ACUTE ADVERSE REACTIONS TO DRUGS OF ABUSE AND MANAGEMENT

INTRODUCTION

Drug abuse is a general term referring to the use of any drug or chemical substance which is either illegal or restricted and which results in harm to the individual and or society in general. The term also refers to the use of banned or restricted drugs in sport.

Drug dependence is a term used when a person has a compulsion to take a drug in order to experience its psychic effects or to avoid the discomfort of withdrawal symptoms. It is a condition arising from repeated administration of a drug on a periodic or continuous basis. The characteristics of drug dependence will vary between individuals and are also determined by the agent involved, route of administration and pattern of drug taking. The common feature of drugs, which can cause dependence, is that all produce a rewarding effect (positive reinforcement). Virtually all dependence-producing drugs activate the mesolimbic-dopaminergic reward pathway in the brain.

Drug dependence is either physical or psychological, but usually a combination of the two.

Physical dependence is a (neuro)adaptive state that manifests itself by intense physical disturbances when the administration of the drug is terminated, or when its action is reversed by the administration of a specific antagonist. These disturbances i.e. the so-called withdrawal or abstinence syndromes are made up of specific arrays of symptoms and signs of a physical and psychic nature that are characteristic for each drug type. The clinical features of the withdrawal symptoms may be severe and even life threatening (e.g. convulsions), necessitating hospitalisation. Most of the drugs that cause physical dependence also induce tolerance (acquired).

Tolerance is a phenomenon that occurs with chronic administration of a drug. It is an adaptive state, characterised by the necessity to progressively increase the dose of the drug to produce the same level of intensity as the original effect. The mechanisms of acquired tolerance may be divided into 3 categories, namely
pharmacokinetic (e.g. increased metabolism), pharmacodynamic (e.g. neuro-adaptive changes in the brain) and thirdly, learned tolerance (which refers to a reduction in the effects of a drug due to compensating mechanisms that are learned).

**Psychological dependence** is a psychic condition where either a search (or longing, craving) for pleasure or euphoria, or a need to escape unpleasant mood states or the harsh realities of life, are the primary drives. There is a psychic drive to experience pleasure or to avoid discomfort. Psychological dependence, which usually outlasts the physical withdrawal syndrome, is by far the most important component responsible for relapse among treated addicts. Psychological dependence (craving) may also be associated with tolerance-producing neuro-adaptive changes. A person who is dependent on a drug in the above way, may also be described as an addict.

In the realms of the illicit drug trade, quality control is non-existent. The concentration of the “active” ingredient may, therefore, vary widely. The “active” ingredient may be completely absent or a specific “batch” may contain higher concentrations than usual, increasing the danger of overdose. Illicit drugs may also contain impurities and adulterants. Pills are often a heterogeneous mixture of a wide variety of active substances with similar structures and superficially similar effects. Some of the impurities may be more toxic than the mother substance (see later under individual drugs.) Substances which are used to “cut” the amount of the main ingredient(s) include talc and sugars (such as lactose, sucrose and mannitol). The talc in tablets injected IV, may cause pulmonary granulomas.

Another matter of concern with regard to drug abuse is the possibility of drug interactions between therapeutic medications and illicit drugs (e.g. antidepressants and ephedrine containing drugs, combined with amphetamines, cocaine etc). These interactions may increase the incidence of toxic reactions and may play a role in the development of the serotonin syndrome (see page __________ for details on serotonin syndrome).
Rapid delivery systems (i.e. intravenous injection, inhalation) increase the dependence potential. Intravenous injections also increase the dangers of overdose and infection (AIDS, hepatitis, endocarditis, septicaemia, etc).

Although the drugs of abuse are classified into different categories, there is no sharp divide between many of them regarding their acute toxic effects. The same applies to symptoms and signs of withdrawal syndromes for the different groups.

In the discussion that follows only the acute toxic effects of drugs of abuse, as well as clinically serious withdrawal syndromes and their management, will be dealt with.

**CENTRAL NERVOUS SYSTEM DEPRESSANTS**

These agents include ethanol, the barbiturates, the benzodiazepines, chloral hydrate, meprobamate, methaqualone (mandrax, boggel, star, flowers,), gamma-hydroxybutyrate (GHB, liquid ecstasy) and various inhalants. The newer sedative-hypnotics, namely zopiclone and zolpidem, may also be included in this group.

All these agents cause dose related central nervous system depression, ranging from inebriation to coma, and in severe cases, respiratory and cardiovascular collapse. Concurrent ingestion of other CNS depressants increases the likelihood of complications. **Barbiturate and benzodiazepine overdoses** are discussed in more detail elsewhere (page ….). Symptoms and signs of severe ethanol poisoning include hypothermia, hypoglycaemia (especially in children), metabolic acidosis and electrolyte disturbances. Cardiac arrhythmias may also occur. Aspiration of vomitus may result in pneumonitis and pulmonary oedema. Blood ethanol levels above 400 mg% may be fatal. (The potential lethal dose of ethanol in the non-tolerant adult is 5 – 6 g/kg and 3 g/kg for children.) Chronic ethanol abusers may survive considerably higher blood ethanol levels.

Certain sedative-hypnotics, such as **gamma-hydroxybutyrate** (GHB, also known as liquid ecstasy) and **flunitrazepam** (Rohypnol), have become popular as so-called ‘date rape’ drugs. GHB, in addition to its CNS depressant effects, may also cause vomiting, bradycardia, agitation, ataxia, hallucinations, uncontrollable shaking and
seizure-like activities. Anterograde amnesia may be experienced. Adverse reactions have been reported for a wide range of doses (2-30 grams), indicating variable individual responses to the drug. GHB is well absorbed orally (within 15-30 minutes) and its effects last up to 2 hours. A dose as low as 5 g may cause significant CNS depression. Respiratory arrest and deaths have been reported in severe poisonings, with or without ethanol and other intoxicants. Flunitrazepam is, on a mg to mg basis, 5 – 7 times more potent than diazepam. It has a rapid onset of action (within 15 – 20 min) and a duration of 4 – 6 hours. Its use is also known to be associated with anterograde amnesia.

The inhalants of abuse include the aliphatic hydrocarbons, such as petrol, benzine and mineral turpentine; butane and propane (simple asphyxiants); toluene, xylene and benzene (aromatic hydrocarbons); and dichloroethylene, trichloroethane, carbon tetrachloride and chloroform (chlorinated hydrocarbons); and the general anaesthetic agent, nitrous oxide. Most inhalants are found in freely available commercial products such as glues, cleaning agents, nail polish remover, hair spray, type writer correction fluid, lighter fluid, deodorant propellants and other aerosols (e.g. Spray ‘n Cook), air fresheners, paints, paint thinners, paint removers and strippers. Intentional inhalation (“sniffing”) of high concentrations may produce unconsciousness, cardiac dysrhythmias, respiratory depression, seizures, and sudden death. Hypoxia and hypercapnia increase the risk of dysrhythmias (especially when re-breathing the mixture from a bag). Poppers are volatile nitrates (amyl, butyl, and isobutyl nitrite) used to improve sexual performance. These agents induce smooth muscle relaxation, including vasodilatation. The user may experience headache, flushing and postural hypotension. Seizures have been reported following high doses. Continued abuse may cause methaemoglobinaemia.

The mainstay of treatment of CNS depressant overdose remains symptomatic and supportive. Management includes assisting ventilation, administering oxygen and treating the cardiovascular complications. The specific management of severe ethanol poisoning includes fluid replacement and correction of electrolyte and acid/base disturbances. IV glucose should be given if the blood sugar is low. In chronic alcoholics, thiamine (Vitamin B₁) should be administered prior to IV glucose to prevent precipitating Wernicke’s encephalopathy. Hypothermia must also be
corrected. As haemodialysis effectively removes ethanol, it should be considered in severe cases where, despite conventional therapy, the condition deteriorates.

CNS depressants are known to cause physical dependence and acute withdrawal syndromes are often encountered in the addict when the drug is withdrawn. Characteristics of the withdrawal syndromes of all the sedative-hypnotics are markedly similar and may be life-threatening. When the syndrome is severe it may include disorientation, agitation, persecutory hallucinations, restlessness, tachypnoea, course tremors, adrenergic hyperactivity (tachycardia, hypertension), hyperthermia, delirium (delirium tremens in alcoholics) and tonic-clonic seizures. The benzodiazepines are the drugs of choice to block or diminish the above effects. Beta-adrenergic blockers, clonidine, carbamazepine, and (in certain situations) the neuroleptics, may be useful as adjunctive therapy. Phenytoin is not effective in alcohol related withdrawal seizures.

**OPIOIDS**

**Heroin** is the most commonly abused opioid and its popularity over morphine in the illicit drug trade, is due to its greater availability and higher potency. (Street names for heroin include brown sugar, H, Thai white, China white, dope, smack, junk). In its pure form, heroin is an almost white to off-white crystalline powder. In this form, it is more soluble in water than morphine, which makes it preferable for parenteral administration. Heroin (diacetyl morphine) is rapidly hydrolysed to 6-monoacetylmorphine (6-MAM), which in turn is hydrolysed to morphine. Evidence suggests that 6-MAM and morphine are responsible for the pharmacological actions of heroin. Street heroin is generally sold as an impure, brown powder. In addition to heroin, the brown powder may contain other opioids, adulterants (such as quinine, strychnine, PCP, scopolamine and cocaine.) and fillers (such as sugars and talc). The concentration of heroin in street heroin powder may vary widely. It may typically contain 20 - 60% heroin, but concentrations may even be as high as 70 - 90%. Overdose is likely to occur when the concentration of the street sample is unexpectedly high. Heroin is usually injected intravenously, but is also injected subcutaneously or intramuscularly. Heroin powder can also be smoked or snorted. “Chasing the dragon” refers to a method whereby it is heated on tin-foil and the
heroin vapours inhaled. Overdose may present with respiratory depression and failure, bradycardia, hypovolemia, pulmonary oedema, coma, aspiration pneumonia and rhabdomyolysis. Heroin is relatively short acting and its effects wear off in 3 – 5 hours.

Overdose with pethidine (meperidine), propoxyphene, tramadol, and pentazocine may be associated with seizure activity. In addition, pethidine toxicity may be associated with other excitatory effects such as hallucinations, tremors, muscle twitches, and dilated pupils. These effects are probably due to nor-meperidine, a toxic breakdown product of pethidine. (The management of opioid overdose is discussed in more detail on p____ of SAMF)

Physical dependence develops readily with prolonged, regular use of opioids. Although withdrawal symptoms and signs may be severe, as is the case with other CNS depressants, they are usually not life threatening. Severe withdrawal effects include nausea, vomiting, sweating, myalgias, arthralgias, shivering, rhinorrhea, rigidity, hyperpyrexia, agitated behaviour, dilated pupils, abdominal pain, mild tachycardia and raised blood pressure. Uncontrollable violent diarrhoea is common and may result in dehydration. Episodic clonic convulsions have been reported. Methadone (Physeptone), a long-acting opioid with properties similar to that of morphine, is effective as a substitute in suppressing the heroin withdrawal syndrome. An added beneficial property of methadone is that it is well absorbed by the oral route. Buprenorphine (Temgesic) may also be used to suppress withdrawal symptoms. Clonidine, an α2-agonist which decreases sympathetic outflow from the brain, may alleviate many of the autonomic symptoms and signs of opioid withdrawal, e.g. nausea, vomiting, abdominal cramps, sweating, tachycardia and hypertension.

**PSYCHOMOTOR STIMULANTS**

The psychomotor stimulants include the amphetamines and chemically related substances (methamphetamine, ecstasy, mescaline and khat), cocaine, nicotine and caffeine.
The amphetamines are indirect acting sympathomimetic amines, which means that a large proportion of their effects are due to the release of endogenous aminergic neurotransmitters, such as noradrenaline, serotonin and dopamine. Due to their high lipid-solubility, they are well absorbed in the gastrointestinal tract. The amphetamines also cross the blood-brain barrier readily and have prominent CNS effects. Examples include amphetamine and methamphetamine (crystal meth, speed, ice, tik). The amphetamines are usually taken orally, but are also smoked, snorted or injected. Other indirect acting sympathomimetic amines which may be abused include ephedrine, methylphenidate (Ritalin), and various appetite suppressants, such as d-phendimetrazine (Obex-LA), and diethylpropion (Tenuate Dospan).

Acute amphetamine toxicity is similar to that of cocaine toxicity, but has a longer duration of action. Cardiovascular effects such as palpitations, cardiac dysrhythmias, anginal pain and raised blood pressure are common. CNS effects include hyperactivity, agitation, delusions and paranoia. Hyperthermia, rhabdomyolysis, renal failure and subarachnoid haemorrhage may occur in severe cases. Fatal poisoning may terminate in convulsions and coma. Methamphetamine is potentially more toxic than amphetamine and severe psychosis has been reported after its use.

3,4-methylenedioxymethamphetamine (MDMA) is a hallucinogenic amphetamine, known as ecstasy (other street names include smiley, diamonds, safe sex, Durex, Bacardi, and 9/11). Incorporation of a methylenedioxy ring structure onto methamphetamine confers hallucinogenic properties to the compound. More than 50 hallucinogenic amphetamines have been synthesized. Although the term “ecstasy” was originally the street name for MDMA, it is now a loose term used for various substituted amphetamines, either alone or in combination with other drugs. A typical ecstasy tablet may contain 30 – 150 mg of MDMA. The usual dose of ecstasy is 1 – 2 mg/kg. It is important to note that contaminants and adulterants are frequently present. Large doses of ecstasy-like agents produce agitation, hypertension, tachycardia, mydriasis, sweating, and dehydration. The toxic effects are primarily due to release of serotonin, noradrenaline, and dopamine, centrally and peripherally. Severe poisoning is characterized by delirium, cardiac dysrhythmias,
hyperthermia, rhabdomyolysis (For a detailed discussion on rhabdomyolysis, see pg ----), hyponatraemia (due to inappropriate antidiuretic hormone secretion), seizures, hepatic and renal failure. DIC has also been reported. The clinical picture may resemble the neuroleptic malignant syndrome (See page ___ for serotonin syndrome).

Abuse of para-methoxyamphetamine (PMA), another hallucinogenic amphetamine, is reported to be associated with significantly higher incidence of morbidity and mortality than MDMA (ecstasy). PMA, also known as “Dr Death” or “Death”, increases extra-cellular serotonin levels to a greater extent than that produced by MDMA. The initial rush from the ingestion of PMA, however, is not as great as that experienced with MDMA, and this may lead to a greater tendency to overdose on PMA to achieve the initial “rush”. It is further reported that hypoglycaemia and hypokalaemia may be an additional complication of PMA poisoning. PMA may be used on its own (sometimes deceptively sold as ecstasy), or in a mixture together with other amphetamines. The use of PMA has resulted in several deaths in recent years.

Although ecstasy and other amphetamines are widely abused, serious toxicity and death are not as common when compared to alcohol intoxication. Acute toxic effects are related to dose, frequency of dosing, co-ingestion of other drugs, as well as environmental factors (e.g. hot night clubs, increased sweating, dehydration, etc).

Mescaline, a methoxylated amphetamine, is a hallucinogen found in the peyote cactus. Its effects are similar to that of the hallucinogenic amphetamines.

Khat (the name used for the leaves of Catha edulis), contains two sympathomimetic amines, cathinone and cathine, which are structurally related to ephedrine. The leaves are chewed. Because of the relatively low lipid-solubility of the two active ingredients, as compared to amphetamine, serious acute toxicity is not expected.

The treatment of intoxication with amphetamine and related agents (ecstasy) is mainly symptomatic and supportive. The benzodiazepines are the drugs of choice for the treatment of agitation and convulsions. Temperature should be monitored,
urine tested for myoglobin, adequate fluid intake assured, and electrolyte and acid-base disturbances corrected. Rapid cooling (fan and/or tepid water) and dantrolene are essential in the management of hyperthermia. Mechanical ventilation, with muscle relaxants, may be necessary to decrease heat production by overactive skeletal muscles. The serotonin antagonist, cyproheptadine, may be useful as adjunctive treatment. (See treatment of serotonin syndrome on page _________)

As is the case with most CNS stimulants, withdrawal from the amphetamines is usually mild and not directly life-threatening. Anxiety, abdominal cramps, gastro-enteritis, increased sweating, lethargy, and depression are common.

**Cocaine** (crack, rock, candy, snow) is an alkaloid extracted from the leaves of the shrub, *Erythroxylon coca*. Cocaine hydrochloride is usually snorted, but may be administered intravenously. Free-base cocaine is usually smoked, mixed with tobacco, or inhaled on its own by heating it in a special pipe. Crack is prepared by mixing a solution of cocaine hydrochloride with baking soda (NaHCO₃), which is then heated to produce a soft mass that dries into a hard ‘rock’. Crack is smoked in a pipe, often mixed with tobacco or marijuana.

Cocaine inhibits catecholamine re-uptake in both the central and peripheral nervous systems, thereby producing marked psychomotor stimulatory effects, such as euphoria (specifically through blockade of dopamine reuptake) and enhancing sympathetic nervous system activity. (Stimulation of the sympathetic nervous system appears to play an integral part in the pathogenesis of cocaine toxicity.) Applied locally to mucous membranes, it causes intense local vasoconstriction. Cocaine is also a potent local anaesthetic agent, which acts by blocking neuronal voltage sensitive sodium channels. Cocaine has a short plasma half-life (30 – 90 min). Snorted, as cocaine hydrochloride, it takes 3 – 5 minutes to reach the brain. Due to its local vasoconstricting action, cocaine may take up to 60 minutes to reach its peak effect, with a duration of action of up to 6 hours. Inhaled (smoked) crack cocaine reaches the brain within 6 – 8 seconds and has a shorter duration of action.

Factors predisposing individuals to acute cocaine poisoning include high, continuous dosing (especially if administered intravenously), co-ingestion of excessive amounts
of alcohol, tobacco smoking, pre-existing cardiovascular diseases (such as hypertension and advanced arteriosclerosis) and patients on drug therapy for psychiatric diseases. Cocaine toxicity may occur hours after peak plasma levels are reached.

Clinical features of acute cocaine poisoning can be summarized as follows: CNS toxic effects include anxiety, restlessness, delirium and seizures. Brain and spinal cord infarction, intracerebral and subarachnoid haemorrhage have been reported. A wide range of cardiovascular (CVS) complications may occur, including self-limited chest pain (without ECG signs of myocardial ischaemia), tachycardia, high blood pressure, and supraventricular or ventricular dysrhythmias. Sudden death is often due to ventricular tachycardia and fibrillation. Severe high blood pressure may lead to cerebral haemorrhage or aortic dissection. Acute myocardial ischaemia, and/or infarction, may occur in patients with normal or diseased coronary arteries, hours to several days after the last dose of cocaine. Cocaine can also cause myocarditis. Cardiogenic pulmonary oedema may be a feature in fatal cases. Ischaemia, due to peripheral vasoconstrictive effects of cocaine can occur in fingers, toes, kidneys, gut and spinal cord. Acute respiratory symptoms (often after smoking crack) may include bronchospasm, pleuritic chest pain, interstitial pneumonitis and haemoptysis. Inhaled impurities can cause direct bronchial and alveolar injury. Metabolic acidosis, hyperthermia, and rhabdomyolysis may develop. Renal failure has been reported. Inhalation of hot cocaine vapours may cause thermal burns of the upper airways.

The benzodiazepines are the drugs of choice for the treatment of agitation, dysphoria and seizures. Transient hypertension and sinus tachycardia can also be treated with the benzodiazepines, because these effects are largely mediated by CNS mechanisms. Symptomatic and supportive treatment includes monitoring the urine for myoglobin (rhabdomyolysis), lowering body temperature in hyperthermic patients, and correcting acid-base and electrolyte disturbances. Dantrolene is essential in hyperthermic patients who fail to respond to diazepam and conventional cooling methods. Myocardial ischaemia usually responds to diazepam, probably through the reduction of central sympathetic stimulation. Aspirin may be given prophylactically to reduce the likelihood of thrombus formation. Sodium nitroprusside is indicated for refractory hypertension. Nitroglycerine or Ca-channel blockers may
be used to reverse vasoconstriction of coronary arteries. Although life-threatening ventricular arrhythmias have been shown to respond well to propranolol, the use of beta-adrenergic blockers alone could lead to unopposed alpha-adrenergic effects, which could aggravate coronary vasoconstriction. Labetalol is preferable (over propranolol) due to its additional selective alpha₁-adrenergic blocking activity. Symptoms of cocaine withdrawal are usually mild and the treatment is symptomatic and supportive. Diazepam may be given for dysphoric agitation.

**Nicotine** is arguably the most dangerous dependence-producing drug with regard to chronic toxic effects. Smoking of tobacco, chewing, and its use as snuff are, however, not likely to cause acute nicotine poisoning. Symptoms and signs of nicotine withdrawal, such as anxiety and irritability, do not warrant any specific treatment.

The management of cocaine body packers is discussed elsewhere (p………).

**THE PSYCHOTOMIMETICS, ALSO KNOWN AS THE PSYCHEDELIC AGENTS (LOOSELY REFERRED TO AS HALLUCINOGENS)).**

The primary effects produced by the psychedelic drugs include distortion of perception (such as hallucinations and illusions), and disorders of thinking (such as paranoia). Drugs in this category include LSD (lysergic acid diethylamide), phencyclidine, ketamine, the anticholinergic drugs and the hallucinogenic (magic) mushrooms. The hallucinogenic amphetamines, such as ecstasy (MDMA) and the chemically related mescaline may also be included in this group (see also page__________).

**LSD** (acid, candy, blotter, microdots) is odourless, colourless and has a slightly bitter taste and is usually taken by mouth. Acute toxic effects include restlessness, anxiety, tremors and incoordination, dilated pupils, depersonalization, hallucinations (usually visual), illusions (often more pronounced than hallucinations), paranoia, panic reactions, and mild hypertension. Severe anxiety, panic reactions and paranoia, often combined with depression and suicidal thoughts, may be perceived as a “bad trip”. The above symptoms and signs may superficially resemble a
psychotic illness. Although rhabdomyolysis, malignant hyperthermia, seizures and coma have been reported, these complications are uncommon. LSD is rapidly absorbed orally, as well as through other mucous membranes, e.g. through the nose ("snorting"). The psychedelic effects begin within 40 – 60 minutes, peaking in 2 – 4 hours and subsiding within 6 – 8 hours. Acute psychotic reactions, however, may last for up to 2 days or longer. LSD, commonly referred to as "acid," is sold on the street in the form of tablets, capsules and, occasionally, as a liquid. LSD is often impregnated onto blotting paper, which is then divided into small, decorated squares, each of which represents one dose.

**Phencyclidine** (PCP, angel dust) is a white crystalline powder that is readily soluble in water or alcohol. It is available on the illicit drug market in the form of tablets, capsules, and coloured powders. It is usually snorted, smoked, or ingested. When smoked, PCP is often mixed with material such as mint, parsley, oregano, or marijuana. The effects of phencyclidine can last for days and are characterised by marked paranoia and violent behaviour. Hypertension and increased sweating are common. Seizures, muscle rigidity, respiratory depression, stupor and coma may occur. PCP coma may last for 7 to 10 days. Hyperthermia and rhabdomyolysis, with renal failure, have been reported. Ketamine, a dissociative anaesthetic agent, has similar effects to that of phencyclidine.

The most common form of deliberate **anticholinergic intoxication** is by ingestion of plants containing belladonna alkaloids (atropine-like substances). *Datura stramonium* (chewing the ripe seeds) and the *Brugmansia* species (drinking a decoction of the plant material) are most commonly involved. This type of poisoning occurs mostly amongst teenagers experimenting with drugs. The anticholinergic effects include a hot, dry, flushed skin, dry mouth, dilated pupils, urinary retention, tachycardia, fever, disorientation, hallucinations, aggressive behaviour and delirium. Seizures, coma and rhabdomyolysis may occur in severe cases.

The **“magic” mushrooms**, such as the *Psilocybe*, *Conocybe* and *Panaeolus* species, contain hallucinogenic indoles, such as psilocybin and psilocin. Symptoms and signs of intoxication are similar to that of the other hallucinogens.
Management of the above acute psychedelic intoxications is symptomatic and supportive. Calming down and reassuring the patient is a good first-line approach. The benzodiazepines are the drugs of choice for agitated and aggressive states. Temperature should be monitored, urine tested for myoglobin and adequate fluid intake ensured. Patients should also be closely observed to prevent injuries, and those with dilated pupils should be managed in a darkened room.

As is the case with other CNS stimulants, chronic abuse of psychedelic agents is usually not associated with any significant withdrawal syndromes.

Marijuana (dagga, hashish, cannabis, Durban poison, Malawi cob) is also classified under the psychotomimetic agents. Δ-9-tetrahydrocannabinol (Δ-9-THC) is the cannabinoid responsible for most of its pharmacological effects. Marijuana is usually smoked (often mixed with other drugs of abuse, such as methaqualone). Absorption from the oral route is relatively poor (only 5 - 20% of THC is bioavailable). Acute effects are usually mild, but heavy daily use may be associated with acute anxiety, panic attacks, hallucinations and even acute psychosis. Tachycardia and orthostatic hypotension have been observed. Withdrawal symptoms and signs are not typically seen. Abrupt discontinuation in heavy users, however, may present with cramping, restlessness, irritability, agitation and aggressiveness. Specific treatment is usually not required. The benzodiazepines are recommended for the treatment of severe reactions.

BODY PACKING (INTERNAL CONCEALMENT OF ILLICIT DRUGS)

Body packers are individuals who ingest multiple packets containing illicit drugs, for the purpose of smuggling them across international borders. The packets may also be found inserted into the rectum or vagina. The drug is most commonly packaged in latex sheaths, such as condoms, and sealed with wax. Cocaine, heroin, and occasionally the amphetamines, are the main drugs transported in this manner.

Body packers are usually asymptomatic. Occasionally, however, severe acute poisoning can develop quite rapidly due to leakage or rupture of a packet. Each of
the packets may contain many times the lethal dose of the drug. Bowel obstruction may be an additional complication of body packing.

General Management: (See sections on individual illicit drugs for more detail.)

- Abdominal X-ray should be performed to establish diagnosis and location.
- The body packer should be carefully monitored in a medical facility until all the packets have been expelled.
- Activated charcoal is recommended, unless surgery is anticipated.
- In the absence of obstruction, whole bowel irrigation with polyethylene glycol-electrolyte solution (Go-Lytely) is recommended. Irrigation should be continued until clearance of all packets is confirmed. Oil-based laxatives should be avoided, since they may damage latex products.
- Leaking cocaine packets should be removed surgically as soon as possible (indicated by signs and symptoms of acute poisoning), since no specific antidote is available which significantly antagonizes the potential lethal effects of cocaine.
- The amphetamine-like agents, including ecstasy, may cause a clinical syndrome similar to that of cocaine poisoning. Management is, therefore, the same as that of cocaine. Prompt surgical removal of leaking packets is also indicated.
- Opioid poisoning should be treated conservatively with continuous infusion of naloxone. Very high doses may be necessary. (see management of opiates on page -------)

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G.J. Müller, MBChB, Hons. BSc (Pharm. and Tox.), MMed. (Anes.), PhD.
Department of Pharmacology
Faculty of Health Sciences
University of Stellenbosch
PO Box 19063
Tygerberg 7505.