CHAPTER 10

SUBSTANCE ABUSE AND DEPENDENCE

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Humans have abused drugs for thousands of years. Wine has been used from at least the time of the early Egyptians, narcotics from about 4000 BC and cannabis was first used medicinally around the third millennium BC in China. These substances are still used and abused today, but the list of abused substances has grown and new "designer drugs" are still being developed. It is essential that every doctor has a good working knowledge of the substances of abuse and the management of those who abuse them.

How big is the problem of substance abuse in South Africa?

South Africans consume 4-billion litres of alcoholic drinks a year, representing two litres a week for every man, woman and child. South Africa is ranked 34th in the world in terms of per capita consumption of alcohol. Since these statistics do not include sorghum beer, a popular home brew in rural South Africa, and most of the countries listed are developed countries (in which a higher proportion of the population drinks), and these statistics do not include sorghum beer, a popular home brew in rural areas our true consumption per drinking person is likely to be even higher that these estimates suggest. Studies showed that two thirds of 12-17 year olds in Cape Town admitted to an alcohol binge (episode of excessive use) within the previous month.

The use of illicit drugs, especially the so-called "hard drugs", has increased in South Africa. This is reflected by the increased demand for treatment, especially marked in younger people. There is especially an increase in demand for in the under-twenty years age group. (There was an 18% point increase in demand for drug treatment for children under 20 years in Cape Town between 1996-2003.)

Substance abuse is associated with a high morbidity for the client, the family, the health care services and society at large. As many as 8% of men and 3% of women in primary care present with alcohol related problems. In 69% of cases of abused women in the Western Cape, alcohol or drugs were the main cause of conflict. Over half of the un-natural deaths in Cape Town and a third of trauma patients were found to have blood alcohol levels above the previous legal drink drive limit of 0.05g/100ml. More than 50% of cadavers in mortuaries have positive blood alcohol. A study in Wellington of Grade 1 learners showed a rate of Foetal Alcohol Syndrome of 40/1000 (18-141 times higher than that found in the various states of the USA). A study found 61% of trauma unit patients in South Africa to have a blood alcohol level above the drink drive limit and 40 % tested positive for drugs. Seventy percent of people arrested for motor vehicle theft or house breaking drug testing was positive for drugs other than alcohol. A third admitted to being intoxicated at the time of the crime and felt it had given then courage to commit the crime. Studies from abroad confirm the huge financial burden on society associated with substance abuse.

A few definitions

Intoxication: A reversible, substance-specific syndrome due to the recent use of a substance.

Abuse/harmful use: When the use of a substance causes biological (physical), psychological or social damage. (E.g., the addict fails to fulfil important obligations at work, school or home, has legal problems or social or interpersonal problems due to substances, etc.)

Tolerance: Physical adaptation of the body to heavy and repeated use of a substance where more of the substance is required for the same effect or the same

amount has a reduced effect.

Withdrawal: A substance specific syndrome due to cessation of or a reduction in substance use after heavy and prolonged use.

Reinstatement: When a sober addict, after a period of abstinence (that may be years), uses the substance again, tolerance and withdrawal develops again very rapidly.

Dependence: A maladaptive pattern of substance use which can be recognised by a cluster of signs and symptoms, including signs of

- physical adaptation of the body to the drug (tolerance, withdrawal),
- loss of control over taking of the drug (taking more or for longer than intended, desire or unsuccessful efforts to cut down or control use),
- and where the drug has taken over the person's life (a great deal of time is spent obtaining or using the drug or recovering from its effects; important activities are given up because of the drug; and the drug is used despite knowledge that it is harmful).

Classification of substances of abuse

- Depressants e.g., alcohol, benzodiazepines, barbiturates, Mandrax
- Cannabis
- Opiates (narcotics) e.g., heroin, morphine, pethidine, opium, methadone, codeine
- Stimulants e.g., cocaine, amphetamines, caffeine, nicotine, appetite suppressants
- Hallucinogens e.g., LSD, PCP (angel dust), psilocybin (magic mushrooms), mescaline
- Solvents
- Designer drugs e.g., ecstasy
- Prescription drugs e.g., antipsychotics, anti-cholinergics, antihistamines, antidepressants.
- Over-the-counter drugs e.g., analgesics, anti-diarrhoea drugs, laxatives
- Other e.g., steroids

Why do people become addicted?

There are various reasons why people end up addicted to a specific substance. Although the decision to experiment with drugs is usually voluntary, it is important to remember that no one uses the drug with the intent of ending up an addict. The reasons for drug use are a complex interplay between factors relating to the specific person, his or her environment and the specific drug. See Figure 1 for some examples.

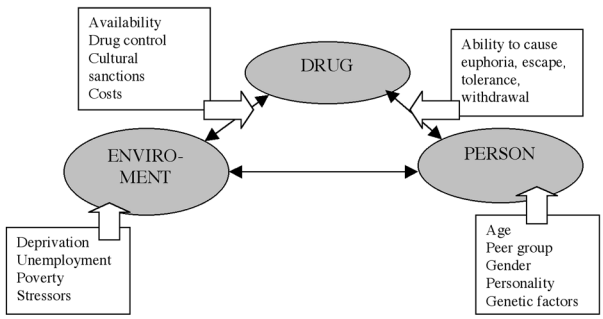

Figure 1 Some of the reasons for addiction

What happens in the brain?

- Neurons communicate with other neurons at synapses. To send a message, a neuron releases a chemical messenger, or neurotransmitter, into the synaptic cleft. From there, the neurotransmitter crosses the synapse and attaches to key sites called receptors on the next neuron in line. When neurotransmitters attach to these receptors, they cause changes inside the receiving neuron and the message is delivered. The chemicals in drugs can disrupt this complex communication system within the brain.
- Drugs of abuse activate a part of the brain called the reward system (see Figure 2), and that makes you feel good. Activation causes neurons to release large amounts of dopamine (see Figure 3). Over time, drugs damage this part of the brain. Because of this damage, things that used to make you feel good no longer feel as good. The reward pathway starts in the ventral tegmental area (VTA). The information travels from the VTA to the nucleus accumbens and then up to the prefrontal cortex. This is the final common path for all substances of addiction.
- Neuromodulators are chemicals that can help to enhance or inhibit the neurotransmission that is controlled by neurotransmitters such as dopamine. Endorphins are examples of neuromodulators. Endorphins bind to endogenous opiate receptors that are found on the post-synaptic cell or, in some cases, on the terminals of other neurons. Opiate receptor activation decreases GABA release, which normally inhibits dopamine release, so dopamine release is increased. This is how opiates, like heroin, have their effects on the reward path.
- Stimulants increase dopamine release by inhibiting the re-uptake transporter responsible for removing dopamine from the synaptic cleft, thereby increasing the dopamine available in the synaptic cleft. (Other catecholamines are increased in a similar way. This

explains the sympathomimetic clinical signs of stimulant intoxication, like tachycardia and hypertension).

- Some drugs of addiction cause long-term structural changes in the brain. Long-term alcohol abuse can cause permanent changes in the lipophilic neuronal cell membrane. Stimulants can cause nerve terminals to be "cut back" with limited regrowth.
- Drugs of abuse act not only on the reward pathway, but also disrupt important brain functions like movement, balance, coordination, memory, judgment and sensations. This explains some of the symptoms of intoxication and some of the long-term effects.
- This is a simplified explanation of what happens in the brains of addicts. Depending on the substance used, various other neurotransmitters (e.g., serotonin, norepinephrine and glutamate) can also play a part.
- Dependence occurs when the neurons have adapted to repeated drug exposure and can only function normally only in the presence of the drug. When the drug is not taken, a physiological reaction (withdrawal) takes place.
- After repeated drug use, you reach a point where deciding to use drugs in no longer voluntary. The prolonged drug use has produced long-lasting changes in the brain. Eventually it becomes difficult to experience other pleasures.
- Craving is increasingly being recognised as an important part of the addictive process. If the addict is exposed to a trigger (something associated with the drug use, like the alcoholic being near his old pub) craving may develop. Rational thoughts are overwhelmed by the uncontrollable desire for drugs. The amygdala, (a part of the limbic system critical for memory and emotions) is activated when craving occurs.
- Addiction is thus a brain disease. It is a chronic relapsing disease characterised by compulsive, often uncontrollable, drug seeking and drug use in the face of negative consequences.

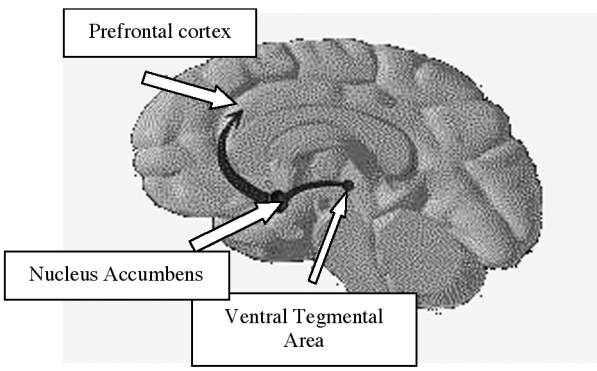

Figure 2 The reward pathway

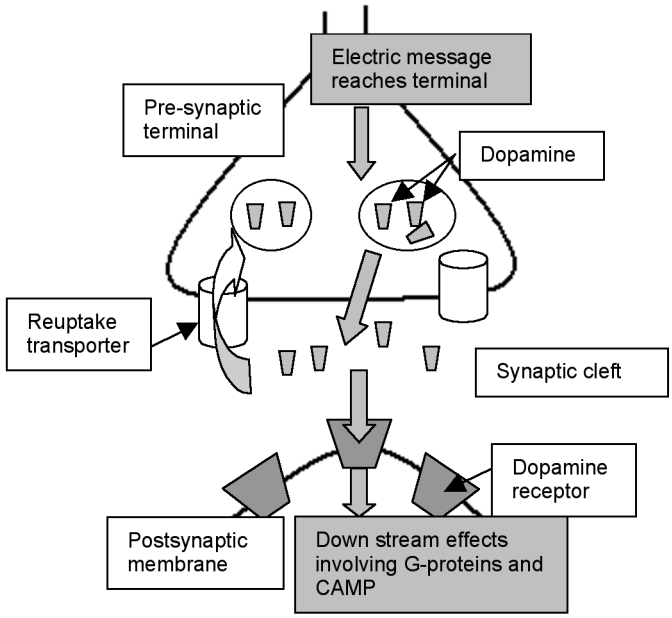

Figure 3 The Dopamine synapse

The spectrum of disease progress from experimentation to addiction

Although people may inherit a vulnerability to becoming addicted, they do not start off addicted. They start by trying out a substance, liking its effects and using it recreationally. They may move on to using it in a manner that is harmful to them, but can stop or use it in a controlled manner if they decide to do so. Once they become dependent, they are never able to use in a controlled manner ever again. (See Figure 4.)

If we take alcohol as an example, roughly 95% of the adult population in the Western Cape experiment with alcohol. More or less 50% of them use in a harmful manner, and 10-15 % become dependent on alcohol.

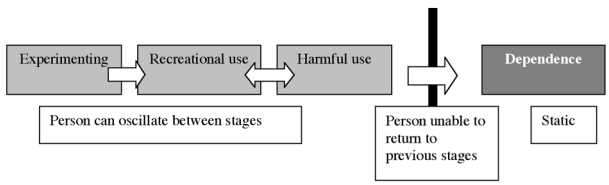

Figure 4 Spectrum from experimentation to addiction

Poly-substance abuse

Many substance abusers misuse more than one substance, but usually have a substance of choice. Some addicts abuse whatever drug is available while others choose combinations for a specific desired effect. With some combinations, one substance is used to counteract the side-effects of the other. An example of this is the abuse of depressants (like alcohol or benzodiazepines) is abused to counteract the activating effect of stimulants (like cocaine or amphetamines). Other addicts may replace one drug with another in the same class if the first drug is unavailable. Examples include the abuse of methadone when heroin is unavailable, benzodiazepines when the client wants to stop drinking alcohol. Poly-substance misuse can be dangerous and may complicate detoxification and rehabilitation.

Comorbidity

Substance abuse and addiction are associated with very high rates of comorbid medical or psychiatric disorders (subjects suffering from the latter being referred to as having dual diagnoses). There are various factors that may account for the high rates of comorbidity between drug abuse and psychiatric disorders. First, substances can be used to alleviate psychiatric symptoms or side-effects of psychiatric medications; second, the drug may actually cause the psychiatric disorder; third, because both problems are relatively common, the co-occurrence of the two disorders may be coincidental. A fourth possibility is that an underlying genetic or biochemical defect might predispose someone to both drug abuse and a mental disorder.

In dually diagnosed subjects, it may be difficult to decide what came first, the substance abuse disorder or psychiatric disorder (i.e., which is primary and which is secondary). A careful and detailed history and collateral history may help. It is increasingly recognised that patients with dual diagnoses require a special approach to the management of both their addiction and their psychiatric disorder, and if both are not managed, relapse of either disorder is more likely. Common comorbid disorders include mood disorders, anxiety disorders, psychotic disorders, delusional disorders (like morbid jealousy), sleep or sexual disorders and personality disorders.

As mentioned, substances can cause psychiatric disorders. These psychiatric symptoms can occur during intoxication or withdrawal or may even persist for weeks or months after the substance has been withdrawn. These disorders are generally managed by withdrawal and avoidance of the offending substance. If symptoms persist, medication is required.

Evaluation of the addict

- Research has shown that doctors do not take adequate drug and alcohol histories, do not identify chemical dependency as a medical problem, and do not involve themselves in treatment recommendations when a substance problem is identified. Researchers feel the reason for this is that addicts represent an "exercise in futility" for doctors who specialise in treating acutely ill or injured patients. These patients makes them feel hopeless and in order for them to deal with these feelings of hopelessness, cognitive restructuring takes place so that addicts are viewed as a "management problem" rather than "ill" and the physician therefore feels justified in dismissing their need for diagnosis and medical management. In the doctor's mind, the addict has been dealt with successfully when he or she is dismissed and staff time and resources are held to a minimum. This attitude does not help our addicted patients! It is thus essential for every doctor to have a good working knowledge of the evaluation and management of addiction in order to combat these "hopeless" feelings and manage patients with confidence.
- We all have feelings towards addicts that stem from our own childhood and prior experiences, our own habits, our ideas about morality and our unique personalities. These feelings are often negative and interfere with our ability to care for our addicted patients. It is thus important to always remember that addiction is a brain disease. No one becomes an addict on purpose. In order to treat our patients we have to recognise these feelings and make sure that they don't interfere with our helping of patients.
- Remember that addicts usually do not have a realistic understanding of the extent of their problems and denial is not devious manipulative lying. It is simply too difficult for them to consciously accept what they have become as a result of their habit. They use various defence mechanisms to "protect" themselves from this awful reality. They may minimise their problem, intellectualise, rationalise or project the problem onto something or someone else. It is therefore important to get collateral history in order to truly understand the extent of the problem.
- The interview with the addict should be constructive, not destructive and should be aimed at improving insight. Insight motivates change. Punitive criticism increases denial. Through a trusting relationship the doctor can gradually increase awareness of the problem and nudge the addict in the right direction.
- Evaluation should include a complete medical, surgical and psychiatric history. Remember to ask about risky behaviour for HIV (needle sharing or prostitution). Also enquire about social problems or forensic problems as a result of the abuse.
- A detailed physical examination is always indicated. Many drugs can be injected intravenously (called

"spiking"). Injection of drugs with needles that are not sterile leads to the potential for contracting a wide variety of infections, including HIV Hepatitis B and C and bacterial infections. Look for needle puncture marks, tracts (scars located along veins, see Figure 5), tattoos to hide tracts, hand oedema (using veins on hands and fingers to "spike"), thrombophlebitis, abscesses and ulcers, ulceration or perforation of the nasal septum (from snorting drugs), cigarette burns/scars (due to drug-induced drowsiness), cheilosis (especially with amphetamines and opiates), contact dermatitis ("glue-sniffer's rash"), jaundice and monilial infections.

- Medical complications of drug abuse include endocarditis, myocarditis, arrhythmias, arteritis, pulmonary oedema, aspiration pneumonia, hepatitis, cirrhosis, menstrual irregularities, seizures, rhabdomyolysis, tetanus, aplastic anaemia, pancreatitis et cetera. Heroin can produce a nephropathy that resembles focal segmental glomerulosclerosis. A "talc granulomatosis" can occur because many injected drugs have been adulterated with an inert substance (such as talcum powder) to "cut" or dilute the amount of drug. Intravenous drug abuse is also associated with an increased incidence of tuberculosis.
- Special investigations may be performed if indicated.
- Addicts often use bravado, aggression and an uncooperative attitude to cope with their poor self esteem. Don't get angry, frustrated or over-involved. A respectful and caring attitude may evoke a much better response.

Figure 5 Tract marks on the arms of a chronic intravenous heroin addict

Laboratory investigations for drugs

Breathalysing the patient or sending blood to the laboratory for alcohol levels can confirm alcohol consumption. Most other drugs are tested using urine analysis. Dipsticks tests are generally less reliable than GC-MS (gas chromatography linked with mass spectrometry). Saliva and hair analyses are also available and tests for sweat are being developed.

Approximate durations of detectability after use:

- Amphetamine 2-3 days
- MDMA (Ecstasy) 30-48 hrs
- Cocaine 6-8 hrs
- Heroin 2-4 days
- Methadone 7-9 days
- Codeine/morphine /dihydrocodeine 24 hrs
- Cannabinoids: May vary from 3 days after single use to 36 days after heavy consumption
- Benzodiazepines: Depends on $T^1/_2$: short-acting 24 hrs, long-acting >7 days

Management of the addict by the primary care practitioner

1. Don't miss the addict:
   - Always routinely ask about alcohol, tobacco, prescription drugs, over the counter drugs and illicit drugs
   - Use screening tools (like CAGE for alcohol)
   - Obtain a collateral history if you suspect a problem
   - Routine laboratory tests (such as an elevated MCV or abnormal liver function tests in alcohol abuse) may raise suspicion
   - Urine, blood, saliva or hair analysis can be done.
2. Medical management - treat physical complications, overdoses.
3. Screen for and treat comorbid psychiatric disorders.
4. Encourage treatment for the addiction - use brief interventions or motivational interviewing (see below)
5. If treatment is refused, consider harm reduction (see below) or committal (see below).
6. Detoxification may be indicated if the patient is determined to change (see below).
7. Have a directory of specialised agencies available to refer for more specialised care if indicated. Most patients that reach the stage of dependence are unable to stop without further help and need rehabilitation to sustain sobriety. It is important to refer patients on for this.
8. Ensure that you don't enable patients to continue their habits by, for example avoiding addressing the problem because you feel embarrassed.
9. Involve the family if indicated.
10. Avoid the iatrogenic creation of addicts by reckless or inappropriate prescribing of benzodiazepines or analgesia. It is best to avoid prescribing addictive substances to patients with a history of addiction. (Opiates are over-prescribed and often not very

Table 1 Brief interventions as described by Bien

Feedback	**Feedback** of personal risk or impairment	"Your liver is enlarged and not functioning properly and the tests show that too much alcohol is the most likely cause for this"
Responsibility	Emphasis on personal **responsibility** for change	"I feel concerned about you, but it is really up to you to do something about this"
Advice	Clear **advice** to change	"I suggest that you stop drinking"
Menu	A **menu** of alternative change options	"If you want help to quit you can phone this number. Alternatively, you can try to stop by yourself"
Empathy	Therapeutic **empathy** as a counselling style	
Self-efficacy	Enhancement of patient **self-efficacy** or optimism	"I believe that you can do this"

successful in chronic pain. Anxiety disorders are better managed with antidepressants such as SSRI's and Cognitive Behavioural Therapy than by repeated benzodiazepine prescriptions.)

Brief interventions

Studies have shown that structured brief interventions in primary care can be of great value in the treatment of addiction. Doctors in primary care have more opportunities to intervene (addicts consult doctors more frequently). Studies have shown that 5-10 minutes of advice leads to a 25-35% reduction in drinking at 6-12 months.

Bien described the elements of effective brief interventions by using the acronym FRAMES (see Table 1). He also suggested that doctors should offer patients a brief follow up appointment after this intervention to monitor progress and encourage and support the patient.

Motivational interviewing

Motivational interviewing (MI) is increasingly recognised as an effective tool in interventions for substance abuse and addiction (and for changing other undesired behaviours e.g., non-compliance, over-eating). It is important for all doctors to ensure that they understand the principles involved and can use them.

The first principle in MI is respect, warmth, sympathetic understanding and positive regard by the therapist for the patient, allowing the patient to make his or her own choices.

MI is based on the "Stages of change" model described by Prochaska and DiClemente. (See Figure 6.) Interventions are likely to have a better outcome if they are appropriate to the client's stage of readiness for change. Rehabilitation from addiction will work better if the client is determined to change and the idea with MI is to get the patient to that stage.

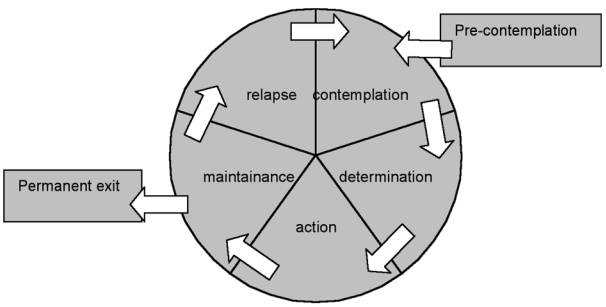

Figure 6 The cycle of change (Prochaska and Di Clemente)

Stages in the cycle of change:

- Pre-contemplation: The stage at which people are unaware that they have a problem and thus do not feel that there is any point in changing.
- Contemplation: The stage at which the patient begins to weigh up pros and cons of his or her habit and feels somewhat ambivalent about his or her behaviour.
- Determination: The stage at which the balance is tipped and the patient decides to do something (or nothing) about the habit.
- Action: The stage at which a strategy for change is chosen which the patient pursues.
- Maintenance: the stage at which efforts are made to maintain the gains made in order to avoid returning to previous undesired behaviour. A patient can either maintain gains and exit the cycle permanently or lapse/relapse.
- Relapse: Relapse is recognised as a frequently occurring event. If viewed as a positive learning experience, it increases the chances of success the next time round.

MI was described by Miller and is especially useful in the pre-contemplation and contemplation stages of change. It embraces Egan and Rogers' behavioural philosophy "I learn what I believe as I hear myself talk". The patient is helped to discover for himself that his habit is harmful and this motivates change.

If a person perceives his behaviour as being discrepant with his beliefs, attitudes and feelings, there is a tendency to restore this discrepancy by changing one or more elements of this behaviour. MI aims to create such a state of ambivalence, which the patient would then (hopefully) resolve by way of changing the undesired behaviour (stop drinking or drugging). There is a de-emphasis on labelling. ("You are an alcoholic") Emphasis is rather placed on creating ambivalence regarding his substance habit, thus hopefully generating an internal desire to change. The therapist actively recognises and respects the client's capacity to make his own decisions.

Miller described five elements to MI:
1. Express empathy (acceptance facilitates change, use skilful reflective listening)
2. Avoid arguments (they are counterproductive and increase resistance, don't label e.g., "Alcoholic")
3. Develop discrepancy (discrepancy between behaviour and goals motivate change, the patient should present the arguments for change)
4. Roll with resistance (use momentum, new perspectives are invited, not imposed, the client finds solutions to problems; "How would you know that you are in trouble?")
5. Support self efficacy

Early strategies used include:
- Reflective listening ("if I understand correctly, you feel that...")
- Ask open ended questions ("how would this make you feel..." rather than "does this make you feel unhappy")
- Affirm ("you can do this if you want to...")
- Summarise (especially feeling of ambivalence: "On the one hand you feel...; on the other hand you want to...")
- Elicit self-motivational statements ("What would be the best thing that would happen to you if you were to stop using substance X?")

Avoid:
- Just asking questions and getting answers
- Confrontation - this just increases denial
- Approaching the patient from a position that you are superior and the expert
- Labelling the patient (i.e., alcoholic, addict)
- Allowing the patient to blame others or blaming him/her

Useful steps to follow:
1. Assess the patient's perceptions of the good things about his or her substance use. ("What are the nice things that drinking does for you?")
2. Facilitate the generation of the patient's inventory of the less good things about the behaviour ("What are the things that make you worried about your drinking?")
3. Elicit the patient's current satisfaction with his lifestyle vs. that previously envisaged and anticipated for the future. ("Where did you plan to be today ten years ago?")
4. Which of the elicited problems are real concerns to the patient? ("Which of the things we discussed makes you really worried?)
5. Compare and contrast the benefits and costs of continuing the behaviour (a cognitive evaluation of the current situation) ("your social life is arranged around drinking and you don't want to lose that, but it is preventing you from achieving what you have planned for your life and, furthermore, the doctors say that your liver is being affected by it")
6. Reflect on areas of greatest concern and discrepancy, thereby generating discomfort for the patient with current behaviour (an emotional overview) ("So although you like the way that alcohol makes you feel relaxed, you feel worried that you cannot stop once you start and the way you treat your family when intoxicated makes you feel really guilty")
7. Elicit and agree on future intentions regarding the behaviour. ("I agree that stopping the drinking is the right choice for you. How will you do this?)

Harm reduction

If treatment is refused, it may be of value to reduce the harm from the addiction until the patient is ready for help. Examples include a vitamin prescription; a social worker referral to look at housing, family support and other social needs; education about the risks of infections and overdoses; testing for HIV and Hepatitis B and C. There are needle exchange programs and methadone clinics abroad that aim to engage patients into treatment. (This is not available in South Africa at present.) Patients, who use drugs and are unwilling to stop, can be encouraged to stop injecting. If they are unwilling to do this, they can be encouraged to stop sharing needles and if they are unwilling to do this, they can be encouraged to clean the equipment first.

Committal

Substance abusers who cause harm to themselves or their families, but are unwilling to receive help may be legally committed for treatment under Section 22 of the Prevention of Drug Dependency Act. This is different from certification under the Mental Health Care Act. It is a lengthy process (can take a month or longer) and also involves a court of Law. The relative must first obtain an application form from the magistrate's office and this is then handed in at the nearest social services

office. A social worker examines the case and makes a recommendation to the magistrate regarding treatment.

Detoxification

Detoxification is indicated if withdrawal symptoms are poorly tolerated (thus predisposing to relapse) or dangerous, and a substitute drug is available that is less harmful than the original drug. Substitute drugs usually have a longer half-life than the misused drug. Adequate doses of the substitute drug are given to alleviate symptoms and signs of withdrawal. This is then slowly reduced over days until it is stopped. The substance itself may also be taken in gradually reducing quantities. Substances that usually require detoxification include alcohol, opiates and benzodiazepines. Details of detoxification regimes are described in more detail under the individual drugs.

ALCOHOL

"Noah, who was a farmer, was the first man to plant a vineyard. After he drank some of the wine, he became drunk..." Gen 9: 20-21 G N Bible

Pharmacokinetics

Beverage alcohol (ethanol) is almost always taken orally. It is absorbed quickly from mainly the small intestine into the bloodstream. The bioavailability is somewhat reduced by the first bypass effect. Most of the alcohol is metabolised in the liver to acetaldehyde by alcohol dehydrogenase and this is further metabolised to acetate. The metabolism of alcohol follows first-order elimination at levels below 0.1mg ml^{-1} and then via zero-order elimination at higher levels.

Table 2 Physical complications of alcohol abuse

System	Early consequences	Late consequences
Liver	Elevated liver enzymes	Fatty liver, alcoholic hepatitis, cirrhosis
Pancreas		Acute pancreatitis, chronic pancreatitis
Cardiovascular system	Hypertension	Cardiomyopathy, arrhythmias, stroke
Gastrointestinal system	Gastritis, gastro oesophageal reflux disease, diarrhoea, peptic ulcer disease	Oesophageal varices, Mallory-Weiss tears
Neurological system	Headaches, blackouts, peripheral neuropathy	Seizures, Wernicke's encephalopathy, Korsakoff's psychosis, dementia, cerebral atrophy, cognitive deficits, impaired motor functioning
Reproductive system	Fetal alcohol syndrome	Sexual dysfunction, amenorrhoea, anovulation, early menopause, spontaneous abortion
Malignancies		Neoplasms of the liver, pancreas, oesophagus

Safe drinking

The consumption of alcohol is lawful, and society should therefore learn to use it responsibly. The Royal

College of Psychiatrists (UK) gives the following guidelines for safe drinking:

1. Men less than 21 Units/week and women less than 14 Uunits/week
2. Not daily (at least 2 alcohol free days/week)
3. Not all on one day (avoid binges)
4. Not during pregnancy
5. Never before or during driving, swimming, active sport or use of machinery, electrical equipment, ladders or in other potentially dangerous situations

- 10 ml Alcohol = 8 g Alcohol = 1 Unit (1U)
- Number of Units = volume of alcohol in ml x alcohol percentage/1000
  (Examples: Bottle of spirits (40%) (700ml) = 28U, Bottle of wine (11%) (750ml) = 8U)

Screening tests for alcohol abuse

Various quick screening tests can be used to determine if someone has an alcohol problem. An example of such a test is the CAGE. If you score more than 2/4 on the cage, your drinking requires further investigation.

CAGE CRITERIA:
1. Do you ever feel you should Cut down on drinking?
2. Do you get Annoyed when people talk about your drinking?
3. Do you ever feel Guilty about your drinking?
4. Do you ever need an Eye opener (drink first thing in the morning)?

Medical complications resulting from alcohol
Medical problems due to alcohol are common. Not only are these patients popular as exam cases, but it is also important in practice to be able to recognise these problems. Table 2 gives some examples. This is not an exhaustive list and you should supplement it further.

Psychiatric disorders resulting from alcohol
1. Alcohol abuse and dependence
Please refer to definitions of abuse and dependence above.

2. Alcohol intoxication
Please see Table 3 for a rough guide to the association between the amount of alcohol consumed and clinical picture. The effects of alcohol are very variable, and depend on many factors, such as dose, rate of drinking, gender, bodyweight and fat, and the time since previous dose.

3. Alcohol withdrawal
Within hours of a single heavy bout of drinking, someone can experience nausea or tremors ("hang over"). When someone is dependent on alcohol, he or she will experience a withdrawal syndrome characterised by nausea, sweating, tachycardia, insomnia, craving and a profound tremor. There is a high risk of seizures (found in up to one third of those who stop abruptly). This risk is highest in the first 48 hours, but persists for more than 2 weeks. Remember that focal seizures suggest additional pathology (e.g., subdural haemorrhage). Patients with alcohol dependence should therefore never be told to stop drinking abruptly without being detoxified first. (Details of how to detoxify someone are discussed under management.)

4. Alcohol intoxication delirium
In alcohol intoxication delirium, the degree of intoxication is such that a disturbance of consciousness occurs. It usually lasts only a few hours. An example of this is the so-called "blackout" where memories are not captured during intoxication and the person cannot recall the "drunken memories" after regaining consciousness.

Table 3 The association between blood alcohol concentration (BAC) and clinical picture of intoxication

Units consumed	BAC mg/ml	Signs and symptoms
0-2	30	Euphoria, sense of well- being, mildly disinhibited, talkativeness, mildly reduced visual reaction time
3	50	Joviality, judgement impaired, impaired driving ability
5	80	Co-ordination impaired, inattention, loss of social judgement
10	150	Ataxia, obvious drunkenness
12	200	Diplopia, amnesic, staggering
25	400	Respiratory depression, Coma
30+	500+	Death

Women are more susceptible, liver disease or drug interactions may affect tolerance

5. Alcohol withdrawal delirium
This is a complicated form of alcohol withdrawal and is a medical emergency. It can occur in up to 5% of people who stop drinking abruptly. The onset is usually within 2-5 days after stopping and often starts at night. Symptoms and signs include clouded consciousness, anxiety, disturbed sleep, autonomic hyper-arousal with tachycardia, sweating and possibly fever, a coarse irregular tremor and vivid hallucinations which are usually tactile or visual ("the horrors"). It lasts an average of 3-5 days. There is a risk of death (about 5%) and these patients should be treated in a general medical unit. There is also an increased risk of concomitant infections, trauma, severe hypertension, malnutrition and vitamin deficiencies, bleeding tendencies, seizures or metabolic disturbances (especially electrolyte disturbances).

6. Alcohol-induced persisting dementia
We do not understand the precise mechanism whereby alcohol abuse causes dementia. There are various possible causes for the condition including the direct effect of longstanding alcohol abuse on the brain, peripheral organ involvement (e.g., liver, kidney, pancreas, et cetera), malnutrition, and vitamin deficiencies. If this diagnosis is suspected, one should treat any vitamin deficiencies and allow the patient at least three months for recovery, to allow his or her physical condition and nutritional status to improve, before finally making the diagnosis.

7. Alcohol-induced persisting amnestic disorder
Amnestic disorder differs from dementia in that the former affects mainly short-term memory. The patient's ability to retain new information and learn new tasks is impaired. Long-term memory, however, remains unimpaired. These patients "fill" in the gaps in their recent memories by means of a practice known as "confabulation." They "make up" memories and stories in order to compensate for their inability to recall things. They are either unaware of their memory deficit, or appear not to be concerned about their inability to acquire new memories. This disorder is also known as Korsakoff syndrome. The treatment of alcohol-induced persisting amnestic disorder is oral thiamine 100mg two

or three times a day for 3 – 12 months. Only 20% of people with alcohol-induced persisting amnestic disorder respond to thiamine therapy.
Chronic thiamine deficiency, a consequence of malnutrition and malabsorption, is common in alcohol dependence and is the cause of alcohol-induced persisting amnestic disorder. Wernicke's acute encephalopathy is due to acute thiamine deficiency and presents with the following triad of symptoms: delirium, ataxia, and ophthalmoplegia (nystagmus, paralysis of the 3rd, 4th and particularly 6th cranial nerves supplying the eye). It is a medical emergency requiring parenteral thiamine.

8. Alcohol-induced psychotic disorders
Alcohol-induced psychotic disorders are less common than the abovementioned disorders. Alcohol hallucinosis and alcohol-induced delusional disorder are the two most important categories of these disorders.

- Alcohol hallucinosis can occur during intoxication and/or withdrawal and is usually short-lived (days to weeks). However, it can last for longer periods, even after the patient has stopped drinking. This disorder may recur with each subsequent episode of intoxication. It is characterised by auditory hallucinations occurring in a state of clear consciousness. The voices usually curse, malign and intimidate the afflicted persons.
  It is important to distinguish alcohol hallucinosis from alcohol withdrawal delirium (also known as "DT's", "horries" or "klontjies"). In the latter, hallucinations are mainly tactile or visual, and the person's consciousness is impaired, while it remains clear in alcohol hallucinosis.
  Therapy consists of the initiation of complete abstinence, and the use of antipsychotic medication (e.g., haloperidol) for up to 3 months after complete resolution of symptoms. Patients who refuse admission often need to be certified, because they are a danger to themselves and others.
- Alcohol-induced delusional disorder: In this disorder the chronic alcoholic develops a delusional system. It usually consists of delusions involving suspicion or jealousy and can certainly endanger the life of the person against which it is directed. The treatment is the same as that used in alcohol hallucinosis.

9. Other alcohol-induced disorders
- Alcohol-induced mood disorder
- Alcohol-induced anxiety disorder
- Alcohol-induced sexual dysfunction
- Alcohol-induced sleep disorder

Detoxification

Alcohol is short acting and withdrawal starts around 6-8 hours after the last intake of alcohol. Withdrawal may be complicated or uncomplicated.

- Uncomplicated withdrawal

Patients with uncomplicated withdrawal can undergo detoxification on an outpatient basis: First perform a thorough physical examination. If no complicating factors are present, prescribe a long-acting benzodiazepine (i.e., a GABA-agonist, like alcohol), such as diazepam (Valium®) 5mg, two or three times a day in gradually reducing doses for 5 days and oral thiamine 100mg twice a day and multivitamins for 10 days. Arrange follow-up and motivate for further treatment. If the patient is unable to maintain a sober lifestyle, rather refer him or her for inpatient treatment. The patient should not only remain abstinent, but must also learn to achieve a sober lifestyle.

- Complicated withdrawal

Patients with complicated withdrawal include those with a history of withdrawal delirium (DT's) or convulsions, patients with psychosis, those with suicidal ideation, patients with severe liver pathology, who are physically severely compromised, those who have very severe withdrawal symptoms and persons who have repeatedly failed outpatient detoxification.

These patients should be admitted to hospital for detoxification. Perform a very thorough physical examination and appropriate special investigations, and treat the medical complications. These patients are often confused and restless, and one should therefore nurse them in a safe environment. Place the mattress on the floor, if necessary, and try to avoid over-sedation. Request detailed nursing observations and monitor fluid intake and output. For the withdrawal state, prescribe 5-10mg diazepam (Valium®) two- to four-hourly orally or intravenously (not more than 60mg or diazepam equivalents per 24 hours). Should the patient be very restless initially, one could give 2-4mg of intra-muscular lorazepam (not more than 8 mg per 24 hours) as an adjunct to the Valium®. Note that 1 mg of lorazepam is equivalent to 5 mg of diazepam. Beware of over-sedation and respiratory suppression and be careful not to over-hydrate. During alcohol withdrawal, a state of inappropriate ADH secretion often exists, leading to over-hydration (check serum sodium). Rehydrate only if the patient is clinically dehydrated. If the intravenous fluid contains glucose, be sure to add intravenous thiamine to the infusion. (Glucose metabolism requires thiamine and if this is not added, Wernicke's encephalopathy may be precipitated.) Initially administer thiamine 100mg (together with the infusion, or intra-muscularly) and thereafter 100mg twice a day for 10 days by mouth. In the past, complicated withdrawal was associated with a high mortality rate. For that reason, re-evaluate the patient

regularly. Acute withdrawal will not usually last longer than 5 to 7 days. The general practitioner should possess the skills necessary to detoxify an alcohol dependent patient in the local hospital.

Medication used to help maintain sobriety in alcohol addiction

Disulfiram (Antabuse®): During the first stage of sobriety, patients often still feel very unsure of themselves and may therefore readily accept Antabuse® if is offered to them. Antabuse® can be effective if used as follows: The patient takes Antabuse® first thing in the morning, while motivation for abstinence is still high. The patient then knows that he or she will certainly not be able to drink, because alcohol would cause severe physical discomfort, such as palpitations, headaches, abdominal cramps, nausea, dizziness and flushing. Disulfiram blocks the enzyme acetaldehyde dehydrogenase. This produces an accumulation of acetaldehyde, which produces the physical discomfort. Patients must be thoroughly instructed regarding the mode of action of disulfiram, and must use it of their own free will. Antabuse® remains an external motivator. It should not be given to people surreptitiously. Implants of this product are not recommended. Antabuse® can be very effective, but only when combined with an ongoing good doctor-patient relationship.

Naltrexone: It may be useful to administer an opiate antagonist while the person is still drinking. The opiate antagonist blocks the dopamine/endogenous opiate positive reinforcement of alcohol and this result in a decrease in drinking behaviour. Alcohol therefore loses its positive or "kick" effect.

Acamprosate: Long-term alcohol abuse up-regulates NMDA receptors in the glutamate system and down regulates GABA receptors. This reduces craving and a return to drink in the presence of old drinking stimuli. Acamprosate is associated with longer periods of sobriety.

Pharmacotherapy certainly does not replace psycho-therapy and rehabilitation, but can serve as an adjunct to help maintain sobriety.

CANNABIS ("DAGGA")

Cannabis is the most commonly used illicit drug in South Africa. According to Interpol, South Africa is one of the top four cannabis suppliers in the world. Its street names include "marijuana", "dagga", "weed", "dope", "pot", "slow boat", "ganja", "herb", "boom", "bung", "Durban poison", "groengoud" and others. Cannabis contains more than 400 compounds, including over 60 cannabinoids, substances unique to the plant genus Cannabis. The most potent agent is delta9-tetrahydrocannabinol (THC).

Sources

THC is present in stalks, flowers, leaves and seeds of the Cannabis sativa plant (see Figure 7) and in resin that is secreted by female plants. It is smoked as cigarettes (joints) or in pipes or buckets, or else is taken orally either baked in cookies or cakes, or else taken as an extract. It is water insoluble and thus unsuitable for intravenous use.

It was first believed that cannabis is not physically addictive. This was based on research done in the 1970's on THC doses of 5-25mg. An average joint in the 1960's contained about 10 mg THC. Plant breeding techniques have greatly increased the potency of cannabis. Nowadays a potent joint contains about 150-300mg of THC, rendering obsolete the 1970s research on the risks and consequences of cannabis use. Tolerance and withdrawal have been clearly described for cannabis. It is thus physically addictive.

Figure 7 The cannabis sativa plant

Pharmacokinetics

When smoked 50% of the joint is inhaled and nearly all of this is absorbed. Bioavailability is about 25-30% via oral ingestion. Cannabinoids are lipid soluble and thus accumulate in fatty tissues. The tissue elimination half-life of THC is about 7 days. Complete elimination of a single dose may take up to 30 days. High concentrations are reached in the neo-cortical, limbic, sensory and motor areas. Cannabinoids are metabolised in the liver and major metabolites are also psychoactive and have long half-lives.

Pharmacodynamics

Endogenous cannabinoid receptors have been discovered. Neuronal receptors are called CB1 receptors (brain, peripheral nerves) and immune cell receptors are called CB2 (macrophages, spleen, other immune cells). The endogenous ligand for these receptors is called anandamide (after the Sanskrit word ananda, which means "bliss"); its physiological function is not yet confirmed.

Clinical picture of cannabis use

- Acute signs of intoxication include red eyes (vasodilatation), tachycardia, postural hypotension, motor in-coordination, impaired driving skills, impaired estimation of time and distance, impaired judgment, increased appetite, dry mouth, various psychological reactions, such as euphoria, anxiety, perceptual distortions/hallucinations, paranoid thoughts, impaired short term memory and other abnormalities. (Research on rats has demonstrated that THC destroys cells in the hippocampus, a part of the brain that is important in laying down new memories.)
- Chronic heavy use is associated with long-term impairment in performance- especially involving attention, memory and the ability to process complex information- which can last for weeks, months or even years. Researchers suspect that it may even lead to permanent cognitive impairment. Chronic use is also associated with an "amotivational syndrome" which causes sufferers to function poorly and not achieve their full potential.

Cannabis withdrawal

The withdrawal syndrome of cannabis is similar to that of alcohol, benzodiazepines and opiates, though less severe. It includes symptoms like restlessness, insomnia, anxiety, aggression, anorexia, muscle tremor and autonomic effects. Patients rarely need pharmacological intervention for withdrawal. If symptoms are severe, a short course of benzodiazepines may be used cautiously, for example, diazepam 5mg 3 times a day, tapered and stopped after 3 days.

Medical complications

- Cardio-vascular: Postural hypertension and fainting, acute cardiac incidents
- Respiratory: cannabis smoke contains the same constituents as cigarette smoking: carbon monoxide, bronchial irritants, tumour initiators and promoters and carcinogens. Furthermore, three times as much tar is retained with cannabis smoking compared to cigarette smoking and cannabis has a higher combustion temperature than tobacco. Cannabis is therefore more likely to cause bronchitis, emphysema and lung cancer (3-4 joints roughly equivalent to 20 cigarettes)
- Immunosuppressant
- Endocrine effects

Psychiatric complications

- Acute "toxic" responses including panic, anxiety, depression or psychosis
- Acute functional psychosis
- Possibly chronic psychosis that persists after abstinence
- Amotivational syndrome (impaired attention, memory, learning, drive)
- Effects on pre-existing mental illness
- Cannabis is a risk factor for mental illness
- Withdrawal syndrome

Medicinal use

There has been a resurgence of interest in the therapeutic benefits of cannabis. Cannabis was first used 5000 years ago in China for malaria, constipation, rheumatic pains, childbirth and as a surgical analgesic. Reynolds (Queen Victoria's physician) described "Indian hemp" as "one of the most valuable medicines we possess". It has been shown to be of possible benefit in controlling nausea and vomiting in chemotherapy patients, but side-effects were common. It may help in multiple sclerosis and similar neurological conditions and may increase appetite in cancer and AIDS patients. Its effects on the immune system have not been fully described and this will need monitoring. Cannabis has been shown to be superior to placebo in the treatment of neuropathic pain. THC reduces intra-ocular pressure and is a potent bronchodilator. Its usefulness was shown in studies to be limited by its side-effects. Research is ongoing and the verdict on its benefit as a medication is not yet out. Cannabis remains an illegal substance at this stage and doctors are not advised to recommend it to their patients.

METHAQUALONE ("MANDRAX")

The French pharmaceutical giant Roussell Laboratories initially marketed Mandrax® as a sedative or sleeping tablet. Mandrax® was thought to be safe and non-habit forming, but was later found to be highly addictive and was banned in 1977. Mandrax® was originally a white tablet with the letters Mx written on it. Illegally produced Mandrax has various additives and its colour can vary from beige, pink, blue, purple, black, brown and green. It can also be a light and dark blue capsule. Illicit Mandrax is sometimes sold as 1cm thick tablets, called "double barrels". Cape Town is reportedly considered the Mandrax capital of the world. It is the drug of choice in the ganglands of the Cape Flats.
Mandrax can be swallowed or injected, but is usually smoked. The tablets are usually crushed and mixed with cannabis and then smoked using a pipe called a "bottle neck" (See Figure 8). This combination is known as 'white pipe'. It is unique to South Africa and its use is particularly common in the Western Cape. Street names include "buttons", "henna", "Mx", "sproetjie", "pille", "whites", "witpyp", "mandies", "cremora", "Volkswagen", "Macarena", "cream", "gholfsticks", "doodies", "lizards", "germans", "flowers", "omo", "hits", and others. Many impure or "dirty" drugs are available.

Figure 8 "Bottle neck" or Mandrax pipe

Intoxication

Mandrax initially causes feelings of relaxation and euphoria. The person feels less inhibited and "witty". It is a depressant and users may become drowsy and have impaired co-ordination and slurred speech. The user may have a dry mouth and very little appetite and may have bloodshot red or glazed or puffy eyes (especially if used with cannabis). Nausea, vomiting and stomach pains are can also occur. The effects last for several hours. Some people will feel aggressive as the effects start wearing off. In many cases, users feel tired after taking Mandrax and may go to sleep for protracted periods. Depression is common and occurs as part of the Mandrax "hangover".

Overdose

A person who has taken an overdose may present with ataxia, respiratory failure, hypotension and coma, and may even die.

Withdrawal

Withdrawal symptoms include anxiety and restlessness, nausea and vomiting, abdominal cramps, poor appetite, headaches, insomnia, tremors, weakness, and seizures. Withdrawal symptoms start 12-24 hours after the last dose and are worse after 24-72 hours. Mild withdrawal does not usually require detoxification, but more severe withdrawal may require medication. Diazepam (Valium®) is usually used at doses of about 5mg three times per day. This is then tapered over 3-5 days.

Chronic abuse

Long-term use may be associated with aggressive behaviour, craving, mood swings or depression. The patient may exhibit distinctive yellow discolouration of the palms (from smoking "bottle necks", see Figure 9) reduced libido, weight loss, anaemia, impaired liver function or a chronic cough. There is an increased risk of lung cancer, TB, chronic bronchitis and arrhythmias. There are inconclusive reports of fits, cerebral atrophy, birth defects and impaired fertility. In South Africa, Mandrax use and trafficking is associated with vehicle theft, violent and property-related crime, organised crime, and gang-related activities.

Figure 9 Typical stains caused by a Mandrax pipe

THE STIMULANTS

1. Cocaine

Cocaine is derived from the Coca plant .The leaves of the Coca plant were used by the Incas in the Andes Mountains of South America as a stimulant and a traditional medicine.

Coca plants are cultivated in an organised way by the crime syndicates of South America. The leaves are harvested and taken to laboratories where they are processed into cocaine powder. It is then sold at an enormous profit and dispersed worldwide. The cocaine powder is mixed with other substances to increase its volume and this is called "cutting". Cocaine can be mixed with substances such as tooth powder, sugar and various other starches, but cases have been reported of cocaine being mixed with harmful substances, like foot powder, drain cleaner, Vim or other poisonous substances.

The street names for cocaine include "coke", "Charlie", "snow", "C" and "dust". Cocaine preparations can be eaten (coca leaves or paste), sniffed ("snorting"), injected alone ("mainlining") or with heroin ("speedballing").

"Crack" is the street name given to cocaine that has been processed from cocaine hydrochloride to a free base for the purpose of smoking it. It is manufactured by mixing cocaine hydrochloride and ammonia or bicarbonate of soda and allowing it to crystallise. It produces rapid and intense psychoactive effects. Other street names include "rocks", "bananas" or "golf balls". Crack is usually smoked in a pipe called a "Rock pipe". (See Figure 10). This is a glass or metal tube that contains a copper wire filter. The crack is placed on the filter and then heated with a lighter. The released vapour is then inhaled. The term "crack" refers to the crackling sound heard when the mixture heated, presumably as a result of the sodium bicarbonate. "Crack" is often the drug of choice among South African prostitutes.

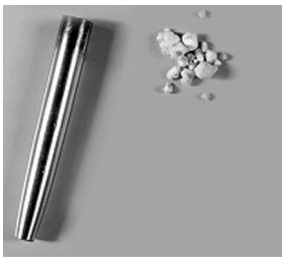

Figure 10 Crack cocaine and a crack pipe

Common pattern of abuse

Cocaine can cause profound subjective feelings of well-being and alertness and tolerance develops very quickly. It has a very short half-life (less than 90 minutes) and is often taken in binges in which repeated use causes overwhelming compulsive urges to take more. (It can be taken up to 10 times per hour!) Binges last from several hours to several days. The binge is usually followed by a crash, which lasts from 9 hours to 4 days. During the early stages of this phase agitation, anorexia, depression and severe craving occur, followed by exhaustion and insomnia but with the desire to sleep. Hypersomnia and hyperfagia occur late in the crash phase. The crash phase is followed by 1-10 weeks of withdrawal. During the withdrawal phase anhedonia, anergia, anxiety and severe cravings are prominent. Note that this often builds up to a binge, which can perpetuate the cycle. An extinction phase eventually ensues, with the return of normal hedonic responses together with episodes of craving brought on by conditioned triggers.

Intoxication

The effects of cocaine occur within minutes after its administration.

- The psychiatric symptoms of cocaine intoxication include euphoria, hyperarousal, heightened self-esteem, agitation, impulsivity, reduced appetite, rapid and excessive speech, over activity, anxiety or panic, violence, paranoia and formication (the sensation of insects creeping on the skin, known as "cocaine bugs").
- Physical symptoms of intoxication include tachycardia, hypertension, chest pain, dilated pupils, seizures, nausea, chills, teeth grinding, weight loss, cardiac arrhythmias and nasal septum perforation or ulcers.

When people mix cocaine and alcohol, they are compounding the danger of the two drugs. Researchers have found that the human liver combines cocaine and alcohol, manufacturing a third substance, cocaethylene, which intensifies cocaine's euphoric effects, while possibly increasing the risk of sudden death. Alcohol ingestion can be a very powerful trigger for relapse these patients.

Intoxication rarely requires treatment. Symptoms of intoxication are usually too brief to treat and patients commonly require only support, or, at most, a benzodiazepine for severe anxiety, restlessness or convulsions.

Withdrawal

The symptoms of withdrawal were discussed under the pattern of abuse. Withdrawal does not usually require medication.

Psychiatric presentations

- Intoxication delirium
- Manic symptoms especially during intoxication.
- Violence
- Substance-induced psychotic disorder
- Substance-induced anxiety disorder
- Formication
- Protracted dysphoria
- Substance-induced depressive disorder (patients can become severely depressed and a suicide risk especially during the withdrawal phase)
- Substance-induced sexual dysfunction
- Substance-induced sleep disorder
- Permanent dopaminergic degeneration has been described with persistent anhedonia and anergia for more than 10 years after stopping.

Medical complications

Cocaine produces increased catecholamine levels, which can produce vasoconstriction. It can cause various medical complications, including hypertensive strokes, hyperpyrexia (with a risk of rhabdomyolysis, renal failure, DIC, hypotension, and death), myocardial infarction, cardio-respiratory collapse, cardiac arrhythmias, seizures, diffuse cerebral micro-infarcts, hepatotoxicity, movement disorders such as Parkinsonism, neonatal complications, rhinitis and mucosal excoriation with snorting, et cetera.

Medications used for relapse prevention

In spite of extensive research, there is currently no available medication that can prevent relapse, but studies have suggested that antidepressants may be of benefit.

2. Amphetamines

Amphetamines were first synthesised in 1887 by Edeleau as part of a systematic program to manufacture

aliphatic amines. Amphetamine is thus a man-made drug (as opposed to cocaine that comes from a plant). It was first introduced in the 1930s as a remedy for nasal congestion. It was found to be useful in the treatment of obesity, narcolepsy, and hyperactivity in children, but clinical use has been limited by its abuse potential.

Amphetamines can be taken orally, snorted, smoked or injected IV. Illicit amphetamine appears as crystals, chunks, fine to coarse off-white to yellow powders and capsules or tablets of various sizes and colours. Its street names include "Speed", "Uppers", "chalk", "Crystal", "Ice" or "glass".

Methamphetamine is amphetamine to which one methyl-group has been added to make it more potent by enabling it to cross the blood brain barrier more rapidly. It was apparently used by the Nazi's as a combat drug to elicit aggressive behaviour and to keep them fighting in the absence of food or rest. It was recently discovered that Hitler was probably an IV methamphetamine user. Its use is on the rise in the Western Cape, especially among schoolchildren and gangsters on the Cape Flats. Here it is used in its crystallised form (crystal methamphetamine). This is smoked in the glass portion of a light bulb. It is vaporised over a flame and inhaled through a straw. It is known locally as "straw", "globe", "tik", or "tuk-tuk" (because of the clicking sound it makes when smoked). It is manufactured locally in clandestine laboratories from easily available substances like ephedrine and is thus relatively cheap.

Clinical picture of intoxication

Amphetamine intoxication is similar to cocaine, but it has a longer duration of action and can last from 4 to 24 hours, depending on the drug and mode of use. Methamphetamine is sometimes called the "poor man's cocaine". In common with cocaine and other similar stimulants, amphetamine is most often used in a "binge and crash" pattern. Tolerance develops quickly. (The pleasurable effects disappear before the drug concentration in the blood falls significantly and therefore users try to maintain the high by binging on the drug).

After the initial "rush" there is typically a state of heightened agitation, which in some individuals can erupt into violent behaviour. Amphetamines that are taken for days on end (in a binge) cause patients to become sleep deprived and this often leads to aggression. After about 5 days of heavy use, severe sleep deprivation will set in, and patients can begin to see so called "shadow people" (almost as if the shadows were coming alive). This is sometimes referred to as "the meth monster". It is frequently associated with irrational thinking and paranoia.

Withdrawal

Withdrawal is similar to that of cocaine, but the degree of craving appears to be less marked.

Long-term effects

In addition to causing medical and psychiatric complications similar to those found in cocaine, amphetamine abuse is more likely to be associated with out of control rages and severe violent behaviour. Psychotic disorders can result in homicidal as well as suicidal thoughts and behaviour.

Animal research shows that high doses of methamphetamine damage neuronal endings. The neurons do not die, but their nerve endings ("terminals") are cut back and re-growth appears to be limited. This may explain why recovering amphetamine addicts feel depressed, fuzzyheaded, and that think life is not as pleasurable without the drug for months or even years after withdrawal. This is called the "wall" period. (Some people never recover, remaining unsatisfied with life because of the permanent brain damage). Over time, methamphetamine appears to cause reduced levels of dopamine, which can result in syndromes such as Parkinsonism.

Treatment

Acute intoxication can often be managed by observation in a safe, quiet environment. In cases of extreme excitement or panic, treatment with anti-anxiety agents such as benzodiazepines can be helpful.

There is no replacement medication for the treatment of amphetamine withdrawal. Medical complications such as hyperthermia and convulsions are treated symptomatically. There are currently no particular pharmacological treatments for dependence on amphetamine or amphetamine-like drugs such as methamphetamine. Antidepressant medications may be helpful in some cases with depressive symptoms.

3. Over-the-counter and prescription stimulants

Appetite suppressants containing pseudo-ephedrine and related substances are frequently abused for their stimulant effects. They are sometimes called "little E" (because their stimulant effects are similar to that of Ecstasy). Drugs used for the treatment of attention deficit hyperactivity disorder (ADHD) and narcolepsy, like methylphenidate (Ritalin®) can be abused for similar reasons. Patients wishing to lose weight should receive advice regarding healthy non-pharmacological alternatives and great caution.

DESIGNER DRUGS: ECSTASY

Ecstasy, or 3,4-methylenedioxymethamphetamine (MDMA) (also known as "Adam" or E's), is part of a new family of drugs, called the designer drugs. These drugs are designed and manufactured to have specific effects. Ecstasy's chemical structure is similar to that of the stimulant, methamphetamine, and the hallucinogen, mescaline, and it can produce both stimulant and psychedelic effects.

MDMA affects the brain by increasing the activity of different neurotransmitters, but has the greatest effect on serotonin and dopamine. Serotonin is a neurotransmitter that plays an important role in the regulation of mood, sleep, pain, emotion, appetite, and other behaviours. The excessive release of serotonin causes the mood elevating effects experienced by MDMA users, but in releasing large amounts of serotonin, the brain becomes significantly depleted. This contributes to the negative behavioural after effects users often experience for several days after taking the drug. Of even greater concern is that MDMA has been shown to damage serotonin-containing neurons, an effect that may be long lasting.

MDMA is most often found in tablet form although it is sometimes found in powder, liquid or capsule form. It is usually administered orally, or less frequently, intranasally (snorted), inhaled (smoked), or inserted per rectum ("charging").

Intoxication

A "High" develops 30-90 minutes after ingestion (orally). This consists of CNS stimulant effects (enhanced sense of pleasure, increase in self-confidence and energy) and psychedelic effects (feelings of peacefulness, acceptance, empathy, alterations in consciousness with sensual and emotional overtones, and perceptual and visual distortions). Physical effects include increased heart rate and blood pressure, nausea, dry mouth, decreased appetite, jaw clenching, grinding of teeth, muscle aches, gait disturbances and insomnia.

Complications of intoxication include urinary retention, seizures, strokes, heart attacks, cardiac arrhythmias, paranoia, and hallucinations. A potentially fatal complication is heatstroke (hyperthermia). This is caused by changes in thermoregulatory processes caused by disruption of normal serotonin function. It is characterised by a raised body temperature, and may lead to seizures, acute renal and hepatic failure and DIC (disseminated intravascular coagulation).

Dehydration is also a possible complication, because of the heat and the strenuous dancing that takes place at "raves" parties where the drug is usually taken. Some dancers try to compensate for this by taking in large amounts of fluid, thus becoming over-hydrated. This may result in a low sodium level, which may cause brain oedema that can lead to unconsciousness and death.

A malignant neuroleptic-like syndrome can result, possibly from a rapid increase in serotonin levels. Symptoms include confusion or agitation progressing to rigidity, hyperthermia and autonomic dysfunction (fluctuating blood pressure, tachycardia and urinary incontinence). This may be fatal.

Withdrawal symptoms

Withdrawal symptoms include fatigue, loss of appetite, depressed feelings, and concentration difficulties.

Long-term complications

With chronic use, tolerance to the effects of MDMA develops. Regular MDMA abuse can cause major depression, anxiety and panic disorders, paranoid ideation, increased impulsiveness, and sleep disorders. MDMA use may precipitate the onset of psychosis in predisposed individuals. Studies have shown that some heavy MDMA users experience long lasting confusion, depression, and selective impairment of working memory and attention processes. This impairment may be permanent.

Treatment

There is currently no pharmacological treatment for dependence on MDMA. Antidepressant medications might be helpful in combating the depressive symptoms frequently seen in MDMA users who have recently become abstinent.

OPIATES/NARCOTICS

Psalm of a Heroin addict:
Heroin is my shepherd, it will always be wanting
He lays me down in a sewage ditch
To troubled waters he leads me
He destroys my sole
He leads me in the footstep of wickedness,
Yes, I go through poverty and will fear all calamity
Because you are with me -
You're needle and pellet comforts me
You pillage the table of my family
You make my head empty
My cup runs over with grief
Heroin addiction will follow me to the ends of my life
And I will live in the house of doom until the length of day

Author unknown
From reality media website:http://www.drugaware.co.za

Opium is derived from the opium poppy or Papaver somniferum. (See Figure 11). The Opium poppy was already known by 3000BC and was called the "joy plant". After the plant has flowered, small cuts are made in its seedpod, and a white substance seeps out. This liquid becomes dark and sticky and is known as raw opium.

Opiates have been used both medically and non-medically for centuries. A tincture of opium called laudanum was widely used since the 16th century as a remedy for "nerves" or to stop coughing and diarrhoea. Morphine was extracted in a pure form suitable for solution, by 1805. By the mid-19th century, the hypodermic needle was introduced and intravenous use became the route of administration of choice. Heroin was synthesised from morphine in Germany during 1874 and introduced as a remedy for morphine addiction, but it soon became apparent that it was even more addictive than morphine. In the last century, many synthetic drugs have been developed with essentially the same effects as the natural opium alkaloids.

Figure 11 The poppy plant

Types of opiates

Opiates can be divided into opioids, which are derived from opium, (examples include morphine, heroin, codeine), and synthetic opiates (examples include Pethidine®, Wellconal®, Doloxene®, Valoron® and methadone).

Opium appears either as dark brown chunks or in powder form, and is generally eaten or smoked. Heroin usually appears as a white or brownish powder (called "Thai white", "white sugar", "brown sugar", "smack", "H", "skag", "junk", "smack", "horse", "dreamer" or "herries"). This is dissolved in water for injection, smoked with dagga, snorted or heated over tinfoil and then inhaled ("chasing the dragon"). Opiate solutions are injected under the skin ("skin popping"), directly into a vein ("mainlining") or into a muscle. Typically, a heroin abuser may inject up to four times a day. Most street preparations of heroin contain only a small percentage of the drug, since they are diluted with sugar, quinine, or other drugs and substances.

Other opiates appear in a variety of forms, including capsules, tablets, syrups, elixirs, solutions, and suppositories. Wellconal® is a synthetic opiate that was popular in South Africa during the eighties and was known as "South African heroin", "pinks" or "pink heroin". It is a pink tablet that was dissolved in water and injected. Its popularity declined in the nineties when heroin became more freely available. Pethidine (called "peth" on the street) is a schedule 7 opiate that is frequently abused by medical practitioners. Urine toxicological screening generally tests positive for opioids but negatively for related synthetic analgesics.

Mode of action

Once an opiate such heroin is taken, it rapidly distributes through the body and crosses the blood-brain barrier. In the brain, heroin is converted to morphine, binding rapidly to opiate receptors. Abusers typically report feeling a surge of pleasurable sensation (a "rush", "scoring"). The intensity of the rush is a function of the dose of the drug and how rapidly it enters the brain and binds to the natural opioid receptors. Opiate antagonists, like naloxone or naltrexone reverse this action.

Intoxication

Symptoms and signs of intoxication include pupillary constriction, constipation, drowsiness, relaxation, dulling of pain, slurred speech, euphoria followed by apathy, poor memory and attention, respiratory depression, cough suppression, difficult micturition, nausea and vomiting, sweating, warm flushing of the skin, itching, dry mouth, loss of libido, menstrual irregularities and rarely convulsions.

The treatment of mild to moderate intoxication requires general measures such as observation, gastric lavage within 1 to 2 hours of oral ingestion and the use of activated charcoal.

Overdose

Large doses of heroin may result in a potentially lethal overdose. Due to the depressant action of heroin on the CNS, these patients may present in a coma, with respiratory depression. There is also a risk of hypoglycaemia and seizures. The risk of overdosing increases with the concurrent use of other CNS depressants (such as alcohol), greater heroin dependence, and a longer history of heroin consumption.

Overdoses are managed by giving naloxone (Narcan®) 0,4mg IV slowly at 5-minute intervals. If there is no response after five doses, one should reassess the diagnosis of opiate toxicity. (Maximum dose - 6mg Naloxone; this is rarely needed.) It is important to remember that the duration of action of naloxone is much shorter than most opiates of abuse and thus careful observation and repeated doses of naloxone

may be necessary. If the patient is opiate dependent, naloxone may precipitate severe opioid withdrawal.

Withdrawal

Withdrawal symptoms include abdominal cramps, anxiety, craving, irritability, dysphoria, fatigue, hot and cold flushes, muscle aches, nausea, restlessness and yawning.

Signs of opiate withdrawal include diarrhoea, increased blood pressure and pulse, lacrimation, rhinorrhoea, sweating, muscle spasms ("kicking the habit"), dilated pupils, goose bumps ("cold turkey"), vomiting and fever. (Interested students may want to see the film "Trainspotting", where this is demonstrated.) Other symptoms may include depression, anxiety and personality changes.

The duration of withdrawal depends on the half-life of the drug. Pethidine addicts start to withdraw within 4 – 6 hours, and this peaks after 8 – 12 hours, heroin addicts start after 6 – 8 hours and peak at 36 – 72 hours, morphine addicts after 8 – 12 hours, codeine after 24 hours and methadone after 36 – 72 hours, peaking after 72 - 96 hours. Withdrawal symptoms usually last about 4-5 days.

Heroin withdrawal is considered reasonably safe, but sudden withdrawal in heavily dependent users who are in poor health can occasionally be fatal. Withdrawal early in pregnancy may precipitate miscarriage and these patients are best maintained on methadone during their pregnancy or at least during the first trimester, then slowly and carefully withdrawn in their mid- or last trimester. (Methadone has a longer half-life than heroin and this is possibly safer for the fetus than the repeated intense highs and withdrawals associated with continued heroin during the first trimester.) Heroin abuse can cause serious complications during pregnancy, including miscarriage and premature delivery. Children born to addicted mothers are at greater risk for SIDS (sudden infant death syndrome). Although infants born to mothers taking prescribed methadone may show signs of physical dependence, they can be easily withdrawn from this drug.

Medical management of heroin withdrawal

Withdrawal is generally very poorly tolerated by addicts and is a common cause of early relapse. Withdrawal is thus best managed in a hospital. Mild withdrawal can be managed conservatively with an antispasmodic like hyoscine butylbromide (Buscopan ®) for the abdominal cramps, a non-steroidal anti-inflammatory drug like ibuprofen (Brufen®) for the muscle cramps and aches, and diazepam (Valium®) for irritability, dysphoria and anxiety.

Moderate to severe withdrawal requires a methadone detoxification. This necessitates hospitalisation and good supervision, with restrictions being imposed by the hospital staff to ensure that no opiates, other than prescribed medication, are taken by the patient as overdose may occur. Methadone (Physeptone® syrup 2 mg/5ml) is used only when objective clinical signs of withdrawal are present. There is a risk of overdose if the patient's subjective need for methadone is used as a guideline for treatment.

The first step is to determine the patient's opiate requirements, which depend on many factors including the level of tolerance, the heroin dose and the quality of heroin used. This is done by giving a small dose of methadone to a patient with objective signs of withdrawal and watching its effects (signs of continued withdrawal or of intoxication). This is repeated until the minimum dose that suppresses withdrawal symptoms, but does not induce signs or symptoms of intoxication, is identified. This dose is then gradually reduced and stopped.

In practice, methadone (Physeptone® Syrup 2mg/5ml) is given in the presence of clinical signs of withdrawal. A starting dose of Physeptone® syrup 10mg (25ml syrup) is given orally. The patient is observed and if, after one hour, withdrawal symptoms are still present, another 5-10mg (12-25ml syrup) can be given orally. This can be repeated. In the majority of cases, the total 24-hour dose should not exceed 30mg. The total daily requirement is then repeated on the second day of withdrawal in a single or divided twice-daily or BD dose. This dose is then decreased by 5mg/day to a total daily dose of 10mg, and then reduced by 2mg/day until it is stopped. If the patient's withdrawal symptoms allow, the withdrawal regimen may be shortened. Patients should never be given more than 30 mg of methadone in a single dose unless the current level of physiological dependence is very high and is clearly documented as such.

Buprenorphine (Temgesic®) is a partial opioid receptor agonist and can be used for detoxification as can the alpha-2 agonist clonidine. The process and outcome of long-acting opioid agonist (methadone) tapering may be improved through the addition of a calcium channel blocker such as nimodipine.

A possible way of reducing the risk of relapse into illegal opiate use is the long-term prescription of an opioid antagonist such as naltrexone. At present, there is not sufficient evidence of efficacy of naltrexone to justify its routine use in the maintenance treatment of opiate dependence. Naltrexone maintenance is usually reserved for those patients who are highly motivated to achieve complete abstinence and who are otherwise in a stable and productive life situation with sufficient social support (e.g., opioid-dependent physicians). There is a potential overdose risk following discontinuation of naltrexone treatment. There are also

unconfirmed reports that naltraxone can cause depression.

Prescription and over-the-counter opiate abuse

Prescription and over-the-counter analgesic abuse and addiction (especially codeine addiction) is of great concern since these are often iatrogenic disorders resulting from careless prescribing by doctors or poor patient education by doctors and pharmacists about the dangers of these drugs. These addicts often conform to a different profile than do the classic addicts. They are often middle-aged, "white-collar" workers or house-wives who become dependent unwittingly and don't view their addiction as a problem since the drug is "legal". They frequently somatize stress and possess poor coping skills and there is an emphasis on immediate relief. They therefore pressurise doctors into providing prescriptions instead of improving their coping skills and functioning. Chronic analgesic use for stress headaches may lead to analgesic induced headaches.

This problem is best managed by prevention. Analgesics should always be prescribed for a time-limited period along with good patient education about their dangers. Once the patient has become addicted, the addict should not be "enabled" by the doctor by continuing repeat prescriptions.

It is important to remember not to withdraw one drug by using a more dangerous and addictive drug. Codeine should therefore not be withdrawn with methadone. Instead, one should use diminishing doses of codeine.

HALLUCINOGENS/PSYCHEDELICS

The hallucinogens are a group of drugs that are characterised by their ability to cause perceptual distortions, and alter mood and thought processes. They are a chemically diverse class. One group is structurally similar to serotonin (the indolealkylamines) and includes drugs like LSD and Magic mushrooms (psilocybin). The next group is the phenylethylamines. This group includes mescaline, MDA and MDMA or ecstasy. We have classified ecstasy separately under "designer drugs" (see above). Theses drugs are structurally related to amphetamines. The next group is the dissociative anaesthetics (the acrylcycloalky-lamines) and includes phencyclidine (PCP or angel dust) and ketamine. The final group is the atropine-like family and includes drugs like atropine and scopolamine. Some people also classify cannabis as a hallucinogen, but we have considered it separately in this chapter.

LSD is possibly the best-known hallucinogen and was discovered in 1938 by Dr Albert Hoffman, who was doing experiments on a fungus found on rye grass. He was looking for a medical cure for respiratory and circulatory illnesses. Since then it has captured the imagination of drug cults, especially during the "hippy era". Some well-known songs from that era allegedly referring to LSD include "Lucy in the Sky with Diamonds" by the Beatles and "Purple Haze" and "Are you experienced" by Jimi Hendrix. LSD made its comeback in South Africa in the 90's with the increased popularity of the "Rave Scene".

Street names for LSD include "acid", "sunshine", "candy" and "smarties". LSD is generally placed on blotting paper and other street names are based on pictures on these carriers (examples include Superman, Smiley Faces, Garfield, Bart Simpson, etc.) These blotters are placed under the tongue and the LSD is released. It is also occasionally taken in liquid form with drinks or as gel tablets ("Window panes") or as small tablets known as "Microdots".

Ketamine (Ketalar®) is occasionally abused in South Africa, possibly because of its availability, as it is a commonly used as anaesthetic in rural areas, especially for children. It is either injected intramuscularly in low doses or heated to obtain a white powder, which is smoked or snorted. Street names include "Vitamin K" or "Special K".

For thousands of years before Europeans set foot in the New World, the "sacred mushroom" was in use in native rituals. Today psilocybin-containing mushrooms (so called "magic mushrooms") are still used for their psychedelic effects. They can be eaten fresh or dried.

Phencyclidine (also known as PCP, "angel dust", "hog", or "horse tranquilliser") was first used in the 1950s as a surgical anaesthetic. Its use was quickly discontinued because it produced highly undesirable side-effects, such as convulsions during operations and post-operative hallucinations. In the 1960s it was marketed again, this time to veterinarians, but strictly as an animal anaesthetic and tranquilliser. Fortunately, it is not commonly abused in South Africa.

Intoxication

Someone described his experiences after using magic mushrooms (psilocybin) as follows:

"There are no apt words ... to characterize your state when you are, shall we say, 'bemushroomed.' ... How do you tell a man born blind what seeing is like? ...to see, more clearly than our perishing mortal eye can see, vistas beyond the horizons of this life, to travel backwards and forwards in time,... What you are seeing and what you are hearing appear as one: the music assumes harmonious shape, giving visual form to its harmonies, and what you are seeing takes on the modalities of music - the music of the spheres ... All

your senses are similarly affected: the cigarette with which you occasionally break the tension of the night smells as no cigarette before had ever smelled; the glass of simple water is infinitely better than champagne..."

With psychedelic drug intoxication, there are altered perceptions that include an intensification of perceptions, derealisation, illusions, hallucinations or synaesthesia (when one sensation is experienced as another sensory modality, so well described by the psilocybin user above). There may also be anxiety, depression, delusions or the feeling of losing one's mind. Unpredictable behaviour can occur, with impaired judgment. Physical symptoms include dilated pupils, tachycardia, hypertension, goose bumps, salivation, nausea, tremor, lacrimation, flushing, sweating, blurred vision, tremor and incoordination. Adverse experiences are called "bad trips".

PCP is a dangerous drug that characterised by bad trips, psychotic reactions and extreme violence in its users. It produces a brief dissociative psychotic state that resembles schizophrenia. Clinical signs of intoxication include nystagmus, hypertension, tachycardia, diminished pain sensation, ataxia, dysarthria, muscle rigidity, seizures and delirium. Behavioural changes can include impulsive, unpredictable behaviour, psycho-motor agitation, impaired judgment, and impaired functioning.

Hallucinogen intoxication is managed by applying general life support measures and ensuring that the patient and the treating team are kept safe. A "babysitter" or guide is used to "talk the patient down" in a safe and quiet environment. Struggling on the part of the patient should be avoided, as this may cause rhabdomyolysis, hyperpyrexia and death. Benzo-diazepines, like lorazepam (Ativan®) may be used if tranquillisation is required.

Long-term complications

Patients develop rapid tolerance, but there is no withdrawal syndrome. Flashbacks of experiences during intoxication can occur in up to 80% of long-term users. Flashbacks of "bad trips" are called "bummers". They may occur as late as 20 years after initial ingestion. Longer-term use is also associated with mood, anxiety or psychotic disorders and permanent personality changes. Long-term PCP use is also associated with memory loss, difficulties with speech and thinking, depression, and weight loss.

VOLATILE SOLVENTS (INHALANTS)

Volatile solvents are a group of volatile substances that are intentionally inhaled for their psychoactive effects. Younger adolescents and the destitute (like street children) commonly abuse this group of drugs. It includes chemicals such as petrol, glue, lighter fuel, varnish remover, thinners, rubber cement and aerosols (spray paint).

Intoxication

An initial period of disinhibition is experienced. This may be accompanied by feelings of euphoria and excitement. This is followed by depression of the central nervous system, with sedation. Other effects include dizziness or light-headedness, disorientation, visual disturbances, incoordination, slurred speech, an ataxic gait, a red face, aggression, impaired judgment, apathy, stupor or psychotic symptoms like hallucinations or delusions. There is a risk of respiratory depression, cardiac arrhythmias, aggression, asphyxiation and accidents during intoxication.

Long-term effects

Tolerance is described but its incidence has been difficult to estimate. There is no withdrawal syndrome, but the withdrawal period is associated with an increased susceptibility to seizures. Chronic use may lead to a persistent cough, lethargy, a runny nose or a rash around the mouth. Dementia, cerebellar damage, deafness, neuropathy and leukaemia have been described. There is also the risk of liver cell damage, kidney failure and neuromuscular damage.

Treatment

The treatment of volatile substance abuse is difficult. During intoxication, treatment is supportive and the focus is on damage limitation. Patients can be referred for further rehabilitation, once the intoxication has resolved. They often do not do very well in traditional rehabilitation services. A supportive doctor-patient relationship, where the pros and cons of drug usage are discussed and sobriety is encouraged, may be of more benefit.

BENZODIAZEPINES

Benzodiazepine addiction is usually an iatrogenic disorder. Doctors who are unable to correctly diagnose anxiety and stress or who do not know how to manage their patients may prescribe benzodiazepines to make the patients (and themselves) feel better. This does not make the problem go away and creates a further problem with dependence. Always remember that benzodiazepines should only be used only in the short term and for specific purposes.

Intoxication

Benzodiazepines are sedative-hypnotics that act as central nervous system depressants by increasing the

action of the inhibitory neurotransmitter, GABA. Side-effects include sedation, motor incoordination, psychomotor retardation, cognitive dysfunction, disinhibition, memory impairment and emotional blunting.

Withdrawal

Tolerance develops rapidly and withdrawal is similar to alcohol. Signs and symptoms of withdrawal include arousal, restlessness, anxiety, tremors, nightmares, insomnia, anorexia, nausea, vomiting, postural hypotension, seizures, delirium and hyperpyrexia.

Long-term effects

Prolonged use not only results in dependence, but is also associated with memory impairment.

Management of benzodiazepine withdrawal

- The therapeutic relationship between the patient and doctor is most important in initiating benzodiazepine withdrawal. There is usually great reluctance on the part of the patient. Take time to explain to the patient concepts such as tolerance and withdrawal and then convince him or her that stopping the benzodiazepines is the best thing to do. Encourage him or her not to seek medication from other doctors. Negotiate each reduction with the patient.
- Withdrawal from benzodiazepines takes time. Be patient.
- Replace short-acting benzodiazepine with long acting benzodiazepine.

Equivalents to diazepam 5mg =
chlordiazepoxide (Librium®) 15mg
lorazepam (Ativan®) 1mg
alprazolam (Alzam®, Zanor®) 0.25mg
bromazepam (Lexotan®) 1.5mg
flunitrazepam (Rohypnol®, Insom®) 0.5mg
nitrazepam (Mogadon®) 5mg
oxazepam (Serepax®) 15mg
temazepam (Normison®) 15mg

- Decrease the diazepam by 2-2.5mg every 1-2 weeks, but stick to a dose for a while if symptoms reappear, or go up a notch and reduce more slowly.
- When 20% of initial dose is reached, taper more slowly: 0.5-2mg per week.
- The patient will require regular monitoring and motivation.
- Never stop benzodiazepines without tapering. It can be dangerous.

Carbamazepine (Tegretol®) in therapeutic doses may be of value for benzodiazepine withdrawal symptoms.

NICOTINE, CAFFEINE, AND OTHER SUBSTANCE ABUSE

Nicotine is one of the most heavily used addictive drugs. It is most often abused through cigarette smoking and this is associated with severe medical complications such as cancers and pulmonary disease. Nicotine is a stimulant and its effects during smoking include arousal, increased attention and concentration, enhanced memory, reduced anxiety and suppressed appetite. These effects are caused by its agonist actions at the nicotinic cholinergic receptors. There is a high degree of tolerance. During periods of abstinence and/or craving, smokers have shown impairment across a wide range of psychomotor and cognitive functions, such as language comprehension. Withdrawal is accompanied by symptoms like irritability, hostility, anxiety, dysphoria, depressed mood, increased appetite and craving.

It can be difficult for smokers to maintain abstinence. Studies have shown that pharmacological treatment combined with behavioural treatment, including psychological support and skills training to overcome high-risk situations, results in some of the highest long-term abstinence rates. Generally, rates of relapse after smoking cessation are highest in the first few weeks and months and diminish considerably after about three months.

Pharmacological treatments include nicotine replacement via nicotine chewing gum, transdermal patches, sprays or inhalers. The only non-nicotine based pharmacological treatment currently available is the antidepressant, bupropion (Zyban®). It has been shown to improve abstinence rates, especially is combination with nicotine replacement and psychotherapy.

Caffeine is another socially acceptable stimulant. Overuse caffeine is a common preventable cause of anxiety and sleep disorders. Intoxication is associated with jitteriness, irritability, anxiety, tachycardia, dysrhythmias, hypertension, tremor, increased gastric acid secretion and even convulsions or delirium. An oral dose in excess of 5000mg may be fatal. Caffeine withdrawal symptoms include headaches, drowsiness, fatigue, anxiety, depression, nausea or vomiting. Withdrawal from caffeine should be gradual (say about 10% every few days) to prevent a withdrawal syndrome.

Athletes and body-builders sometimes use anabolic steroids illegally. These synthetic substances are related to male sex hormones. They are taken orally or injected, typically in cycles of weeks or months (referred to as “cycling”), rather than continuously. Side-effects of abuse may include acne, premature balding, hypertension, increased LDL and reduced HDL, malignancies, testicular atrophy, premature skeletal maturation in adolescence and liver dysfunction. Women may experience masculinisation. After a period

of elevated mood, steroids can lead to irritability, anxiety and depression. Steroid abuse may precipitate mania, paranoid jealousy, extreme irritability and rage reactions. Depression and anxiety may occur during withdrawal.

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