UNIVERSITY OF PORT HARCOURT

EAT RIGHT, LIVE RIGHT THAT YOUR LIVER WILL BE RIGHT

An Inaugural Lecture

BY

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DEDICATION

This work is dedicated to the Glory of God for all those sufferers of liver disease who have not been fortunate enough to overcome their disease.

ACKNOWLEDGEMENTS

Mr Vice Chancellor Sir, I want to start by giving thanks to the Almighty God who has kept us alive till date to witness this event, and Who guided me into studying Medicine when I was set to go to the Civil Aviation training School in Zaria to train as a Pilot at the end of the Nigeria hostilities. I never thought of studying medicine on completion of High school in 1970, but toyed with the idea of becoming a Pilot or a Geologist. My Principal at the Saint Augustine's Grammar School, Nkwerre impressed us when he made Geography very interesting and introduced terminologies which hitherto were unheard of, such as Geomorphology and Geomorphogeny.

My late father, who instilled discipline in all who passed through him, and as President General of my town union, and late Mayor of Port Harcourt was a father to all and sundry. He'raided' virtually every house in Nkwerre, where the young men gathered to gamble, seized their cards and overturned their tables- a feat no one will ever try these days. He also taught us honesty in service and truthfulness in all we did. To my late mum Esther, and late eldest brother Dr Victor 'de Empire' Ihekwaba (born on British Empire day hence his name) both of whom saw us through those teething years when our father died soon after the Nigeria civil war. To late uncle Sir E.C.O. Uzoukwu who offered words of wisdom in those days.

I will not fail to mention my teachers at the University of Lagos, who simplified the various subjects. These include Dr. J.A. Ekundayo (Biology), Prof. Akinsanya, the son of the late famous Odemo of Ishara (Chemistry), Prof. Felix Dosekun (Physiology), Prof. Alex Eyimofo Boyo (Chemical Pathology),

Dr. V. P. N Mordi (Anatomical Pathology), Prof. Deji Femi-Pearse, Dr. O. O. Elegbeleye, and Prof. Thomas Johnson (Internal Medicine) and Prof. Amechi Anumonye and Dr. Abua Nwaefuna who taught us and demonstrated to our utter consternation the science and art of Hypnosis and Abreaction.

I wish to also acknowledge the contribution of Prof. Gilbert Onuaguluchi, who was a Professor of Clinical Pharmacology and Therapeutics at the University College Hospital, Ibadan, later Administrative Head of Enugu Campus of University of Nigeria, and Vice Chancellor University of Jos. He advised me to abhor desk-top publishing-(the art of publishing scientific papers in the office without performing any experiments), for which he was hated by the young Lecturers who wanted to become professors before they were due. He taught me diligence, intuition and keeness in Experimental Research, as well as how to present papers at conferences.

I must not fail to mention two Physicians who played a major role in my career as Lecturer / Physician. These are Dr Sylvester Oguguo Ukabam, who encouraged me to take to Gastroenterology, and put me through the sub-specialty some twenty five years ago at the University of Nigeria Teaching Hospital, Enugu; and Prof. Chris Anah, whom I first worked under in 1978 at the UBTH, Benin. He was Provost, College of Health Sciences, University of Port Harcourt when the Medical School was on the verge of being closed down because of inadequacy of teachers. He came to fetch me from Enugu, having secured the kind approval of the then Vice Chancellor Prof. Sylvester Cookey, to join him and the then Dr. Osaretin Odia (now Prof. Odia).

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My thanks go to the Vice Chancellor, by whose special permission granted me this opportunity to deliver this lecture, and the financial support to put this lecture together and in print; and to the University press for an excellent work.
I also acknowledge the assistance of Residents in my department who contributed to the generation of some of the data especially Dr. Madubuike who worked closely with me since 2005 before moving over to FMC, Yenogoa; and Dr. Mrs. Kike Oyeleke who is now in Lagos.

I must not fail to acknowledge the support I have had from my siblings; notably amongst who are: Prof. (Mrs.) Mabel Uk. Akpuaka, Prof. (Mrs.) Pat. Nwamuo; Sir Arc Emenike Ihekwaba, Admiral Chinaka Ihekwaba, Prof (Mrs.) Ola Nnadi; Dr. Zerry Ihekwaba, and the Spiritual assistance of Pastor Charles Ezenwa Ihekwaba of Living Faith Church (aka Winners Chapel).

Finally, to my family, by the special grace of God I have been married to Ijeoma, the love of my life, my help mate and the mother of our children who has been with me these thirty years in thick and thin, who provided the enabling environment for me since 1986 when I ventured into

experimental and academic medicine. To those lovely children of ours-Chinedu, Ezinne, Nkechi, Kelechi, Afoma and Sochi; our daughter in-law Bunmi, son in-law Ikenna, and grandchildren Geovanni and Tara, to God be all the Glory, Amen.

Mr. Vice Chancellor Sir, Deputy Vice Chancellors, other Principal Officers of the University, Provost College of Health Sciences, Dean, School of Postgraduate Studies, Deans of Faculties, Directors of Institutes, Heads of departments, Distinguished Scholars, members of the academia, Ladies and Gentlemen.

INTRODUCTION

It is a thing of joy and pleasure for me to deliver my inaugural lecture today the 27th day of October 2011. For me to deliver this lecture, I know that I shall be faced with the task of translating/ breaking down some medical terminologies into simple everyday terms to enable a good number of members of this audience to understand the subject matter, while at the same time not bore my medical and paramedical colleagues.
I have chosen to share my thoughts and experience on a subject matter that I have had the singular opportunity of being associated with over time.

Interest and decision to specialize in the medical specialty
Some 30 years ago, while undergoing my junior residency in Internal Medicine at the University of Nigeria Teaching Hospital, I came into contact with a man who knew nothing other than academics and Research, whom I regarded as a Teacher of Teachers, in the person of Gilbert Onuaguluchi, a Professor of Clinical Pharmacology and Therapeutics at the University of Ibadan in 1965,Administrative Head of Enugu campus of the University of Nigeria in 1970, and later Vice Chancellor of University of Jos. I had the singular opportunity of working in his laboratory on *Polyadoaumbellata* (ERIN), a local herbal preparation used by the Yorubas to de-worm Children. The work was a subject of Research for my Master of Science degree in Clinical Pharmacology (Ihekwaba A. E, 1986), while undergoing training as a Physician in the gastroenterology unit of Sylvester Ukabam, who convinced me to go into the sub-specialty of Gastroenterology and Hepatology.

What is Gastroenterology and Hepatology:
As the name implies it is the science of the study of Alimentary system which covers the mouth, oesophagus, stomach, intestines (small and large), rectum and anus, and the appendages arising from the gut which are the Liver, gall bladder, spleen and pancreas. With time a sub-specialty was carved out of it, called Hepatology which deals with the liver alone. Thus, it is now Gastroenterology and Hepatology.

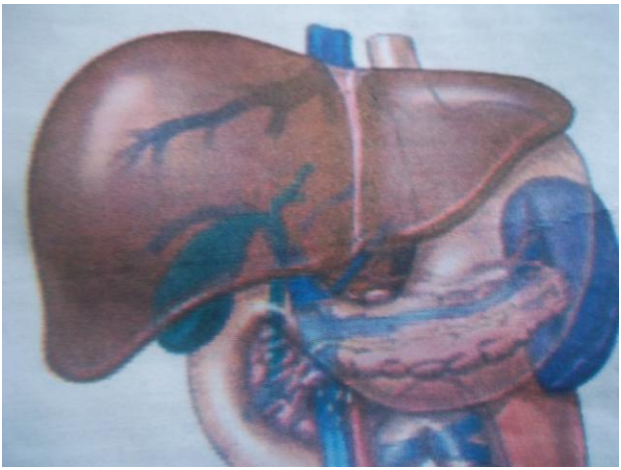


Fig. 1 Parts of the upper Gastrointestinal system excluding the oesophagus.

The beginning of Gastroenterology.
One of the commands God at creation gave Adam (Genesis 2: 16) had to do with **Gastroenterology**.

*"Of every tree of the garden**, you may freely eat**, but the tree of knowledge of good and evil, **you shall not eat**. For in the day that you eat of it **you shall surely die**".*

It means that there are foods we eat and will live, and there are those which if we eat we will die. Every food that enters the mouth gets to the stomach and will then pass through the liver before getting into the blood stream.

I do not know if death as prescribed by God was through Liver disease, but from our experience in medicine we know that liver disease is not uncommon, for approximately one out of every five patients admitted into the medical wards of the University of Port Harcourt Teaching Hospital over a four year period had liver disease (Madubuike O. C, Oyeleke G. K, Ihekwaba A. E 2007).

I have therefore chosen the topic **EAT RIGHT, LIVE RIGHT THAT YOUR LIVER WILL BE RIGHT**.

I shall deal with this along the following headings.

I. The liver- its structure.
II. The Right Liver
III. Eating Right.
IV. A Liver that is not Right: (a) from not eating right.
(b) from not Living Right
V. The way forward?
VI. Recommendations and Conclusions.

I. THE LIVER

Structure.

The liver is a football-sized organ sitting in the right upper quadrant of the abdomen directly under the right rib cage. It is

the largest organ in the body. Though single, it is subject to attack by agents that can render it weak and diseased; but it has the capability of functioning normally even when seven out of eight parts of it are removed. It is located strategically between the outside world (inner lining of the gut-portal circulation) and the interior of the body (blood stream- systemic circulation). It is this location of it that explains one of its major functions of protecting the body from attack by microorganisms.

II. The Right Liver.

When the liver is right, it will function optimally such that it will fulfill its obligations to the body. It is regarded as the power- house of the body, and has the following functions:

1. The liver cell metabolizes nutrients preparing them for storage (**Glycogen synthesis**) within itself, and for distribution to the rest of the body. By this, it is capable of pushing into the blood stream 100 to 200 grams of glucose per day, which is sufficient to prevent hypoglycaemia in the absence of food.
2. It **Catabolizes** (breaks down) and **Detoxifies** (renders inactive) a wide range of noxious (dangerous) substances including therapeutic drugs and potential poisons.
3. It is a major biosynthetic organ that provides the body with proteins-**Albumin**- thereby preventing the legs and abdomen from being swollen as you find in Kwashiorkor;

   Clotting factors- making the blood to clot following the slightest injury; **Lipoproteins**- which carry many of the hormones; and **Plasma proteins components** involved in fighting infections. Thus, damage to the Liver cell may result in the diminished capability of the body to use nutrients,

synthesize the needed proteins, inability to detoxify noxious substances and failure to defend the body from attacking microorganisms.

III. EATING RIGHT

There are six classes of food, namely Carbohydrates, Proteins, Fats, Minerals, Vitamins and Water. Thus, eating right is determined by food availability, affordability and knowledge of which component(s) is/are right to eat.

Food intake and the major problems arising from lack or abundance of supply of food have been with man over the ages, but it varies with technological development.
The various stages of technological development (Truswell, AS 1995) were:

1: **The hunter-gatherers**: at this stage, man collected a wide range of vegetable foods; ate meat and fish. He did not have access to salt, alcohol, milk (other than mother's milk), and there was little cereal or sugar (honey).
People at that stage remained lean, malnutrition was unlikely, and coronary artery disease (heart attack) and high blood pressure were unknown then.

2. **The Pastoralists**- man at that time followed his grazing animals where ever adequate pasture was available. Such people had a diet high in animal foods and milk. Man at that time was tall, but passage of lose watery stool following eating of certain sugary foods in adults (intestinal lactase deficiency) was the order of the day.

3. The **Peasant agriculturists** tended to rely on one crop which yielded the best result. There were crop diseases

and crop toxin resulting in drought due to seasonal shortages.

The risk of malnutrition was high due to milling and refining of cereals. At that stage, unreliable rainfall in some areas caused famine with malnutrition and diseases due to deficiency of certain vitamins especially the B group-Beriberi (swollen legs and abdomen, with skin changes) and Pellagra (skin changes, diarrhoea, memory and intellectual derangement in young and middle-aged persons). Grains stored during harvest were afflicted by fungal toxins (mycotoxins).

4. **The urban slum and peri-urban shanty**.

There were the rapidly growing cities with the poor masses in and around those cities. That was the situation in London, New York and other present-day big cities of the Western world in the 19th century. There was loss of food traditions. No home gardens, mothers often had to work, there was poor food hygiene and food was expensive. Children became very vulnerable (were not breast-fed) developing gastroenteritis and Rickets (bow legs, K-legs), the adults resorted to excessive drinking of alcohol, and became obese and develop high blood pressure.

5. **Affluent societies**: Favourite foods are available all the year round, high fat diet which is processed, convenient to prepare and eaten in form of take-away foods (Genesis, Promise, King Fisher, Mr Biggs of our time).

Many health foods are marketed, and there is increased intake of vitamins. Malnutrition is unknown except in hospitalized patients and the elderly, thus Obesity though unfashionable became increasingly common and difficult to avoid.

Such was the evolution of life and diets. What do we have today? It is a combination of stages 3, 4, and 5 depending on where one is.

A situation akin to Stage 5,is what we have presently, in which people are predisposed to being overweight from abnormal digestion.

Thus, **EATING RIGHT** involves eating the right **type** of food and the right **quantity**, and a standard diet must contain carbohydrate, fat, protein, minerals, water and vitamins.

The **quantity** of food eaten is determined by **hunger**, while the **type** of food eaten is determined by **appetite.**

Body weight and composition are maintained over time by a balance between intake of energy and expenditure, any excess energy is stored as fat, and body weight increases giving rise to **overweight**.

A major part of the energy for body functions is provided by **carbohydrates**. Each gram of carbohydrate metabolized by the body produces 4 Kilo calories, whilst one gram of fat produces 9 Kilo calories of energy. The major sources of carbohydrates are found in sugars (fruits, milk, sucrose, starches-wheat, rice, root foods (such as cassava, yams, potatoes), and legumes.

Protein

The distribution of calorie/energy among carbohydrates, fat and protein is determined by physiologic factors, and partly by taste and economic capability. A daily protein intake of 1gm

per kilogram body weight is desirable to supply the eight nutritionally essential amino acids, and other amino acids. The protein source is important as not all proteins supply the essential amino acids. Animal proteins (meat, fish, dairy products and eggs) contain these amino acids and are thus called Grade 1 proteins, unlike most plant proteins which are grade II, because they supply different proportions of amino acids and some lack one or more of the essential amino acids.

Each gram of protein gives 4kcal of energy hence in communities where protein consumption is in excess of carbohydrates as in Western countries there is the tendency for the excess energy to be converted into fat. This is worsened where the protein source contains a large amount of fat as in the following: torso, offals-(intestines, stomach), skin – (whether of cow, goat or chicken), kidney, liver, egg, and pork.

Fat. This is the sweetest of the food types, yet the greatest culprit for weight problem. Sources of fat vary in different societies depending on availability and affordability. Whereas in industrialized countries pork and goat meat may be the predominant sources, but in resource poor countries the commonest sources are the intestines, the skin of cows, brain, with their attendant complication of cholesterol deposition in the arteries.

There are other foods we eat without realizing what harm we do to the body due to their fat content.

The foods and their percentage fat contents are as follows: table i.

Table i. Fat content of certain foods.

s/n	Food type	Fat content(%)
1	Walnut	64.4
2	Pea nuts	44.2
3	Chocolate	52.9
4	Cashew nuts	47.2
5	Butter	81

Vitamins

There are 13 vitamins, essential or probably essential to human nutrition. These are: Vitamin A, Thiamine (B1), Riboflavin (B2), Niacin, Ascorbic acid (Vitamin C); Vitamin D, Vitamin E; Vitamin K; Folic acid; Vitamin B 12; Pyridoxine, Pantothenic

Minerals

These are necessary for maintenance of health. The common ones are Sodium, Potassium, Calcium, Chloride, Phosphorus and Magnesium. However, there are others called trace elements that are found in tissues in minute amounts, which are believed to be essential for life, at least in experimental animals. These include Arsenic, Chromium, Cobalt, Copper, Fluorine, Iodine, Iron, Manganese, Molybdenum, Nickel, Selenium, Silicon, Vanadium and Zinc. Their deficiencies are known to cause diseases in man.

Table ii. Trace Elements and Deficiency Diseases

Trace element	Deficiency disease
Iron	Anaemia
Cobalt	Is part of vitamin B12- anemia
Iodine	Thyroid disorders
Zinc	Skin ulcers, hypogonadal dwarf, reduced immune responses
Copper	Anaemia and changes in ossification
Chromium	Insulin resistance
Fluorine	Increases incidence of dental caries.
Selenium	Cardiomyopathy
Lithium	Abortion

IV. THE LIVER THAT IS NOT RIGHT

The Liver is subject to attack by a wide array of substances that may cause disease either in the acute state or result in chronic liver diseases.

Several aetiological agents are known to attack the liver causing various liver diseases. Generally, they are shown in **table iii**; but not all are commonly encountered in every day clinical practice.

Table iii. Aetiological Agents in Liver Disease.

S/n	Aetiological Agent	Disease caused	Remarks
1	Bacteria	Pyogenic abscess	
2	Viruses- A,B*,C,D,E,F,G	Acute & chronic hepatitis, Cirrhosis, HCC	B,C, & D causes chronic disease
3	Protozoa-E. histolytica*	Liver abscess	
4	Alcohol	Fatty liver ,Hepatitis- acute and chronic, Cirrhosis, HCC	
5	Excess fat in Obesity*	Fatty liver, Steatohepatitis, Cirrhosis. HCC	

6	**Paracetamol.***	**Acute liver failure**	
7	**Excess copper**	**Wilson disease**	
8	**Excess iron**	**Haemochromatosis**	
9	**Isoniazid, Rifampicin, Aldomet**	**Hepatitis**	
10	**Comfrey, Senna,Vit A.Mistoletoe, Anabolic steroids**	**Elevated transaminases**	

At the University of Port Harcourt Teaching Hospital, the major liver diseases we encountered include acute viral hepatitis, amoebic liver disease, chronic liver disease due to hepatitis B virus and alcoholic liver disease; fulminant hepatic failure and liver malignancy particularly primary liver cell carcinoma associated with alcohol or the hepatitis group of viruses.

Liver disease can either be acute or chronic. The commonest acute liver diseases are the Infectious hepatitis and amoebic liver disease. These usually respond to therapy and the patients recover without any complications, but chronic liver disease is problematic.

Thus, these liver diseases can be as a result of wrong eating habits or wrong lifestyle.

(a). A wrong liver from not eating right.

This can be as a result of:

1. Eating more than the normal quantity.
2. Eating in the wrong environment.
3. Eating the wrong quality.

1. Eating more than the normal quantity.

Gluttonous eating: For one to maintain an ideal weight there must be a balance between energy intake and expenditure. Once intake exceeds expenditure, the excess is stored in body stores- in the muscle (as glycogen) and in the fat depots mainly the subcutaneous tissues (as adipose). For each 9.3 calories of excess energy entering the body, one gram of Fat is stored in the fat depots.

Large quantities of fat are stored in two major tissues/organs in the body: adipose tissue (skin- the largest fatty tissue) and the Liver.

Other areas in the body containing fats include brain, intestines kidneys, muscle. There are several factors which determine the balance between energy intake and expenditure. These include heredity, chemical messengers in the body (Hormones), socio-economic and environmental.

Excess energy stored as fat results in **OBESITY**. The magnitude of obesity was estimated by the World Health Organization (WHO) in 2008, that 1.5 billion people above the age 20 years were overweight. Of these, 200 million men and 300 million women are obese. Body mass index (BMI) is a simple index of weight- for-height that is used to classify overweight and obesity. It is defined as a person's weight in kilogram divided by the square of his height in meters (kg/m^2). Overweight is defined as a BMI equal to or greater than 25, and obesity as a BMI equal to or greater than 30.

The fundamental cause of obesity and overweight is an energy imbalance between calories consumed (increased intake of energy-dense foods that are high in fat, salt and sugars but low

in vitamins, minerals and other micronutrients), and calories expended (a decrease in physical activity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization).

What are the health consequences of obesity? An elevated BMI is a major risk factor for Diabetes mellitus, Hypertension, Arthritis (especially of the knees and waist), Gall bladder stones, Atherosclerosis, Stroke, Sudden death and **lately Fatty liver.**

Fatty liver is a situation whereby the liver contains more than the normal amount of fat thereby resulting in abnormal liver function. A normal liver has a tawny brown colour unlike a fatty liver that is yellowish due to abnormal fat accumulation

Fatty liver can also be associated with excess alcohol intake in which case it is called alcoholic fatty liver, but the fatty liver that is associated with obesity is called Non-Alcoholic Fatty Liver Disease (NAFLD).

Both conditions cannot be differentiated except for the history of excess alcohol consumption in the former.

Fatty liver is three to five times more common in males than females, and it has been shown that 30% of obese people have fatty liver, whilst 80 percent of morbidly obese people(BMI.>35 Kg/m^2) have fatty liver.(Anderson T and Gluud C 1984; Luyckx FH, Desaive C, Thiry A et.al,1998).

The danger of fatty liver is that the excess fat in the liver may cause steatohepatitis in which fatty acids affect mitochondrial

oxidation and thereby generating free radicals resulting in accumulation of fats within the cell.

The free radicals cause release of mediators –tumour necrosis factor, cytokines and interleukins resulting in cell death. Ten percent of individuals with fatty liver will develop steatohepatitis, with 5 to 25% of the later, progressing to fibrosis. The natural course of fatty liver obeys the rule of tens: 10% will progress to steatohepatitis, and 10% of the steatohepatitis progressing to liver cirrhosis. Thus, fatty liver is a frequent and known cause of Chronic Liver Disease and it has been demonstrated that 17% of cases of liver cirrhosis are due to excess body weight. The other danger of obesity is that in patients with cirrhosis there is an increased risk of decompensation in those who are obese than in those with normal body weight. (Garcia-Tsao G et al 2011) Fortunately, one month of dieting to reduce weight is effective in reducing the fat stored in the liver.

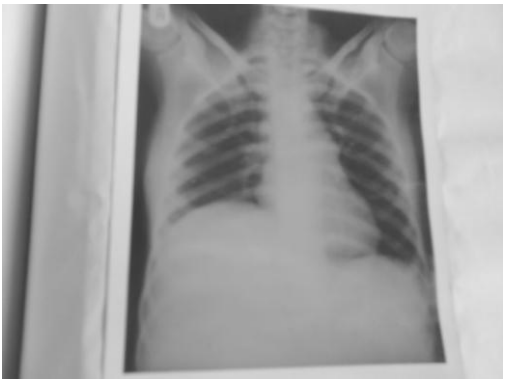
Doctors have had to rely on ultrasonography for diagnosis, or the diagnosis is suggested following ultrasound examination of the abdomen as part of a routine general physical examination**.**

a. **Eating the right quality of food but in an unsanitary environment.**

AMOEBIC LIVERABSCESS (ALA). One common disease acquired by eating in unsanitary conditions is **intestinal amoebiasis (Amoebic dysentery)**. It is due to the protozoon *Entamoeba histolytica*, which resides in the large bowel of man without causing any illness in the cystic form, but is shed intermittently and constantly in the stool to contaminate food and water.

Intestinal amoebiasis presents with passage of loose stool containing blood and mucus (catarrh-like material). A simple passage of blood and mucus in the stool which will respond to metronidazole or tetracycline may later become devastating.
It causes ulceration of the inner lining of the large bowel; which if untreated may result in perforation of the bowel.
Although amoebic colitis may resolve without treatment, but it can become recurrent, and when the organism enters the blood stream it lodges in the liver to cause amoebic Liver abscess (ALA). The infected liver becomes big and painful, and more than half or three-quarters of the liver can become a mass of pus which resembles anchovy sauce or 'egusi soup'.

ALA is a common acute liver disease in the tropical environment whose incidence depends on the incidence and relative control of the primary intestinal infection. It is one of the common causes of an enlarged, tender liver in the tropical environment. In the past, the impression was that visitors to the Tropics more often have ALA than natives. That this was due to partial acquired immunity from constant re-infection from childhood of the natives, hence the natives had comparatively milder symptoms and fewer hepatic complications. Because it can mimic other diseases, certain criteria were suggested as being diagnostic of amoebic liver abscess. These were: a tender hepatomegaly, serological evidence of amoebiasis, ultrasonographic demonstration of an abscess cavity, response to specific anti-amoebic therapy, elevated right hemi-diaphragm, and demonstration of pus in the liver (Nigam et al 1985).



Elevated right hemi-diaphragm due to ALA.

Using these criteria, we evaluated a series of sixty patients at the University of Nigeria Teaching hospital, Enugu, and were able to show that some of the teachings about amoebic liver abscess written in Standard textbooks of medicine were not applicable to our environment. For instance, jaundice was said to be rare or uncommon in amoebic liver abscess. (Adi FC 1965;Salako 1967; Cain GD et al 1968), and that it is a feature of pyogenic liver abscess rather than of amoebic aetiology. But we were able to show that jaundice was a prominent presenting feature, occurring in 28.3 percent of our series (**Ihekwaba** 1988**; Ihekwaba AE,** Mgbor SO, Ukabam SO 1993))

Table iv: Major clinical features in 60 cases of liver abscess.

s/n	Physical sign	No. (%)
1	Pyrexia	52 (91.7)
2	Tender hepatomegaly	44 (73.3)
3	Jaundice	17 (28.3)
4	Ascites	14(23.3)
5	Mass in the liver	10 (16.6)
6	Pleural effusion	8 (13.3)

Another finding from our study was the presence of leukocytosis in 58.3% of our series of patients. Previously, leucocytosis was used as a distinguishing parameter between pyogenic and ALA.

Lately, we at UPTH were able to show that ALA is as common in south-south as in the south-east, for we found that ALA was responsible for more than one-third of all liver diseases seen over a four year period. (Madubuike O. C, Oyeleke G. K**, Ihekwaba A. E** 2007).

Because ALA classically presents with tender hepatomegaly, and that jaundice was not a common feature, misdiagnoses by non-gastroenterogists have been recorded. While working as a young Physician we had several referrals from colleagues, of patients with GI disorders which turned out to be ALA. The initial diagnoses were hepatitis, tuberculosis or pyrexia of unknown origin, before arriving at the definitive diagnoses of ALA. (**Ihekwaba AE**, Ukabam SO 1991).

Table v. Unusual presentations of 7 cases of ALA.

s/n	Age (yrs)	Sex	Main features	Admission diagnosis
1	50	M	Fever, jaundice	Hepatitis
2	22	M	Fever, weight loss, 6 weeks	PUO
3	60	F	Fever, drowsiness	Coma
4	32	M	Fever, jaundice	Hepatitis
5	29	M	Abdominal pain & swelling, drowsy, jaundice, ascites	Hepatic pre-coma
6	37	M	Jaundice, splenomegaly	Jaundice ?cause
7	47	F	Fever -4 weeks	PUO

It is of note that one laboratory test suggestive of ALA is an elevated alkaline phosphatase. This is also seen in patients with intra-hepatic mass lesions such as Liver cancer especially

at the initial stages of the abscess when the liquefaction of the liver tissue is yet to occur. Ultrasonography may not be able to differentiate between these two conditions. However, to the gastroenterologist and Sonologist, typical ultrasonographic characteristics are discernible in majority of cases.

Standard textbooks of Medicine, and experience of earlier workers have it that amoebic liver abscess typically presents with a solitary cavity on the right lobe near the dome of the diaphragm, and on the inferior surface near the hepatic flexure of the colon, and that the presence of multiple abscess cavities suggests a pyogenic cause (Knight R 1987)).

But while at UNTH, we did show that multiple abscess cavities were not only common in amoebic liver abscess occurring in 58% of patients (**Ihekwaba AE**, Mgbor SO, Ukabam SO 1993), but that its occurrence and the location of the abscess in the hilum of the liver were responsible for the development of jaundice (**Ihekwaba AE**, Ukabam SO, Mgbor SO 1995).
Amoebic liver abscess is amenable to medical treatment, but there is the danger that it can rupture into the chest, general abdominal cavity, into the blood stream to reach the brain. Full recovery is the rule in majority of cases, but its prognosis may be grave in those patients who are jaundiced, who have a higher morbidity than non-jaundiced cases. This finding was first reported by Lamont and Pooler in 1958, and then corroborated by Salako at UCH, Ibadan. We at UNTH, Enugu confirmed this finding and went ahead to determine why such patients developed jaundice.(**Ihekwaba AE** 1988).

b. Infectious Hepatitis.
Very often, eating the right food prepared by infected food handlers in unsanitary environments who are carriers of the

organism that causes Infectious hepatitis may be a source disease of the liver. Infectious hepatitis is the commonest liver disease world-wide, affecting all age groups but commoner in childhood especially in developing countries where most children are exposed to the virus early in life. Such children have life-long immunity, hence the antibody is detected in the blood of 90% of residents; unlike in developed countries where with good sanitation there is low population immunity in young adults with the antibodies detected in 30 to 40 percent of adults. Those at increased risk are children in day care centres, health care workers and lately homosexuals.

In most adult infections, symptoms are mild to moderate and are non-specific, such as malaise, fatigue, anorexia, nausea, vomiting and a low grade fever, resembling malaria or may look like early stages of amoebic abscess (**Ihekwaba** A. E, Ukabam S. O & Mgbor S. O 1995), hence the name amoebic hepatitis. During this period, the virus is shed in the stool and this may last up to two weeks before the individual becomes jaundiced. Right upper quadrant abdominal discomfort is common, but severe abdominal pain is not a feature. In 90 to 95% of acute cases, recovery is the rule without any specific treatment, but in less than 5% of cases the illness may run a fulminant course resulting in death within a few days from raised intracranial pressure associated with cerebral oedema. At the University of Port Harcourt Teaching hospital, we managed 42 patients with Acute Viral hepatitis over a twelve month period, eight of whom presented in grade III (2 cases) to IV hepatic encephalopathy (six cases), using intravenous mannitol. All but one regained consciousness within seven days, and thereafter had normal liver function tests. **(Ihekwaba AE 1992)**.

3. **Eating a poor quality food**.

It is a known fact that the quantity of food we eat is determined by hunger, whilst the type/quality is determined by our appetite. Appetite is usually controlled by the resources available thus; those who can afford luxury foods tend to eat all manner of food, why the poor will purchase the cheapest food.

Carbohydrates, fats and proteins are available in most of our daily foods though in variable quantities. Good protein sources are usually not within reach of the poor except for legumes such as beans. Beans, the cheapest plant source of protein may become stale and mouldy from the effect of a fungus *Aspergillusflavus.* This fungus has widespread distribution and contaminates groundnuts, beans and other stored grains. Its attack on stored grain produces aflatoxins, the most toxic of which, is aflatoxin B1.

Administration of as little as 15ug per kilogram daily of this toxin will produce cancer in rats. Other foods which have been shown to be contaminated by aflatoxins include smoked fish and virtually all grains which have a tendency to become mouldy.

Thus, consumption of mouldy grain or mouldy smoked fish is likely to predispose one to aflatoxin accumulation in the body
Aflatoxins are one of the agents which interact with other aetiological agents to cause Liver cancer.

LIVING RIGHT

Living involves not only eating, but also one's life style. There are life-styles that are likely to affect the liver. These are:

i. Alcohol consumption,

ii. Sexual relationships.

Alcohol Consumption.

Alcohol intake has been with mankind since the Biblical times, and had resulted in disastrous consequences. It uncovered Noah's nakedness (Gen.9:21).

*'**Then he drank of the wine and was drunk, and became uncovered in his tent'.***

Two of the enemies of the children of Israel in Biblical times were the descendants of Lot: the Moabites and the Ammonites as a result of the incestuous relationship between Lot and his two daughters after an alcoholic intoxication of the Lot.(Genesis 19:31-38)

……..'***Come, let us make our father drink wine, and will lie with him, that we may preserve the lineage of our father'…….***

It brings woes, causes contentions and inflicts injuries. *(Prov. 23:29-30).*

*'**Who has woe? Who has sorrow? Who has contentions? Who has complaints? Who has wounds without cause? Who has redness of eyes? Those who linger long at the wine. Those who go in search of mixed wine'**.*

Kings were warned about it, because it can cause them to deliver unrighteous judgments against the less privileged. (Proverbs 31:4-5).

'It is not for kings to drink wine, nor for princes to intoxicating drink; lest they drink and forget the law, and pervert the justice of all the afflicted'.

It is a mocker and will lead the unwise astray.

'***Wine is a mocker. Strong drink is a brawler, and whoever is led astray by it is not wise'. (Proverbs 20:1).***

It is not all woes with it but it has some good qualities as Uncle Paul did practice Gastroenterology when he recommended alcohol to his spiritual son Timothy.
'No longer drink only water, but use a little wine for your stomach's sake and your frequent infirmities' *1 Tim. 5: 23.*

I am aware that Cardiologists do recommend a little wine for the heart in line with the Biblical recommendation by the Teacher for those with a bitter heart:
'Give strong drink to him who is perishing, and wine to those who are bitter of heart. Let him drink and forget his poverty and remember his misery no more'. (Prov.31:6-7).

It has been estimated that alcohol abuse in the USA cost the government over 185 billion dollars yearly, and over 2 million people have some form of alcohol liver disease. Alcohol is readily available in every nook and corner in our villages and cities. Is obtained from natural sources, and easily produced by both the local populace (gin brewing), and Breweries in Port Harcourt, Calabar, Aba, Umuahia, Benin City, Enugu, Ibadan, Kano, Lagos.

It comes in various forms and concentrations, and is given different names by different cultures according to type- the local gin is called **Kai kai**, **Ekpeteshi**, **Ogogoro**, **Ijaw water** and **Akamiri**, while the Ibos call the Palm wine **Ngwo** from raffia palm and **Nkwuenu** from the palm tree. A special brew in Northern Nigeria is called **Burukutu.**

It is consumed by the old and young, educated and uneducated, in palaces, motor parks, and local huts called 'bush bars' or 'joints', Officers' mess, University Staff clubs, be it in the mornings and at nights. Most times some delicacies go with it

either as ‘pepper soup’ of goat meat, fish (either fresh or dried), goat head or cow tail, depending on the appetite of the consumer.

No matter its name, alcohol remains the commonest liver toxin, and is almost extensively metabolized in the liver. The danger of alcohol to the liver is that it induces the activity of an enzyme in the liver called **Mixed-function oxidases**, which metabolizes itself and other toxins. By enhancing its own metabolism, the tendency is for one to consume more of it, thus a vicious cycle occurs whereby as you drink it, you crave for more.

Unfortunately it causes a spectrum of liver diseases, ranging from Fatty liver to alcoholic hepatitis, liver cirrhosis and finally liver cancer.

Ten to thirty percent of people who consume alcohol will develop a fatty liver, and they need to consume 30grams of alcohol (in the male) and 20grams (in the female) for a fatty liver to develop. This translates to one and half bottles of lager beer; a glass of wine for males, and two glasses of beer or a bottle of half stout, or two shots of full-proof whiskey for females.

Alcohol undergoes oxidation by alcohol dehydrogenase to the acetaldehyde; the latter is converted to the acetate by acetaldehyde dehydrogenase. There is genetic pleomorphism of these enzymes resulting in different rates of alcohol elimination and susceptibility to alcohol induced damage.

Those individuals with reduced activity of aldehyde dehydrogenase develop alcohol induced liver damage at a

lower cumulative intake of alcohol. Note that alcohol has a direct hepatotoxic effect and does not require pre-existing malnutrition, but malnutrition may play a prominent role in producing alcohol liver damage. There is a threshold of alcohol toxicity beyond which no dietary supplements can offer protection.

Fatty liver is the most frequent abnormality of the liver following alcohol ingestion and it develops within 7 to 10 days of excessive intake. Alcohol causes increased triglyceride synthesis and a reduced oxidation of lipids and impaired secretion of lipids by the liver. The resultant effect is the accumulation of triglyceride within the liver cells which initially is localized but later becomes diffuse. Individuals with fatty liver are usually asymptomatic with enlargement of the liver, but in severe cases they lose appetite, are nauseated and tend to vomit. At this stage it will take four to six weeks of complete abstinence and adequate nutrition for the fat to disappear from the liver. Where the fatty liver of alcohol fails to resolve, it may result in either alcoholic hepatitis following an alcoholic binge; or to liver cirrhosis following continuous alcohol ingestion.

Thus, the natural history of alcohol abuse is fatty liver, steatohepatitis, alcoholic hepatitis, chronic alcoholic liver disease, cirrhosis and liver cancer.

But, it is not all who consume alcohol that are at risk of developing alcoholic liver disease, because the tendency to abuse alcohol runs in families. People with a family member who has alcohol abuse have a more positive view of, and a more positive experience with alcohol. Such people are at an increased risk of resorting to alcohol abuse and they

experience a stimulating effect from alcohol and want to take more alcohol.

Alcohol is a major cause of chronic liver disease in the USA, as ninety percent of chronic liver disease cases in USA are due to alcohol abuse. At the UPTH between 2001 and 2004, we looked at a series of 398 patients with liver disease and found the prevalence of alcoholic liver disease to be 12.5%. (Madubuike, Oyeleke, **Ihekwaba** 2007) (**Table vii.)**

Table vii. Diagnostic considerations amongst 398 cases of Liver diseases at UPTH (2001-2004)

S/n	Type of Liver disease	M	F	Total (%)
	Acute Liver disease **(215 cases)**			
1	**Acute viral hepatitis**	**83**	**36**	**119 (55.6)**
2	**Fulminant hepatic failure**	**6**	**5**	**11 (5.1)**
3	**Amoebic liver disease**	**58**	**26**	**84 (39.3)**
	Total	**148**	**67**	**214 (100)**
	Chronic liver disease **(184 cases)**			
1	**Alcoholic liver disease****	**16**	**7**	**23(*12.5*)****
2	**Chronic liver disease(non-alcoholic)**	**55**	**45**	**100 (54.3)**
3	**Primary liver cell carcinoma**	**41**	**20**	**61(33.2)**
	Total	**112**	**72**	**184 (100)**

But in 2010, in another study of chronic liver disease patients over a twelve month period in which specific aetiologies were sought for, we found that the prevalence of alcoholic liver disease had increased to 23 percent. (Madubuike O. C 2010)

Table vii. Aetiological factors in100 cases of Chronic Liver Disease.

S/n	Type of Liver disease	(percent)
1	HBV	(46)
2	Alcohol	(23)
3	HCC without HBV or Alcohol	(11)
4	HBV with Alcohol	(4)
5	Others	(16)

At times it may be difficult to determine if the liver disease is due to other causes other than alcohol, as more than one aetiological factor may be involved as can be seen in the above study where we found that 4percent of the cases had both HBV and alcohol. In the 2007 series of ours, where we evaluated 100 patients with chronic liver disease, (half of whom were HBs antigen positive), and we found that 71% of them admitted to regular alcohol consumption of variable quantity.

Alcoholic hepatitis is usually indistinguishable from viral hepatitis at the bed side, except for a strong alcohol history. Mild cases of alcoholic hepatitis are diagnosed following a liver biopsy, but in severe cases, the individual is malnourished, nauseated, does not have appetite for food and has lost a lot of weight.

In such situations, about two out of every five are jaundiced and have all the stigmata of chronic liver disease. These individuals are susceptible to lapsing into coma following vomiting, diarrhea or should they develop an infection. At this stage resolution may take up to six months following abstinence.

More than five years of continuous daily alcohol intake (usually ten years) of more than 60 grams in males (2 bottles of small stout; half bottle of Kai kai; or a glass of Gin or Whiskey); and 40 grams in females (two-thirds of male consumption) will result in alcoholic cirrhosis.

Many patients with cirrhosis may remain asymptomatic but following an alcoholic binge or inter-current infection will decompensate and manifest with the features of chronic liver disease**. (Table ix).**

Table ix. Major Clinical manifestations of chronic liver disease

Systemic.

Anorexia, malaise, fatigue, Fever,
*General deterioration, weight loss
Cholestasis,* xanthomas,* malabsorption problems

Jaundice.

Enlarged liver with/or without pain

Portal hypertension

Fluid derangements

*Ascites, with/without edema,
Electrolyte disturbances;
Functional renal failure (‘hepatorenal failure’)

Hepatic encephalopathy

***Cutaneous and endocrine changes**

Spider naevi, palmar erythema, Dupuytren’s contractures, Gynaecomastia, testicular atrophy, Impotence; Amenorrhoea; female hair distribution in the male.

Parotid enlargement

Coagulopathy

Thrombocytopenia, Dysfibrogenemia;

Hypoprothrombinaemia.

Circulatory changes

Hyperdynamic circulation,

*Arterial desaturation, Clubbing of fingers and toes

* implies chronicity

DRUG INDUCED LIVER DAMAGE

Very often following an outing over alcohol, one develops headache and decides to take an analgesic, the commonest of which is acetaminophen (paracetamol).

Most of the paracetamol ingested is normally rendered harmless in the liver through a process known as conjugation (complexed with sulphate and glucuronide) the product is excreted in the urine. However, about 5 to 10 percent of the ingested paracetamol undergoes oxidation by the group of enzymes of the Cytochrome P phosphatases in the liver (which also oxidizes alcohol) to form a toxic metabolite.

This metabolite is subject to either conjugation with glutathione to form a harmless product for excretion into the urine; or it may undergo co-valent binding to the liver macromolecule. The latter pathway results in damage to the liver cell. When large quantities of paracetamol are ingested, the glutathione stores will be depleted, therefore there is increased co-valent binding to the liver macromolecules resulting in liver cell damage. Paracetamol induced-damage is not a common problem except in attempted suicides.

The liver damage is accentuated if both alcohol and large doses of paracetamol are ingested at the same time, and this may result in coma.

VIRAL LIVER DISEASE

When God created Adam he decided to find a companion suitable for Adam in the person of Eve. With time, one 'Eve' was no longer enough for man; hence Man began to acquire more daughters of Eve. In Genesis 6:1-2, we are told that

'when men began to multiply on the face of the earth, and daughters were born to them; that the sons of God saw the daughters of men, that they were beautiful; and they took wives for themselves of all whom they chose'.

Man's heart is insatiable even with the acquisition of more wives, some men decided to practice sexual relations which was contrary to God's injunctions. He resorted to trying other sexual activities as we can read from Genesis 19:6, where we see men desiring to have sexual relations with fellow men not knowing that they were angels.

Where are the men who came to you tonight? Bring them out to us that we may know them carnally?

It is this vile passion of men lusting after fellow men which Uncle Paul warned the people in Rome (Romans 1: 27):

'Likewise also the men, leaving the natural use of the woman, burned in their lust for one another, men with men committing what is shameful, and receiving in themselves the penalty of their error which was due'

This has resulted in one of the greatest tragedies of this century- human immunodeficiency viral (HIV) infection.

The Liver is subject to attack by several viruses designated by the letters of the alphabets. The first which was identified is hepatitis A. It causes Infectious hepatitis and was initially known to be acquired by ingesting faecal contaminated food or

water (vide supra), but lately shown to be transmitted by **analingus**- (anal-oral sex**). What an anomaly of life**!!!.

The other hepatitis viruses that cause identical illnesses as Infectious hepatitis are **Hepatitis B, C, D and E.**
Although all these Hepatitis viruses cause similar liver diseases, the **B, C, and D** viruses cause more severe illnesses than A, and are not acquired by consumption of faecal contamination of food and water, but have similar routes of infection as the HIV.

The known routes of transmission of Hepatitis B, C, and D viruses are:

1. From mother to child during delivery and breast feeding,
2. From person to person as in children playing together in schools,
3. Transfusion of blood or its products,
4. Use of unsterilized instruments in manicure, tattooing, ear piercing,
5. Intravenous drugs abusers
6. Use of unsterilized surgical instruments, and
7. Sexually (whether normal-male to female, or abnormal-male to male or female to female).

HEPATITIS B VIRUS
Of the three viruses (B C D), **Hepatitis B** is the commonest affecting approximately **2 billion** people worldwide, with **300 to 350 million** chronic carriers, and accounting for **50 million new cases** annually, and one million people die annually of hepatitis B virus (HBV) related causes.

Its prevalence varies depending on the areas**.**
Low prevalence areas with rates of one to two percent are: the United States, United Kingdom, Australia, New Zealand and parts of Europe; **Intermediate prevalence** areas with rates of

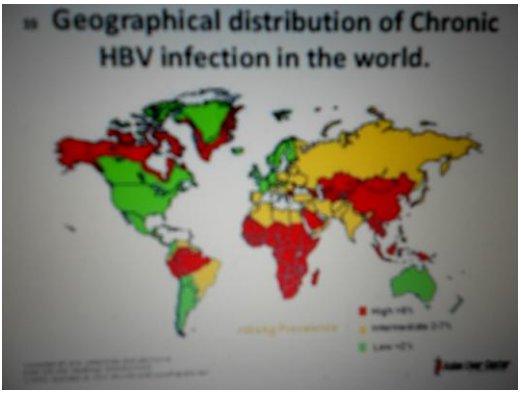
3 to 5 % are: Japan, Central Asia, Middle East, Latin and South American. **High prevalence** areas with 10-20 % include South East Asia and sub-Saharan Africa.

In India, 40million people are infected with HBV, whilst in Hawaii 1-3% are affected. Amongst Americans the prevalence is higher in foreign born Americans than those born in the country, and even among the foreign born Americans; African Americans had the highest prevalence of 11.8%, followed by the Asians - 7.9%.

In Nigeria the carrier rate varies from 8 to 20%, and varies from one geo-political zone to another.

The rates are: South-South- 4.6 - 26%; South East- 9-18.4; South West-10.3 - 84%; North Central-11-27.8%; North East-26 - 49%; and North West-70.3% (Ola SO 2010).

Fig. 24 Geographical distribution of HB virus in the World.



These wide variations in the figures obtained is due to the different populations studied, being higher amongst medical and paramedical personnel than the general population. The lowest prevalence of 2.89% was found amongst pregnant

women in south-south Nigeria (Obi R. K, Umeh S. C & Okurede O. H 2006).

Some two and half decades ago, it was estimated that the national average prevalence of HBV carrier rate was 12% (Olumide 1976). Recently, we surveyed about 500 hospital workers at the UPTH, and we found that 4.2% of them tested positive to the HB surface antigen, more amongst the clinical staff than administrative personnel (Wokoma I. S**, Ihekwaba A. E**, and Uchenna DI, 2007); which was not different from the 4.6% reported from UPTH earlier on. (Ibiama 1976). With a carrier rate of 4 to 20% and a population of Nigeria of 150 million, we can estimate that 6 to 30 million Nigerians are carriers of this virus.

HBV is more fastidious than HIV, surviving on the ordinary surface for four hours unlike the HIV that dies within seconds of exposure to sunlight. The antigen is secreted into all body fluids. We had earlier looked at a group of patients with Liver disease with ascites and discovered that some of them also had the antigen in their ascitic fluid, (**Ihekwaba A. E** and Aniemeka J. I, 2001) thus exposing the medical personnel who handle such specimens to danger.

Despite these statistics, the commonest route of transmission of this virus worldwide and in the advanced countries is the sexual route. But in sub-Saharan African, horizontal transmission amongst siblings and mother- to -child-transmission are the major routes of transmission in children, whilst heterosexual is the major route in adults.

CHRONIC HBV DISEASE

Chronic liver disease (comprising chronic hepatitis, Liver cirrhosis and hepatocellular carcinoma), is a common disease worldwide. Its incidence varies from geographical region to another depending on the prevalence of the dominant aetiological factor. In Western industrialized countries, hepatitis C and alcohol abuse are common causes, while in sub-Saharan Africa and south-east Asia, Hepatitis B is the commonest cause.

At the UPTH, out of 398 cases of liver disease, 100 had chronic liver disease presumably due to HBV, although we did not at that time determine the incidence of hepatitis B infection in them.(Madubuike O. C, Oyeleke G. K, **Ihekwaba A. E** 2007). However, some three years later we surveyed another group of 100 chronic liver disease patients, to determine the prevalence of HBV in them. We found that 50 percent of them had markers of HBV infection in their sera. (Madubuike 2010).

The role of the sexual route in acquiring HB surface antigen is reflected in the later series, where 70% of them admitted to having multiple sexual partners of two or more.

Further analysis of the of 50 cases who had evidence of HBV infection, in which we looked at the risk factors for acquiring the virus, we found that 78% had multiple sexual partners, and almost all were not vaccinated against the HBV.

The features of chronic liver disease are almost the same irrespective of the aetiology. At the UPTH the commonest features we encountered in our series were abdominal/leg swelling followed by abdominal pain and jaundice.

LIVER CANCER

Liver cancer is a common cancer in sub-Saharan Africa with the highest incidence seen amongst the Bantu males of Mozambique.(Kew M. C, Geddes E. W (1982). It is the 6thcommonest cause of death Worldwide, and the single most important cancer death in some countries. (Higginson J, Svoboda D. J 1970). It is the leading cause of death among patients with cirrhosis, and its incidence is expected to increase until 2020 in Europe and America, because of HCV related post-transfusion hepatitis. But in Nigeria, chronic HBV is the predominant risk factor with little contribution from alcohol abuse, HCV very rarely, and fatty liver.

The prevalence of HCC tends to vary, decreasing in incidence as one moves down south from the Savannah belt following the same prevalence pattern as hepatitis B surface antigenaemia. In Port Harcourt, south-south Nigeria we reported a Liver cancer prevalence of 17.1% of all cancers presenting in our medical wards over a four year period, and 3.97% per 1000 patients per year **(Ihekwaba A. E**, Nwankwo NC, 2003), as against a value of 11.2 % per 1000 patients per year reported from Maiduguri (Gashua 1988)

The incidence is also dependent on whether the study is clinical or pathological. Abdulkareem and her colleagues in a pathological study of liver biopsy specimens found it to be 33% (Abdulkareem et al 2006); but we in UPTH in 2010 reported a clinical prevalence of 11% amongst Chronic Liver disease cases (Madubuike 2010).

The incidence of HB surface antigen in Liver cancer also varies from one locality to another. In some series as much as 89.6 % were reported, (Tacke F, Trautwein C, 2006), while

Otu (1987) reported a prevalence of 90% in Calabar, we in UPTH reported an incidence of 36.8% in our series in 2003 (Ihekwaba A. E Nwankwo N.C 2003).

With such figures it has been estimated that the risk of a chronic carrier of the HBs Ag progressing to Liver cancer is 12 to 300 times that of a HB surface antigen negative individual (Ajayi BB, Nggada HA, Moses AE 2007). The risk is higher in the presence of Aflatoxin B1, alcohol consumption, hepatitis C virus, Diabetes mellitus, Fatty liver and iron over load.

Primary liver cancer being a common malignant tumour in the sub-Saharan Africa, and a major cause of death from cancers has been shown to account for about 200,000 deaths each year. (Boyle P.2002).

It affects young black men more than women, attacking them in their prime of life. It carries a grave prognosis with an average survival from onset of symptoms being as short as two weeks (Kew MC & Geddes E. W. 1982; Ndububa D. A, Ojo O. S, Adeodu O. O et al 2001). Its diagnosis is usually straight forward in the presence of a hard nodular and tender hepatomegaly in the setting of weight loss and abdominal swelling. An ultrasound finding of coarse irregular internal echoes which are hypo or hyperechoeic with posterior acoustic enhancement adds to the diagnosis. In most centres, tissue diagnosis is not possible because of lack of well trained personnel to perform liver biopsy and lack of relevant laboratory services. In such situations an elevated alpha foeto-protein level is seen in 90% of Black African patients, but more importantly in about 75% of cases, values of greater than 400ng/ml was said to be diagnostic (Alpert E, 1976;

Kew MC, 1989), though recently a value of > 200ng/ml has been suggested (Ola and Odaibo 2007)

HCC is one cancer that occurs in the context of well recognized and potentially preventable risk factors as can be seen from its route of transmission.

TREATMENT OF HEPATITIS B, AND ITS RELATED DISEASES

1. The HB surface antigen positive- the options here are: where the HB surface antigen positivity is less than six months-either following routine testing or contact tracing: monitor patient with serial liver function tests, abdominal ultrasonography, viral load quantitation and e antigen estimation.
2. Acute viral hepatitis. Supportive care is the treatment of choice in acute viral hepatitis and does not require antiviral therapy.

Antiviral treatment is required in the patient who has chronic infection to prevent progression of the disease to cirrhosis and liver failure.

Some five to ten years ago, there were two main drugs approved for the treatment of chronic hepatitis B. These were Lamivudine and Interferon. But recently several drugs are available but the cost of treatment is well beyond the reach of the average patient. Whereas the cheapest oral drug- Lamivudine costs 14,000 naira monthly, treatment may last for 5 years, Interferon which is administered by injection costs between (but costs 29,000 monthly Oct, 2011) 46,000 and 52,000 naira monthly with a minimum duration of treatment of six months. Unfortunately with stoppage of treatment with

Lamivudine, there is the tendency for the infection to recur, and long term treatment results in development of drug resistance.

But for Interferon,which is expensive, it is administered as an injection and has more side effects

Other newer drugs have been introduced such as Adefovir, Entecavir and Tenofovir, but are expensive and not within reach of the average patient within the sub-Saharan Africa.

For liver cancer its treatment in sub-Saharan Africa is largely unrewarding because by the time the diagnosis is made the tumour is far advanced due to the short doubling time of ten days compared to 60 to 120 days in in other regions (Purves LR 1976; Chen DS, Sung JL, Sheu JC et al 1984; Okazaki N, Yoshino M, Yoshida T et al 1989); and the occurrence of early metastasis to the lungs. (Levy JL, Geddes EW, Kew MC 1976)
A major hindrance to early diagnosis of liver cancer is the fact that the liver is a big organ and has a considerable functional reserve; hence its function is preserved until most of the organ has been replaced by tumour.Experience from South Africa has shown that black African HCC are unusually large weighing almost one and half times those of South African whites and the Japanese.(Kew MC, Paterson AC 1985; Davies JNP 1961; Okuda K 1997)

Surgery (Resection) has remained the main treatment modality for HCC in most centres. The decision to operate is determined by whether the tumour is resectable, and also if transplantation would be curative (if feasible).
For us in Nigeria, Surgery is an unlikely option because no tertiary centre has the equipped facilities- trained and

experienced Surgeons, Anaesthetists, and Nurses experienced in post- transplantation care.

Even less invasive treatment modalities such as trans-arterial chemoembolization (TAE), percutaneous ethanol injection (PEI) and radiofrequency ablation (RFA- which has succeeded in 70% of patients (Yao FY,2008) is not available.

One option that may be available to us in Nigeria is the use of drugs. **Sorafenib,** is an oral multi-kinase inhibitor which inhibits the tumour cell proliferation and tumour angiogenesis. This drug increases the rate of apoptosis (cell death), but experience so far, has reported a median survival of 10.7 months as compared to 7.9 months with placebo (Llovet JM, Ricci S, Mazzaferro Vet al. 2008); and some troublesome side effects have recently been reported. However, there is hope for recently Chinese Clinicians have shown that individuals who drank large quantities of Green tea and for a long time (more than 30 years) had the lowest risk of developing hepatocellular carcinoma (Li Y,Chang-Shen C, Goldestein BH, et al 2011).

Other drugs include: Interferon-some improved survival.
Ocreotide-some survival obtained, Tamoxifen-no effect obtained. Retinol - preventive benefit after resection. That is the dilemma in the treatment of Liver cancer.

Thus, the solution to all we have said so far is found in the old adage that:

PREVENTION IS BETTER THAN CURE.(PREVENTIVE HEPATOLOGY), RECOMMENDED MEASURES

1. Ear Right

The right **quality** of food which is nutritious (healthy foods) containing vegetables and fruits are ideal. It is highly recommended that meat, saturated fat and products with less complex carbohydrates should be avoided. A mixture of food comprising two-thirds- plant based, and the remaining one-third of animal protein is recommended. The food which Daniel requested for in Dan 1: *12***.**

''Please test your servants for ten days, and let them give us vegetables to eat and water to drink''

After ten days their features appeared better and fatter…..vs 15.

Diets which are non-nutritious containing high fibres and complex carbohydrates that have low glycaemic index should also be part of the diet.

In some situations, food additives are part of the meal as these add colour, make the food look appetizing, and makes the individual feel full with less quantity of food.

The right **quantity**. Eating the right quantity of food based on the ideal weight for the age and sex of the individual is recommended. This involves life style modification such as increase in physical activity and abstinence from alcohol

(a) Weight reduction can be achieved via the following means:

1. Should limit the energy intake from total fats;

2. Increase consumption of fruits and vegetables, as well as legumes, whole grains and nuts;
3. Limit intake of sugars;
4. Engage in regular physical activity;
5. Achieve energy balance and a healthy weight.

Weight loss should be slow and moderate, aiming at losing less than 10% of body weight over 6-12 months. Should avoid rapid weight loss from very low calorie diet, or starving because of the increased risk of progression of liver disease and even liver failure.

(b) Special diets- there has been the suggestion of special diets whose effects are very doubtful.

(c) Other measures that have shown promise and of some benefit are

i. the use of drugs that induce weight loss, when dieting and life style modification do not work, such as Orlistat

ii. Those who are massively obese in which all other measures have failed, have had to have Surgery in which their intestines are reduced (By-pass surgery)

iii. Antioxidants.

These are substances which act within the cell to prevent the action of free radicals on the cell mitochondria. They have cyto-protective properties. Examples include vitamin C, Vitamin E, glutathione, acetycysteine and ursodesoxycholic acid.

Many have been tried, but multi-centre and Cochrane meta-analysis have shown that they are of no significant benefit.

However, it was shown that **Grapes** and **Grape juice** especially **purple** and **red** grapes contain a substance called **Reversaterol** which has strong antioxidant and anti-

inflammatory properties in animal experiments where it prevented tissue damage that trigger cellular cancer formation.

Other fruits shown to have antioxidant properties are **Strawberries** and **Blue berries**. Whereas Strawberries contain the phytochemical-Ellagic acid, a powerful antioxidant that deactivates cancer cells and slows the growth of cancer cells; Blue berries contain antioxidants that rid the body of free radicals.

Some authors have suggested that **Tomatoes** reduce the risk of developing cancers and increase the cancer potential, but they are not sure if **Lycopene**, the pigment that gives tomatoes its red colour is the agent that helps to fight cancer.

Also it was recently shown in rats that a mitochondrion-targeted ubiquinone intercepted and neutralized free radicals before they could damage mitochondria, thereby preventing the cascade of effects that ultimately lead to steatosis caused by excess alcohol.

Live Right

Alcohol- complete abstinence for those who are able; or moderate intake for others. What is moderate for you may be over dose for another, although the Cardiologists recommend a little wine for the heart.

HBV

This requires an integrated approach involving the Clinician, the Health care institutions and the Government of the land:

1. The Clinician: the clinician needs to educate the patient on
    i. The natural history of the disease,
    ii. What the treatment options are, and

iii. What to expect in future.

2. The health institution: the health authorities in collaboration with stake holders to:
   i. Organize health talks on preventive strategies to reduce the risk of contacting the hepatitis B virus (based on the knowledge of the routes of transmission of the virus).
   ii. Educate the population on dietary measures to reduce the incidence of obesity; intake of foods shown to reduce the incidence of Liver cancer such as drinking of coffee and Green tea.
   iii. Organize mass vaccination campaign with and without screening
   iv. Integrate immunization of all newborns into the National immunization program (already in place)
   v. Screen all pregnant women, and vaccinate all hepatitis B surface negative mothers before delivery
   vi. Vaccinate all health workers, at risk patients and HB surface antigen negative contacts of infected persons.

3. A national policy to be formulated for compulsory screening at the following times:
   i. Pre-school,
   ii. Pre-employment and
   iii. Pre-marital

4. Surveillance of HBV positive persons using Ultrasonography and alpha foeto protein estimation to detect hepatocellular carcinoma at its early stages when Surgery is likely to be curative.

5. Finally, training of the necessary man power and establishment of a Liver centre to look after the patients with the disease.

CONCLUSION

Mr. Vice Chancellor Sir, we have seen that not eating right (either in quality or quantity) and not living right is an invitation to liver disease.

Gluttonous eating without adequate exercise will result in obesity which in turn will cause a fatty liver whose natural history results in hepatitis, chronic liver disease and in some instances to Liver cancer.

Chronic ingestion of alcohol in whatever form (with as little as one bottle of Lager beer daily) will cause not only a fatty liver but also alcoholic hepatitis and liver cancer.

That of all the viruses that cause viral hepatitis, hepatitis B is sub-Saharan African number one enemy, in that it is more infectious and fastidious than HIV; it causes chronic liver disease, which is the commonest cause of liver cancer.

Its transmission can be controlled, and it is amenable to treatment, unfortunately treatment is expensive, long lasting, and the incidence of drug resistance is common for the cheaper available drug. Unfortunately it suffers the same fate as eating the wrong food and chronic alcohol ingestion- development of liver cancer which is responsible for the death of most young men in sub-Saharan Africa; whose treatment is expensive and the outcome dismal.

The key to a **RIGHT LIVER** is **PREVENTIVE HEPATOLOGY: EAT RIGHT AND LIVE RIGHT.**

THANK YOU FOR LISTENING 27/10/2011

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CITATION

ON

PROFESSOR ANELE EJIKEME IHEKWABA

MBBS (Lagos); MSc. Clinical Pharmacology, (UNN); FMCP (Nig.)

Introduction

Professor Anele Ihekwaba's attainment to the professorial chair has been a journey fuelled with dedication, vision, innovation, courage and tough decisions. Born in Port Harcourt on the 19th of July 1951 to the late Francis U. Ihekwaba (the then Mayor of Port-Harcourt) and Esther E. Ihekwaba (Nee Uzoukwu), both of Nkwerre in Imo State of Nigeria.

Education:

His educational voyage started in January 1956 from Wesley Methodist primary school Harbour Road, and later Banham Memorial School, Port Harcourt. This expedition took him to Saint Augustine's Grammar School, Nkwerre in 1964, where he completed his secondary education after the civil war in June 1971.

With a Distinction in his Primary school education, and a Division One in the West African School Certificate Examination, Anele Ihekwaba was admitted to read Medicine at the University of Lagos in 1972. As if the barrels of the gun at war had stimulated an inert academic, his performances at all professional examinations at the University of Lagos were exceptional. Apart from the A.G. Leventis Group of Companies scholarship and the East Central State scholarship in his early University education, he won the Federal Government National Award, following his brilliant

performance at the 2nd MBBS professional examinations. At his graduation with the MBBS degree in 1978, Anele bagged:- the KB club prize for the best student in the 2nd MBBS examinations having passed with distinctions in Anatomy and Physiology, the Nicholson prize for the best student in Pathology; the Glaxo Pharmaceutical prize for the best student in Pharmacology; and the Biode prize for the best student in Medicine as a Subject, at the final MBBS examinations.

Professional/Academic career

A visionary scholar of extraordinary insight, he started his housemanship at the Lagos University Teaching Hospital in 1978, and moved after six weeks to the University of Benin Teaching Hospital. Here he was also appointed a demonstrator in Anatomy.

Following his National Youth Service in Niger State, and a brief spell as a Medical Officer at the Queen Elizabeth Specialist Hospital, Umuahia, he started his residency training in Internal Medicine at the University ofNigeria Teaching Hospital (UNTH) in 1981. While doing his residency, this multi-talented and focused doctor found time to read and eventually bagged a Master of Science degree in Clinical Pharmacology and Therapeutics at the University of Nigerian in 1986.

Upon completion of his residency programme with a Fellowship of the National Post Graduate Medical College in 1988, he was employed as a Clinical Research Fellow in the WHO/UNDP/World Bank sponsored Onchocerciasis research programme at the University of Nigeria Enugu campus. Seven months later, the Anambra State University employed him as a

foundation staff to develop the department of Pharmacology and Internal Medicine.

It was while waiting for the commencement of the State University in 1989, that the established unique University of Port-Harcourt employed him as Lecturer 1, the third academic staff in the internal medicine department.

Mr. Chairman Sir, This was an answer to a Macedonia call as the University of Port-Harcourt was about to lose its accreditation with the Medical and Dental Council of Nigeria due to poor staff/student ratio.

He rose from lecturer I to Senior lecturer in 1992, then to a Reader in 2003 and subsequently to the Chair of Medicine in 2006. Although he was in the Department of Internal Medicine, he has had to assist the department of Pharmacology and Physiology without remuneration.He was also a part-time lecturer in Physiology to the Abia State University in 1990.

Administrative responsibilities and academic leadership

Professor Anele Ihekwaba is a multi-talented, multi tasked administrator who served the University of Port Harcourt and the University of Port Harcourt Teaching Hospital (UPTH) in various capacities. He was appointed an Honorary Consultant Physician to the UPTH in August 1989. The following year, he was appointed Coordinator of Mental Health department, and the Head of General Out–Patient Department. He served as Acting Head of Department of Medicine from 1993 to 1997, and Head of department from 2006 to 2009.

He was Associate Dean, Faculty of Clinical Sciences from 2002 to 2006.

Professor Anele Ihekwaba has been a member of the Senate of the University of Port Harcourt since 1990 to date, first as coordinator of Mental Health, acting head of department, College representative and then Professor of Medicine .

He was Senate Representative in the boards of the University Demonstration Primary School in 2005, and UPTH in 2006/2007. He served in various University committees including Post–graduate School, Housing loan, Committee to determine the criteria for graduation of students.

Professor Anele Ihekwaba is a man noted for his forthrightness. He insists on playing according to the rules, little wonder that Resident doctors call him 'Mr. Protocol'. In the College of Health Sciences, he chaired various investigation committees on acts of misdemeanor by staff and students. At the UPTH, he also chaired many investigative panels on acts of insubordination and fraud involving staff. He has been the Chairman of the Anti-Corruption and Transparency Unit from 2006 till date.

Research career

Professor Anele Ihekwaba cut his teeth in Scientific inquiry early in life while working with the late doyen of Clinical Pharmacology and Therapeutics in Nigeria in the person of Professor Gilbert Onuaguluchi. They both showed that a herbal preparation the Yorubas use to deworm children, had some deleterious effect on the heart.

He is a Clinical Trialist to several pharmaceutical companies on Anti-retroviral drugs and Proton Pump Inhibitors. He has proven himself to be a formidable International scholar having published over 30 articles in local and international journals,

read several papers at local and international conferences and written a book. Most of his publications are in his sub-specialty of Gastroenterology and Hepatology.

He is a supervisor of Graduate students in Physiology and has trained three Gastroenterologists who are now Consultants in various hospitals.

Professional Activities

A tested and known professional of an inestimable value, Professor Anele Ihekwaba was Vice President of the Association of Resident Doctors, UNTH, Enugu 1984/85. He is a member of the Nigeria Medical Association; member, Association of Physicians of Nigeria; member, Medical and Dental Consultant Association of Nigeria, past Chairman, Port-Harcourt branch of the Association of the Study of the Liver in Nigeria, past Chairman West African Society of Gastroenterology, and Chairman, Rivers State branch of the Society of Gastroenterology and Hepatology in Nigerian (SOGHIN).

Professor Anele Ihekwaba is an External Examiner in Internal Medicine to various Universities including the University of Nigeria, University of Calabar, Abia State University, Imo state University, Nnamdi Azikiwe University, University of Benin, and the University of Jos.

He is an examiner to the National Post-Graduate Medical College of Nigeria.

He is a member of the Faculty Board of Internal Medicine of the Post-Graduate Medical College of Physicians of Nigeria.

Private Life

An ardent believer in family unit, Professor Ihekwaba is happily married to Ijeoma, a Lecturer in our Nursing department. They are both blessed with six children, a daughter in law, a son in law and two lovely grand- children.
His community and the church has also benefited from his huge reserve of knowledge and experience. He was chairman of his Town Union (Nkwerre Aborigine Union), Enugu branch (1986 to 1989), Sole administrator of its Port Harcourt branch 1989 to 1994.

He was Financial Secretary, Harvest Committee, Assistant Secretary, Parish Council, Emmanuel Anglican Church Enugu (1987 – 1988); and Chairman, Medical Consultative Committee, St Matthew's Anglican Church Nkpogu, Port Harcourt (2000 till date). He is presently an Un-Ordained Preacher of the Gospel of the Lord

Conclusion:

Mr. Vice chancellor Sir, distinguished ladies and gentlemen, I present to you our 80th inaugural Lecturer, a man of Noble birth, an Award winner, , a Multi-talented and gifted Scholar of extraordinary insight, a human Physiologist, Anatomist, Clinical Pharmacologist, Internist, Gastroenterologist Hepatologist, a Preacher of the Word of God, a Moral and Intellectual mentor, and Role model, a courageous, forthright visionary administrator and researcher, who has served this University, his community and mankind – PROFESSOR ANELE EJIKEME IHEKWABA.

Thank you

Professor John Ikimalo. (27th October 2011)