

National Centre for Education and Training on Addiction



The National Methamphetamine Symposium

This resource is part of NCETA's methamphetamine resource package developed for the National Methamphetamine Symposium, 12 May 2015.

This resource and other methamphetamine related materials are accessible from NCETA's website:

www.nceta.flinders.edu.au



Alcohol & Other Drugs:

A Handbook for Health Professionals

Alcohol and Other Drugs:

A Handbook for Health Professionals

© Commonwealth of Australia 2004

ISBN 0 642 82312 X

This work is copyright. Apart from any use as permitted under the Copyright Act 1968, no part may be reproduced by any process without prior written permission from the Commonwealth available from the Australian Government Department of Communications, Information Technology and the Arts. Requests and enquiries concerning reproduction and rights should be addressed to the Manager, Copyright Services, Info Access, GPO Box 1920, Canberra ACT 2601.

Australian Government Department of Health and Ageing Publication approval number: 3315

Suggested Citation:

National Centre for Education and Training on Addiction (NCETA) Consortium. (2004), Alcohol and Other Drugs: A Handbook for Health Professionals. Australian Government Department of Health and Ageing.

www.nceta.flinders.edu.au

Amphetamines

MPHETAMINES are the second most commonly used illicit drug in Australia after cannabis. There is evidence of increasing use and purity, and of serious harms associated with regular use. Health workers can expect to see increasing numbers of amphetamine users.

Although there are few specific interventions or treatment options for those experiencing problems related to their use of amphetamines, engaging individuals in harm reduction measures and responding to their specific needs can substantially reduce harms.

PHARMACOLOGY

Amphetamine is a closely related family of drugs with psychostimulant properties. This group of drugs includes:

- amphetamines used for recreational purposes, produced in illegal or 'clandestine' laboratories e.g. amphetamine sulphate/amphetamine and methamphetamine/methylamphetamine
- pharmaceutical quality amphetamines available on prescription for the treatment of obesity, narcolepsy, and Attention Deficit Hyperactivity Disorder (ADHD)/(ADD). These drugs include:
 - □ phentermine (Duramine®)
 - □ diethylpropion (Tenuate®)

- □ dexamphetamine
- □ methylphenidate (Ritalin®) (Victoria Police, 2002)

Amphetamines (including methamphetamine) are synthetic substances structurally related to naturally occurring adrenaline and ephedrine. Amphetamines activate the central nervous system (CNS) and sympathetic nervous system (SNS), increasing synaptic concentrations of excitatory neurotransmitters and inhibiting their reuptake. The monoamines affected by amphetamines are:

- dopamine
- noradrenaline
- serotonin

Through stimulating neurotransmitter release, and preventing their reuptake, amphetamine use results in:

- CNS effects: euphoria; increased wellbeing, confidence and physical activity; improved cognitive and physical performance; suppression of appetite and need for sleep
- SNS effects: increased blood pressure, tachycardia or reflex bradycardia, increased temperature (Victoria Police, 2001, Latt et al., 2002)

Distribution

Amphetamines are concentrated in the brain, lungs and kidneys.

Metabolism

Between 30–40% of amphetamines are metabolised by the liver, with the remaining 60–70% excreted by the kidneys. The half-life of amphetamine and methamphetamine are 12–36 hours and 8–17 hours respectively. Although amphetamines can be more rapidly eliminated if the urine is artificially acidified (Victoria Police, 2001; Latt et al., 2002), the practice of acidifying urine is

believed to increase risk of renal failure from rhabdomyolysis (Wickes, 1993). Some amphetamine users deliberately exploit this fact by alkalinising their urine to prolong the effects.

Availability and Quality

Methamphetamine. Commonly known as 'speed' or 'whiz'. The term speed previously referred to amphetamine sulphate, however the powder form has been superseded in recent years by the more potent methamphetamine. Speed varies in:

- texture (fine to crystallised or coarse powder)
- colour (white to yellow, brown, orange or pink), and
- purity

Variation in production techniques and chemicals used ensure that it is virtually impossible to estimate drug quality or purity through taste, smell or appearance. Speed is usually snorted or injected, and less often mixed with drinks (including alcoholic drinks). Speed is usually purchased in grams or ounces, but contains only around 5–20 mg of amphetamine, the remainder comprised of bulking agent (e.g. ascorbic acid). Prices during 2000/01 ranged from \$50–100 per gram of powder (Topp & Churchill, 2002).

Crystal methamphetamine (ice, crystal meth) is the crystalline form of high purity methamphetamine. It originates in Asia, and has a 'crushed ice' appearance (large translucent to white crystals or coarse crystalline powder). Crystal meth is usually smoked, although it is also snorted, swallowed or injected (it dissolves in water to break down into smaller particles). Snorting may cause significant nasal damage. Most often sold in 'points' (0.1 gram; \$50 per point in 2000/2001) (Topp & Churchill, 2002).

Free base methamphetamine (base, wax, paste, point, pure) is a damp, sticky, gluggy powder, of a yellow or brown colour which results from imperfect manufacturing processes. It can be swallowed, smoked, snorted or injected. Due to its oily consistency it is difficult to dissolve without heat, and hence is associated with vein problems. It is also sometimes mixed with a dry substance (e.g., vitamin powder) for snorting. In 2000/01, base cost between \$30–50 per 'point' (0.1 gram) (Topp & Churchill, 2002).

Methamphetamine pills currently make up approximately 80% of tablets marketed as ecstasy (MDMA), and are deliberately manufactured to appear similar to ecstasy tablets. Drugs such as ketamine may be included in the manufacturing process to produce hallucinogenic or MDMA-like effects. These pills vary widely in purity, tend to be available in most jurisdictions, and during 2000/01 cost around \$30–40 each (Topp & Churchill, 2002).

PATTERNS OF USE

The 2001 National Drug Household Survey found that 8.9% of the population (aged 14 years and over) reported having ever used amphetamines, with 3.4% reporting use in the last 12 months. One in nine people aged 20–29 years have used amphetamines in the past 12 months. In general, males are more likely to use amphetamines, although there is little gender difference amongst teenagers who use amphetamines. Of all illicits, amphetamines are most likely to be the first drug ever injected, and the drug most recently injected (AIHW, 2002).

Many people take small amounts of amphetamines in specific social settings (e.g. dance parties, 'raves') and never meet the criteria for dependence. However, there are trends in patterns of use that suggest:

- heavy users tend to use amphetamines in binges often lasting days (called a 'run'), followed by a period of abstinence (see Figure 6-1)
- heavy users will often use amphetamines concurrently with other drugs (especially alcohol, cannabis, benzodiazepines and heroin), and may use CNS depressants to help 'come down' after a binge

Routes of Administration

Amphetamines can be administered in a number of ways, depending on the form of the drug, desired effect, dose required and previous experience in mixing and injecting. Level of effect and risks according to route are outlined in Table 6–1.

PHYSICAL AND PSYCHOLOGICAL EFFECTS

Methamphetamine is more potent than amphetamine. It is considered to be more addictive, and responsible for greater harm. Users of methamphetamine are more likely to report anxiety, aggression, paranoia and psychotic symptoms compared with amphetamine (Topp & Churchill, 2002). Physiological effects are similar to cocaine, but longer lasting.

Acute Physical and Psychological Effects

See Table 6–2 for an overview of acute physical and psychological effects of amphetamines.

Long-term Physical Effects

- weight loss, malnutrition, lowered immunity, although with re-establishment of self-care and eating habits, likely to resolve over time
- eating disorders, anorexia or nutritional deficiency

Table 6-1 Routes of administration, effects and risks

Route	Effect	Risks		
Intravenous	Intense peak effect within seconds of administration lasting a few minutes, then reduction in intensity over the next 4–6 hours.	Intoxication with any drug may lead to risk taking behaviour such as sharing needles or equipment, hence increasing risk for contracting blood borne viruses (BBV).		
		Injection risks include:		
		 inflammation, infection, scarring, or abscess at IV site 		
		 introduction of contaminants, which may result in thrombosis 		
		 increased risk of developing tolerance and dependence 		
		 acute intoxication risks from IV use such as psychosis, seizures, cardiovascular complications (incl. arrhythmias, cerebrovascular accident), hallucinations, accidents and injury. 		
Smoking/ inhalation	Slightly less intense onset and duration of effect.	Best route for controlling dose, though relatively uncommon. This route is second to injection for rapidity of effect.		
'chasing the dragon'	GIIGOL	May have sore throat, bloody sputum, and potential exacerbation of asthma.		
Snorting	Weaker onset and slower reduction in intensity relative to injecting but slightly longer lasting.	Damages epithelium and nasal septum, potentially causing nasal ulcers, runny nose, sinusitis, and septum perforation.		
Swallowing or 'bombing'	Delayed absorption (about 30 minutes to 'come on', slower peak, slower reduction, lasting	Impatience waiting for effect, inability to control the dose, or seeking a stronger or more intense effect may result in taking more drug/s, possibly increasing intoxication risks, and duration of effects.		
	around 6 hours).	Variable effect depending on presence of food and rate of gastric emptying (speed can inhibit this process to produce an anorexic-like effect).		
Anal (shelving)	Effects unpredictable, vary with quality and quantity of drug, and form (powder, capsule, wrapping).	Highly acidic forms may irritate mucosal lining.		
		Time is required for absorption to occur before effect is experienced (see oral use above).		

Table 6-2 Potential acute physical effects from using low and high doses of amphetamines

	Low doses	High doses	
CNS, neurological, behavioural	 overstimulation, insomnia dizziness, mild tremor euphoria/dysphoria, restless, talkative, excited with need to speak increased confidence, self-awareness mild confusion, panic (rarely psychotic episodes) appetite suppression pupillary dilatation increased energy, stamina and reduction in fatigue heightened alertness and psychomotor activity with improved performance or concentration on simple fatigue impaired tasks with increasing doses, may increase libido headache teeth grinding 	stereotypic or unpredictable behaviour violent or irrational behaviour, mood swings, including hostility and aggression pressured or slurred speech paranoid thinking, confusion and perceptual disorders headache, blurred vision, dizziness psychosis (hallucinations, delusions, paranoia) cerebrovascular accident* seizures coma teeth grinding gross body image distortions	
Cardiovascular	 tachycardia (possibly brief bradycardia), hypertension palpitations, arrhythmias 	cardiac stimulation (tachycardia, angina, arrhythmia*, MI) vasoconstriction / hypertension cardiovascular collapse*	
Respiratory	increased respiration rate and depth	respiratory difficulty/failure*	
Gastrointestinal	nausea and vomitingconstipation, diarrhoea or abdominal cramps	dry mouth nausea and vomiting abdominal cramps	
Skin	pale sweaty skinhyperpyrexia	flushing or pallor hyperpyrexia, diaphoresis	
Skeletal	 increased deep tendon reflexes 		

(Adapted from Gourlay, 2000; Latt et al., 2002; Victoria Police, 2002)

- possible cerebral atrophy and impairment of neuropsychological functioning
- poorly maintained injection sites (e.g. infection) may cause callusing, scarring or abscesses
- vascular and organ damage may occur due to blockages caused by particles blocking small blood vessels in organs (e.g. kidneys). Contaminants present in the blood stream (from acute injection or due to longer term accumulation) may result in lung or cardiac emboli, cardiac valve infections, or stroke
- sexual dysfunction
- cardiovascular symptoms consistent with shorter term use patterns (such as hypertension and cardiac arrhythmias)

(Gourlay, 2001; Latt et al., 2002; Victoria Police, 2001)

Long-term Psychological Effects

psychological problems associated with amphetamine intoxication include delirium, paranoia, acute anxiety, and tactile hallucinations, which tend to readily resolve upon resolution of intoxication. Some people may experience a brief psychotic reaction of a few week's duration that was precipitated by amphetamine use. Amphetamine-induced psychosis tends to resolve on cessation of drug use and with short-term pharmacological treatment (usually haloperidol and diazepam). Reinstatement of amphetamine use may increase the likelihood of further psychotic episodes, however, repeated episodes may not necessarily cause, nor be related to schizophrenia-like disorders. Some people may experience a schizophrenia-like illness that appears to be precipitated by their use of amphetamines, however it remains unclear whether the drugs are responsible for the condition or rather increase the likelihood of its occurrence in susceptible individuals (Latt et al., 2002; Todd, 2002).

- depression, other mood disorders (e.g. dysthymia), or eating disorders may be features of protracted withdrawal or become long standing problems post-drug cessation. Also consider the context of multiple losses experienced by people changing long established drug-oriented behaviours (loss of, or damaged relationships, lack of employment, financial insecurity, homelessness etc.), and take care not to overdiagnose concurrent psychiatric disorders that may be based on lifestyle factors associated with drug use (e.g. involvement in criminal activities or prostitution in order to obtain money for drugs) (Latt et al., 2002; Saunders & Young, 2002).
- highly dependent individuals show poorer performance on tests of cognitive functioning, especially with memory and concentration (McKetin & Mattick, 1998).

Amphetamine-related Harms

Like other drugs, effects extend beyond the subjective physical and psychological. A practical way to engage patients may be to consider the range of potential amphetamine-related harms and implications of use, from acquiring and using the drug to symptoms of withdrawal (see Table 6–3) (Pead, Lintzeris & Churchill, 1996, p. 36).



Table 6-3 Amphetamine-related harms

Acquisition	Administration	Intoxication	Intoxicated behaviour	Withdrawal/ crash
Not enough money Police and jail Underworld Poor relationships Unknown drug quality Ripped off by dealers Dealing Supplying Alienation Secrecy/stigma	Vein abscesses and scarring Thrombosis Contaminants BBV Nasal infections Needle sharing	Agitation Weight loss Tachycardia Dehydration Hyperthermia Poor immunity Paranoia Delusions Hallucinations Restlessness Sleeplessness Seizures Teeth grinding Stroke Cardiovascular problems Death	Aggression/ fights Alcohol use Driving Parenting Risk taking Accidents/injury Unsafe sex Social avoidance Other drug use Relationship problems	Depression Restlessness Cravings Suicidal ideas Lapse to drug use Job issues Bizarre thoughts Flat mood Dependence Poor social functioning

MANAGEMENT AND INTERVENTION STRATEGIES

Acute Adverse Effects: Uncomplicated Intoxication

Management strategies for acute amphetamine and cocaine intoxication are the same. Focus on the management of psychosocial aspects (reassurance and support) and manage somatic complaints as they emerge.

Generic strategies for managing clients who are intoxicated and uncomfortable include:

- provision of non-stimulating environment
- provision of support and reassurance
- preventing harm to self and others
- keeping the person safe

Other general measures include:

 avoidance of confrontation or arguments whilst allowing the user to satisfy their need to talk

- creation of a sense of security and confidence that the situation is under control
- encouragement of supportive friends and relatives to stay with the person
- reduction of environmental stimuli as much as possible
- monitoring of vital signs (urine drug screening may be useful if there is uncertainty regarding drugs used)
- provision of food and fluids with encouragement to maintain fluid intake
- benzodiazepines may be indicated if agitation and anxiety are the most prominent symptoms and are not controlled by environmental measures (diazepam 10–20 mg orally, repeated every 1–2 hours until symptoms settle). Higher doses may be required if the person is dependent on benzodiazepines
- antipsychotic agents e.g. haloperidol, may be indicated for psychotic episodes where sedation from benzodiazepines is insufficient (Wickes, 1993)

Acute Adverse Effects: Intoxication with Complications

Complications are rare, but when they occur they can be life threatening, requiring intensive care. Management strategies for acute psychostimulant intoxication with complications include:

1. Obtain accurate diagnosis

- include in differential diagnosis as a history of drug use may not be volunteered, or where the 'patient may be unconscious or acutely anxious, paranoid and belligerent'
- obtain a history from patient (where possible) or others (friends, relatives, onlookers, dealers, ambulance officers, police etc.)
- initial symptoms may include nausea, vomiting, general malaise, excessive diaphoresis, chest or abdominal pain
- evidence of drug administration (injection site, nasal septum damage)
- high arousal states may mimic psychostimulant toxicity (tachycardia, increased blood pressure, temperature, dilated pupils)
- life-threatening conditions associated with psychostimulant toxicity include acute myocardial infarction (MI) and ischaemia (without pre-existing heart disease), arrhythmias (ventricular tachycardia or fibrillation, asystole), hypothermia, convulsions, subarachnoid haemorrhages and cerebral infarctions, aortic dissection, bowel ischaemia and infarction, rhabdomyolysis, renal failure
- violence may be an outcome of psychostimulant induced paranoid ideation (with or without psychosis) (Wickes, 1993)

2. Management strategies

Treat signs and symptoms as they arise, but where appropriate refer for further medical or psychiatric assessment. In general:

- correct and monitor fluid and electrolyte disturbances and hypothermia
- extreme agitation: sedate with benzodiazepines
- conduct mental state assessment where the clinician is concerned about a patient who appears overly suspicious, appears to be experiencing delusions, hallucinations, or is misinterpreting their surroundings or interactions with other people. These features may manifest in behaviours such as significant concern about personal safety (checking doors, windows, hiding). Check whether the person is carrying a weapon. Where possible, identify previous occurrence of these behaviours, whether they are related to previous episodes of intoxication, and prior mental health history (Pead et al., 1996)
- choose haloperidol over phenothiazines for psychosis if present, as phenothiazines lower seizure threshold. May require referral and admission to a psychiatric institution for short-term management of psychotic symptoms
- monitor vital signs. ECG monitoring may be indicated to assist in detecting cardiac disturbances
- hyperthermia: if the temperature rises rapidly or above 39°C implement rapid cooling measures, sedation and hydration, with intensive care if temperature continues to increase
- rhabdomyolysis: all patients at risk (postseizure, prolonged agitation, or hyperthermia) should have regular creatine kinase (CK) analysis, receive sedation for agitation, be fully hydrated and closely monitored. Intensive care may be required (Wickes, 1993)

If unconscious, general measures include:

- observation of airway, breathing and circulation
- check evidence of injury
- screen urine or blood to confirm diagnosis or use of other drugs that may complicate presentation
- if suspicious of significant ingestion of alcohol, administer intravenous thiamine (100 mg) prior to using glucose to prevent onset of Wernicke's encephalopathy (50 ml of 50%). If opioid overdose is suspected, naloxone (0.4–2.0 mg) would be appropriate
- CT scans or lumbar puncture may be warranted to diagnose subarachnoid or cerebral haemorrhage, infarctions or infections (Latt et al., 2002; Wickes, 1993)

USING AND STOPPING AMPHETAMINES

Identification and Detection of Amphetamine Use and Related Problems

Expressing health-related concerns about the possible effects and consequences of amphetamine use (e.g. grinding teeth, increased heart rate, insomnia, etc.) may have little relevance or impact on the subjective experience of the user.

Many amphetamine users are not dependent and only use occasionally. More regular users frequently adopt a 'binge' pattern. As seen in Figure 6–1, a typical pattern of speed use commences with the intoxication phase, or 'run' (a single session of a few days to weeks),

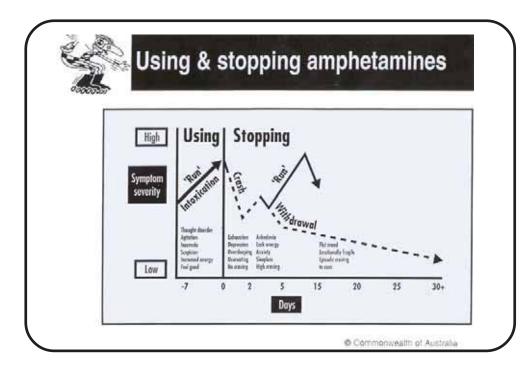


Figure 6-1 Using and stopping amphetamines (Pead et al., 1996, p. 30)

followed by a short period of abstinence, or the 'crash' (feeling flat, tired, withdrawn, poor appetite, few cravings). For dependent users, reinstatement of use (another 'run') may occur, however, if use is ceased, withdrawal may be experienced.

While amphetamines may result in, or exacerbate health, social or mental health problems, many people will not link these problems with their drug use. Triggers to assist discussion about lifestyle factors incorporating amphetamine use may include features of intoxication, withdrawal or crash, such as:

- overwhelming tiredness at the beginning of the working week
- otherwise unexplained irritability, agitation or mood swings
- difficulty concentrating, poor work or study performance
- mental health problems, such as paranoia, delusions, feeling generally flat or depressed
- apparent unconcern about otherwise serious matters
- health problems, such as palpitations, infected injection sites or lesions

Other discussion triggers may include:

- drug seeking behaviour (benzodiazepines, opioids, codeine)
- occupation (e.g. shift workers, transport, medical and hospitality industries, students and musicians)
- age (young adults)

Prolonged or high dose use, and injecting use, tend to be associated with dependence. For assessment of dependence use DSM-IV or ICD-10 criteria, the Severity of Dependence Scale (SDS) for psychological dependence (Gossop et al., 1995) or the Leeds Dependence Questionnaire (LDQ) (Raistrick et al., 1994).

ASSESSMENT

1. Take a lifestyle approach

- ask about needs, lifestyle, current stresses, and role of drug use
- encourage patient to talk about problems
- elicit motivation for change
- focus on feelings and behaviours rather than referring to 'your drug problem' or 'addiction'

2. Identify patterns of drug use

- pattern and duration of use (binge patterns are more common than patterns of daily use)
- quantity (measured in grams, points (there are 10 'points' in a gram) or dollars)
- route(s) of administration
- recent history of use (past 2–3 weeks)
- other drug use
- physical, social and psychological issues
 - ☐ Has the patient linked problems with their speed use?
 - ☐ Has use continued despite evidence of problems?
- tolerance/severity of dependence
- assess value of additional information sources (amphetamines are detectable in urine for about 48 hours after use)

3. Obtain evidence of medical/ psychiatric illness

- existing medical care
- current medications

4. Identify psychosocial factors

- social and family supports
- living arrangements and accommodation
- employment/finances
- relationships, dependents
- legal issues

(Pead et al., 1996)

WITHDRAWAL

The 'typical' pattern of 'Using and Stopping', as illustrated in Figure 6–1, varies across individuals and with previous withdrawal experiences. Most withdrawal signs and symptoms dissipate over the course of two weeks to a month, however, withdrawal may be protracted, lasting a few months or more.

During the crash phase (days 1-4 post cessation of use) common complaints may include:

- fatigue and exhaustion
- hunger
- emotional lability (irritable, agitated, depressed)
- overwhelming desire to sleep, or sleeping difficulties
- cravings

During the crash phase, advise carers to ensure that adequate food and fluids are provided and encouraged.

During the next week, typical complaints include:

- strong cravings or urges to use
- disrupted sleeping patterns and sleeping difficulties
- mood swings
- headaches, and generalised aches and pains
- increased appetite
- irritability, possibly paranoia or misinterpretation of surroundings

During the following weeks, most signs and symptoms tend to subside, with mood swings, sleeping problems and cravings causing patients the most difficulty. After 1–3 months, sleeping patterns, health and interest in other activities should return to normal.

Non-pharmacological Management of Withdrawal

Psychosocial management is crucial in providing support for people withdrawing from alcohol or other drugs. Supportive care is crucial to reducing the incidence and severity of somatic complaints, for example:

- organising a safe environment
- organising supports
- non-pharmacological means of coping with cravings
- tips to improve sleep
- relaxation techniques
- coping with mood swings, strange thoughts and aches and pains
- eating properly
- concentrating only on the immediate future
- identifying high risk situations
- obtaining counselling (Lintzeris et al., 1996)

Abstinence from all psychoactive drugs is the preferred treatment goal, as other drugs may trigger reinstatement or reduce ability to cope with cravings. There is no evidence to suggest that either inpatient withdrawal management or tapered withdrawal with amphetamines or other drugs is any more effective in achieving long-term cessation of the use of amphetamines.

Inpatient withdrawal management

Inpatient treatment may, however, be appropriate in the following circumstances:

- evidence of polydrug dependence
- where severe withdrawal is anticipated
- for medical complications requiring close observation or treatment
- psychiatric complications (e.g. psychotic, suicidal)
- absence of social supports
- previous failed outpatient treatment
- for specific therapies e.g. introducing cue exposure

Where inpatient treatment is necessary, programs should be tailored to the specific needs of the patient, focusing on management of emotional lability (mood change) and cravings. Patients should ideally remain in inpatient care until the main withdrawal symptoms subside, however, days 3-5 following cessation of use are often risky, and for many, may result in early self-discharge. Encourage usual sleeping patterns, dietary and self-care habits, and provide distractions from drug using activities. Because of the protracted nature of amphetamine withdrawal, encourage involvement in outpatient programs for additional support and relapse prevention. For further information refer to Lintzeris et al. (1996) 'Getting through withdrawal — amphetamines'.

Pharmacotherapies for Managing Withdrawal and Relapse

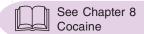
Evidence is inconclusive regarding the efficacy of pharmacotherapies in managing amphetamine withdrawal or relapse, however trials with dexamphetamine show promise as a replacement therapy (see Shearer et al., 2001). For a review of the literature see Kamieniecki, Vincent, Allsop & Lintzeris (1998). Medications that may assist in reducing the severity of withdrawal symptoms include:

- Somatic symptoms: mild analgesics (such as paracetamol)
- Anxiety and insomnia: a short low dose course of benzodiazepines may reduce irritability and promote sleep (e.g. diazepam, p.r.n. for a week or less)
- Gastrointestinal complaints such as diarrhoea, cramps, nausea and vomiting: loperamide, hyoscine butylbromide, metoclopramide (Victoria Police, 2001). (Whilst these symptoms are normally associated with heroin withdrawal they may not necessarily be unusual in a person tolerant to amphetamines, who is a polydrug user with a recent history of poor self-care.)

 Cravings and dysphoria: desipramine, bromocryptine, amantadine



For patients undergoing home withdrawal management, ensure that an appropriate person is available to monitor medications. The treatment outcome literature for managing relapse in cocaine users is also relevant for amphetamine users and much more extensive.

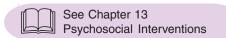


Intervention Strategies Post-withdrawal

There are a number of strategies health workers can use to intervene with problematic amphetamine use. These should be individualised. There are few randomised controlled trials of counselling (e.g. Baker et al., 2001) and much of the outcome literature is based on cocaine users. A review of the evidence (Kamieniecki et al., 1998) recommended:

- cognitive-behavioural therapy/relapse prevention (particularly for heavy users)
- cue exposure therapy
- multi-faceted behavioural treatment involving family support, addressing the antecedents and consequences of use, employment counselling and recreation

The principles for intervening with amphetamine users are similar to those employed with other drugs. Engaging clients, planning withdrawal management, skill development (goal setting, relapse prevention etc.) within the context of the client's readiness to change are important.



Harm Reduction Measures

For people likely to experiment with amphetamines

Discuss advantages and disadvantages of oral versus other forms of amphetamine administration. Discuss hazards of injection, without exaggerating risks of occasional oral use of low doses of amphetamines, and discourage injecting.

For current amphetamine users

Advise:

- against daily use
- against injection, or to use other forms of administration
- if injecting, encourage use of new injecting equipment, and awareness of locations of services that provide new needles and equipment. Stress that all injecting paraphernalia and the using environment must be sterile to avoid local infection risks and transmission of blood borne viruses (e.g. hepatitis C)
- practising safe sexual behaviours

For people using large amounts of amphetamines on single occasions, or over short periods of time

Encourage awareness of:

- techniques for moderating use and minimising potential harms, such as
 - □ planning use earlier in the weekend to allow for recovery
 - ☐ use routes of administration other than injecting
 - □ avoid high doses in any one episode

- avoid using for extended periods, and before important events, or before work or study
- □ avoid using with other drugs whilst using speed, including alcohol
- ☐ general health care, such as getting enough sleep, drinking plenty of water and eating before, during and after using
- symptoms of heavy use, such as:
 - preoccupation with obtaining and using speed
 - □ increased tolerance
 - □ continued use despite evidence of problems associated with use
 - emergence or exacerbation of social, physical or mental problems
 - □ transition to other methods of administration (e.g. IV use)
 - polydrug use to exacerbate the effect of amphetamine or to modify withdrawal symptoms
- 'the false sense of psychomotor competence' that amphetamines may produce, and especially when used in combination with alcohol (e.g. avoid driving when using amphetamines)
- strategies to reduce harmful side effects, e.g. to obtain the drug from the same, or reliable sources; to use smaller amounts per occasion; only use in company of others (Pead et al., 1996; Lintzeris et al., 1996).

RESOURCES

Alliance of NSW Divisions of General Practice



- Jarvis, T.R., Tebbutt, J. & Mattick R.P. 1995, *Treatment Approaches for Alcohol and Drug Dependence. An Introductory Guide*, John Wiley & Sons Ltd., Chichester, England.
- Lintzeris, N., Dunlop, A. & Thornton, D. 1996, *Getting Through Withdrawal Amphetamines*, Turning Point Alcohol and Drug Centre Inc., Fitzroy, Victoria.
- McKetin, R. & McKenna, S. 2000, 'Amphetamine dependence and withdrawal', *GP Drug and Alcohol Supplement*, No. 12, www.health.nsw.gov.au/public-health/dpb/supplements/supp12.pdf
- Topp, L., McKetin, R., Hando, J. & Dillon, P. no date, *A User's Guide to Speed*, National Drug and Alcohol Research Centre, Sydney, www.ndarc.med.unsw.edu.au/ndarc.nsf

Counselling strategies

Jarvis, T.R., Tebbutt, J. & Mattick R.P. 1995, *Treatment Approaches for Alcohol and Drug Dependence. An Introductory Guide*, John Wiley & Sons Ltd., Chichester, England.

REFERENCES

- AIHW (Australian Institute of Health & Welfare) 2002, 2001 National Drug Strategy Household Survey: First Results, Drug Statistics Series No. 9, AIHW cat. no. PHE 35, AIHW, Canberra.
- Baker, A., Boggs, T. & Lewin, T. 2001, 'Randomised controlled trial of brief cognitive—behavioural interventions amongst regular users of amphetamine', *Addiction*, vol. 96, pp. 1279–1287.
- Gossop, M., Darke, S., Griffiths, P., Hando, J., Powis, B., Hall, W. & Strang, J. 1995, 'The Severity of Dependence Scale (SDS): psychometric properties of the SDS in English and Australian samples of heroin, cocaine and amphetamine users', *Addiction*, vol. 90, pp. 607–614.
- Gourlay, D.L. 2000, 'Chapter 6.2: Amphetamines' in B. Brands (Ed.) *Management of alcohol, tobacco and other drug problems*, Centre for Addiction and Mental Health, Toronto, Canada.
- Kamieniecki, G., Vincent, N., Allsop, S. & Lintzeris, N. 1998, *Models of Intervention and Care for Psychostimulant Users*, Monograph Series No. 32, Commonwealth Department of Health and Family Services, Canberra.
- McKetin, R. & Mattick, R.P. 1998, 'Attention and memory in illicit amphetamine users: Comparison with non-drug-using controls', *Drug & Alcohol Dependence*, vol. 50, pp. 181–184.
- Latt, N., White, J., McLean, S., Lenton, S., Young, R., & Saunders, J. 2002, 'Central nervous system stimulants' in Hulse G., White, J. and Cape G. (Eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 8, Oxford University Press, South Melbourne, Victoria, pp. 124–140.
- Lintzeris, N., Dunlop, A. & Thornton, D. 1996, *Getting Through Withdrawal Amphetamines*, Turning Point Alcohol and Drug Centre Inc., Fitzroy, Victoria.
- Pead, J., Lintzeris, N. & Churchill, A. 1996, From Go to Whoa, Amphetamines and Analogues, The Trainer's Package for Health Professionals, Commonwealth Department of Human Services and Health, Canberra.
- Raistrick, D., Bradshaw, J., Tober, G., Weiner, J., Allison, J. & Healey, C. 1994, 'Development of the Leeds Dependence Questionnaire (LDQ): A questionnaire to measure alcohol and opiate dependence in the context of a treatment evaluation package', *Addiction*, vol. 89, pp. 563–572.
- Shearer, J., Wodak, A., Mattick, R.P., Van Beek, I., Lewis, J., Hall, W. & Dolan, K. 2001, 'Pilot randomised controlled study of dexamphetamine substitution for amphetamine dependence', *Addiction*, vol. 96, pp. 1289–1296.
- Saunders, J. & Young, R. 2002, 'Chapter 3: Medical and psychosocial problems' in Hulse G., White J., and Cape G. (eds.) 2002, *Management of Alcohol and Drug Problems*, Oxford University Press, South Melbourne, Victoria, pp. 32–44.

- Todd, F. 2002, 'Coexisting alcohol and drug use and mental health disorders' in (ed.) Hulse G., White, J. & Cape G. 2002, *Management of Alcohol and Drug Problems*, ch. 20, Oxford University Press, South Melbourne,: Victoria, pp. 359–373.
- Topp, L. & Churchill, A. 2002, Drug Trends Bulletin, June 2002, NDARC, Sydney.
- Victoria Police 2002, Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems, 2nd edn., Custodial Medical Unit, Mornington, Victoria.
- Wickes, W. 1993, Amphetamines and Other Psychostimulants: A Guide to the Management of Users, AGPS, Canberra.