINUE IV ANTIBIOTICS

Being essentially very kind at heart, I empathise with patients who have to keep buying expensive intravenous drugs. Besides, IV drugs cause the patient more pain, and thereby expose me to a greater risk of being attacked by the patient or his caring relations. (One other reason I prefer oral drugs is that I'm not the one that gives them.)

NPO

A patient being on nil per oris means the nurses physically disrupt your activities and remind you in a loud voice every four to five minutes if the patient's IV line is in tissue. It also means you will monitor electrolytes and urea more frequently than the good Lord in his wisdom intended them to be.

GROUP AND CROSSMATCH

On the one hand, grouping and cross matching throws you into the ethical dilemma of drawing blood from an anaemic patient whom you are far from guaranteeing blood for. On the other hand, it sets you up for a later not-likely-to-be pleasant encounter with the folks at the blood bank when you attempt to "ensure" blood for your patient.

MONITOR

Another sweeping instruction that could apply to a number of things, ranging from serial PCV's to vital signs or hourly urinary output. The only problem is your boss doesn't want to believe that there are differences, even if subtle ones, between you and electronic monitors, hence the disparity in accuracy, efficiency and tirelessness.

LATEST PCV

When my heart misses a beat when I'm asked a patient's latest PCV, it's either I can't quite remember if the "19" in my head is the patient's PCV, age or urea value; or I'm certain the "19" is the PCV, but it was done 6 days ago and the patient has upper G.I. bleeding.

FULL SEPSIS SCREENING

Usually a paediatrics affair. The key word here is "full". A full sepsis screen *fully* drains the patient's resources: it typically costs well over two thousand naira, which is often a third to one half of the monthly income of the patient's father. It takes its toll on the full complement of body fluids; no body compartment is spared. Rumour has it that aqueous humour and synovial fluid will soon begin to be subjected to analysis. Finally, by the time the results of the various aspects of the screen are *fully* out, the patient has either died or been discharged home.

Life, thank goodness, is not always so bleak. There are those precious few but joyful occasions when the Consultant strolls in, takes one look at the patient, and declares, "Commence feeds as tolerated, change to oral antibiotics, withhold investigations and discharge home to see in clinic in two weeks' time!"

COLLEGE OF MEDICINE, UNIVERSITY OF IBADAN
BACHELOR OF MEDICINE AND BACHELOR OF SURGERY DEGREE

LIST OF GRADUANDS

DOKITA Vol. 29 No. 1

OCTOBER 2002

NAME (SURNAME FIRST)

ABU, HYACINITH-JOHN OSHIOMHA
ABDUL, MUTIAT MOJIBOLA
ABDULMALIK, JIBRIL OMUYA
ABDULSALAM, TUNDE SHUAIB
ABE, OMOTOMILOLA ANNE-MARY
ABIAHU, JOSEPH AMAUZO
ABITOYE, OLUTOYIN OLUDARE
ABODUNDE, OLUBUNMI
ADAJI, AKUH
ADEDUN, ADEDAYO
ADEGBAYIBI, YETUNDE ADEJOKE
ADEGOKE, DANIEL ADEWALE
ADEJUMO, OLUROTIMI
ADELAJA, ABIOLA OLADAPO
ADELEKE, ADEGOKE OLAOLU
ADENEYE, OLUBUNMI ADETUTU
ADETUNJI, ADEBOLA OLUTOYIN
ADEWALE, ADEPOJU SOLOMON
ADEWUYI, YETUNDE OLUBUSAYO
ADEYEMO, OLAITAN TOLULOPE
ADIGUN, KEHINDE GANIYAT
ADIGUN, OLUSEYI ABOLARIN
ADIGWEME, IKECHUKWU NNAEMEKA
ADIO, OLUWAFEMI AJOKE
AFOLABI, AKINWALE IBRAHIM
AFOLABI-BROWN, OLAYINKA ORIYOMI *
AFOLAYIN, ODUNAYO OLUWASEUN
AGBEDEYI, GERTRUDE OGHENEPRIYE
AGORO, KAMALDEEN OYEBOLA
AJAKAIYE, OLUBUKOLA ADESOPE
AJALA, OLUBUKOLA
AJAYI, ADEBAYO OLUWATOYIN
AJAYI, SAMUEL OYEWOLE
AJIBADE, ADEDOTUN TEMITAYO
AJIBOYE, AYODEJI GAFAR *
AJOSE, SEMIU OLALEKAN
AJUFO, CHRISTOPHER ANIKWE
AJUMOBI, OLUFEMI OLAMIDE
AKHIDENOR, OSARGIE PATRICK
AKINDELE, AKINBAYO ANTHONY
AKINKUGBE, KIKELOMO ABOSEDE
AKINMURELE, OLUSHOLA OLADOTUN
AKINPELU, FOLAKE OLUFUNMILOLA
AKIODE, AKINTUNDE OLADIMEJI
AKUSU, ADERONKE VIVIAN
AKUTE, TOLULOPE TOKUNYORI
ALAKE, OLUWASEYI MOSUNMOLA
ALAWODE, TOLULOPE THERESA
ALLWELL-BROWN, ENEIMI
ALONGE, OLAKUNLE OLADUNJOYE
ALUKO, BOLANLE FEYISAYO
ANANI, ANTHONY JAMES OMOKHEONA
ANIMASAUN, OLALEKAN NURUDEEN
APAKAMA, OKWUCHI IHEOMA
ARANSIOLA, CLEMENT OLUKAYODE
ARCHIPONG, RICHARD BASSEY
ARIFALO, GREGORY OLAWALE
ASOJO, OLUYOMI ADEBOLA
AWORINDE, OLADIPO AKINLABI
AYENI, OLUWATOYIN AYODEJI
AZIZA, MARIAN AVIONAH OMOKINENE
BABALOLA, KEHINDE MOBOLANLE

BABATUNDE, MAYOWA
BABATUNDE, TAIWO OLABIMPE
BADRU, ADEWALE ISMAHIL
BAKARE, OLADIPO
BETIKU, ANTHONY OLAWALE
BETIKU, TEMIWOLUWA MONISOLA
BIAKOLO, NUVIE
BIDMUS, RASHEEDAT MOBOLAJI
BUSARI, KAFAYAT TEMITOPE
DANIA, SIMPA ENDURANCE
DANIEL, ABOYAWOH NJINGEH
DANIEL, ADEKUNLE
DANIEL, OLUGBENGA KAYODE
DIEJOMAOH, ESEOGHENE TEMITOPE
EBIE, UZEZI ISAIAH
EFFIONG, AMANA OKPOVO
EGBABGE, AUGUSTINE SUNDAY
EMEGWALU, PATIENCE NGOZI
EHIMIYEIN, EFEARUE TEMITOPE
EJIKUNLE, SAMUEL DAYO
EKANEM, SUNDAY OKON
EKONG, DONALD EFFIONG
EKRIKPO, UDEME EKPENYONG
ELEMILE, MOYOSORE TAIWO
ETSU-NDAGI, RAKIYA
EVESI, UFUOMA
EZEASHI, EJIME VALENTINE
ABOYA, IBUKUNOLUPO ODUNAYO
ADIPE, FUNKE FOLASHADE
AKUNLE, OLUBUNMI ADERIYIKE
ASIPE, MOJISOLA ADEJOKE
ASORO, OLANIKE OLUFUNMILAYO
HINTOLA, AKINTUNDE OLUSEGUN
RAHIM, OLUWAFEMI ADEKUNMI
RAIMOH, AMODU AKINLAWON
RAHEEM, GBADEBO HAKEEM
OWU, ADEKEMI OLATOKUNBO
TOKUN, OLUFEMI MICHAEL
HAKEEM OLAKUNLE
MOYAN, ARIYO AYOMIDE
DUOLA, FOLUSHO EMELDAH
IJAROTIMI, OLUWAMUYIWA KEHINDE
IKE, ROSEMARY IFEOMA
IKOMI, JOLOMI TOBOGBANMI
IKPEME, IWO KOFI
ISHOWO, RAHMAT BOLA
ITAKPE, SHOPEKHAI EMMANUEL**
KOLAWOLE, OLAIDE OLUTOYIN
KEHINDE, RAZAK OLALEKAN
KUPOLUYI, OLALEKAN ADEDOTUN
KUYE, BABAJIDE AYOADE
LASAKI, OLUWASEYI FOLARIN
LAWAL, ADEKUNLE OYEYEMI
LAWAL, FOLUKE MAGARET
LOHOR, MICAH DASKYES
MAKINDE, OLUSESAN AYODEJI
MAKINDE, OLADIMEJI
MBACHU, IKECHUKWU INNOCENT
MERENU, CHIKA ONYINYECHI
MAMBULA, CHRISTOPHER
MOHAMMED, SHAMSUDEEN
MOKONOGHO, JOSEPHINE BEMIGHO
NDEKWU, CHUKWUNONYELOUN
NKANOR, EMMANUEL YAKEDOHO
NKWONTA, CHINWENDU IFEYINWA
NSOEDO, ADAEZE CHINEYE
NWAOMU, SAMUEL UGOCHUKWU
NWIZU, TOBENNA IGWEONU
NWOKO, ODUNLAMI ANTHONY
NYA, GLORIA ATIM
OBADINA, OLUBUSOLA TANIMOWO
OBAMUYIDE, HENRY ADEBAYO
OBIANWA, IKENNA MICHAEL
OBIOHA, UGOCHUKWU GEORGE
ODAFE, SOLOMON FRANCIS
ODELOLA, OLAIDE ADEBOMI
ODOMEJA, EDWIN ENEJI
ONOJA, AMINA
ODUNEYE, OLUSEUN OLUWAKEMI
ODUYOYE, OLUKEMI ADEBOWALE
OGAN, SOALA DAEREFAKA
OGBU, UZOR CHIGOZIE

OGBECHE, GRACE ECHA
OGBUOJI, OSONDU RAPTURE SUNDAY
OGUNDEJI, OLAJUMOKE ABOSEDE
OGUNTOYINBO, BOSEDE ESTHER
OJEMUYIWA, OJEDARA FAYOFUNMI
OKE, OLATUNBOSUN AYOKUNLE
OKEKE, CHINEDU DANIEL
OKELANA, MOSOPE ABIODUN
OKEWOLE, ADENIRAN OLUBUNMI
OKONJI, CHINYEMIKE JAMES
OKONKWO NGOZI STELLA MARIS
OKUSANYA, ABOSEDE ADEKEJI
OLA, OLUFUNKE CAROLINE
OLAH, FRIDAY GEORGE ECHE CRISPIN
OLABODE, ADESOJI AYOBAMI
OLABODE, JOSEPH AYOBAMI
OLADELE, EDWARD ADEKOLA
OLAFISOYE, OLAWOLE OLADELE
OLAJIDE, ABAYOMI OLUSEYE
OLAJIDE, OLUWABUNMI
OLALEKAN, OLALEYE EMMANUEL
OLALEYE, OLADEJO AYINDE
OLALEYE, OLALEKAN
OLAWOYE, MOJISOLA OLUTAYO *
OLOPADE, IDRIS OLADELE
OLUBANIYI, BABAJIDE OLUSOLA
OLUKINNI, OLUMIDE OLUDARE
OLUGBODI, TEMITOPE OLUWATOSIN
OLUPITAN, ORITSEGBUGBEMI EGHOSA
OLU-TIMEHIN, OLUKEMI IFEOLU
OLUSUNMADE, OPEYEMI IDRIS
ONAOLAPO, OLAKUNLE JAMES
ONASANYA, AYOOLA FOWOWE
ONASANYA, OLUSEYI
ONIMODE, YETUNDE AJOKE
ONIWIDE, TUNDE RICHARD
ORAGBADE, ADEDUNNI WUMI
ORANYE, BENJAMIN CHIKWUKA
ORETADE, JAIYEOLA ADEOLA
OSEROHWOVO, ISRAEL ONAJITE
OSHIAME, OMOLE AFISHIETU
OSHIONEBO, WINIFRED OMOAREGHA
OSISIOGU, CHUKWUEMEKA UCHENNA *
OSUNKUNLE, TAIWO IBUKUNOLUWA
OSUNKUNLE, KEHINDE BUKOLA
OYETUNDE, OYEDAPO OLALEKAN
OYENUGA, OLUSEGUN ADEWOLE
OYENIRAN, OLUWASEUN OYEYEMI
PETERS, OLUKEMI
SAHEED, ADEOLA RUKIYAH
SALAKO, OYEWOLE AKINOLA
SALAWU, OLUDÓLAPO SHERIFAT
SHOBAYO, FRANCIS OLADAPO
SHOBODE, MUTALEEB AYODELE *
SOBOẆALE, YEWANDE FADEKE
SODE, ABIMBOLA OLAJUMOKE
SOLADEMI, AKINNIYI OLUSEYI
SOTONADE, OLUWAFEYISETAN
SOYEMI, OLUFEMI MICHAEL
TAIWO, AOFOḶAJUWONLO ADEJUMOBI
TAIYE-AKINSUN, AKINWALE AKINTOMIDE
TAWOSE, ADETUNJI MAPADERUN
TIJANI, ARAMIDE MIKHAIL
TIJANI, HAKEEM GBOLAHAN
UDOH, EMEM SYLVANUS
UDUCHUKWU, CHIKAEZE OKECHUKWU
UGOLEE, JERRY CHINEDU
UGWUEKE, THADDEUS NKEMAKOLAM
UKOYEN, ENO EKAETE
UMOH, IMO OFONIME
UMAR, FATIMA ONOZARE
UMORU, BENEDICT
UTUTU, CHIDINAM IFECHI
UZODIKE, CHRISTOPHER UZOMA
UZOH, CHIZOBA CHRISTOPHER
YEKINNI, SAKIRU ABIODUN
YUSUF, RASAQ ADEKUNLE

KEYS

* Former Board members

**Editor-in-Chief

Don't Toy With Your Life

Life Is Precious

At Goldlink, our operations
are designed to protect
the sanctity of life
in all ramification

GICO

GOLDLINK INSURANCE COMPANY LIMITED

15TH Floor, Western House Broad Street, Lagos
Tel: 01-2601070-4, 2645449-51, Fax: 01-2637339

...innovation driven by experience!

NIA
A MEMBER OF THE NIGERIAN INSURERS ASSOCIATION