

Lecture Notes

Last updated 24.10.07

Alcohol & Alcoholism

1. [Absorption, Distribution, Elimination Effects](#)
2. [Alcohol Dependence](#) (chronic alcoholism)
3. [Alcohol-related deaths](#)
4. [Drinking & Driving](#)

INTRODUCTION

Alcohols: a group of organic liquids which have a particular chemical grouping (OH). Named according to the length of the carbon backbone

Methanol (methyl alcohol)

Ethanol (ethyl alcohol) = "alcohol"!

Propanol (propyl alcohol)

Butanol (butyl alcohol)

Ethanol is by far the commonest alcohol. Moderate use of alcohol is socially acceptable and medically beneficial. Alcohol is used throughout most societies to affect mood and to alleviate discomfort. It is an [addictive drug](#).

<i>Recommended intake</i>	Men	Women
Safe	3-4 units/day (21-28 u/week)	2-3 u/d (14-21 u/w)
Hazardous	21-50 u/w	14-35 u/w
Dangerous	> 50 u/w	> 35 u/w

SPECTRUM OF ALCOHOL USE / ABUSE (ABC of Alcohol, 1994; Naik & Lawton, 1996):

Teetotal - 10% of population.

Social drinker - drinks some form of alcoholic beverage occasionally or regularly in moderation, i.e. within sensible limits. 75% of those who drink come to no harm. Benefits probably outweigh hazards.

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 causes physical, psychological and social problems. Continues to drink in spite of developing difficulties. Criteria for alcohol dependence are not met.

Dependent or addicted drinker ("alcoholic") - has subjective awareness of compulsion to drink; exhibits prominent drink-seeking behaviour; becomes tolerant to alcohol; 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.

UK adult population 43m; 36m Regular drinkers; 7.3m drinking above sensible limits, 4m Heavy drinkers; 800 000 problem drinkers; 400 000 dependent drinkers.

Average GP list of 2000 will include 186 heavy drinkers, 37 problem drinkers and 19 dependent drinkers.

In USA the third largest health problem, is on the increase, reduces life expectancy by 12 years.

Causes: serious problems in 1m drinkers, 500 deaths under 25.

Assoc. with: 1/2 million hospital admissions, 17 000 psychiatric admissions, 80% fire deaths, 65% serious head injuries, 50% murders, 40% road traffic accidents, 30% fatal accidents, 30% domestic accidents.

Contributes to: 33% divorces, 33% child abuse cases.

Costs: 8m working days per year, £1600 m per year to society.

Attitudes to alcoholism vary.

PHYSIOLOGY OF ALCOHOL

Convenient to consider consumption in terms of **units** of alcohol.

1 unit = a standard drink = approx 8 g 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, i.e. 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 = **9.5 g**)

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

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

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

alcohols and aldehydes.

ABSORPTION OF ALCOHOL

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

20% of ingested alcohol absorbed in the stomach

80% absorbed in the upper small intestine.

Absorption is most rapid when the stomach is empty. Accelerated **gastric emptying** also accelerates 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).

Gastric emptying **decreased** by food in stomach, certain drugs. The apparent sobering effect of food probably due to the delay in gastric emptying.

Gastric emptying also affected by the type of beverage consumed (different beverages have different alcohol concentrations and congeners), emotional state, drugs, food.

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 upto 20% of ingested alcohol may be oxidised 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 rapid from 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; BAC will not rise any further.

Absorption is generally complete in **one to three hours**.

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 (eg Barbour, <http://home.lightspeed.net/~abarbour/jfss.htm>)

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.

$$C_0(\text{mg}/100\text{ml}) = \frac{\text{Alcohol consumed (g)} \times 100}{\text{WF} \times \text{Body weight (kg)}}$$

C_0 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 $\frac{2}{3}$ of this theoretical C_0 , since absorption is incomplete in the presence of food.

Values of C_0 can also be extrapolated back from the linear elimination phase of experimental Blood Alcohol Concentration – Time curves (see Powerpoint slides)

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

More individual values are obtained from Barbour's chart, <http://home.lightspeed.net/~abarbour/jfss.htm>.

After one unit (8 g alcohol):

E.g.1: Average 70 kg male, $C_0 = (8 \times 100) \div (70 \times 0.68) = 16.8 \text{ mg}\%$

e.g. 2: Obese 90 kg male. $C_0 = (8 \times 100) \div (90 \times 0.6) = 14.8 \text{ mg}\%$

e.g. 3: Lean 80 kg male $C_0 = (8 \times 100) \div (80 \times 0.72) = 13.8 \text{ mg}\%$

e.g. 4: Average 60 kg female $C_0 = (8 \times 100) \div (60 \times 0.55) = 24.2 \text{ mg}\%$

e.g. 5: Obese 75 kg female $C_0 = (8 \times 100) \div (75 \times 0.5) = 21.3 \text{ mg}\%$

e.g. 6: Lean 55 kg female $C_0 = (8 \times 100) \div (55 \times 0.6) = 24.2 \text{ mg}\%$

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 (C_0):

$$C_0(\text{mg}/100\text{ml}) = \frac{\text{Number of units consumed} \times 8 \times 100}{WF \times \text{Body weight (kg)}} \quad (\text{using WF obtained from Barbour's chart})$$

Working out the likely BAC after consumption of a known amount of alcohol is called “**forward estimation**” of BAC. Sources of error are the uncertainty in knowing exactly how much alcohol was consumed, how much was absorbed, and not knowing the individual's own Widmark Factor.

To allow for ongoing elimination since the start of drinking, the amount eliminated during that time interval must be subtracted. 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 C_t :

$C_t = C_0 - (\beta \times T)$ (where β 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, C_0 is the starting level of alcohol and ($\beta \times T$) 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 alcohol was consumed in order to account for the measured BAC or BrAC:

$$\text{Alcohol consumed (g)} = \frac{\text{BAC (mg/100 ml)} \times WF \times \text{Body Weight (kg)}}{100}$$

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 approx. 1.07.

During the **elimination phase** (sobering up phase) the BAC is dropping 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 Conc. (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. 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:

$$\text{BrAC} = \text{BAC} / 2.3 \quad (80 / 2.3 = 35)$$

$$\text{BAC} = \text{BrAC} \times 2.3 \quad (35 \times 2.3 = 80)$$

$$\text{UAC} = \text{BAC} / 0.75 \quad (80 / 0.75 = 107)$$

$$\text{BAC} = \text{UAC} \times 0.75 \quad (107 \times 0.75 = 80)$$

$$\text{VHAC} = \text{BAC} / 0.8 \quad (80 / 0.8 = 100)$$

$$\text{BAC} = \text{VHAC} \times 0.8 \quad (100 \times 0.8 = 80)$$

ELIMINATION OF ALCOHOL

Alcohol is eliminated through all bodily routes of excretion. 5% is excreted in the breath; 5% in the urine. This is only of practical relevance at high BAC. 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 (**B**) by liver is 15 mg alcohol per 100 ml blood per hour (the equivalent of one unit per hour). However, the range is from 10-40 mg per 100 ml per hour. Convicted drunk drivers average 20 mg/100ml/hour. Genetic and racial differences exist. Liver disease may reduce metabolism.

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**:

- a. The alcohol concentration rises steeply to a distinct maximum (**absorption phase**).
- b. 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.

(c) 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 8 am next morning!

The Blood Alcohol Curve:

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

THE EFFECTS OF ALCOHOL ON THE BODY (PATHOPHYSIOLOGY)

Alcohol is completely miscible with water, enters all cells (except adipocytes) and is toxic to all cells. Metabolism of alcohol generates aldehydes, which are also water-soluble and toxic. Alcohol and aldehyde disturb many biochemical pathways. Alcohol has no nutritive value, depresses appetite and prevents the absorption of nutrients.

Clinical Features of Alcohol Intake:

1. Acute alcohol intoxication
 2. Pathological intoxication
 3. Alcohol abuse
 4. Alcohol dependence
 5. Alcohol withdrawal:
 - a) uncomplicated
 - b) alcohol withdrawal fits
 - c) alcohol withdrawal delirium
 - d) Wernicke's encephalopathy
 - e) Korsakoff syndrome
 - f) alcoholic hallucinosis
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1. Acute alcohol intoxication

This is a transient condition due to acute ingestion of alcohol. Its effects on the brain are of most importance. Psychological and physical effects diminish as alcohol is eliminated from the body. In dependent drinkers, withdrawal syndromes will appear as they begin to sober up.

Alcohol is a nervous system **depressant**. Depression of the brain centres is from above downwards, i.e. affecting first the most recently developed brain functions and at higher levels the vital respiratory and cardiovascular centres in the brainstem.

Cortex - recently evolved brain functions associated with orderly social behaviour (inhibitory).

Depression causes disinhibition, talkativeness, show off ('stimulant' effect). Higher doses reduce anxiety and pain.

Limbic System - memory functions. Depression causes loss of memory, confusion and disorientation.

Cerebellum - muscular co-ordination and speech. Depression causes inco-ordination and slurring.

Reticular formation (upper brain stem) - control of consciousness. Depression causes stupor and coma.

Lower brain stem - vital centres controlling breathing (respiratory centre) and blood pressure (vasomotor centre). Depression may cause death.

Stages of Intoxication

1. Excitement (<100)- loquacious, vivacious, a sense of well being often fostered by the jocular conversational stimulus of friends. A tendency to lose emotional restraint, to forget animosities, converse with abandon, to be less critical and to lose control over one's moral integrity. Feeble jokes and easy affection mark the hour. The ability to pull oneself out of it and to sober up by the force of will (e.g. when confronted with an officer of the law) still exists.

2. Confusion (100-200)- a tendency to come to grief over longer words owing to slight inco-ordination, to slurr speech, to lose control over finer movements, slight blurring of vision, and inability to perform co-ordinated acts, such as writing or driving a car with its usual quick appreciation of danger. Confusion exists over any problem requiring clear thinking and concentration. Emotional upsets become more marked, boasting, loud laughter, coarser jesting, anger and violence begin to appear. The stimulus of surrounding persons usually stir these emotions.

3. Stupor (>200)- the dead drunk stage from which the subject can only be aroused in response to strong stimuli. The subject tends to be anaesthetic and unfeeling to injury and may lie with his face flushed, dribbling from the lips and snoring loudly. Stupor may be followed by coma and death. It is the stuporous stage which is likely to be simulated by cerebral disease or head injury. Indeed, head injury and drunkenness are commonly co-existent.

Physical clues are blood-shot eyes, dilated pupils, rapid bounding pulse, physical inco-ordination and nystagmus (jerking eye movements) on lateral gaze.

There are considerable **inter-individual and intra-individual variations** in the effects of alcohol. Effects also depend on kind of beverage taken, the presence or absence of food, the body weight and build, the rate of elimination. The same BAC may cause different effects in the same individual on different occasions. Possible explanations include induction of liver enzymes, which accelerate alcohol breakdown after repeated social exposure to alcohol, and mood!

Hangover is due to the reversible, toxic effects of ethanol on the brain, gastro-intestinal tract and liver. The clinical features are familiar and include malaise, headache, tremor and nausea. These symptoms are self-limiting and respond to antacids and simple analgesics.

Reasons for legal intervention:

Driving while alcohol exceeds legal limit; drunk in charge of a motor vehicle, ship, cattle, horse, child under 7 years; drunk and disorderly; drunk and incapable; using abusive language; assault (either as assailant or victim).

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.

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

2. Pathological intoxication

A rare condition characterised by a marked change in behaviour occurring within a few minutes of

drinking a small amount of alcohol, one which would not produce intoxication in most people. Behaviour is often aggressive.

3. Alcohol abuse ("problem drinking")

Drinking causes physical, psychological and social problems. Individual continues to drink in spite of the developing difficulties but the criteria for alcohol dependence are not met.

4. Alcohol dependence ("alcoholism")

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.

Features which distinguish the dependent or addicted drinker from the alcohol abuser:
liable to withdrawal symptoms following cessation or reduction in alcohol intake;
use alcohol to avoid or relieve symptoms of withdrawal;.

Criteria for alcohol dependence:

drinking >10 units per day
tolerance to the effects of high blood alcohol concentration
withdrawal syndromes on stopping or reducing intake
repertoire narrowed by drink
compulsion to continue drinking in spite of problems
abnormal laboratory tests

A useful 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. Many have problems in all four areas.

Extreme dependence, with severe physical, psychological and social degeneration, may have two extreme effects:

1. Resorts to increasingly cheap and toxic forms of alcohol, e.g. meths (a cheap combination of ethanol, methanol, methyl violet and pyridine). Methanol is more toxic.
 2. Lapses into petty crime, in order to obtain and conserve supplies.
The criminal alcoholic thus develops. Must be distinguished from the intoxicated criminal, i.e. the person with pre-existing criminal inclinations which are potentiated by alcohol consumption.
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The Addictive Process

Alcohol ingestion provides temporary euphoria and relief from physical and psychological stress. Chronic abuse produces a psychological increase in the desire for more alcohol.

Thus the substance which satisfies the need also increases the need.

Tolerance to the effect of alcohol develops at three levels:

1. Metabolic (liver enzymes are increased).
2. Intra-cellular (nerve cells of CNS undergo metabolic change).
3. Behavioural (learning to maintain control whilst intoxicated).

Tolerance is followed by physical dependence. An early feature is the alcoholic blackout which occurs when the BAC rises above a certain threshold level. May last several hours or even days with complete amnesia afterwards.

Neurones become more excitable to compensate for the depressant effect of chronic alcohol intake. A sharp drop in the BAC may cause the CNS cells to become irritable with subjective craving for alcohol and the onset of withdrawal symptoms. Individuals become comfortable only when the BAC is rising. Impending withdrawal symptoms maintain alcohol intake.

5. Alcohol Withdrawal

Falling BAC after prolonged heavy intake causes cortical and autonomic nervous system excitability.

a) Uncomplicated alcohol withdrawal

Onset follows 6-12 hours after stopping drinking.

Peaks at 48 hours.

Subsides over next few days.

The clinical features include: coarse tremor, nausea & vomiting, malaise, headache, insomnia, weakness, sweating, tachycardia, hypertension, anxiety, depression, irritability, transient hallucinations.

b) Alcohol withdrawal fits

Onset within 12-24 hours

Generalised fits, of unknown cause.

c) Alcohol withdrawal delirium (delirium tremens)

Severe withdrawal syndrome due to cortical and brainstem hyper-excitability.

Onset follows within a week of stopping drinking.

Lasts 2-4 days

The clinical features include disorientation, agitation, visual hallucinations, delusions, tremor, sweating, tachycardia, hyperthermia, cramps, paraesthesiae, nausea and vomiting. Symptoms are typically worse at night. Dehydration and electrolyte disturbances are frequent. Mortality is 15% untreated. Confusion and disorientation may persist.

d) Wernicke's encephalopathy

An acute, potentially reversible, neurological disorder due to thiamine deficiency in alcoholism.

The clinical features include disorientation, clouding of consciousness, ocular abnormalities, nystagmus, ataxia and peripheral neuropathy. Untreated it may be fatal or may progress to chronic Korsakoff psychosis.

e) Korsakoff psychosis

A chronic, irreversible psychotic neurological disorder due to the combined effects of alcohol toxicity and metabolic derangement due to thiamine deficiency. May follow Wernicke's encephalopathy.

The main clinical features are profound impairment of short term memory, inability to learn new information and compensatory confabulation.

f) Alcoholic hallucinosis

A rare condition which may occur while the individual is still drinking heavily or upon stopping drinking.

The main clinical features are vivid and persistent unpleasant auditory or visual hallucinations occurring in a setting of clear consciousness. May last several weeks or months.

Recovery is in three phases

- 1) Drying out period of 1-10 days
- 2) Physical rehabilitation over 10 days to 2 months
- 3) Personality recovery takes months or years

COMPLICATIONS OF EXCESSIVE ALCOHOL INTAKE

Physical, psychological and social complications are not confined to alcoholics, they can affect any individual who drinks heavily for a prolonged period.

a) Physical

1. Gastro-intestinal tract : Alcohol is a direct irritant to the mucosa of the oesophagus, stomach, small intestine. Impairs the nervous supply of the bowel and absorption of many vital nutrients. Causes oesophagitis, gastritis, duodenitis, peptic ulcer, small bowel malabsorption acute and chronic pancreatitis.
2. Liver: fatty liver; alcoholic hepatitis; alcoholic cirrhosis.
3. Cardiovascular System: hypertension; cardiomyopathy and wet beri-beri (thiamine deficiency).
4. Central Nervous System: cerebral atrophy (alcoholic dementia); Wernicke-Korsakoff Syndrome due to thiamine (vitamin B deficiency); cerebellar degeneration, central pontine myelinosis, and peripheral neuropathy.
6. Metabolic Effects: imbalance of metabolism of many bodily compounds including glucose, uric acid, phosphate, magnesium, potassium, fats and proteins.
7. Endocrine Effects: male impotence; female infertility.
8. Others: Severe bruising of various ages due to frequent, unprotected clumsy falls. Typical distribution is over bony prominences of limbs, torso & head.
Microcytic, hypochromic anaemia.
Infections: lobar pneumonia, bronchopneumonia.

b) Psychological

Anxiety, depression, high suicide risk, dementia, pathological jealousy, alcoholic hallucinosis, sexual dysfunction

c) Social

Marital & family problems, including domestic violence
Work problems, unemployment
Road accidents and crime.

CAUSES OF DEATH IN CHRONIC ALCOHOLICS (Clark, 1988)

The cause is not always clear cut. Clark studied the cause of death in 500 chronic alcoholics. 66% were male in the age range 22-83 (average 56); 45% lived alone and 17% held down a regular job. Only 10% could be described as 'down and outs'. 15% had previously received hospital treatment.

Fairly equal distribution between trauma, natural disease, acute intoxication and other alcohol-related disease.

1. **Trauma.** The largest group (26%).

Fire deaths were the most common.

Drunken falls were frequently followed by fatal head injury.

Murder,

Road traffic accidents (pedestrians),

Drowning,

Railway line accidents,

Accidental poisonings, and

Accidental hangings.

Minor Head injury in the course of an assault

Traumatic Basal Subarachnoid Haemorrhage

Hypothermia may follow severe intoxication or injury (Hirvonen 1976). Paradoxical undressing & injuries create suspicion. External bright pink bruising & abrasions on knees & elbows, oedema. Internal pathological features include haemorrhagic stomach erosions, pancreatitis. Microscopic heart muscle damage (myocardial necrosis). Urinary adrenaline & other catecholamines are markedly elevated due to prolonged agonal stress.

Suicide formed a distinct subgroup of 39 cases (8% of all alcoholic deaths). Most were intoxicated at the time of death and were of a younger average age (47 years). Preferred method was a drug overdose (50% of cases).

2. **Incidental Natural Disease** (25%). Ischaemic heart disease, cerebral haemorrhage, chronic obstructive airways disease and malignancy.

3. **Alcohol Related Disease** (22%). Bronchopneumonia and lobar pneumonia are the commonest. Cirrhosis of the liver due to ruptured varices or hepatic failure. Many of these deaths occur in hospital and are excluded from forensic practice. Alcoholic cardiomyopathy and pancreatitis are other rarely reported causes of death.

4. **Acute Intoxication** (24%). Simple intoxication causing respiratory depression was responsible for 72 cases (seventh of the total). The BAC was 261-612 mg% (average 450). Those with a BAC below 318 mg% had co-existing ischaemic heart disease, chronic obstructive airways disease or cirrhosis. Inhalation of vomit was the primary cause of death in 18 cases. The BAC was as low as 152 mg% in these cases.

Possible mechanisms of death from simple intoxication:

1. Simple depression of the respiratory centre in lower brain stem by alcohol itself.

2. Inhalation of vomit due to coma.

3. Postural asphyxia (obstruction of the upper airway by the swallowed tongue during coma).

Heatley & Crane (1990) found the mean BAC amongst 175 fatal acute intoxications to be **355 mg%**. The levels were lowest in cases complicated by aspiration (inhalation) of stomach contents into the respiratory tract (bronchial tree). Levels were highest in chronic alcoholics, due to acquired tolerance.

The level measured at autopsy does not necessarily reflect the level causing death (Johnson, 1985). The post mortem level reflects the level at the time of death, but death may have occurred after a long period of coma, up to several hours after the peak BAC was reached.

Prolonged coma prior to death may be followed by postural asphyxia or inhalation of vomit. A 7 hour delay may allow BAC to drop by $7 \times 20 \text{ mg\%} = 140 \text{ mg\%}$. The UAC may be higher than the BAC in these cases.

5. '**Obscure**' cause of Death is noted in a variable proportion of cases, up to 10%. Many cases show fatty liver only and no convincing cause of death. Death can be attributed in these cases to the numerous severe metabolic and biochemical disturbances caused by alcoholism (Thomsen, 1996).

ECG changes are common in alcoholics and death may be due to a disturbance in the electrical rhythm of the heart (Day et al, 1993).

Interpretation of post mortem blood alcohol levels is complicated by 2 important artefacts.

1. **Post mortem redistribution of unabsorbed alcohol in the stomach** (or in the airways following aspiration of stomach contents) passing by diffusion into central blood vessels (heart, inferior vena cava, pulmonary artery, pulmonary veins, aorta). There can be up to 400% difference between central & peripheral sites. Blood should be taken from peripheral veins (femoral vein in pelvis) to avoid this possible artefact.

2. **Post mortem Microbial alcohol production.** Activity due to bacteria & yeast present in the bloodstream, acting on glucose and lactate, can result in spurious alcohol production in the the body (in vivo?!) or within a specimen tube (in vitro).

Production favoured by warm environment, septicaemia (bacteria in bloodstream at time of death), hyperglycaemia (substrate for fermentation), severe disruptive trauma (allows spread of bacteria). Production inhibited by refrigeration, use of Fluoride/Oxalate preservative in collection tube (prevents further fermentation). Levels up to 70 mg/100 ml are commonly seen. Rarely upto 150!

These artefacts make it necessary to corroborate the measured BAC by simultaneous measurement of Urinary & Vitreous alcohol concs.

(Reference: D.J. Pounder. Dead Sober or Dead Drunk; May be hard to Determine. BMJ; 316: 87 (10 January 1998)

DRINKING AND DRIVING (McLay, 1990)

10% of all accidents involving injury result from driving with excess alcohol.

33% of drivers and motor cyclists killed are over the legal limit.

66% between 10 p.m. and 4 a.m. on Friday and Saturday nights.

1,000 people are killed every year.

30,000 are injured.

Cost to community is over £360 million per year.

Effect of alcohol on performance

The unfortunate paradox is that alcohol gives a feeling of physical well-being whilst actually depressing brain function, lessening muscular control and co-ordination, and lengthening reaction time. Vision is blurred and awareness decreased, especially in the dark. The ability to judge speed and distance and to deal with the unexpected are impaired. These factors adversely affect driving performance. Impairs judgement so that driver believes that he is driving well.

Accident Risk

Research done in the 60's. The risk of being involved in an accident increases sharply beyond the legal limit. The likelihood of an accident, and of it being serious, increases disproportionately at higher levels. For young or inexperienced drivers, the risk increases from well below the legal limit, even after the first drink.

At the legal limit the accident risk is doubled. For inexperienced or young drivers, the increased risk is five-fold.

At twice the legal limit the relative accident risk is 20 times that of the sober driver.

This relationship led to the introduction of a legal limit in the Road Safety Act of 1967. This was a major legislative landmark.

HISTORY

The offence of being drunk and incapable was first recognised in statute during 18th & 19th centuries. Drunkenness in charge of dangerous machinery, animals and the like was prescribed. e.g. carriage, horse or steam engine. 1872 penalties imposed for drunkenness in persons having responsibility for others, i.e. engine drivers, ships masters, taxi drivers and service personnel.

The **Criminal Justice Act 1925**, penalised a person drunk in charge on any highway or any public place of any mechanically propelled vehicles. Definition of "drunk" was open to interpretation and only those obviously drunk were prosecuted.

The **Road Traffic Act of 1930** recognised that it was necessary to be properly "drunk" and Section 15 states

" any person who when driving or attempting to drive or when in charge of a motor vehicle on a road or other public place is under the influence of drink or drugs to such an extent as to be incapable of having proper control of a vehicle [shall be guilty of an offence]

In 1954 the BMA committee produced a report "**The Recognition of Intoxication**",. Many accidents caused at alcohol levels insufficient to be noticed by the police. It outlined:

1. A model for clinical examinations, (to exclude illness), and
2. considered the techniques available for alcohol analysis in body fluids.

The **1956 RTA** gave the expression "unfit to drive" as being under the influence of drink or drugs to such an extent as to be incapable of having proper control of a motor vehicle.

1960 BMA report "Relation of Alcohol to Road Accidents" discussed the growing evidence of the effects of relatively small amounts of alcohol on impairment. Recommended a fixed BAC above which it would be an offence to drive

The **1962 RTA** required Courts to have regard for the proportion of alcohol or drugs in the body as evidenced by blood or urine levels.

The **1967 Road Safety Act** introduced the Breathalyser as a screening test which if positive was followed by an evidential blood or urine test. Made it an offence to drive or be in charge of a motor vehicle in a public place if the alcohol level was above 80 mg/100 ml of blood or 107 mg/100 ml of urine

The **RTA 1972**, amended by the **Transport Act of 1981** introduced evidential breath analysis. If a roadside breath screening test was positive there was a requirement to provide two breath samples at a police station for direct alcohol analysis. Also became an offence to refuse to provide such specimens of blood or urine when required to do so .

35 µg of alcohol per 100 ml of breath became the legal limit

A further report in **1976** made two recommendations, implemented in **1983**:

1. Breath testing instead of blood or urine sampling as the routine evidential specimen.
2. The recognition of the high risk offender who exceeded the legal limit on two occasions with levels greater than 200 mg per 100 ml. Driver would have to convince the DVLC that drinking was under control before restoration of licence.

Current Legislation

The 1972 Act was replaced by the **1988 Road Traffic Act**, The Road Traffic Offenders Act and the Road Traffic (Consequential Provisions) Act.

Section 4(1) of the Act states

1. A person who when driving or attempting to drive a motor vehicle on a road or other public place, is unfit to drive through drink or drugs.

Used when it is not possible to determine alcohol levels or when the driver is suspected of being under the influence of drugs. The definition of Drugs is any intoxicant other than alcohol. e.g solvents, tranquillisers etc.

Section 5(1) *If a person :-*

a) drives or attempts to drive a motor vehicle on a road or other public place, or

b) is in charge of a motor vehicle on a road or other public place, after consuming so much alcohol that the proportion of it in his breath, blood or urine exceeds the prescribed limit then he is guilty of an offence.

The Legal Limit

35 µg per 100 ml of **breath**.

80 mg of alcohol per 100 ml of **blood** (7.4 mmol/l).

107 mg of alcohol per 100 ml of **urine**.

It is an offence for a person without reasonable excuse to fail to provide:

- (a) a roadside screening sample of his breath under the required circumstances (section 6, RTA);
- (b) two specimens of breath for analyses by means of an approved device. The current approved devices are the Lyon Intoximeter 3,000, and the Camic Breath Analyser.
- (c) A specimen of blood or urine for laboratory testing (section 7, RTA).

It is an offence to ride a pedal cycle, not being a motor cycle, on a road or other public place while unfit to ride through drink or drugs (section 30, RTA).

Police Procedure

A constable in uniform may require a person driving, or attempting to drive, or in charge of a motor vehicle, on a road or other public place, to take a breath test if he suspects that that person

- (a) of having been drinking, or
- (b) of having committed a moving traffic offence
- (c) where an accident has occurred,

a constable in uniform may require any person whom he has reasonable cause to believe was drinking and attempting to drive or was in charge of a vehicle at the time of an accident, to take a screening breath test.

A person who (a) is unfit to drive, or
(b) has provided a positive breath test, or
(c) has refused/failed to take a breath test
will be arrested and taken to a police station.

At the police station, driver will be required to provide two evidential specimens of breath or, if the machine is not available, a specimen of blood for analysis. The subsequent procedure depends on which offence appears to have been committed.

Section 5, RTA, (driving while alcohol exceeds the prescribed limit). The police request the driver to provide two specimens of breath. The lower of two breath tests is used in evidence. In Scotland the Camic breath analyser is used. Analysis carried out by trained police officer at a police station. The machines are self-calibrating. Calibration and zero checks are printed on the analysis form, a copy of which is given to the driver.

If BrAC is 35-50 µg/100 ml driver has the option to replace the breath result with a laboratory test on blood or urine. Which option is at the discretion of the police officer.

The police surgeon is called to take a blood sample, and must have the driver's consent. No clinical examination is carried out to assess the driver's fitness to drive. Part of the blood sample may be handed to the driver for private analysis.

The blood samples are placed into a vial containing preservatives such as sodium fluoride to prevent changes in alcohol concentration by bacterial activity. Urine samples are treated in the same way. Blood and urine samples are then sent for laboratory analysis usually using gas chromatography.

Section 4, RTA (driving while unfit through drink or drugs).

When a driver gives a negative breath test at the police station, but the custody officer still considers that there is still evidence of impairment, the subject will be examined by a police surgeon.

In these cases the police surgeon normally has two tasks, both requiring consent.

1. to assess by examination the subject's fitness to drive, and
2. to take a specimen of blood for analysis if he concludes that fitness is impaired.

The driver is made aware that the police surgeon is examining the driver on behalf of the police. Driver may call another doctor on his own behalf, whose fee he must pay.

Failure to provide any specimen constitutes an offence tantamount to having alcohol in the specimen above the prescribed limit.

Specimens required in hospital can only be obtained with authority of the medical practitioner in charge of the case who may object if undertaking the test, or the warning given, would be prejudicial to the care of the patient.

If urine is required it must be taken within one hour of the incident, after previously providing a specimen which is discarded. This avoids the specimen being contaminated with urine secreted into the bladder by the kidneys at some time before the accident and possibly containing more alcohol than was exerting its influence at the time of the accident.

Whenever a result is less than 100 mg/100 ml, 6 mg/100 ml is automatically deducted. Levels above 100 mg/100 ml have 6% of the actual figure deducted. The result is then expressed as "not less than". This eliminates the argument of experimental error analysis.

The top Ten Defences offered by drivers (Jones, 1991):

- 1) Drink taken after the offence "to calm the nerves" **The Hip Flask Defence or Post Accident Drinking**
- 2) Drinks laced
- 3) Inhalation of alcohol vapours at work
- 4) Natural disease or trauma affecting alcohol breakdown
- 5) Contamination by use of skin antiseptic containing alcohol
- 6) Alleged mix-up of specimens (at police station or lab)
- 7) Post-sampling alcohol production in container
- 8) Alcohol breakdown impaired by prescribed drugs
- 9) Consumption of alcohol-containing tonics, elixirs
- 10) Infusion of fluid in hospital

The Hip Flask Defence or Post Accident Drinking

Section 15(3) of the Road Traffic Offenders Act 1988 provides

If the proceedings are for an offence under section 5 of the Act or for an offence under section 4 in a case where the accused is alleged to be unfit to drive through drink that assumption shall not be made if the accused proves-

- a) *that he consumed alcohol after he had ceased to drive, attempt to drive or be in charge of a motor vehicle on a road or other public place and before he provides a specimen and*
- b) *had he not done so the proportion of alcohol in his breath, blood or urine would not have exceeded the limit and if proceedings are for an offence under section 5, would not have been such as to impair his ability to drive.*

BAC can be estimated from the amount of alcohol consumed using the Widmark equation.

Back Calculation: variable popularity. Widely used in Germany. 100 calculations per year in Glasgow. Need to know amount consumed before & after crash, times of drinking & crash, BAC, rate of elim., weight & build.

Example: two 125 ml glasses of 12% vol wine at 8 pm; third at 9 pm, fourth at 10 pm. Accident at midnight. Triple whisky to steady nerves at 0030 (85 ml of 43% vol). Police breath tests 0100. Blood test

at 0130.

Calculate alcohol content in each drink. Calculate what BAC it is responsible for. Construct a BAC curve.

Assume elimination at 15 mg/100 ml/hour.

Extrapolate the line back to the time in question and subtract BAC due to post accident drinking.

Providing the BAC does not reach zero it is possible to construct a simplified curve by assuming all alcohol was taken at time at once!

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