UNIVERSITY OF PORT HARCOURT

WHO HATH WOE? WHO HATH SORROW? WHO HATH CONTENTIONS? A CHRONICLE OF THE CONSEQUENCES OF ALCOHOL MISUSE AND INTERVENTIONS

An Inaugural Lecture

By

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DEDICATION

This inaugural lecture is dedicated to my beloved parents (both of blessed memory) Hon. Emmanuel Friday Stanley and Mrs Esther da Sunday Stanley for their unwavered support and uncompromised believe in academic excellence.

And

To my darling wife, Dr (Mrs) Catherine Stanley, our five lovely children and my twin brother, Dr H.O. Stanley for their unity of purpose towards excellence.

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JUTH, 1990-1997), Prof G.A Ubom (Unijos), Dr B.G. Fente, Prof I. Siminialayi, Prof and Prof (Mrs) C. Akani,

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PREAMBLE

Vice Chancellor, Sir, it is with immense pleasure and gratitude to God that I stand before you and this distinguished audience to deliver the 108th inaugural lecture of the University of Port Harcourt, my Alma mater.

Today marks an important academic landmark in the pursuit for excellence.

Firstly, it is the day that marks the fulfilment of God's promise that I will stand before Kings and not mean men nor women (Proverb 22:29).

Secondly, it is a day of great reassurance that our diligence in establishing a functional Department of Neuropsychiatry and a drug treatment unit is rewarding. This indeed is the first inaugural lecture from the Department.

Thirdly, it marks the actualization of a dream to make a branded statement against Drug and Alcohol misuse at the Centre of Excellence, the home of Academic enthusiasts.

Consideration for the Title: Vice Chancellor, Sir, having been driven by sufficient empathy to understand the etiopathogenesis of mental illness-a debasing disorder, that often demeans man and takes away the pride of human dignity-; I became quickly convinced that apart from genetic predisposition, drugs and substances of abuse contribute substantially to this.

In this regard, I further understood the dangers of one of these drugs, Alcohol, so innocuous it could look, so admired a companion it could be, so alluring it can be, so admissible into our homes and families, yet, so devastating.

Indeed, the greatest enemy of man are the members of his household (KJ), Alcohol being the first, hence I refer to it in my first book in 2003, "As the Silent Killer." Time has therefore come for us to remind ourselves of the devastating effects of alcohol misuse in our society today (Stanley and Jumbo, 2005). It will indeed afford us another opportunity to save humanity, particularly our most endangered group, the so called leaders of tomorrow.

If Professor Wole Soyinka the Noble Laureate could refer to his, as a wasted generation, the generation of our youths could be likened to a desolate one, so devastated by drug abuse, maligned by perpetual deprivation and companioned by violence.

Hence, this lecture has become auspicious and could not have come at a better time than this.

Vice Chancellor, Sir, and our distinguished audience, it is in this vain that I have considered the topic of this lecture from a Bible passage-"***Who hath woe? Who hath sorrow? Who hath contentions?*** Coincidentally, it is quoted on the Preliminary page (iv) of my first book-Alcohol: the Silent Killer.

INTRODUCTION

The Holy Bible (the word of life) says in Genesis 2:16 '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'. This death here may be physical, spiritual, emotional, psychological or economic.

Vice Chancellor Sir, I was miffed the day I saw this passage in the Bible, Proverb 23: 29-30 which says "***Who hath woe? Who hath sorrow? Who hath contentions? Who hath babbling? Who hath wounds without cause? Who hath redness of eyes? They that tarry long at the wine; they that go to seek mixed wine.*** This is in keeping with the Diagnostic and Statistic Manual (DSM-III-R) third revised edition of the American Psychiatric Association which classifies Alcohol misuse (Alcoholism) into alcohol abuse and alcohol dependence.

Alcohol abuse indicates psychological dependence, that is the need to use alcohol for adequate functioning, along with occasional heavy drinking and continuation of drinking despite social and occupational problems.

Alcohol dependence comprises similar impairment, coupled with evidence of increased alcohol tolerance and physical signs of withdrawal from alcohol.

Vice Chancellor Sir, permit me to share a recent survey experience from the Department.

Background of Treatment Centre

The Department of Mental Health and Neuropsychiatry was established alongside 9 other Departments in the College of Health Sciences at the inception of the University in 1977.

However, the Mental Health Department then operated under Internal medicine at the Teaching Hospital, ran clinics twice a week and had no admission facility. However, in the year 2000, two years after I was appointed a consultant, I made a strong request for us to obtain the status of a full fledged Department at the Teaching Hospital based on the increasing needs of our Clinical Services.

This request was granted in 2001 and we were given 12 bed spaces (6 each for males and females) at the temporary site. The need for admission was so overwhelming that we began to admit into other wards (but with much difficulty). It was also interesting to note that one quarter (25%) of these patients at the clinic who needed admissions were necessitated by drugs and substances of abuse. Hence, in the year 2006 when the hospital moved to its permanent site, we got a whole floor with two wings with a bed capacity of 120 (60 each for males and females). Our outpatient consultation rate per annum increased from 1480 in 2000 to 1988 in 2013 with the number of clinic days increased from 3 to 5.

Forty percent (40%) of patients seen usually have drug related problems. This trend inspired the formation of a drug unit in January, 2010, under the headship of Prof PC Stanley. Sixty percent (60%) of these drug related cases were found to have at least one major psychiatric co-morbidity.

The most common being Severe Depression 54%, Schizoaffective 16%, Schizophrenia-like 14%, Anxiety Disorders 10%, Personality Disorder 6%.

Drug Use Problem in the Catchment Area

Port Harcourt is the capital of Rivers State, usually referred to as the Treasure Base of the Nation, with a population of 3.3 million people (NPC, 2006). A cosmopolitan city attracting and accommodating people of most nations of the world, being the hub of oil and gas business in Nigeria.

It is located within the South-south geopolitical region of Nigeria, and shares boundaries with other neighbouring South-south states such as Delta, Bayelsa, Akwa Ibom, and the South-East states of Abia and Imo.

With the influx of persons of diverse cultures and orientation one understands the enormity of challenges associated with drug misuse in Port Harcourt and its environs. Our department, which is within the University of Port Teaching Hospital has a capacity of 120 bed spaces, a quarter (30 bed

spaces) of which is dedicated to the drug unit. Currently, a spirited effort is being made to obtain a stand-alone facility for the Unit. Consultations are on going and promises made with undertakings.

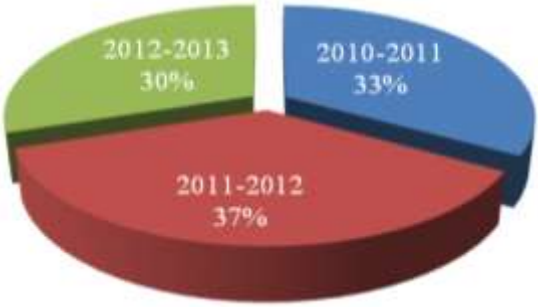


Fig. 1: Admissions due to drug related disorders in Mental Health Department, UPTH (2010-2013)

Table 1: Pattern and Prevalence of Male and Female Admitted Between 2010-2013

Age Group	N	Male %	Female%
< 13	108	98 (90.7)	10 (9.3)
13-19	284	221 (77.8)	63 (22.2)
20-26	398	301 (75.6)	97 (24.4)
27-32	452	398 (88.1)	54 (11.9)
33-39	346	303 (87.6)	43 (12.4)
40-46	280	211 (75.4)	69 (24.6)
47-52	162	159 (98.1)	3 (1.9)
53-59	94	94 (100.)	0 (0)
> 60	76	75 (98.7)	1 (1.3)
Total	2,200	1860	340

N: Total Number of patients admitted between 2010-2013

Table 2: Socio-Demographic Attributes

Educational Status	**M**	**F**	**Total**
No formal	325	132	457
Primary	1005	95	1100
Secondary	422	102	524
Post Secondary	108	11	119
Total	1860	340	2200

Table 3: Ethnicity/States of Origin/Nationality

States of Origin	**Number**	
Rivers	544	
Bayelsa	502	
Akwa Ibom/ Cross River	255	
Edo/Delta	300	
Imo/Abia/Anambra	298	
Oyo/Ondo/Osun	293	
Other Nationals	British	2
	American	1
	Ghanaian	2
	Lebanese	3
	(All males)	
Total	2200	

Fig. 2: Occupational Status

Table 4a: Commonly Abused Drugs

Drugs	**Age Group Most Susceptible**	**Use (%)**
Alcohol	10-19	47
Cannabis	20-26 33-39	25
Hypno Sedative	27-32	13
Opiate	33-39 40-46	8
Cocaine	27-32	7
Total		**100**

Table 4b: Commonly Abused Drugs

Drugs	Age Group Most Susceptible	Use (%)
Poly Substance Use	20-26 33-39	67
Intravenous Drug Use (IDU)	27-32 33-39	7

Outreach Programmes

We have organized several awareness and advocacy programmes agaisnt substance abuse among the following groups in collaboration with NDLEA.

- Niger Delta Youths
- Tertiary-Students Associations
- Military and Paramilitary such as Nigeria Army, Naval, Air Force, police, customs, FRSC etc.
- Secondary Schools

What is Special about the Centre?

The centre is strategically located and easily accessible to all south-south and south-east states and to other regions of the country by land sea, and by air. Thus, making it a suitable centre catering for a sizeable proportion of youths who were deeply involved in militancy in the south-south region. Hence, the centre is in need of funding and facilities for effective service delivery.

It is obvious that drug related problems in Nigeria, particularly the Niger Delta area of the Nation, portend great danger not only to security and economy but to the health of individuals involved. It is therefore a welcome idea that we are not only a model treatment centre but additionally a training centre.

Indeed this encapsulated the strong feelings I had developed towards the misuse of alcohol, hence, the title of my inaugural lecture "***WHO HATH WOE? WHO HATH SORROW? WHO HATH CONTENTIONS? "***

This aptly reveals how the impact of knowledge I have gathered in the course of my research has shaped my perception.

This chemical compound called Ethanol makes a ridicule of God's own creature, made in his own image and likeness, and brings him to public odium, and disrepute. It fractures, fragments and disrupts the unity of the Trinity of human being: the body (physical), the soul (social), and the spirit (psychological).

Definition of Alcohol

In chemistry, an alcohol is an organic compound in which the hydroxylfunctional group (-OH) is bound to a carbon atom. In particular, this carbon centre should be saturated, having single bonds to three other atoms (IUPAC, 1997). An important class of alcohols are the simple acyclic alcohols, of the general formula $C_nH_{2n+1}OH$. Of these, ethanol (C_2H_5OH) is the alcohol found in alcoholic beverages.

Alcohols are hydrocarbon derivatives in which one or more hydrogen atoms have been replaced by the hydroxyl (OH) group (Caputto *et al*., 1967). The locally brewed beers are of cultural significance in their geographical localities. For instance, in the South-South and South-East, we have kai-kai, sapele water, ogogoro, mia-ngwo (ibo), izon wuru (Ijaw),-North Central and other parts of the North, Burukutu and Pito (Hausa), and in the South-West, Paraga (Yoruba). The local brews among them (Kai-kai, Pito, Burukutu, Izon-wuru, and Paraga) are usually produced by catabolic reaction (in the absence of oxygen) (Wood, 1961; Horecker, 1962; Barker, 1965).

History of Alcohol: Use and Misuse

The word alcohol appears in English as a term for a very fine powder in the 16th century. It was borrowed from the French, who took it from medical Latin.

Ultimately the word is from the Arabicكحل (al-kuḥl, ‘kohl, a powder used as an eyeliner). Alcohol was originally used for the very fine powder produced by the sublimation of the natural mineral stibnite to form antimony sulfide Sb_2S_3 (hence the essence or ‘spirit’ of the substance), which was used as an antiseptic, eyeliner, and cosmetic. The word's meaning became restricted to “spirit of wine” (the chemical known today as ethanol) in the 18th century and was extended to the class of substances so-called as ‘alcohols’ in modern chemistry after 1850.

The deliberate creation of drinkable alcohol is thought to date back roughly ten thousand years, and most of the ancient world was very familiar with alcoholic drinks (Austin 1985; Sournia 1990). Beer without hops and drinks made from honey (‘mead’) are likely to have been the first alcoholic beverages, but other drinks were quickly discovered and produced from whatever was locally available. Wine cultivation came later but still dates back at least 5000-6000 years (Burnett 1999), although wine was still a relatively scarce commodity in Ancient Greece and the early Roman Empire. Viticulture only became widespread in Italy from the 2nd century BC, but wine-making then spread quickly through the Roman conquests (Jellinek 1976).

Several positive references to wine are contained in the bible, while many monasteries have produced alcoholic drinks, although the clergy have also issued condemnations of drunkenness which was seen as a form of gluttony (Edwards 2000). The Torah also describes how alcohol can lead to violence, loss of consciousness, and intoxication which is generally stigmatized in the Jewish community (Sournia, 1990).

The existence of abuse of alcohol, or excessive drinking or alcoholism, suggests the existence of normal drinking norms or customs (Effron, 1965). These norms differ widely from society to society, hence what is considered an abuse in one society, ethnic group or culture may not be in another.

These ethno-racial and societal differences have been independently highlighted by Hyde and Chisholm (1944), Varela and Marconi (1952), Jellinek (1960) and Efron (1965). Today in Nigeria, alcohol remains the elixir of life that it was for our forefathers (Odejide *et al*, 1989).

Keller (1989) noted that "in prehistoric times, fermentation of fruits and berries occurred in natural settings; and that although the resulting mash might not have been particularly palatable to our ancestors, upon consumption of it, they experienced relief of fatigue or pain, enhancement of bravery, greater warmth in friendship, and greater ease in communicating with the spirit, in order to be in fuller control of mankind's fate."

In its traditional forms alcohol has been a feature of African social life for a long time. Alcoholic beverages produced from cereal grains (e.g Maize, millet, guinea corn and rice) date back to early agricultural settlements. As early as 4241 B.C. barley beer was consumed in Egypt (Obot, 1993).

Lei (1975) documented many accounts of the preparation and use of alcohol in pre-colonial Africa. These locally produced alcoholic beverages include palmwine from raffia and oil palm trees; and native gin (ogo-goro) from fermented palmwine are produced and commonly used in southern Nigeria. On the other hand, Burukutu and pito are produced from the fermentation of grains, including millet and maize, guinea corn and rice. They are commonly produced and used in the northern Nigeria, particularly the middle belt region (Obot, 1993).

Lynn Pan (1975) had shown that in colonial Nigeria, even as early as 1840, the native leadership sought to uphold the health-promoting cultural attitudes of locally brewed alcohol and tried to discourage the populace from using the imported European brews. Netting (1964) in his anthropological survey of native Kofyar people in Jos (middle belt) area of the country found that though the people lived by brewing alcohol, drinking played a socially harmonizing role in the traditional culture. This is in support of a report credited to Lord Lugard (the British founder of Nigeria) by Lynn Pan (1975) in which Lugard noted

that drunkenness in Africa during the colonial period was far less evident among the native people than among the Europeans. On the other hand, the mythical story of the founding of the Yoruba kingdom (in the present day south west Nigeria) indicates that alcohol abuse could have played a socially disruptive role in ancient history. Eledumare (the chief god of the Yoruba pantheon) first sent his eldest son to found a kingdom on earth. He failed to accomplish this mission because of his love for alcohol. This was however accomplished by his second son, Oduduwa (the patriarch of Yorubas). He succeeded in establishing an Oduduwa kingdom in Ile-Ife, now a University town.

These conflicting reports about the socio-cutural implications of alcohol in Nigeria are not surprising, because recent reports show that most people especially in the traditional culture do not regard alcohol as a drug (Odejide *et al*, 1989).
It is rather seen as a substance that facilitates social communion and harmony, occupying a cultural and prominent role in our traditional culture.

It is from this later perspective that we understand the history and drinking behaviour of the kegites, the 'palm-wine drinkers club,' found in Nigeria's higher institutions of learning (Ohaeri *et al* 1996). This club is a registered socio-cultural organization which promotes African cultural heritage. It seeks to elevate the native beer, palm wine to sacred heights.

Prevalence of Alcohol Use and Misuse

In these days when scientific attention is focused on alcohol misuse in developing countries, such as Nigeria, it is easy to ignore the fact that certain aspects of the culture of alcohol consumption are held to be glamorous by the general population (Ohaeri *et al* 1996).

Elements of some of these cultural attitudes could be used as tools for primary prevention, especially among the youths whose increasing tendency to misuse alcohol has been documented in recent works (Odejide *et al*, 1987; Ihezue 1988;

Adelekan *et al*., 1992). In several studies, high rates of alcohol consumption and abuse have been found to be reported among students in post-primary institutions (Oshodin, 1981; Mbosowo, 1988; Onwuzurike, 1988). It has been found to be the most widely abused substance in Nigeria (Asuni, 1975; Odejide and Ohaeri, 1988; Obembe, 1988) and remains a widely observed medico-social problem in some parts of the country especially Benue and Plateau States (Obembe, 1988; Obot, 1993; Ohaeri and Odejide, 1993) of Nigeria. This unprecedented trend in the use and misuse of alcohol tend to have promoted and encouraged the rapid growth and proliferation of the alcohol industry, both in the rural and urban areas of the country.

Aluko-Olokun (1988), reported that between 1985 and 1987 the volume of wine and spirit produced in the country jumped from 13,545 to 189,293 hecto-litres with a revenue of ₦ 18.2 million in 1987. Ebie (1988) and Obot (1992) in their studies of prevalence rates of life time use of various psychoactive substance in the Nigeria population, documented that 0.7%, 2% and 4% respectively drank industrially brewed spirit and wine; palm wine and burukutu (Native brews) and industrial beers. Adelekan *et al* (1992) in a recent epidemiological study of a university student population found that 2.7% drank some alcohol everyday (mostly industrial beer and palm wine), while Ohaeri *et al* (1996) in a similar study found that 9% of the subjects scored 1% while 2% scored at least two with CAGE screening instrument.

In a community study at Kugiya, in Jos South, Stanley and Odejide (2002) found a prevalence of 35.2% alcohol abusers. This is primarily an agro-based community, whose natives predominantly produce burukutu. However, a similar study conducted in Port Harcourt and its environs documented a prevalence of 17% for alcohol abusers, 3% alcohol dependent, 1% for alcohol withdrawal syndrome (Stanley *et al*., 2005).

In Nigeria, beer turnover is growing faster than its economy. “At the moment, beer consumption is about 19.5 million hectoliters in 2012 and growing at about 8-9 percent per

annum," said Esili Eigbe, analyst at Stanbic IBTC, who covers the brewery sector. By this, the expected volume of consumption will be at 25.35 hectolitre by 2015. This indeed, is worrisome. More so, it has been documented that Nigeria ranks second only to the United States of America in the consumption of alcohol in the world (Vanguard Newspaper, 2014). The reality therefore calls for urgent intervention.

Chemical Structure and Names of Alcohol

An alcohol is an organic compound in which the hydroxyl functional group (-OH) is bound to a carbon atom. In particular, this carbon center should be saturated, having single bonds to three other atoms.

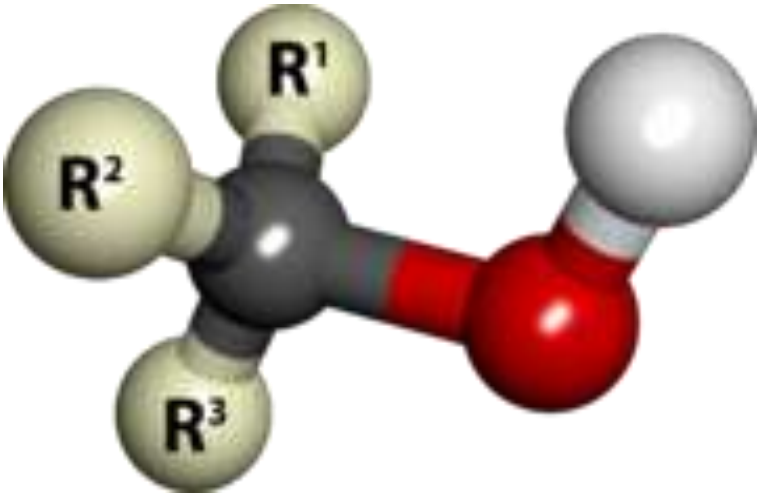


Fig 3: Ball-and Stick Model of Hydroxyl
Source: IUPAC, 2014

Ball-and-stick model of the hydroxyl (-OH) functional group in an alcohol molecule (R_3COH).

The three 'R`s' stand for carbon substituents or hydrogen atoms.

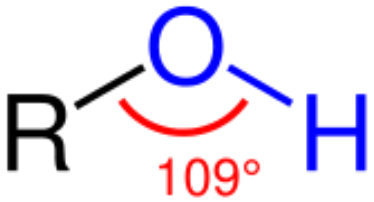


Fig 4: Compound Formula of Hydroxyl
Source: IUPAC, 2014

The hydroxyl (-OH) functional group with bond angle. An important class of alcohols are the simple acyclic alcohols, of the general formula $C_nH_{2n+1}OH$. **Of these ethanol (C_2H_5OH) is the alcohol found in alcoholic beverages.**

There are four major types of alcohols, namely monohydroxyl (primary, secondary and tertiary), dihydroxyl, trihydroxyl and tetrahydroxyl alcohols.

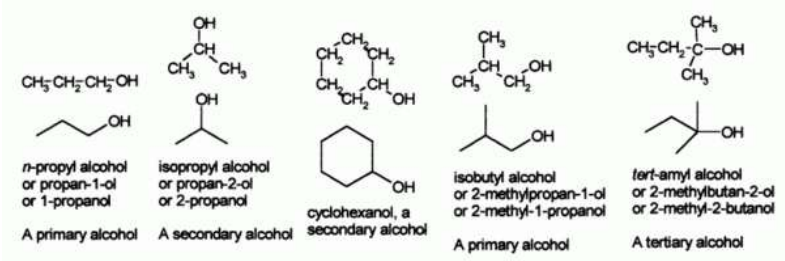


Fig 5: Chemical Compound Major Types of Alcohols
Source: IUPAC, 2014

They may be local or industrially brewed, gin or beer. Alcohols are prepared by various methods, ranging from natural to synthetic. These methods include fermentation, hydration of olefins in the presence of acid, hydrolysis of alkyl halides by water or alkali, hydrolysis of ethers in strongly acidic conditions and hydrolysis of esters (Caputto *et al*, 1967).

Table 5: Chemical formulas, IUPAC names and common names of Alcohol

Chemical Formula	IUPAC Name	Common Name
Monohydric alcohols		
CH_3OH	Methanol	Wood alcohol
C_2H_5OH	Ethanol	Alcohol
C_3H_7OH	Isopropyl alcohol	Rubbing alcohol
C_4H_9OH	Butyl alcohol	Butanol
$C_5H_{11}OH$	Pentanol	Amyl alcohol
$C_{16}H_{33}OH$	Hexadecan-1-ol	Cetyl alcohol
Polyhydric alcohols		
$C_2H_4(OH)_2$	Ethane-1,2-diol	Ethylene glycol
$C_3H_6(OH)_2$	Propane-1,2-diol	Propylene Glycol
$C_3H_5(OH)_3$	Propane-1,2,3-triol	Glycerol
$C_4H_6(OH)_4$	Butane-1,2,3,4-tetraol	Erythritol, Threitol
$C_5H_7(OH)_5$	Pentane-1,2,3,4,5-pentol	Xylitol
$C_6H_8(OH)_6$	Hexane-1,2,3,4,5,6-hexol	Mannitol, Sorbitol

$C_7H_9(OH)_7$	Heptane-1,2,3,4,5,6,7-heptol	Volemitol
Unsaturated aliphatic alcohols		
C_3H_5OH	Prop-2-ene-1-ol	Allyl alcohol
$C_{10}H_{17}OH$	3,7-Dimethylocta-2,6-dien-1-ol	Geraniol
C_3H_3OH	Prop-2-in-1-ol	Propargyl alcohol
Alicyclic alcohols		
$C_6H_6(OH)_6$	Cyclohexane-1,2,3,4,5,6-hexol	Inositol
$C_{10}H_{19}OH$	2 - (2-propyl)-5-methyl-cyclohexane-1-ol	Menthol

Source: IUPAC, 2014

Socio-demographic Factors Influencing Alcohol Use/Misuse

It is known that the onset of alcohol misuse can occur fairly early in life (Lourie, 1943; Mackay, 1961). This has been found to have severe consequences when it is not recognized on time (Mitchell *et al*, 1979).

Parents and Relatives

An association between the early onset of drinking problems and parental alcohol use has been noted by several authors, (Mackay, 1961). In his report of adolescent drinkers, he found that the family constellation was remarkably consistent in that every one of the fathers of the boys was an alcoholic and alcoholism was common among relatives in the families of the girls. Goodwin *et al*. (1972), reported possible multifactorial hereditary influences in many cases of alcohol misuse.

Multiple Family Problems

Families of alcohol misusers tend to be significantly unstable, with recurrent family disruptions, inconsistent discipline and lack of supervision. Mitchell *et al* (1979) reported that about 90% of the biological parents of children with alcohol-related problems were divorced, at the time of their study. Mackay (1961) found that, the fathers of sixteen of seventeen adolescent male drinkers, had left their houses permanently before their child reached adolescence.

Behaviour Characteristics

Several personality traits such as depression, feelings of isolation, alienation and low esteem were reported by Mitchell *et al* (1979). Similarly, Braucht *et al* (1973), in reviewing psychological correlates of deviant drug use in adolescence, found that there was often a coherent cluster of traits shown in these individuals namely, relatively low self esteem, anxiety and depression. Wechsler and Thum (1973) found that heavy drinkers in a teenage population tended to rate themselves as having more personal problem than classmates. Stacey and Davies (1970) found that youths showing delinquent behaviour were more likely to drink than youths not showing this behavior (Delinquency, rebellion and absenteeism).

Peer Relationship and School Performance

Mackay (1961); Braucht *et al* (1973), and Wechsler and Thum (1973) have in different studies reported that peer relationships and influence, were contributory to alcohol abuse. Similarly, school problems including truancy, abstenteeism and academic failure are common among children of alcoholic (Kammier, 1971). In a similar study among secondary school students in Port Harcourt to determine the correlates of alcohol abuse, it was found that alcohol abusers were overrepresented among school dropouts, those with declining academic performanceand school absenteeism (Stanley *et al*., 2005).

Family Culture

Mitchell (1979) found that alcohol was freely used in the homes of most families studied who drink alcohol, hence most parents did not see it as a problem among their children. Therefore, this lends credence to the model of drinking as a family culture (Seifer, 1973).

Guide and the Spectrum of Alcohol Use and Misuse

The quantity of alcohol use was classified by the recommendations of the Royal College of Physicians (1987). That report documented maximum safe levels of 21 and 14 units for men and women respectively, provided that the amount is not taken in one bout and provided that there are occasional drink free days. The same report has 21 to 49 units and 14 to 35 units per week as hazardous levels for men and women respectively. Levels above these are regarded as dangerous. However, in our environment, similar validity studies have not been done. Hence for the purpose of this lecture, levels above 21 and 14 units per week for men and women respectively were regarded as alcohol misuse.

Obembe *et al* (1993) documented that in Nigeria the alcohol content (per cent) of ordinary industrial beer is 3-5% and that of the local brew (Burukutu is about 7 per cent using another measure (units of alcohol, a bottle of industrial beer and an average calabash of burukutu (600ml) each contains nearly 2 units; a bottle of table wine (600ml) contains about 7 units; a bottle of spirit, about 30 units; glass of wine (60mls) contains about 2 units and a glass of gin (15ml), about 2 units, while one unit is about 8 grams of alcohol.

Table 6: Recommended Intake of Alcohol

Recommended intake	Men	Women
Safe	2-3 units/day (14-21 units/week)	1-2units/day (7-21 units/week
Hazardous	21-50 units/week	14-35 units/week
Dangerous	> 50 units/week	> 35 units/week

Source: David, 2007

Teetotal - 10% of population.

Social drinker - drinks some form of alcoholic beverage occasionally or regularly in moderation, that is, within sensible limits. 75% of those who drink come to no harm. Benefits probably outweigh hazards (ABC of Alcohol, 1994; Naik and Lawton, 1996).

Heavy drinker - drinks regularly and heavily (Men >7 units/day, Women >5 units/day).

Binge drinker - drinks irregularly and heavily.
Both of the latter two patterns will cause problems if prolonged.

Alcohol abuser ("problem drinker") - drinking has caused physical, psychological and social problems. This individual continues to drink in spite of developing difficulties, but however has not met the criteria for alcohol dependence.

Dependent or addicted drinker ('alcoholic') - has subjective awareness of compulsion to drink; exhibits prominent drink-

seeking behaviour; becomes tolerant to alcohol with obvious physical, psychological and social problems.

Liable to withdrawal symptoms following cessation or reduction in alcohol intake; uses alcohol to avoid or relieve symptoms of withdrawal

A practical definition of dependent drinking or alcoholism is persistent drinking that interferes with the person's health, legal position, interpersonal relationships, or means of livelihood.

Of UK adult population of 43 million (m) people, 36 million drink alcohol regularly. Of these, 7.3 million drink above sensible limits, 4 million known as heavy drinkers; 800 000, problem drinkers and 400 000 dependent drinkers (David, 2007). Furthermore, of 2000 persons in the United Kingdom that visit the General practitioner, 186 are heavy drinkers, 37, problem drinkers and 19 dependent drinkers. This clearly puts Alcohol misuse at 10% among the adult population.

In United States of America (USA) alcohol misuse remains the third largest health problem. It has continued to rise, while reducing life expectancy by 12 years, as it is found to be responsible for one in every 10 deaths (CDC, 2014). It poses serious problems in one million drinkers, and 500 deaths among those under 25years of age. It is associated with half a million hospital admissions, 17 000 psychiatric admissions, 80% fire deaths, 65% serious head injuries, 50% murders, 40% road traffic accidents, 30% fatal accidents, and 30% domestic accidents.

Furthermore, it contributes to 33% divorces, 33% child abuse cases and cost £1.6 billion (₦ 257.6 billion) per year to society (David, 2007).

Vice Chancellor Sir, a similar data in our environment is still uncertain due to cultural limitations and attitudes of many to alcohol research. However, the most recent National Survey on Alcohol and Drug abuse (NASAD) coordinated by me as the focal person in the south-south geopolitical zone, sponsored by World Health Organisation (WHO) and anchored by

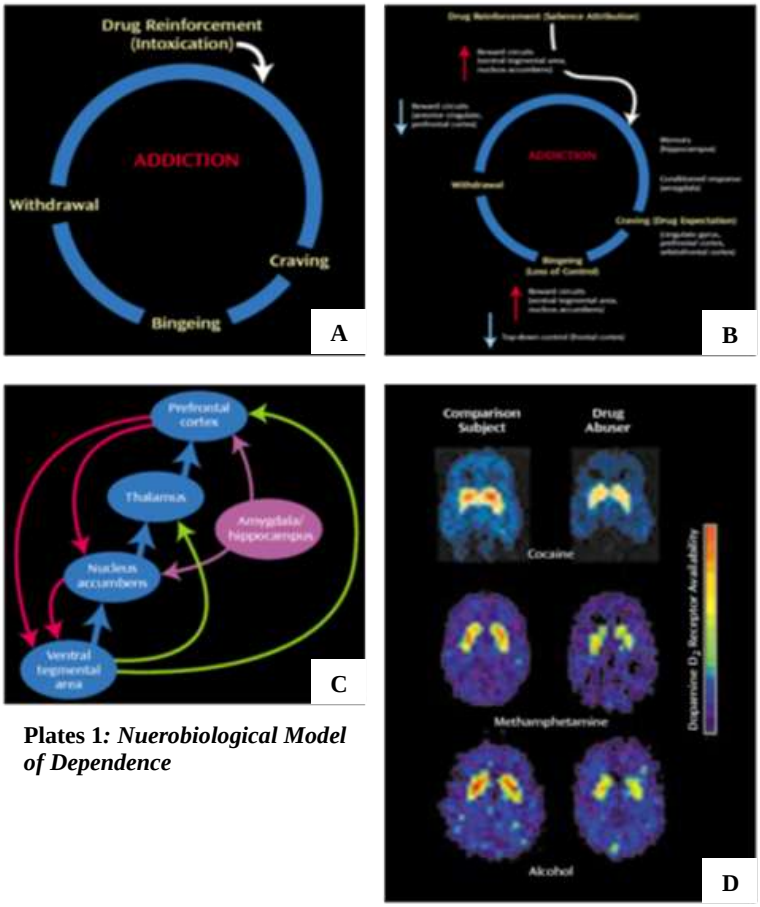
Neuropsychiatry Hospital, Aro, Abeokuta will soon make a comprehensive data available.

Neurobiological Basis of Alcohol

Tolerance, Dependence and Withdrawal

Dependence is a complex disease process of the brain that results from recurring drug intoxication and is modulated by genetic, developmental, experiential, and environmental factors. The neurobiological changes that accompany drug dependence are not well understood. While until recently it was believed that dependence predominantly involved reward processes mediated by limbic circuits (Blum *et al*., 2000), however, results from recent neuroimaging studies have implicated additional brain areas, particularly, the frontal cortex. A summary of the findings from neuroimaging studies, with the pertinent results from preclinical studies strongly help, in the formation of an integrated model of drug dependence.

Most imaging studies have concentrated on the involvement of dopamine in the process of drug dependence because the ability of drugs of abuse to increase brain dopamine concentration in limbic brain regions is considered crucial for their reinforcing effects (Koob *et al*., 1994; Di Chiara, 1999). However, the increase in dopamine only is insufficient to account for the process of dependence, since drugs of abuse increase dopamine in naive as well as in dependent subjects. The findings of several recent structural/volumetric magnetic resonance imaging studies documenting morphological changes in the frontal lobe in various forms of drug dependence, on alcohol is convincing (Jernigan *et al*., 1991a, b; Pfefferbaum *et al*., 1997), and heroin-dependent subjects (Liu *et al*., 1998).



Plates 1*: Nuerobiological Model of Dependence*

A. *Behaviour Manifestation of the t-RISA (Impaired Response Inhibition and Salience Attribution) Syndrome of Drug Addiction*
B. *Integrative Model of Brain and Behavior: the I-RISA (Impaired Response Inhibition andSalience Attribution) Syndrome of Drug Addiction*
C. *Interactions of the Mesocortical and Mesolimbic Circuits in Drug Addiction*
D. *Lower Striatal Dopamine D2 Receptor Binding in Drug Users During Withdrawal From Cocaine, Meth-amphetamine, and Alcohol Than in Normal Comparison Subjects*
Source: Goldstein and Volkow, 2002

These four components can be viewed as intricately related to the four dimensions of our drug-dependence model (Plates 1-4), each potentially predisposing to drug dependence; thus

i. Drug intoxication is associated with the experience of its strong positive and negative reinforcement effects, an association that is strengthened through repeated self administration and that possibly hinders the formation of similar associations; attribution of primary salience to the drug occurs at the expense of less powerful reinforcers.
ii. Impairment in response inhibition is believed to be an underlying factor in the experience of relapse and bingeing. When response-reinforcement regulation is down because of impaired salience attribution, response disinhibition, or impulsive responding to immediately salient, drug-related rewards is expected.
iii. Expectation of the effects of the drug of abuse, whether it is the "high" or a lower negative state, is integral to drug craving.
iv. Dysthymia is a core symptom of withdrawal, possibly reflecting adaptation responses to repeated dopamine enhancement by drugs of abuse in the reward circuits that render the latter less responsive to natural reinforcers (Cassens *et al*., 1999; Barr and Phillips, 1999; Barr *et al*., 1999). Behaviorally, this lower sensitivity in the reward circuits may represent a generalized impairment in the ability to derive pleasure from non-drug-related stimuli, leading to a state of anhedonia, which puts drug-addicted individuals at greater risk for seeking drug stimulation.

Finally, an association between depression and prefrontal abnormalities has been demonstrated in neuroimaging studies conducted in depressed patients, with suggested disruptions of frontostriatal (Robbins *et al*., 1992) and corticolimbic networks (Mayberg *et al*. 1999). Results of these studies revealed resting abnormalities in the dorsolateral, ventrolateral, and medial aspects of the prefrontal cortex and the anterior cingulate, blunted

responses in the anterior cingulate and medial prefrontal cortex to behavioral and pharmacological challenges, and abnormalities localized to the orbitofrontal cortex (Mayberg *et al*. 1999; Elliot *et al*., 1998). Lower activity in the striatum of depressed patients in the resting state and in response to a reaction-time task and feedback have also been reported (Teneback *et al*.,1999; Hickie *et al*., 1999).

Physiology of Alcohol

It is convenient to consider consumption in terms of units of alcohol.

1 unit = a standard drink = approx 8g of pure alcohol.

One unit is *commonly quoted* as being equivalent to a half pint of beer, a glass of table wine, a glass of sherry or port, a single measure of spirits.

The alcohol content is expressed on the label as % alcohol by volume (%ABV). 4% by volume = 4 mls alcohol per 100 mls of solution.

Alcohol content can also be expressed as % weight/weight (4g/100g), % weight/volume (4g/100ml) or % volume/weight (4ml/100g). These are not the same: specific gravity of pure ethanol = 0.79 that is 1 ml ethanol weighs 0.79g.

1 pint = 570 mls = 20 fl.oz. = 4 gills

Beer: a half pint of 4.5% beer = 284 ml x 0.045 = 12.78 ml of pure alcohol

= 12.78 x 0.79 = 10.1 g alcohol. Therefore 1 pint = 20 g = 2.5 units.

Original gravity (OG) is 1000 x the Specific Gravity of the wort before fermentation. SG depends on sugar content. The 3rd and 4th figures, divided by 10 give the approximate alcohol content:

1040 = 4%,

1085 = 8.5%

Wine: 125 ml glass of 12 % wine = 15 ml of alcohol = 15 x 0.79 = 11.85 g (1.5U)

Fortified wine (sherry, port), liqueurs are 20% v/v. (60 ml glass x 0.2 = 12 ml = 9.5g)

Spirits: Whisky: 43% v/v. British Proof value is 1.75 x v/v. US Proof vaue is 2 x v/v!

United Kingdom measure is now usually 25 ml: 25 x 0.43 = 10.75 ml alcohol = 8.5g

The flavour and colour of alcoholic beverages is largely a function of congeners which are higher alcohols and aldehydes.

Absorption of Alcohol

Ethanol is a weakly charged molecule that moves easily through cell membranes, rapidly equilibrating between blood and tissues. The effects of drinking depend in part on the amount of ethanol consumed per unit of body weight; the level of alcohol in the blood is expressed as milligrams or grams of ethanol per deciliter (that is, 100mL or o.1000g/dL). Congeners found in alcohol beverages may contribute to body damage with heavy and progressive drinking. These include low-molecular-weight alcohols (for example methanol and butanol), aldehydes, esters, histamine, phenols, tannins, iron, lead, and cobalt.

Ethanol is a central nervous system (CNS) depressant that decreases activity of neurons, although some behavioural stimulation is observed at low blood levels. This drug has cross-tolerance and share a similar pattern of behavioural problems with other brain depressants, including the benzodiazepines, barbiturates, and other sedatives and hypnotics. Alcohol is absorbed from mucus membranes of the mouth and esophagus (in very small amounts), from the stomach (20%) and large bowel (in modest amount), and from the proximal portion of the small

intestine (80%) (the major site). The rate of absorption increases with rapid gastric emptying; the absence of proteins, fats, or carbohydrates (which interfere with absorption); the absence of congeners; dilution to a modest percentage of ethanol (maximum absorption is seen at about 20 percent by volume); and carbonation (champagne).

Blood Alcohol Concentration (BAC); Urinary Alcohol Concentration (UAC); Vitreous Humour Alcohol Concentration (VHAC); Breath Alcohol Concentration (BrAC).

Absorption is most rapid when the stomach is empty and accelerated gastric emptying also quickens the rate of alcohol absorption due to rapid passage of alcohol into the small intestine where absorption is more rapid. Gastric emptying accelerated by tolerance (habituation) to alcohol, operative reduction in the stomach size (e.g. gastrectomy), and decreased by food in stomach, certain drugs and the type of beverage consumed. Different types of beverages have varying alcohol concentrations and congeners, and these could affect emotional state, drugs, and food absorption. Moreso, there is considerable intra and inter-individual variation in the rate of absorption.

The Peak BAC reached when alcohol is taken with a meal may only reach 50% of that reached when alcohol is taken on an empty stomach. In the presence of food in the stomach up to 20% of ingested alcohol may be oxidized before it can be absorbed. Absorption is by passive diffusion and proceeds as long as the alcohol concentration in gastrointestinal tract (stomach and duodenum) exceeds that in the blood.

Absorption is most rapid when the alcohol concentration in the stomach is 10%-20% (fortified wine, beer & 'chaser'). Higher concentrations of alcohol (neat spirit) irritate the gastric mucosa, causing increased secretion of mucus and delay in gastric emptying and absorption. Absorption is more rapid with carbonated drinks (champagne). Alcohol in beer is absorbed

more slowly (low alcohol concentration, carbohydrate rich). If alcohol is taken slowly it can be eliminated as fast as it is being absorbed, hence, BAC will not rise any further. Absorption is generally complete in one to three hours (David, 2007).

Distribution and Equilibrium

Once absorbed, alcohol dissolves in the blood and is distributed by the blood stream to the tissues. Alcohol becomes distributed in the blood and water of the body. Tissues rich in water (muscle) take up more alcohol from the blood than those rich in fat. The amount of water available for alcohol to distribute into depends on body weight and build. A large body weight offers a larger volume for alcohol to be distributed into.

(Concentration of alcohol in the blood = Amount of alcohol consumed / Volume of water in the body)

A lean person has a greater muscle bulk which provides a larger volume of distribution for the alcohol than an obese counterpart of similar weight. This is because adipose tissue (fat) has a poor blood supply and alcohol is water-soluble and not fat-soluble. Women on average have a smaller body mass than men. They also have a higher proportion of body fat. As a result of these 2 factors women have a lesser volume of water in the body (or lean body mass) into which the alcohol can distribute. Because of these two factors, women usually achieve a higher BAC than men do after drinking the same amount of alcohol. It is commonly assumed that 68% of male body weight and 55% of female body weight is available for distribution of alcohol.

Average Widmark Factor for males = 0.68.

Average Widmark Factor for females = 0.55.

Mathematical equations and charts exist to enable estimation of a more individual WF based on height and weight.

Equilibrium between the tissues and blood is obtained within one to two hours.

The blood alcohol concentration (BAC) at any time is determined by many factors apart from the quantity ingested.

Rule of thumb: *One unit of alcohol will elevate the blood alcohol concentration within the first hour by 15 mg per 100 ml in a man and by 20 mg per 100 ml in a woman.*

The Widmark equation gives a rough estimate of peak BAC expected following ingestion of a known amount of alcohol. Co is a theoretical value which assumes 100% absorption and instantaneous distribution, which is never the case. In practice, the peak BAC after food is often less than 2/3 of this theoretical Co, since absorption is incomplete in the presence of food. Values of Co can also be extrapolated back from the linear elimination phase of experimental Blood Alcohol.

Concentration – Time curves

The Widmark Factor (WF) is an estimate of body water content. The mean experimental values are 0.68 for men and 0.55 for women. After one unit (8g alcohol), for Example:

i. Average 70 kg male, Co = (8 x 100)¸ (70 x 0.68) = 16.8 mg%
ii. Obese 90 kg male. Co = (8 x 100)¸ (90 x 0.6) = 14.8 mg%
iii. Lean 80 kg male Co = (8 x 100)¸ (80 x 0.72) = 13.8 mg%
iv. Average 60 kg female Co = (8 x 100)¸ (60 x 0.55) = 24.2 mg%
v. Obese 75 kg female Co = (8 x 100)¸ (75 x 0.5) = 21.3 mg%
vi. Lean 55 kg female Co = (8 x 100)¸ (55 x 0.6) = 24.2 mg%

WF x Body Weight (in kg) is the Lean Body Mass, which is equivalent to the volume of water into which the consumed alcohol is distributed. Individuals with a large lean body mass seem to have "hollow legs", particularly if they drink regularly and therefore also eliminate alcohol more rapidly. Since the number of units of alcohol (a unit being 8 g) is stated on the

bottle/can, there is an even simpler method of estimating BAC (Co): (using WF obtained from Barbour's chart). The individual's own rate of elimination is rarely known (unless 2 or more measurements of BAC or BrAC have been made). The average rate of 15 mg/100 ml/h is often used, although for legal purposes, a range of values calculated upon elimination rates of up to 20 mg/100 ml/h are often quoted. It is often of medico-legal interest to determine the BAC at a material time (T hours) after the start of drinking.

The BAC at time T hours is designated as Ct: (where ß is the rate of elimination (mg alcohol / 100 ml blood / h) and T is the time elapsed since the start of drinking (in hours). Put simply, C0 is the starting level of alcohol and (ß x T) is the amount of alcohol eliminated during the time interval in question. Calculation of the BAC or BrAC which likely existed at an earlier time is called “back estimation” or “back calculation”. The principal source of error is not knowing the individual's own rate of elimination.

The Widmark equation can be rearranged to work out the amount of alcoholconsumed in order to account for the measured BAC or BrAC: The air in the terminal air sacs of the lungs (alveoli) is in intimate contact with the blood in the capillaries which bathe the alveoli.

Therefore in theory, a constant breath to blood ratio of alcohol content should exist, and this is accepted as 1:2,300 (breath to blood ratio). This is the basis of breathalyser test. Breath alcohol concentrations (BrAC) rise faster and fall earlier than venous blood levels. Thus the level in blood (BAC in milligrams alcohol /100 ml blood) is 2300 times higher than the level in Breath (BrAC in micrograms /100 ml breath). Note the much smaller units used in expressing BrAC. 1 milligram (mg) = 1000 micrograms (µg). The conversion is simple in practice: BAC = 2.3 x BrAC and BrAC = BAC / 2.3.

Alcohol also enters the eye fluid (vitreous humour). At equilibrium the ratio of BAC to VHAC is 0.81. However, this ratio depends on whether the alcohol curve is at the absorption or

elimination phase. During the absorption phase, (before equilibrium is attained) BAC rises faster than VHAC. The blood to vitreous ratio under these circumstances is approximately 1.07 during the elimination phase (sobering up phase), the BAC drops slowly, and the VHAC is able to keep pace and remain in equilibrium. The blood to vitreous ratio under these circumstances is approximately 0.81

Estimating the BAC from a known VHAC has been attempted in cases where blood is not available at autopsy. This calculation has recently been shown to be unscientific (Pounder & Kuroda, 1994). Alcohol will be present in the urine which is formed by the kidneys. As urine contains a large proportion of water and very little solid material, urine contains more alcohol per 100 ml than does blood.

At equilibrium ureteric UAC: BAC = 1.3:1 (4:3). When alcohol begins to filter into the urine this new ureteric urine mixes with urine already present in the bladder. The original urine may have a lower alcohol level which will dilute the excreted alcohol. The best estimate of Urinary Alcohol Concentration (UAC) is obtained after emptying the bladder and then testing the next smallest amount of urine which can be naturally voided. This usually represents some 20 minutes of current excretion by the kidneys.

BAC changes constantly and the UAC in the bladder is the average of several hours excretion. It is usual for results to be quoted either as UAC or BAC. It is unreliable to extrapolate one result from the other (Kuroda *et al*, 1995).

Conversions:
BrAC = BAC / 2.3 (80/2.3 = 35)
BAC = BrAC x 2.3 (35 x 2.3 = 80)
UAC = BAC / 0.75 (80 / 0.75 = 107)
BAC = UAC x 0.75 (107 x 0.75 = 80)
VHAC = BAC / 0.8 (80 / 0.8 = 100)
BAC = VHAC x 0.8 (100 x 0.8 = 80)
Blood to Breath Ratio (Q) = BAC / BrAC

UK law uses value of 2300:1 (value of 2.3 takes care of different units as BAC is expressed in milligrams/100 ml and BrAC is expressed in micrograms/100 ml).
However, Arterial BAC rises & fall earlier than Venous BAC (which lags behind)
Arterial BAC correlates closely with BrAC. About constant at 2251:1 Venous BAC / BrAC depends on time since start of drinking

<30 mins = 1800 – 2000 (ABAC & BrAC > VBAC)
60-120 mins = 2100 – 2300 (ABAC = BrAC = VBAC)
Post absorption = 2400 – 3000 (ABAC & BrAC < VBAC)
Other jurisdictions use different values of Q (Austria = 2000)

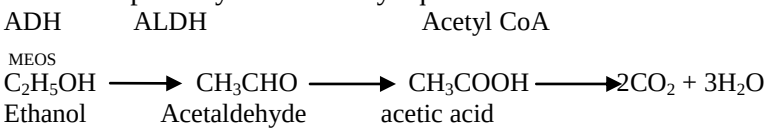
Metabolism

Between 2 percent (at low blood alcohol concentrations) and about 10 percent (at high blood alcohol concentrations) of ethanol is excreted directly through the lungs, urine, or sweat, but the greater part is metabolized to acetaldehyde in the liver. At least two metabolic routes, each with different optimal concentrations of ethanol (K_m), result in the metabolism of approximately one drink per hour. The first and clinically most important pathway occurs in the cell cytosol via alcohol dehydrogenase (ADH) with a K_m of about 2 mmol. This reaction produces acetaldehyde which is then rapidly destroyed by aldehyde dehydrogenase (ALDH) in the cytosol and mitochondria. Each of these steps requires nicotinamide adenine dinucleotide (NAD) as a cofactor, and it is the increased ratio of the reduced cofactor (NADH) to NAD (NADH:NAD) that is responsible for many of the metabolic derangements observed after drinking. Second, microsomes of the smooth endoplasmic reticulum (the microsomal ethanol-oxidizing system or MEOS) with a K_m of about 10 mmol may be responsible for 10 percent or more of ethanol oxidation at high blood alcohol concentrations. Increased activity of this system can be induced after repeated exposure to ethanol (Schuckit, 1991).

All pathways result in the production of acetaldehyde, which is oxidized to acetate. The specific clinical significance of acetaldehyde is not fully known, but low levels of this substance may cause stimulation and behavioural reinforcement. Accumulation of higher levels in liver, brain, or other body tissues may cause organ damage.

Alcohol cannot be stored and obligatory oxidation must take place predominantly in the liver. The metabolism of ethanol is associated with multiple toxic consequences and is currently considered to be the major route through which alcoholic liver disease is produced. Three enzymes systems in the liver are involved. These are peroxisomal cytosolic enzyme-alcohol dehydrogenase (ADH) which is a major route of alcohol breakdown in non alcoholic man and the microsomal ethanol oxidizing system (MEOS). In the chronic alcoholic, the ADH activity decreases, a finding in keeping with the observation that alcoholics may display decreased hepatic ADH activity even in the absence of liver disease (Leevy *et al* 1975). Following chronic ethanol consumption MEOS significantly increases in activity supplementing the cytosolic enzymes.

Metabolic pathway illustrated by equation below:



Elimination of Alcohol

Alcohol is eliminated through all bodily routes of excretion. 5% is excreted in the breath; 5% in the urine. Negligible amounts of alcohol are eliminated in sweat and faeces under normal circumstances. 90% broken down in the body, mostly in the liver, by liver enzymes including hepatic alcohol dehydrogenase (AlcDH). Oxidation of the products (acetaldehyde and acetic acid) finally yields carbon dioxide and water. A small amount is metabolised by microsomal enzyme oxidising system (MEOS), especially in alcoholics whose enzyme levels are induced by chronic abuse.

Ethanol is converted to Acetaldehyde by the enzyme Alcohol Dehydrogenase (AlcDH) Acetaldehyde is converted to Acetate by the enzyme Aldehyde Dehydrogenase (AldDH) In tolerant drinkers, other enzyme systems are activated to help cope with the workload (the Microsomal Enzyme Oxidase System (MEOS) and Catalase).

Acetate is Converted to Carbon dioxide and Water

In experiments and police situations, individual elimination rates can sometimes be calculated from the drop in BAC or BrAC which occurs between two points in time. In a healthy person, the rate of clearance of alcohol from the blood (ß) by liver is 15 mg alcohol per 100ml blood per hour (the equivalent of one unit per hour). However, the range is from 10-40 mg per 100ml per hour. Convicted drunk drivers average 20 mg/100ml/hour. Genetic and racial differences exist. Liver disease usual reduces metabolism (DiCecco and Francisco-Ziller, 2006).

Elimination of alcohol from breath has been less extensively studied. The mathematically predicted level (equivalent to 15 in blood) is 15/2.3 = 6.52 ug/100 ml/h. Experiments on student volunteers in Dundee gave an average value of 5.8 ug/100 ml/h (range 4-9).

Following consumption of a single alcoholic drink, the combined effects of different factors affecting absorption, metabolism and excretion, result in a characteristic blood alcohol curve:

i. The alcohol concentration rises steeply to a distinct maximum (absorption phase).
ii. There then follows an irregularly curved fall due to a period of diffusion within the tissues to equilibrium. This takes place over 15 to 30 minutes. The peak concentration is reached 45 to 90 minutes after ingestion, the majority after 60 minutes.
iii. The BAC then falls progressively in a linear fashion (elimination phase).At very high levels (> 200 mg%) the decrease is not linear due to greater loss in breath and urine.

Over 12 hours are required to eliminate 200 mg%. An individual may still be over the legal limit for driving at, 8hour later.

The Blood Alcohol Curve: The height of the peak BAC, the time taken to reach the peak and the shape of the curve depend depend on numerous factors: sex, size, build, tolerance, amount and type of beverage taken, duration of drinking, presence of food, type of food.

Consequences of Alcohol Misuse

a. Vice Chancellor Sir, permit me to mention that the consequences are 3 fold, namely; psychological, socio-economic and physical consequences.

Let us now undertake a painstaking academic audit into these listed consequences

1. Psychological Consequences

Co-morbidity of alcohol misuse and other psychiatric syndromes are commonly, present in over two-thirds of dependents. The reasons for misuse often provide a clue to underlying psychopathology, most commonly depression and anxiety. The dependent or addicted drinker (alcoholic) has subjective awareness of compulsion to drink; exhibits prominent drink-seeking behaviour; becomes tolerant to alcohol and develops obvious physical, psychological and social problems, and primacy of alcohol over important issues of life.

Alcohol Intoxication

The DSM-IV-TR Diagnostic Criteria for alcohol intoxication are based on evidence of recent ingestion of ethanol, maladaptive behaviour, and at least one of six possible physiological correlates of intoxication (Table 7). The tenth revision of the International Statistical Classification of Disease and Related Health problems (ICD-10) criteria for acute alcohol intoxication are generally similar to DSM-IV-TR, listing seven physiological

signs of intoxication, some of which, such as conjunctival injection, are not seen in DSM-IV-TR.

Table 7: Impairment likely to be seen at different Blood Alcohol Concentration

Level	Likely Impairment
20-30mg/dL	Slowed motor performance and decreased thinking ability
30-80mg/dL	Increases in motor and cognitive problems
80-200mg/dL	Increases in incoordination and judgment errors Mood lability Deterioration in cognition
200-300mg/dL	Nystagmus, marked slurring of speech, and alcoholic blackouts
> 300mg/dL	Impaired vital signs and possible death

Source: Saddock and Saddock, 2007

Alcohol Withdrawal

Alcohol withdrawal, even without delirium, can be serious; it can include seizures and autonomic hyperactivity. Conditions that may predispose to, or aggravate, withdrawal symptoms include fatigue, malnutrition, physical illness, and depression. The DSM-IV-TR criteria for alcohol withdrawal (Table 8). Require the cessation or reduction of alcohol use that was heavy and prolonged as well as the presence of specific physical or neuropsychiatric symptoms. The diagnosis also allows for the specification "with perceptual disturbance." One recent positron emission tomographic (PET) study of blood flow during alcohol withdrawal in otherwise healthy persons with alcohol dependence reported a globally low rate of metabolic activity, although, with further inspection of the data, the authors concluded that activity was especially low in the left parietal and right frontal areas.

The classic sign of alcohol withdrawal is tremulousness, although the spectrum of symptoms can expand to include

psychotic and perceptual symptoms (for example, delusion and hallucination), seizures, and the symptoms of delirium tremens (DTs), called Alcohol withdrawal delirium in DSM-IV-TR. Tremulousness (commonly called the "Shakes" or the "Jilter") develops 6 to 8 hours after the cessation of drinking, the psychotic and perceptual symptoms begin in 8 to 12 hours, seizures in 12 hours to 24 hours, and DTs during 72 hours, although physicians should watch for the development of DTs for the first week of withdrawal. The syndrome of withdrawal sometimes skips the usual progression and, for example goes directly to DTs.

Table 8: DSM-IV-TR Diagnostic Criteria for Alcohol Withdrawal

A. Cessation Of (Or Reduction In) Alcohol Use That Has Been Heavy And Prolonged
B. Two (Or More) Of The Following, Developing Within Several Hours To A Few Days After Criterion A:
(1) Autonomic Hyperactivity (For Example, Sweating Or Pulse Rate Greater Than 100)
(2) Increased Hand Tremor
(3) Insomnia
(4) Nausea Or Vomiting
(5) Transient Visual, Tactile, Or Auditory Hallucinations Or Illusions
(6) Psychomotor Agitation
(7) Anxiety
(8) Grand Mal Seizures
C. The Symptoms In Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The symptoms are not due to a general medical condition and are not better accounted for by another mental disorder.
Specify if:
With perceptual disturbances

Source: Saddock and Saddock, 2007

Table 9: Drug Therapy for Alcohol Intoxication and Withdrawal

Clinical Problem	Drug	Route	Dosage	Comment
Tremulousness and mild to moderate agitation	Chlordiazepoxide	Oral	25-100mg every 4-6hr	Initial dose can be repeated every 2 hrs until patient is calm; subsequent doses must be individualized and titrated
	Diazepam	Oral	5-20mg every 4-6hr	
Hallucinosis	Lorazepam	Oral	2-10mg every 4-6hr	
Extreme agitation	Chlordiazepoxide	Intravenous	0.5mg/kg at 12.5mg/min	Give until patient is calm; subsequent doses must be individualized and titrated
Withdrawal Seizures	Diazepam	Intravenous	0.15mg/kg at 2.5mg/min	
Delirium tremens	Lorazepam	Intravenous	0.1mg/kg at 2.0mg/min	

Source: Saddock and Saddock, 2007

Limbic system: Depression of the limbic functions causes loss of memory, confusion and disorientation, and of cerebellum, loss of muscular co-ordination and speech, leading to in-coordination and slurring (Royal College of Physicians, 1987).

Reticular formation: Its interference with the functions leads to impairment of consciousness, with eventual stupor and or coma.

Lower brain stem: Furthermore, vital centres controlling breathing (respiratory centre) and blood pressure (vasomotor centre), may be severely impaired, leading to respiratory arrest and automatic instability.

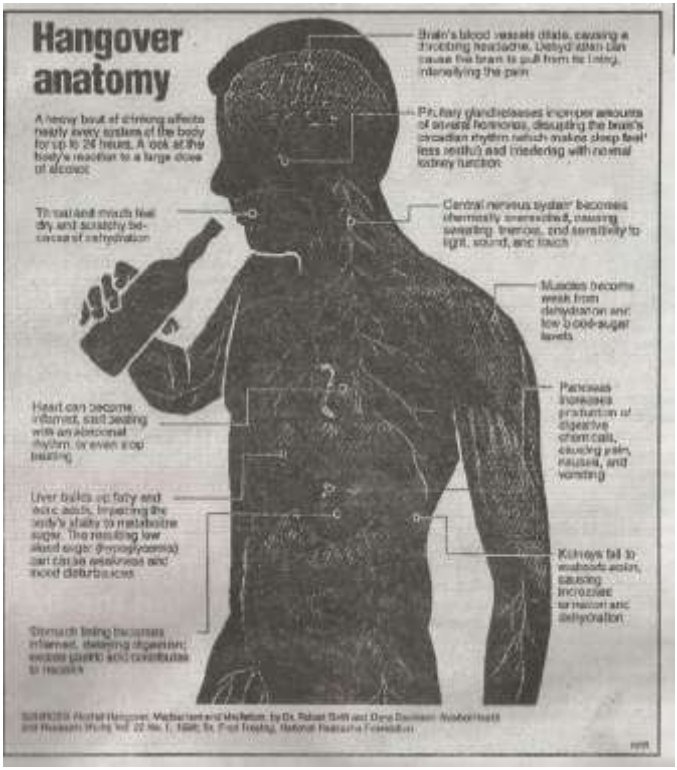


Plate 2: Picture showing the Anatomy of Hangover

Source: The Nation, Saturday, July 5, 2014

Plate 3*: A Woman in coma following Alcohol Intoxication.*
Source: www.youritablets.com, 2014

Plate 4: Showing Death from excessive alcohol drinking (intoxication)
Source: The Nation, Saturday, July 5, 2014

Plate 5: Brands of Alcohol (A) Spirit (Gin) and Wine ; (B) Beers **Source:** *www.lionco.com*, **2014**

Perceptual disturbance

i. Alcoholic hallucinosis (Perceptual deceptions)

These are auditory hallucinations, often in the third person, characteristically derogatory or command, that occur in clear consciousness. They may also take the form of fragments of conversation or music and there may be secondary delusions or perseveration. The symptoms may be associated with a reduction in dose or precipitation of withdrawal and hallucinosis must be differentiated from delirium tremens, during which hallucinatory experiences particularly visual occur in confusional states.

ii. Perceptual distortions-illusions.

Mood Disorders

Up to 70 percent of alcoholics complain of dysphoria during heavy drinking, with depression occurring in about 20 percent. 80-90 per cent of depression was formerly thought to be secondary to alcohol use with the diagnosis of alcohol misuse antedating that of mood disorder. However, recent large epidemiological studies suggested that a diagnosis of depression and or anxiety was likely to precede, the diagnosis of alcohol dependence. Certainly the incidence of depressive symptoms falls with abstinence, but careful assessment is required and treatment should be instituted if a primary diagnosis of depression is made, since if untreated, may precipitate relapse. Alcohol misuse may interfere with treatment efficacy and

compliance, and consumption may rise during a manic episode in some with Bipolar Affective Disorder.

Depression may occur as a direct result of the pharmacological effects of alcohol as well as in response to the complications of alcohol misuse. Reasons for the association between alcohol dependence and depression may include common genetic factors especially in women, shared family environment, poverty, social isolation and unemployment or both conditions may develop as secondary features of other disorders such as personality disorder, polysubstance abuse and Briquet's syndrome (Appleby *et al*., 2001).

Significant percentage of adolescent with alcohol abuse/dependence have been found to be suffering from anxiety and depression (McGee *et al*., 1992; Deykin *et al*., 1987; Stanley *et al*., 2005). With a higher sex predilection for females (Lowinsohn, 1995; Windle, 1996). According to the 'anxiety reduction' theory, alcoholics self-medicate what otherwise might be disabling anxiety. One review concludes that 'anxiety traits do not appear to be an important causal factor in alcoholism.' However, in combination with learning theory it may be regarded as one route to alcohol abuse. Other anxiety disorders such as social phobia also predispose to alcohol abuse, although, as with anxiety, often alcohol is the cause of the problem, with prolonged abstinence often leading to symptom resolution.

This has often been associated with antisocial personality (psychopaths) type, who are known to be selfish, cruel to human and animals, violent and enjoy seeing others suffer the pains of their misdeeds (Stanley, 1997). Furthermore, Cloninger *et al*.(1988) identified two subgroups, type 1 and 2.

Table 10: Subtypes of Alcohol Misuse

	Type 1 (milieu limited)	**Type 2 (male limited)**
Drinking Pattern	Loss of control with high psychological dependency and guilt	Less likely to achieve abstinence
Sex	M and F	M
Family History	Parents mild/non-abusers	High geneticcomponent for alcohol and antisocial behaviour
Age of onset	Usually > 25 years	Usually <25 years
Violence and Crime	No association	Increased
Personality	Traits of dependence and harm avoidance	Impulsive and antisocial traits.

Source: Cloninger et al. 1998

Major psychotic disorders such as mood disorders with psychotic features (Cubson and Becker, 1973; Woodniff *et al*, 1973) and schizophreniform psychosis, alcohol related hallucinosis and delusional disorders, (Hershon, 1977) have been reported, among alcohol abusers.

Pathological jealousy (Othello syndrome)
This is characterized by the abnormal belief of (delusion or overvalued idea) of infidelity of the sexual partner and is twice as common in men than women with alcohol related problems (Appleby *et al*., 2001).

Eating disorders
Up to 30 per cent of young women with serious drinking problem have significant eating disorder, while among those attending

eating disorder clinics heavy drinking is overrepresented (Appleby *et al*, 2001).

Sleeping distortion: This alters the pattern, quality and stages of sleep adversely. Polysomnographic studies have shown evidence of decreased latency of Rapid Eye Movement (REM) sleep among alcohol abusers. Unfortunately, sleep disorders often precede various types of mental disorders.

Other drug use
Dependence upon another drug is the commonest co-morbid disorder in some with an existing dependence syndrome on Alcohol. Many alcoholics abuse other drugs, such as benzodiazepines, cannabis, amphetamine, and cigarettes. Hence, the name Gate-way drug (Obot, 1991; Gelder *et al*, 2006).

2. Socio-economic Consequences

i. Decline in occupational functioning, productivity and job performance (Stanley and Odejide, 2002).
ii. Disinhibition and poor sense of judgment, which prone to lack of self control, diligence, moral conscience, and breakdown of boundaries of acceptable conduct, norms, and behaviour in a given societal setting. This often leads to incestuous behaviour, child and or spousal abuse.
iii. Disruption of family dynamics: loss of normal family function which may lead to separation, divorce, mood disorders and poverty (Stanley and Odejide, 2002; Stanley *et al*., 2005)
iv. School dropout, and poor school records, unemployment and job loss.

Drinking and driving
Over three-quarters of fatal road traffic accidents involves alcohol misuse. The peak age for drink-driving convictions is 21 years, with one-third of convictions in those aged 25 or under. The legal limit is 80mg/100ml. with urinary concentration 1.3 times that of blood.

A survey in 2009, conducted among 2,230 commercial drivers in 6 major Motor Parks in Port Harcourt and Obio-Akpor Local Government Areas of Rivers State, using breathalyzers, on the pattern of alcohol use among commercial drivers in Port

Harcourt and its environs with the physiology students of the university of Port Harcourt, found that 65% of these drivers had Alcohol levels significantly higher than the legally acceptable limits for driving, 13 percent had quite less, and the other 12 percent had no detectable alcohol levels. Of the 65%, 45% had been involved in various degrees of fatal accidentals in the past few weeks leading to various degrees of injuries, deaths and hopitalisation (Stanley *et al*., 2009).

Stanley *et al*. (2014) a survey of the pattern of substance of abuse among patients admitted into the drug unit of the University of Port Harcourt Teaching Hospital (UPTH) from 2010-2013.

Violent Behaviour, Suicide and Crime

This is simply defined as a behaviour that is intended to hurt other people usually physically (Stanley and Jumbo, 2004). Stanley and Odejide (2002) in a study conducted in Jos, found that though Alcohol misuse has been highly associated with unrests and violent crimes, there was no an associated case of suicide. The higher incidence of crime reported in our study, agrees with the findings of the Royal College of Physicians (1987) and Chiswick (1993) except for suicide cases. This was attributable to a possible social norm in most African societies which tends to ostracize members of the family of a suicide victim. This serves as a deterrent and may obviously lead to concealment of such acts even when they occur (Stanley and Odejide, 2002). However, in a similar study by Stanley and Nwosu (2004) among militant youths of the Niger Delta who engaged in Alcohol misuse, high rates of suicide (3%) was reported.

About a quarter of alcoholics attempt suicide, with a rate that is four-fold increased in men and 20-fold in women. Recent studies suggest a lifetime risk of suicide in alcoholics of 2-4 per cent (down from earlier reports of levels of 10-15 per cent) (Appleby *et al*, 2001).

About two-thirds of male and one-fifth of female prisoners have serious drinking problems. About 70 per cent of habitual community criminals have drinking problems, with legal problems most commonly for affray, driving offences and theft.

Alcohol is also implicated in rapists (40-70 per cent), paedophile offences and in over half of both victims and perpetrators of violence (Royal College of Physicians, 1987; Wilkins, 1974; Department of Environment, 1976; Edwards *et al*., 1971; Dunbar *et al*, 1985; Harvard, 1975).

i. Physical Consequences

Gastrointestinal System

Esophagus and stomach

Acute alcoholic intake can result in inflammation of the esophagus (possibly secondary to reflux of gastric contents) and stomach (resulting from damage to gastric mucosal barrier). Esophagitis can cause epigastric distress, and gastritis, the most frequent cause of gastrointestinal bleeding for heavy drinkers, can present with anorexia and abdominal pain. Chronic heavy drinking, if associated with violent vomiting, can produce a longitudinal tear in the mucosa at the gastroesophageal junction-a Mallory-Weiss lesion. Although many gastrointestinal problems are reversible, two complications of chronic alcoholism, esophageal varices secondary to cirrhosis-induced portal hypertension and atrophy of gastric cells, may be irreversible (Schuckit, 1991).

Small bowel

The greater part of the ethanol is absorbed from proximal small bowel, where it may interfere with absorption of the B vitamins and other nutrients. Acutely, ethanol can cause hemorrhagic lesions of the duodenal villi and diarrhea secondary to increased small-bowel motility and decreased water and electrolyte absorption. Chronic alcoholism can contribute to diarrhea through its effects on the pancreas.

Pancreas

Alcoholics commonly develop acute or chronic pancreatitis. This may lead to type 2 diabetic mellitus due to damage of the Islets of Langergans, while increasing the risk of pancreatic cancer

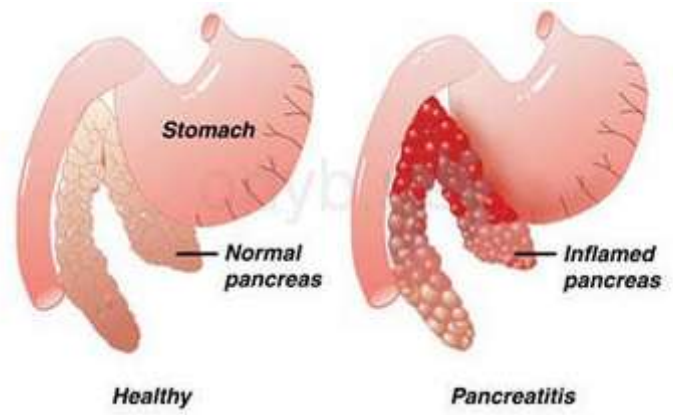


Plate 6: Comparing a normal and inflamed pancreas

Liver

Ethanol absorbed from the small bowel is carried directly to the liver, where it becomes the preferred fuel; NADH accumulates and oxygen utilization escalates, gluconeogenesis is impaired (with a resulting fall in the amount of glucose produced from glucogen), lactate production increases, and there is a decreased oxidation of fatty acids in the citric cycle with an increase in fat accumulation within liver cells. In the healthy individual taking no medications these changes are reversible, but with repeated exposure to ethanol more severe changes in liver functioning are likely to occur. These include, in overlapping stages, fatty accumulation, alcohol-induced hepatitis, and cirrhosis (Okeke, 1991).

Increased cancer risk

Cancer is the second leading cause of death in alcoholics (after cardiovascular disease), who have a rate of carcinoma 10 times higher than that expected in the general population. The sites with the greatest increase over expected rates include the head and neck, esophagus, cardia of the stomach , liver, pancreas, and, according to recent data, breast (Stanley, 1997).

Liver

Acetyldehyde the major breakdown product of alcohol is hepatotoxic. It causes shortening and thickening of the microtubules resulting in impairment of microtubular mediated protein secretion and hepatocellular transport of biliary lipids with hepatic accumulation of lipids and protein resulting in hepatomegaly. Acetaldehyde was recently shown to directly stimulate collagen synthesis by isolating hepatic myofibroblasts. Also the excess NADH produced in the course of alcohol metabolism via the ADH pathway upsets the redox balance of the cells and interferes with the intercellular metabolism of fats, carbohydrates and protein (Leevy *et al*., 1975).

Oxidation by MEOS requires oxygen and thus potentiates anoxic liver injury especially in the zone of hepatocytes near the terminal hepatic venule. The earlier hepatic lesion noticed is alcoholic steatosis (fatty metamorphosis). The liver size is greatly enlarged since every cell is laden with fat. This accumulation of fat is not usually of major pathologic consequence since it may persist without progression or may disappear despite continued intake of alcohol.

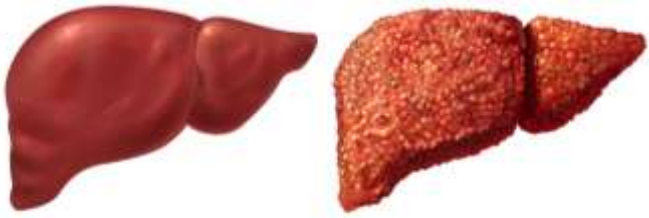
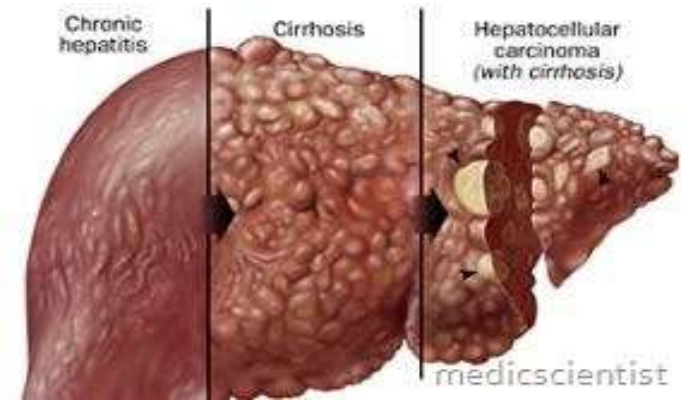


Plate 7*: A: A Normal liver and Liver with Cirrhosis*



B: Disease progression in the Liver following prolonged misuse of alcohol. *Source: medicscientist.com, 2014*

Alcohol steatonecrosis (alcoholic hepatitis) is the key morphologic lesion in alcoholics (Leevy *et al*. 1975). Eighty percent (80%) of patients with the lesion who continue to imbibe alcohol, develop cirrhosis. It is characterized by accumulation of alcoholic hyaline prominent in the central area of the hepatic lobule with polymorphonuclear leukocyte infiltration (Leevy *et al*. 1975). It may progress in some people despite abstinence and correction of dietary deficiency. In some people alcoholic metabolism may directly stimulate fibrogenesis and cause cirrhosis without antecedent alcohol steatonecrosis (Wicks *et al*, 1997; Bell and Nordhagen, 1978).

Four prominent stages in Alcoholic disease and history

1. Fatty liver
2. Alcoholic hepatitis
3. Alcoholic cirrhosis (in 10-20 per cent)
4. Primary liver cell carcinoma (Hepatoma)

Okeke (1991) reported unusually high patterns of cirrhosis of the liver among adult Jos population who were exposed to chronic alcohol misuse. Stanley (1997) reported significant reduction in the liver span of 21% of alcohol abusers in Jos. Furthermore,

Stanley *et al*. (2005) found that a significant percentage of those studied among the alcohol abusers in Port Harcourt had an average liver span of less than 8cm. This indeed was a clinical confirmation of a reduction of the liver size due to exposure to the toxic effect of chronic alcohol intake. Prolonged and heavy alcohol intake has been strongly associated with cirrhosis of the liver in the tropics. Lester (1978) reported 40%, Femi-Pearse and Danisa (1969), 29%, Olumide *et al*, (1978) 36% in Lagos and Madubuike *et al*, (2007) 23% in Port Harcourt.

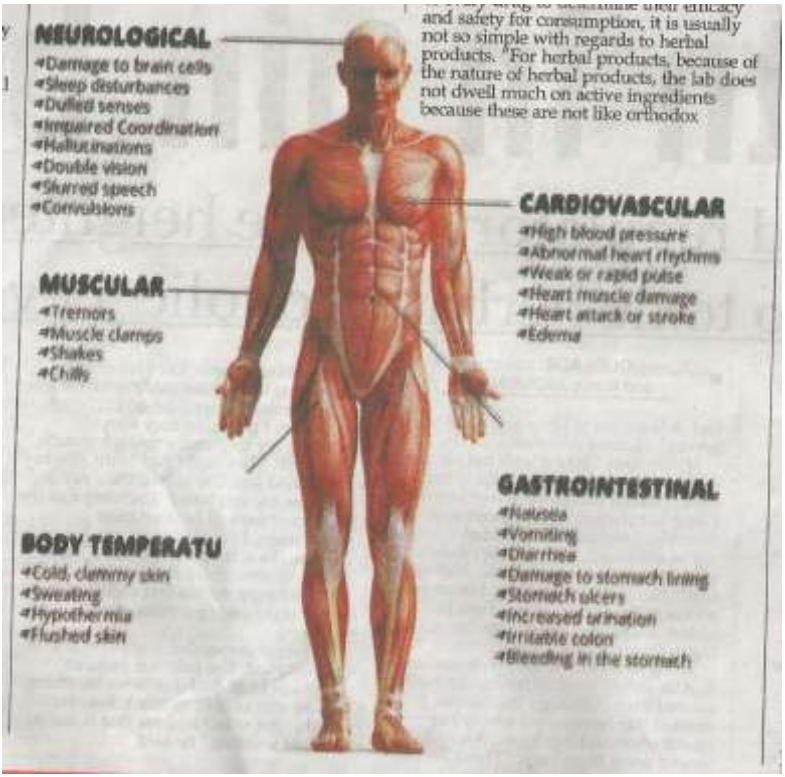


Plate 8: Showing how the body system is affected by alcohol misuse
Source: The Nation, Saturday, July 5, 2014

Genitourinary system

Acutely, modest ethanol doses (for example blood alcohol concentration of 100 mg/dL or even less) increase sexual drive in men. However, modest ethanol doses may simultaneously decrease erectile capacity. Even in the absence of liver impairment, a significant minority of chronic alcoholic men may show irreversible testicular atrophy with concombitant shrinkage of the seminiferous tubules and loss of sperm cells. The repeated ingestion of high doses of ethanol by women can result in amenorrhea, a decrease in ovarian size, an absence of corpora lutea with associated infertility, and spontaneous abortions. Heavy drinking during pregnancy results in the rapid placental transfer of both ethanol and acetaldehyde, which may have serious consequences for fetal development.

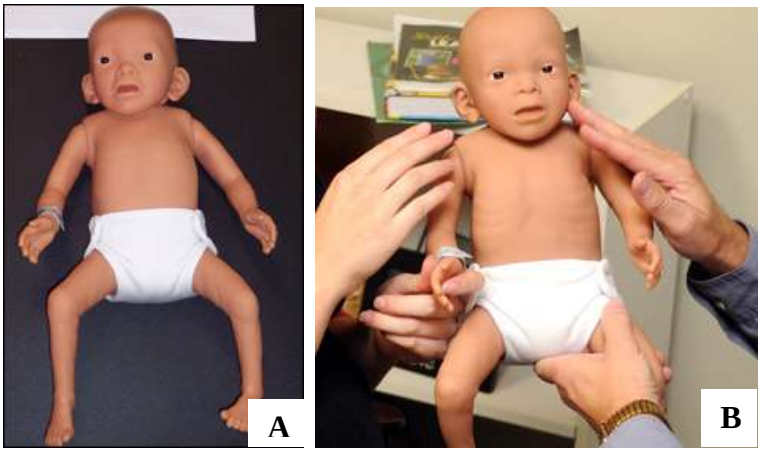


Plate 9: Foetal Alcohol Syndrome (FAS)

The fetal alcohol syndrome can include a mixture of any of the following: facial changes with epicanthal eye folds, poorly formed concha, and small teeth with faulty enamel; cardiac atrial or ventricular septal defects; an aberrant palmar crease and limitation in joint movement; and microcephaly with mental retardation. The specific amount of ethanol and/ or specific time

of vulnerability during pregnancy has not been defined, making it advisable for pregnant women to abstain completely (Schuckit, 1991).

Cardiovascular

Modest doses of alcohol can have both deleterious and beneficial effects in individuals with normal cardiovascular status who take no medications. Ethanol decreases myocardial contractility and causes peripheral vasodilatation resulting in a mild drop in blood pressure and a compensatory increased heart rate and cardiac output. Exercise-induced increases in cardiac oxygen consumption are higher after alcohol. On the other hand, one or two drinks per day over long periods may decrease the risk of cardiovascular death, perhaps through an increase in high density lipoprotein cholesterol (HDL) or changes in clotting mechanisms.

Although ethanol, in low doses causes a mild acute drop in blood pressure, the consumption of three or more drinks per day results in dose-dependent increase in blood pressure which returns to normal within few weeks of abstinence. Hence, heavy drinking is an important contributor to reversible causes of mild to moderate hypertension. Chronic heavy drinking can cause cardiomyopathy with symptoms ranging from unexplained arrhythmias in the presence of the ventricular impairment to heart failure with dilatation of all four heart chambers and hypocontractility of heart muscle. Mural thrombi can form in the left atrium or ventricle, while heart enlargement exceeding 25 percent can cause mitral regurgitation. Finally, there is an association between cerebrovascular accidents and alcoholism, especially within 24 hrs of heavy drinking. Atrial or ventricular arrhythmias, especially paroxysmal tachycardia, can also occur after a binge in individuals showing no other evidence of heart disease-a syndrome known as the “holiday heart” (Schuckit, 1991).

Hematopoietic system and haematological profile

Ethanol exerts multiple reversible acute and chronic effects on all blood cells. Alcohol alters acutely the production of red blood cells (RBC), which reaches clinical significance after days to weeks of heavy drinking. The most common finding is an increase in RBC size (mean corpular volume, MCV) with a mild anemia. If this is accompanied by folic acid deficiency, there can also be hypersegmented neutrophils, reticulocytopenia, and hyperplastic bone marrow. Other forms of anemia including sideroblastic changes, can occur concomitantly, especially in the presence of severe malnutrition.

Chronic heavy drinking can also decrease production of most white blood cells (WBC), decrease granulocyte mobility and adherence, and impair the delayed hypersensitivity response to new antigens (with a possible false-negative tuberculin skin test). While the changes in WBCs themselves are usually temporary, they may contribute to the risk of infections, liver damage, and perhaps to the increased risk of cancers in alcoholics. Alcohol can also cause toxic granulocytosis. Many alcoholics present with mild thrombocytopenia (rarely associated with hemorrhage) due to a decrease in platelet survival and altered function; hypersplenism may occur as a complication of cirrhosis. Alcohol may decrease platelet aggregation and inhibit release of the thromboxane A_2. These problems usually return toward normal within a week of abstinence (Schuckit, 1991).

The haematological profile is usually globally affected resulting in anaemia, usually hypochromic and megaloblastic type, thrombocytopenia and leucopenia. This may be due to direct toxic effect and suppression of the bone marrow, haemolysis, malabsorption due to brush border atrophy and poor nutritional intake (Hasselbalch, 1992; Homman and Hasselbalch, 1992). In a similar study Stanley *et al*. (2005) reported leucopenia, thrombocytopenia and nomochronic red blood cells in the absence of anaemia among the study group who predominantly consumed burukutu. This brand of alcohol made from grain (millet) may have been rich in folate and the iron pots

(used for the preparation) which could explain the absence of anaemia in the cohort. However, raised mean corpuscular volume (MCV) and the gamma-glutamyl transferase (GGT) were reflected both chronic and heavy drinking respectively.

Lipids, Electrolytes and trace elements: the progressive decline of electrolytes, potassium, sodium, magnesium and calcium in 26% total protein and albumin in 23% and trace elements, zinc and manganese 26%, and iron 21% with a progressive rise in GGT and AST in 55%, and urea, creatinine, triglyceride and cholesterol 12% were reported by Stanley and Wakwe (2003).

These findings are in agreement with other past and recent reports (Figueiredo *et al*., 2000; Furman *et al*., 2004; Plauth *et al*., 2006; DiCecco and Francisco-Ziller, 2006; Shimizu, 2012; Wagnerberger *et al*., 2012). These changes have been implicated in various psychotic and mood disorders.

Endocrine and Reproductive

1. Hypercortisolaemia with non-suppression on the dexamethasone suppression test. A 'Pseudo-Cushings' picture may be seen though it tends to resolve on abstinence.
2. Hypogonadism with testicular atrophy, infertility, decreased libido and erectile dysfunction, have been reported (Eggert, 2004).

Musculoskeletal

Heaving drinking can produce an acute alcoholic myopathy charactrised by painful and swollen muscles, high levels of serum creatine phosphokinase (CK), and rarely myoglobinemia and myoglobinuria. Effects on skeletal system include alterations in calcium metabolism with an increased risk for features of osteonecrosis of the femoral head. Hormonal changes include an increase in cortisol levels, which can remain elevated during heavy drinking; inhibition of vasopressin secretion at rising blood alcohol concentrations and the opposite at falling blood alcohol concentrations, with the final result that most alcoholics are

likely to be slightly overhydrated; a modest and reversible decrease in serum thyroxine (T_4); and a more marked decrease in serum triiodothyronine (T_3).

Respiratory

Significant association between alcohol chest infections such as Tuberculosis, Bronchiostasis, and Pneumonias have been reported (Moss *et al*, 2003). This may be due to malnutrition, malabsorption, leucopenia, reduced body immunity and direct toxic effect on respiratory tract associated with alcohol misuse.

Dermatological

Most dermatological signs such as spider naevi, and palmer erythema result from severe liver damage resulting in cirrhosis. The others such as discoid eczema and worsening psoriasis result from immunological responses.

Neurological

Wernicke-Korsakoff syndrome

This is characterized by persistent, prominent impairment of recent memory, while immediate recall and procedural memory are preserved, and orientation in time, place and person often impaired. Disturbances as time sense ordering and memory loss is out of proportion to overall cognitive loss, making new learning difficult. In addition, opthalmoplegia, (abducens (6^{th} nerve palsy) ataxia and nystagmus are often present.

Pathology

Alcohol reduces the absorption of thiamine and the activity of the enzyme that converts it to an active form. The subsequent neurological damage is focused in the brain stem, wall of the third and floor of the fourth ventricles, periaqeductal grey, part of the thalamus, the mamillary bodies, the terminal portion of the fornices and the anterior lobe and superior vermis of the cerebellum. Microscopically, myelinated fibres are affected more

than neurons, with prominent petechial haemorrhages, astrocytic and histiocytic proliferations (Appleby *et al*., 2001).

Clinical features of Korsakoff's syndrome

Korsakoff's syndrome may be defined as 'an abnormal mental state in which memory and learning are affected prominently in comparison to other cognitive functions, in an otherwise alert and responsive subject'. It could be differentiated from bilateral temporal lobe excision by the absence of disturbed social behaviour and emotional and intellectual decline.

Features include:

1. Apathy and reduced initiative
2. Loss of interest in alcohol
3. Thinking is stereotyped, preservative and facile
4. Reduced ability to categorize and form concepts
5. Visuospatial impairment is common
6. Underestimate age and time in hospital
7. Reduced insight and confabulation
8. Impairment of new learning
9. Variable retrograde amnesia
10. Procedural memory and digit span intact

Causes of Korsakoff's syndrome are as follows:

1. Cancer of the upper gastrointestinal tract
2. Malabsorption or malnutrition
3. Heavy metal poisoning
4. Carbon monoxide poisoning
5. Tumours of the third ventricle
6. Head injury
7. Bilateral hippocampal destruction
8. Anaesthetic agents

Imaging in Korsakoff's syndrome

Computerised Tomograph (CT) of the Brain shows frontal lobe atrophy in one-third and convolutional atrophy in quarter of

cases. The Electroencephalogram (EEG) shows diffuse slowing in contrast to (Delirium Tremens (DTs)).

Prognosis

In Victor's classic 1971 study with 245 patients, the following conclusions were reached:

1. 17 per cent died in the acute phase
2. 16 per cent presented with Korsakoff's syndrome
3. Sixth nerve palsies always recovered
4. 66 per cent had residual horizontal nystagmus
5. Of the 186 who were followed up, a quarter showed no recovery
6. Women had a better prognosis

Cerebellar degeneration

The anterior lobe of superior vermis demonstrated purkinje cell loss leading to ataxia of stance and gait.

Amblyopia

Retrobular neuritis which often occurs following few weeks of persistent alcohol intake is associated with peripheral neuropathy, but rarely leads to blindness.

Marchiofava-bignami syndrome

This is characterized by ataxia, epilepsy, dysarthria and severely impaired consciousness. A slowly progressive form involves dementia and spastic necrosis. The neuropathology is demyelination of the corpus callosum, optic tract and cerebellar peduncles (Appleby *et al*., 2001).

Central pontine myelinosis

Clinically, there is pseudobulbar palsy, quadriplegia and loss of pain sensation in the limbs and trunk. Acutely there may be nausea, vomiting confusion and coma and neuropathologically, there is demyelination of pyramidal neurons in the pons.

Epilepsy

This is a common sequelea of chronic alcohol misuse. The occurrence of seizures may arise consequent upon head injuries, or subdural or direct neurotoxicity with alcohol, itself being epileptogenic (Appleby *et al*., 2001)

Dementia

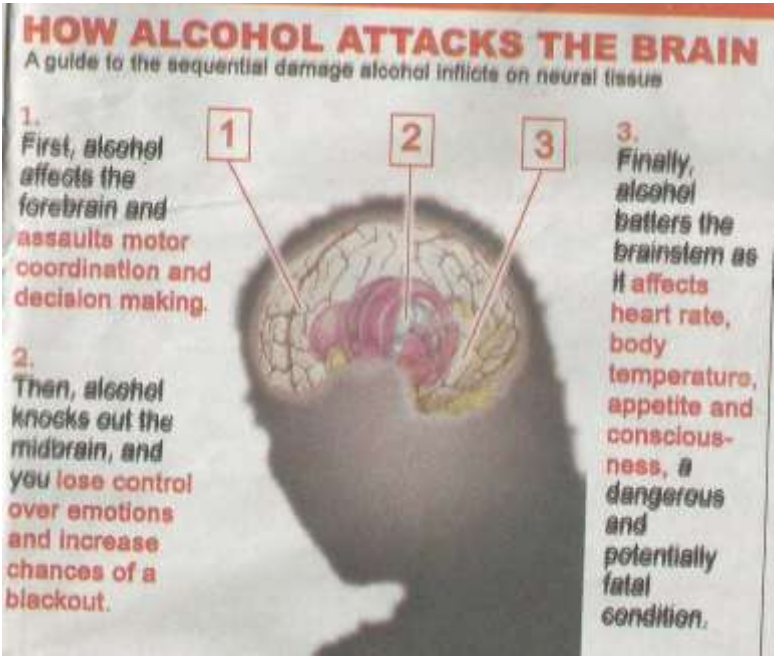


Plate 10: showing how alcohol attacks the brain
Source: The Nation, Saturday, July 5, 2014

Mild cognitive deficits are common in alcoholics, particularly visuospatial, and recent memory. Damage may be due to direct neurotoxicity, head trauma or nutritional deficiency and it would appear that women are more susceptible than men. Computerized Tomograph (CT) and Magnetic Resonance Imaging (MRI) studies show ventricular enlargement and cortical atrophy with some suggesting an association between degree of atrophy, and

cognitive impairment. Some degree of reversibility has been reported with abstinence.

Prevention and Treatment of Alcohol Misuse

This can be discussed at 3 levels namely; primary, secondary and tertiary.

Primary Prevention

This is an effort directed at preventing alcohol misuse. It is imperative to note that this is difficult to achieve if the attitude has been formed. Hence, this effort must be proactive and persistent and aimed at

ii. Health education- in terms of information on health hazards, benefits of not drinking, or minimal drinking.

1. Furthermore, discouraging drinking totally (teetotaler) or preventing harmful drinking, through persuasive and motivational talks
2. Price-control: Prohibitive pricing may assist, but has not been quite rewarding from research experiences.
3. Reduction of advertising reach/control of distribution and restriction of drinking, for example, in beer parlours, entertainment centres and public gatherings.
4. Increase taxation for companies and customers
5. Discourage government agencies from seeking funds and assistance from producers. It is immoral to do this.
    1. Stringent measures/ control on sales

Secondary and Tertiary Prevention of Alcohol Misuse

1. Early Detection and Intervention
1. Identification of Alcohol Misuse
1. History Taking
1. Screening Instruments
1. Laboratory Test
    - Biochemical and Heamatological markers

History of Drinking

If any of the above factors raise suspicion about alcohol misuse, the next stage is to take a comprehensive drinking history. This should be carried out sensitively, with understanding that the patient may have difficulty giving a clear history. The clinician should aim to build up a picture of what and how much the patient drinks throughout a typical day for example, when and where do they have the first drink of the day? The patient should be asked how they feel if they go without a drink for a day or two, and how they feel on waking. This can lead on to enquiries about the typical features of dependence and the range of physical, psychological and social problems associated with it. Questions such as, difficulty at home, work place, job and academic performance, trouble with the law accidents, emotional disorders, self and family care, nutrition and physical health are important. Others include duration, frequency and quantity of alcohol use with types.

Furthermore, when they began drinking in the mornings and when, if ever, they first experienced withdrawal symptoms. It is useful to ask about periods of abstinence from alcohol, what factors helped maintain this state of affairs, and what led to a resumption of drinking. This can lead on to enquiries about past attempts at treatment

1. Describe a typical day's drinking. What time is first drink of the day?
2. When did daily drinking start?
3. Presence of withdrawal symptoms in morning or after abstinence
4. Previous attempts at treatment
5. Medical complications
6. Patient's attitude towards drinking

It is necessary to understand patient's own view of patient's drinking behaviour, because there are a number of possible treatment goals. In this situation the patient's attitude to their

problems plays a key role in determining which approaches are likely to be of likely benefit.

Screening Instruments and Tests
CAGE Assessment for Alcohol Abuse

The **CAGE** is a 4-item, relatively non-confrontational questionnaire for detection of alcohol use disorder. It takes less then 1 minute to administer, is easy to learn, remember and replicate.

1. Have you felt the need to Cut down on your drinking?
2. Do you feel Annoyed by people complaining about your drinking?
3. Do you ever feel Guilty about your drinking?
4. Do you ever drink an Eye-opener in the morning to relieve shakes?

Two or more affirmative responses suggest that the client is a problem drinker.

AUDIT Technique
The **Alcohol Use Disorders Identification Test (AUDIT),** developed in 1982 by the World Health Organization, is a simple way to screen and identify people at risk of alcohol problems. It has high sensitivity and specificity and quite useful for clinicians and researchers. It is a 10-item useful instrument.

1. How often do you have a drink containing alcohol?
(0) Never (Skip to Questions 9-10)
(1) Monthly or less
(2) 2 to 4 times a month
(3) 2 to 3 times a week
(4) 4 or more times a week

2. How many drinks containing alcohol do you have on a typical day when you are drinking?
(0) 1 or 2
(1) 3 or 4
(2) 5 or 6
(3) 7, 8, or 9
(4) 10 or more

3. How often do you have six or more drinks on one occasion?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

4. How often during the last year have you found that you were not able to stop drinking once you had started?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

5. How often during the last year have you failed to do what was normally expected from you because of drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

6. How often during the last year have you been unable to remember what happened the night before because you had been drinking?
(0) Never
(1) Less than monthly

(2) Monthly
(3) Weekly
(4) Daily or almost daily

7. How often during the last year have you needed an alcoholic drink first thing in the morning to get yourself going after a night of heavy drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

8. How often during the last year have you had a feeling of guilt or remorse after drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
(0) No
(2) Yes, but not in the last year
(4) Yes, during the last year

10. Has a relative, friend, doctor, or another health professional expressed concern about your drinking or suggested you cut down?
(0) No
(2) Yes, but not in the last year
(4) Yes, during the last year

Add up the points associated with answers. A total score of 8 or more indicates harmful drinking behavior. It is useful in Primary Health Care services.

MAST Screening Instrument
This is a 10-item shortened version of the Michigan Alcoholism Screening Test (Pokorny *et al*., 1972). A score of five or more points is said to be positive.

Useful Measures of Alcohol-Related Problems
Useful measures of alcohol-related problems (which correlate with severity of dependence) include-self reporting ***Alcohol Problem Questionnaire*** **(APQ)** and ***Severity of Alcohol Dependence Questionnaire*** **(SADQ).**

Laboratory Investigation for Alcohol Dependence
Biochemical investigations such as Gamma-GT (50-60 per cent sensitive), MCV (30-40) per cent sensitive) and most specifically and recently carbohydrate deficient transferring (80 per cent) screens are also useful in identification as well as in monitoring relapse.

Gamma-glutamyl-transpeptidase (GGT): Estimations of GGT in blood provide a useful screening test. The level is raised in about 70 per cent of alcohol misusers, both men and women, whether or not there is demonstrable liver damage. The heavier the drinking, the greater is the rise in GGT, and returns to normal within a week of abstinence.

Mean corpuscular volume (MCV): MCV is raised above the normal value in about 60 per cent of alcohol misusers, and more commonly in women than in men. If other causes are excluded, a raised MCV is a strong pointer to excessive drinking. Moreover, it takes several weeks to return to normal after abstinence.

Carbohydrate Deficient Transferrin: This is a variant of a serum protein which transports iron, and levels are increased in response to heavy drinking. It is probably more specific than GGT (Salaspuro, 1999).

Blood Alcohol Concentration (BAC): A high concentration does not distinguish between an isolated episode of heavy drinking and chronic misuse. However, if a person is not intoxicated when the blood alcohol concentration is well above the legal limit of driving, he is likely to be unusually tolerant of alcohol. This tolerance suggests persistent heavy drinking. Alcohol is eliminated rather slowly from the blood and can be detected in appreciable amounts for 24hours after an episode of heavy drinking. In clinical practice, breath alcohol is often used as a proxy measurement for blood alcohol, breathalyzers.

Treatment Approach in Alcohol Misuse

Psychological Intervention

Provision of information and advice about the effects of excessive drinking is an important first stage in treatment. The information given should relate to the specific problems of the individual patient, both those that have occurred already and those likely to develop if drinking continues.

Early Detection and Brief Intervention

Early detection of excessive consumption of alcohol and alcohol misuse is important because treatment of established cases is difficult, particularly when dependence has occurred. Many cases can be detected early by general practitioners, physicians, and surgeons when patients seek treatment for another problem.

General practitioners are well placed to provide early treatment of alcohol problems, and they are likely to know the patient and his family well. It is often effective if the general practitioner gives simple advice in a frank manner with utmost tact and understanding. Brief intervention studies generally involve simple education and advice about safe levels of alcohol

consumption. The aim is to promote safer drinking rather than abstinence. Generally brief interventions lead to significant reductions in alcohol consumption over the next year but it is uncertain how far these gains are maintained. It is generally agreed that brief interventions are not effective for people with severe problem drinking, particularly those who are alcohol dependent (Room et la., 2005).

Motivational Interviewing

Patients with problems of alcohol misuse, particularly those detected by screening methods, may be unsure whether or not to engage in treatment programmes. An appropriate interviewing style, particularly during the first assessment, can help to persuade the patient to engage in a useful review of their current pattern of drinking. Confrontation is avoided in motivational interviewing, and a less directive approach is taken during which patients are helped to asses the balance of the positive and negative effects of alcohol on their lives. The clinician can help in this exercise by providing feedback to the patient on the personal risks that alcohol poses both to them and to their family, together with a number of options for change. The aim of motivational interviewing is to persuade the patients to argue their own case for changing their pattern of substance use.

1. Express empathy
2. Avoid arguing; don't be judgmental
3. Detect and 'roll with' resistance
4. Point out discrepancies in history
5. Raise awareness about contrast between substance user's aim and behaviour

Group Therapy

The aim of group therapy is to enable patients to observe their own problems mirrored in other problem drinkers and to work out better ways of coping with their problems. They gain confidence, whilst members of the group jointly strive to reorganize their lives without alcohol. Until recently, the most

common plan of treatment in specialist alcohol units was an inpatient programme of group therapy lasting about 8 weeks; this treatment could be preceded by detoxification if required. However, because of lack of evidence that this kind of intensive treatment approach is particularly beneficial, most clinics now offer a broader range of care including outpatient and day patient programmes that utilize variety of psychotherapeutic approaches including marital and family therapy and cognitive-behavioural methods of treatment.

Cognitive behaviour therapy

Cognitive-behaviour methods of treatment tackle the drinking behaviour itself rather than the presumed underlying psychological problems. Such approaches stress the role of education and the improvement of social and interpersonal skills as these relate to alcohol misuse. For example, it may be helpful to identify situational or interpersonal triggers that cause an individual to drink excessively, and then to plan and rehearse new methods of coping with these situations. This is called relapse prevention. The use of cue exposure to alcoholic drinks, without subsequent consumption, usually reduce the risk of subsequent relapse among subjects to enable them to contend with ready availability of alcohol in social settings. Most patients who misuse alcohol have obvious deficiencies in problem-solving skills and appropriate training may help reduce relapse rates. Where patients are in a relatively stable relationship with a partner, couple therapy can produce improvements in drinking behaviour as well as marital adjustment.

Pharmacological Interventions

Disulfiram

Disulfiram (Antabuse) acts by blocking the oxidation of alcohol so that acetaldehyde accumulates. Some patients find it useful because the anticipation of an unpleasant reaction acts as a deterrent to impulsive drinking. The reaction includes facial flushing, throbbing headache, hypotension, palpitations,

tachycardia, nausea and vomiting. In vulnerable patients, cardiac arrhythmias and collapse may occur.

Major contraindications to the use of disulfiram are a history of heart failure, coronary artery disease, hypertension, psychosis, and pregnancy. Patients should be given clear verbal and written instructions along with a list of substances to be avoided that contain alcohol. Common side-effects are drowsiness, bad breath, nausea, and constipation. Disulfiram is given in a single dose of 800mg on the first day of treatment, reducing over 5 days to 100-200mg daily.

Acamprosate (Calcium acetyl homotaurinate)

Acamprosate appears to suppress the urge to drink in response to learned cues, and produces modest but useful reductions in drinking behaviour in alcohol-dependent subjects. It is believed to act by stimulating Gamma-Aminobutyric acid (GABA) inhibitory neurotransmission and decreasing the excitatory effects of glutamate. Abstinence rates appear to be approximately doubled, with some evidence that the benefit continues after stopping the drug. It is unclear, however, which patients are most likely to benefit from acamprosate treatment.

The usual dose of acamprosate is two tablets (each 333mg) three times daily with meals. In lighter subjects (>60 kg). four tablets daily are recommended. Acamprosate is not metabolized in the liver and is excreted by the kidney. Therefore it is unlikely to cause drug interactions. Adverse effects include diarrhoea, and less frequently, nausea, vomiting, and abnormal pain. Skin rashes may occur as can fluctuations in libido.

Naltrexone

The opoid antagonist, naltrexone, is believed to block some of the reinforcing effects of alcohol and in this way decrease the likelihood of relapse after detoxification. A Cochrane review (Srisurapont and Jurasuraisin, 2003) concluded naltrexone is effective in the short-term treatment of alcohol dependence, but there is no clear benefit over acamprosate and disulfiram. It is

possible that the main effect of naltrexone is to prevent a 'lapse' from becoming a full-blown 'relapse': hence it follows that naltrexone should not be stopped if the patient has a drink. Its effects may be enhanced by concomitant cognitive-behaviour therapy (Garbutt *et al*, 1999). Side-effects of naltrexone treatment headache, dizziness, and weight loss.

Antidepressant Drugs

Antidepressant medication is useful in patients who experience persistent symptoms of major depression after detoxification. However, tricyclic antidepressants are not recommended because of potentially serious interactions, including cardiotoxicity and death following overdose (Lingford-Hughes *et al*., 2004). Some studies have suggested that selective serotonin reuptake inhibitors (SSRIs) such as citalopram can reduce drinking in non-depressed alcohol-dependent patients but not all studies are in accord (Chick, 2000). Re-analyses of some trials suggest that the effects of SSRIs may vary according to the Type1' and Type 2' categories described by Cloninger *et al*., (1998). SSRIs may improve drinking outcome in Type 1'alcohol dependence (later age of onset, anxious traits) but may worsen outcome in type 2 alcohol dependence (early age of onset, positive family history, impulsive/ antisocial personality traits) (Lingford-Hughes *et al*., 2004).

Social Intervention

Alcoholics Anonymous (AA)

This is a self-help organization founded in the USA by two alcoholic men, a surgeon and a stockbroker. It has since spread to most countries of the world. Members attend group meetings, usually twice weekly on a long-term basis. In crisis they can obtain immediate help from other members by telephone. The organization works on the firm belief that abstinence must be complete. At present there are about 1200 groups in the United Kingdom (UK).

Alcoholics Anonymous (AA) does not appeal to all problem drinkers because the meetings involve an emotional confession of problems. However, the organization is of great value to some problem drinkers, and anyone with a drinking problem should be encouraged, to access this group..

Alcoholics Anonymous types of treatment.
Al-Anon: This is a parallel organization providing support for the spouses of excessive drinkers, while Al-Ateen does the same for their teenage children.

Non-statutory agencies: These are voluntary bodies that provide a range of services including advice for problem drinkers and their families, counseling, and help with occupational and social activities for those who have recovered.

Hostels: These are intended mainly for homeless problem drinkers. They provide rehabilitation and counseling. Usually abstinence is a condition for residential assistance. In our environment where these agencies, or associations are non-existent, the social work professionals should be adequately and timely engaged.

Aims of Treatment
This includes:
1. Achieve withdrawal, total abstinence or acceptable, harmless and controlled intake / drinking.
2. Harm reduction-this involves efforts at reducing harmful behaviour such as indiscriminate unprotected sex (as they become usually disinhibited) and minimization of hypodermic needle sharing if involved in polysubstance abuse particularly the intravenous drug users (IDU) to avoid human immunodeficiency virus (HIV), Hepatitis B and C infections.

3. Advise on adequate nutrition-balance diets, haematemics, fruits and vegetables, vitamins B complex particularly, thiamines, folates, and other vitamins and minerals.
4. Occupational, domiciliary and family/interpersonal rehabilitation. occupational therapists and social work professionals should be maximally involved

Outcome of Treatment

The prognosis of Alcohol misuse, particularly with dependence is poor. On the overall, relapse rates have been as high as 35% within a month after leaving the treatment centre and another 25% in 3 months. In about 18-36months period, about 75% relapse rates have been reported (Stanley *et al*., 2014). However, on the overall, treatment success rate over 5years is not less than 25%.

Legal Issues

1. Driving while alcohol exceeds legal limit
2. Drunk in charge of a motor vehicle, ship, cattle, horse, child under 7 years; drunk and disorderly
3. Drunk and incapable; using abusive language; assault (either as assailant or victim).
4. It is also an offence for an intoxicated person over 16 to share a bed with a child under 3, as a result of which the child is suffocated by overlaying (David, 2007).

A doctor may be requested to examine a drunken detainee when uncertainty about injury or illness has arisen. Natural disease may mimic alcohol intoxication for example, migraine, multiple sclerosis, stroke, hypoglycaemia or ketoacidosis in diabetics, epilepsy, drug intoxication and Meniere's disease (dizziness). Head injury often mimics intoxication.

RECOMMENDATIONS

1. That the University’s antidrug campaign unit be reactivated and strengthened.

2. The Students' Union executive in league with Medical, Pharmacy, Sciences, Humanities and Social Sciences students should come up with regular enlightenment programmes, aired by uniport radio station, published regularly in the uniport weekly newsletter, while the theatre troupe and music department should produce strongly aversive concerts and repulsive jingos and slogans against alcohol misuse.
3. Regular students' discussions and decent bill boards should speak proudly against Alcohol misuse.
4. Offending billboards such as the one at the Choba East-West road interjection showing 2 youths (male and female) in an offensive position after a bout of intoxicating calypso alcoholic wine should be removed.
5. More accommodation facilities should be made available for students within the campuses as a way of checking the excesses of drug involvement among students.
6. Both academic and non-academic staff of the university should rise up against the scourge of alcohol misuse.

CONCLUSION

Alcohol, an organic compound is as old as mankind, and has been of great service from the time of Jesus Christ. Its importance was demonstrated when Christ heeded his mother's instruction to miraculously produce wine for a marriage ceremony (John 2:1-11). Secondly, Paul recommended wine for his spiritual son as he puts it "no longer drink only water, but use a little wine for your stomach's sake and your frequent infirmities (1 Timothy 5: 23). Thirdly, the Bible further recommended wine for temporary relieve of psychosocial symptoms, "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 (Proverbs 31: 6-7). Fourthly, cardiologists recommend not more than 1.5 units of wine daily for the heart. However, man's insatiable desire for pleasure often leads to a

harmful and hazardous drinking pattern and now makes it safer to advocate total abstinence-teetotallers.

Vice Chancellor Sir, distinguished audience, alcohol misuse disrupts socio-economic, physical and psychological well-being of victims, inspiring both domestic and public violence, poor academic and job performances, prone to accidents at home, workplace and road, and increases tendency to crimes, while targeting the youths for sufficient decimation. Hence, dimming the hope of this group often referred to as the leaders of tomorrow. Of a truth, it makes a spectacle of humanity, and indeed great men and women, bright and brilliant, but alcohol destroyed them all, having contributed substantially to global morbidities and mortalities.

However, sensible and controlled drinking could be helpful, while early intervention for the affected must be aggressive for rewarding results as late intervention are usually futile.

Finally, let me express my great indebtedness to my teachers from the kindergarten to the present day for their rewarding mentorship. Of particular note is my Chief mentor Prof Emeritus Nimi D. Briggs whose mentorship transcends every stratum of human endeavour, for encouraging me to return home from Jos when I did. Further details of this mentorship will be unveiled during my valedictory lecture

Vice Chancellor Sir, I am done. Thank you and God bless you.

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CITATION
ON
PROFESSOR PRINCEWILL CHUKWUEMEKA STANLEY
B. Med Sc. Pharmacology, (UPH); MBBS (UPH); FWACP, FMCPsych (Nig.)

Introduction

PRINCEWILL CHUKWUEMEKA STANLEY's attainment to the professorial chair has been a journey which came through undeterred commitment and dedication, vision, courage and most importantly seems to be a mere response to and fulfilment of destiny. Born to the family of Hon. Emmanuel Friday Stanley and Mrs. Esther da Sunday Stanley (Dede), (both of blessed memory) both of Ndoki ethnic nationality, on the 5th of March, l964 at Ikot Ekpene, Nigeria. The trait of leadership and proactiveness which Prof Princewill Stanley has continued to show in almost all spheres of his life was first exhibited at the time of birth, when out of love, understanding and humility which still continues till today, little Princewill bargained with his twin brother to take the lead to enter this world.

Education

Little Princewill Chukwuemeka Stanley began his journey of his educational career at Opobo Town Junction Kindergarten school in 1968, but was interrupted by the Nigerian civil war. He thereafter started his primary education at the Ogbo Hill primary

school, Aba from 1970-1974. By 1975, he proceeded to Comprehensive Secondary School and later All Saints Secondary School, Ehere, Aba, where he completed his secondary education with a grade 1 certificate in 1979. In 1980, he proceeded to the Prestigious Government College Umuahia, for higher educational programme, and was thereafter admitted into the University of Port Harcourt in 1981 from where he obtained a bachelors degree in Medical Sciences in Pharmacology in 1985 and MB.BS in 1988. Princewill Stanley distinguished himself as he became not only the Best student in ENT, and ophthalmology and psychiatry, but was also the best graduating student in Clinical Medicine.

Professional/Academic Career

A visionary scholar of extraordinary insight, he started his housemanship at UPTH. Following his National Youth Service in Infectious Hospital, Kano State, and driven by the desire for knowledge and academic excellence, Prof. Stanley started his residency programme in June 1992 at the Jos University Teaching Hospital and completed in record time in April 1997 with fellowship in both colleges. Again, Prof Stanley distinguished himself and became the best candidate in Part I in both colleges, and an outstanding Part II fellow in the West African fellowship examinations.

In 1998, Princewill Stanley was appointed by the University of Port Harcourt as Lecturer 1 in the department of Mental Health. Because of his unquenchable desire and interest for medical training, he was appointed the chairman, postgraduate medical training in the department of Internal Medicine barely one year after his appointment. Then the department was a unit of medicine at the University of Port Harcourt Teaching (UPTH) until 2000.

He rose from lecturer I to Senior lecturer in 2001, and subsequently to the Chair of Neuropsychiatry/Mental Health in 2009. Prof Princewill Stanley, as a great scholar, has bias for subspecialties of geriatrics, substance abuse and forensic

psychiatry. Prof Stanley is one person that believes strongly that there is no limit to knowledge. This inspiration made him to enlist for and successfully completed an appreciation course in Electroencephalogram (EEG) in the prestigious Lund University in Sweden in 2006, a rare subspecialty in which he is currently serving as a consultant to Union Diagnostic, Port Harcourt.

Prof Princewill Stanley is currently serving as a Focal Point for training for a national programme on substance abuse organized by United Nation Offices for Drug and Related Crimes (UNODC). For his efforts, the department of neuropsychiatry, university of Port Harcourt has been recognized by the same body as a treatment centre for substance abuse cases.

Prof. Princewill Stanley has also extended his professional and career services to Anti Drug Advocacy as he has been a consultant to NDLEA since 1997 in Jos and 1998 till date in Port Harcourt.

Prof. Stanley has also been a resource Consultant for clinical drug trials and drug presentation to Pfizer, Smith Kline & Beecham and Norvatis.

Professional Activities

With a burning desire to not only excel as an individual, but also to develop his specialty to an enviable height and to provide qualitative training and training ground in the field of psychiatry, hardworking and innovative Prof Stanley was not satisfied with Mental Health only functioning as a unit in the department of Internal Medicine at the Teaching Hospital. This made him mount enormous pressures on the Teaching hospital, until a dedicated department of Mental Health was formed in 2000 at the hospital, barely two years after his appointment. Thus, Prof Stanley became the founding father and pioneer Head, Department of Neuropsychiatry in University of Port Harcourt Teaching Hospital. The "Road Maker" replicated same in Niger Delta University during his one year sabbatical leave, where he equally served as the pioneer HOD, Department of Mental Health and Psychiatry.

He is currently serving as an Examiner in both Colleges (WACP 2001 till date, and NPMC in 2010) and has served as external examiner to many Universities in Nigeria.

Administrative responsibilities and academic leadership

Professor Princewill Stanley is a multi-talented, multi tasked administrator who has served the University of Port Harcourt, and the University of Port Harcourt Teaching Hospital (UPTH) in various capacities.

Professor Princewill Stanley is a man noted for his forthrightness. He insists on playing according to the rules and keeping to standards, and always advocates for moral discipline and the observance of the ethics of medicine in carrying out academic and professional duties.

Obviously due to these rare qualities, the hard-working Prof. Stanley has been found worthy and appointed at various times to serve the University and the Academic community in the following capacity:

Professor Stanley has been a member of the Senate of the University of Port Harcourt since 2001 to date. In the College of Health Sciences, he chaired various investigative committees on acts of misdemeanour by staff and students. At the UPTH, he also chaired many investigative panels on acts of insubordination and fraud involving staff. Other areas where Prof Stanley has been found resourceful include:

1. Hall Warden; Clinical Hostel 2002-2007.
2. Chairman, Continuing Medical Education, College of Health Sciences, 2004-2012
3. Member, College Silver Jubilee planning committee, 2005.
4. Member, Senate appeals committee on certificate verification, 2006 till date.
5. Board Member, University of Port Harcourt Teaching Hospital, as representative of the VC, 2006 to Dec. 2011
6. Member Uniport Medical Audit Panel, 2006 till 2012.
7. Member, Panel of Investigation of Ethical Misconduct, UPTH, 2008.

8. Chairman, UPTH fees review Committee, 2008.
9. Member, Uniport Examination Committee, 2008 till date.
10. Member, College Research Day Activities, 2008 and 2011.
11. Member, UPTH HIV surveillance and control committee, 2008-2012.
12. Member, Implementation committee on American University Medical College in Nigeria (Abuja) 2009 till date.
13. Member, Uniport Honorary degree award committee 2009-2011
14. Member, Uniport Strategic Planning Committee review, 2010.
15. Member University Curriculum Review Committee, 2010.
16. Member, Uniport criteria committee on emeritus professorship, 2011-2012
17. Chairman, fees review committee (UDUTH) 2012

Research Career

Professor Princewill Stanley, to prove his great interest in academic, quickly went into research and began publishing. So far, Prof Stanley has proven himself to be a formidable International scholar having published over 50 articles in local and international journals, and 2 books and a monograph and presented papers at various conferences both local and international including Portugal, Sweden, Italy, France and Lebanon.

The academically minded Prof. Stanley has mentored over 25 residents in Jos and Port Harcourt between 1996 and date. He has also supervised over 40 undergraduates and Masters students in Psychiatry, Psychology, Physiology and other related fields. He has trained seven neuropsychiatrists who are now Consultants in various hospitals while some have been appointed by the University as lecturers.

Life in Medical and Academic Politics

He has held many positions in medical and academic Politics. Prof Stanley manifested his political life quite early, but has

however, limited himself so far to medical and academic politics alone. Thus, he contested and won the following offices. As a medical student,

PUMSA PRO, 1984, PUMSA President, 1986. As a resident doctor, ARD PRO, JUTH, 1993-1994 and later ARD President, 1994-1995. As a consultant, MDCAN, Assistant Secretary, 2004-2006, Secretary 2006-2008, Chairman, 2008-2010. He is currently, the National President of the Port Harcourt University Medical Graduates Association (PUMGRADA), Uniport. He also was elected Chairman, PTA, Federal Government College Nursery and Primary School Chairman, 2011 till date and appointed member, Medical Board NMA, Rivers State, 2011-2012

Humantarian/Community Service

Prof Stanley, as part of his selfless service to humanity, has taken upon himself, in what can aptly be described as "Charity begins at home" to undertake a yearly self-sponsored medical outreach programmes to the Ndoki communities since the last 10 years.

Awards

Professor Stanley selfless and humanity services, have not passed unnoticed, thus he has received awards from almost all well meaning segment of the society.

1. In 2005 and 2007, he was invited for excellent service by This Day Newspaper.
2. 2008 Award for Excellence in Residency Training by the Association of Resident Doctors, UPTH.
3. Prof. N.D. Briggs Award of Excellence in recognition of his selfless service to PUMSA and a worthy Ambassador of the University of Port Harcourt by PUMSA in 2011.
4. Meritorious Service Awards

In recognition of his outstanding contribution in residency Training in UPTH December, 2012 by ARD, UPTH.

5. Award of Excellent Leadership by the ARD Jos in December, 2012, 14 years after he had left Jos.

6. Award for Academic Excellence in recognition of his Giant Studies in Promotion of medical Education in Nigeria and Attachment of the Higher Pedestal in medical Profession by the Nigeria Medical Students Association (NIMSA) at Calabar in 2012.
7. In 2012, Niger Delta Youth Leaders Council "Meritorious Award of Profound Corporate Recognition, in recognition and acknowledgement with contagious conviction that of your altruism Patriotism and outstanding personality with superior sensibility, the Possession of immensity of tact leadership with the development of character and mentality worthy of emulation", 2012.
8. In 2012, received the Roll of Honour Award (the highest awardable to individual) by the Nigerian Medical Association (NMA) Rivers State branch, in 2012.
9. Award of Excellence for Academic and Administration Leadership and most Admired Clinical Science Lecturer by the Pioneer class of Medicine of Niger Delta University during his one year Sabbatical leave at that University in February, 2013.
10. In 2014, Award of Excellence by the (PUMSA) Port Harcourt University Medical students Association.

Private and family Life

His community and the church has also benefited from his huge reserve of knowledge and experience. He served as Assistant Secretary, Full Gospel Businessmen International, Jos Narakuta chapter 1994 till 1997, Men's Fellowship chairman, 1996 till 1997.

An ardent believer in family unit, Prof. Staley is married with 5 children to Dr (Mrs) Catherine Stanley- a pharmacist and Lecturer at the Faculty of Pharmaceutical Sciences, University of Port Harcourt.

Prof. P. C. Stanley is a devout Christian, an Associate Pastor and an Evangelist and he runs a private medical outfit in Port Harcourt.

Conclusion:
Mr. Vice Chancellor Sir, distinguished ladies and gentlemen, I present to you our 108th inaugural Lecturer, a man of Noble birth, a several Awards winner, a Multi-talented and gifted Scholar of extraordinary insight, a seasoned and an accomplished neuropsychiatrist, a Preacher of the Word of God, a Moral and Intellectual mentor, and Role model indeed, a courageous, forthright visionary administrator and researcher, who has served this University, his community and mankind, an advocate of true morals and a crusader of peace. A man with very high level of integrity, an uncompromising man, a man who, in order to speak, defend and stand for the truth, does not fear a lonely path, A liberalist, vocal and workaholic. A man whose friendship defies the law of age and class barriers, An accomplished mentor, and an erudite scholar. A loving husband and a caring father. The founder and pioneer Professor of the department of Mental Health UPTH, The founder and pioneer Professor of the department of Mental Health NDU.

PLEASE, Join me to salute
This amiable academic giant, and a distinguished fellow in the medical profession. This big fish in the ocean of medical knowledge, a true and befitting teacher and lecturer. A man of the people, a true technocrat, an epitome of knowledge and an icon of excellence, a man with a coat of many colours and An all rounder indeed. This unique brand of unique Uniport.
A man with the 4 GIANT Ps -

- A Professor
- A Physician
- A Pastor and
- A Politician

Professor Princewill Chukwuemeka Stanley.

Thank you.

Professor I.M. Siminialayi (31st July, 2014)