

Part 2

The Drugs

Alcohol

ALCOHOL is a licit drug. Its consumption is sanctioned by cultural norms and social practices, and its production contributes significantly to Australia's gross national product (GNP).

Alcohol is a central nervous system (CNS) depressant. Its psychoactive properties contribute to changes in mood, cognition and behaviour. The main psychoactive ingredient in beverage alcohol is ethyl alcohol (ethanol, or C_2H_5OH).

PHARMACOLOGY

Absorption

Alcohol is rapidly absorbed from the small bowel via portal circulation (around 80%), and stomach (around 20%). Alcohol is water soluble, and little or no alcohol enters fatty tissue. It reaches the brain within five minutes of ingestion, with blood concentrations peaking between 30 to 90 (typically 45) minutes. Absorption rate varies with:

- the drug (e.g. beverage type, presence of food in the stomach)
- individual factors (e.g. age, gender, size, drinking rate and experience)

Distribution

Alcohol is rapidly distributed throughout the body water accumulating in tissues with high water content. Alcohol readily crosses blood–brain and placental barriers (Lopatko et al., 2002).

Metabolism

Ninety-five per cent of alcohol is metabolised by the liver into carbon dioxide and water, and 1–5% is excreted unchanged in saliva, urine, faeces and sweat. The enzyme alcohol dehydrogenase (ADH) (and to a smaller extent, cytochrome P450 2E1 (or CYP2E1)) is the catalytic agent for transforming ethyl alcohol into acetaldehyde. The second metabolic process involves aldehyde dehydrogenase (ADLH) as the catalytic agent responsible for oxidising acetaldehyde into acetic acid. Long-term high-risk consumption results in increased production and activity of CYP2E1, thought to be responsible for increasing elimination of alcohol amongst high-risk/dependent users (Victoria Police, 2001; Lopatko et al., 2002).

PATTERNS OF DRINKING

Results from the 2001 National Household Survey (AIHW, 2002) of adults 14 years and over found:

- 8.3% (M: 11%; F: 5.6%) reported drinking alcohol on a daily basis
- almost 40% (M: 46%; F: 33%) consumed alcohol at least once a week
- 35% (M: 29%; F: 40%) consumed alcohol less often than once a week
- 8% (M: 7%; F: 9%) were ex-drinkers
- almost 10% (M: 7%; F: 12%) had never drunk a full glass of alcohol
- 90% of 20–29 year olds were current drinkers

Contrary to common perceptions, Aboriginal and Torres Strait Islanders:

- are more likely to be non-drinkers or ex-drinkers
- are less likely to drink on a weekly (33% compared with 49% of the general population) or occasional (29% versus 32%) basis; and
- are more likely to drink at high or very high risk levels on the occasions they do drink alcohol (82% versus 28%), compared to the general population (CDHAC, 2001; CDHSH, 1994; NHMRC, 2001)

BENEFITS AND HARMS

Benefits

There is good evidence that < 1 standard drink a day for women, and 1–2 a day for men helps prevent heart disease from middle age onwards. The benefit is attributable to alcohol per se, rather than the beverage type consumed. Heavier drinking not only confers no additional benefit, but substantially increases risk of harm. Cardiac protection needs to be assessed against the physiological changes of ageing (e.g. reduced tolerance to alcohol's effects, interaction with prescribed medications). Any benefits can be equally achieved through a healthy lifestyle. There is no evidence that low risk drinking in younger adulthood helps prevent the onset of cardiovascular disease in later life (NHRMC, 2001).

Harms

Most drinkers (73%) generally consume alcohol in ways considered a low health risk (AIHW, 2002). However, harmful/hazardous alcohol use and dependence is estimated to cost the Australian community \$7.6 billion in direct and indirect costs (Collins & Lapsley, 2002). Single episodes of alcohol intoxication contribute to 67% of potential years of life lost (PYLL) due to premature alcohol-related mortality (CDHAC, 2001).

Alcohol contributes to over 3,000 deaths per year, and is implicated in:

- 7% of all male and 2% of all female deaths
- 50,000 hospitalisations
- 20–40% of acute general and psychiatric hospital presentations
- 18% of all injuries presenting to emergency departments
- 50% of assaults
- 44% of fire injuries
- 34% of falls and drownings
- 30% of car accidents
- 16% of child abuse
- 12% of suicides; and
- 10% of industrial accidents

(CDHAC, 2001; CDHA, 2002; NHMRC, 2001; APF, 2001)

Studies suggest that 15–32% of patients presenting to general practice drink at at-risk levels (Sayer et al., 2000). However, fewer than half of all patients routinely undergo screening for alcohol use (Lopatko et al., 2002).

The Australian Alcohol Guidelines: Health Risks and Benefits (NHMRC, 2001, www.nhmrc.gov.au) provide an evidence base for promoting individual and population health in relation to alcohol consumption. The guidelines emphasise the link between 'how much' and 'how often' alcohol is consumed, where the risks are described according to three levels (low, risky and high risk), and two timeframes (short-term and long-term).



See Appendix A

The NHMRC Australian Alcohol Guidelines recommend that to minimise harm:

- *males*: consume 6 drinks or less on any one occasion, or no more than 4 standard drinks per day, with at least 2 alcohol free days per week

- *females*: consume 4 drinks or less on any one occasion, or no more than two standard drinks per day, with at least 2 alcohol free days per week

Abstinence is recommended as appropriate for:

- people with an existing medical or mental health condition that may be exacerbated through drinking
- people taking medications that interact with alcohol (e.g. benzodiazepines, opioids)
- women who are pregnant, planning a pregnancy, or breastfeeding
- people undertaking activities involving skill or risk (e.g. operating machinery, driving, flying, water sports etc.)

N.B. Levels of risk related to the use of alcohol are based on an average or larger body size and a weight of 50 kg or more (NHMRC, 2001).

EFFECTS OF ALCOHOL CONSUMPTION

Blood Alcohol Concentration (BAC) is a reasonable guide to level of intoxication (see Table 3–1). BAC indicates the amount of alcohol in the bloodstream in grams of alcohol per 100 ml blood. A BAC of 0.05 means a person has 0.05 g of alcohol per 100 ml of blood (or a BAC of 0.05% = 11 mmol / L) (Victoria Police, 2001). A person of average build will metabolise alcohol at a constant rate of around one standard drink per hour. One standard drink (see Table 3–2) per hour will cause a rise in BAC of 0.01% to 0.02% in an hour; however:

- small females will have higher blood peak levels than large males for the same volume consumed
- high tolerance to alcohol may result in faster metabolism (hence more rapid reduction in BAC)

Table 3-1
Correlation between BAC* and behavioural/motor impairment

BAC*	Likely effects of intoxication
0.02–0.05 g / 100 ml	<ul style="list-style-type: none"> • cheerful, relaxed, pleasant feelings of happiness and wellbeing • decreasing inhibitions • judgment increasingly impaired • increased chance of accidents • impaired coordination • BAC* 0.05 g / 100 ml = legal limit for driving (if fully licensed) in all Australian States and Territories
0.1–0.2 g / 100 ml	<ul style="list-style-type: none"> • ataxia • decreased ability to appropriately interpret and react to surroundings • poor judgment • loss of 'self-control' • slurred speech • increasingly unpredictable behaviour • labile mood • potential for aggression
0.2–0.3 g / 100 ml	<ul style="list-style-type: none"> • marked ataxia and slurred speech • poor judgment • labile mood • nausea and vomiting • double vision • memory loss
0.3–0.4 g / 100 ml	<ul style="list-style-type: none"> • stage 1 anaesthesia (sleepiness, poor response to external stimuli, oblivion) • memory lapse • labile mood
> 0.40 g / 100 ml	<ul style="list-style-type: none"> • respiratory failure • coma • possible death

*Blood Alcohol Concentration

Source: adapted from Victoria Police (2001, p. 1.8) and Ryder et al. (2001, p. 162).

MEASURING CONSUMPTION: THE ‘STANDARD DRINK’

The ‘standard drink’ concept was designed to assist consumers to monitor their alcohol consumption. One Australian ‘standard drink’ contains about 10 grams (12.5 millilitres) of alcohol. See Table 3–2 for common standard drink equivalents. Whilst legislation requires alcohol producers to label the number of standard drinks in a container, variation in size and type of glass in different environments (e.g. homes, licensed environments) may make it difficult to estimate the actual number of drinks consumed.

Identifying ‘At-risk’ Drinking Levels

Most people tend to be low-risk drinkers, and experience few problems related to their use of alcohol, most of the time (see Figure 3–1).

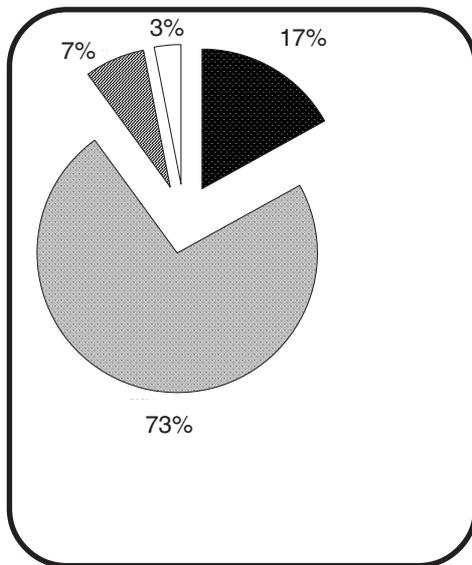


Figure 3–1
Proportion of population at risk of alcohol-related harm
 Source: AIHW (2002)

Groups at High Risk for Alcohol-related Harm

The NHMRC (2001) identified groups who are particularly susceptible to alcohol-related harm, including:

Young people (up to 18 years) and young adults (19–25 years)

Young people’s patterns and levels of drinking place them at significant risk of harm compared with the community in general. Whilst alcohol-related deaths amongst older people can be attributed to *long-term* hazardous or harmful patterns of use (Chikritzhs et al., 1999), approximately 25–33% of 14–24 year olds drink in a high-risk manner (Chikritzhs et al., 2000), increasing the likelihood of serious harm, injury or death due to *acute* conditions resulting from alcohol intoxication. Between 1990 and 1997, 52% of all serious alcohol-related road injuries were sustained by people aged 15–24, with a further 23% of injuries sustained by 25–34 year olds.

Risk of harm amongst young people is increased due to their:

- smaller physical size
- fewer social controls
- peer values and norms that condone intoxicated behaviour
- risk of overdose due to lack of tolerance

Use of alcohol or other drugs at risky and harmful levels may:

- interfere with normal physiological, social and emotional development
- increase risk of suicide
- increase risky sexual behaviour/unwanted sex
- cause blackouts
- contribute to poor academic performance
- contribute to, or cause mental health problems

Table 3-2
Standard drink (SD) equivalents

Beverage	Container	% alc/vol	Standard Drink equivalent
BEER			
Light	375 ml can or bottle	2.7%	0.8
Mid strength	375 ml can or bottle	3.5%	1.0
Full strength	375 ml can or bottle	4.5%	1.5
Light	285 ml glass (middy/pot/schooner)	2.7%	0.5
Mid strength	285 ml glass (middy/pot/schooner)	3.5%	0.7
Full strength	285 ml glass (middy/pot/schooner)	4.5%	1.0
WINE			
White/red	100 ml glass	12%	1.0
	180 ml average restaurant serve	12%	1.8
	750 ml bottle	12%	7.0
	2 litre cask	12%	20.0
	4 litre cask	12%	40.0
Cider	375 ml bottle/stubbie	4.7–7.5%	1.4–2.0
	750 ml bottle		2.8–4.0
FORTIFIED WINES E.g. port, sherry			
	60 ml glass	21%	1.0
	750 ml bottle	18%	11.0
	2 litre (cask/flagon)	18%	28.0
PREMIX COOLERS AND SODAS			
	340 ml bottle	5–8%	1.5–2.4
	375 ml can		
	250 ml–350 ml bottles	3.5–5.5%	0.7–1.4
SPIRITS			
	30 ml (nip)	42%	1.0
	700 ml bottle	40%	22.1

Source: adapted from ATODS (1997) and NHMRC (2001).

Note: a 285 ml glass is a 'middy' in NSW, WA and ACT; a 'pot' in TAS; a 'handle' in NT; and a 'schooner' in SA.

- cause behavioural problems, such as fighting, resulting from feelings of aggression (NHMRC, 2001)

People with mental health problems

The psychoactive effects of alcohol can result in exacerbation of existing mental health problems. Alcohol may also interact with prescribed medications. Always give specific advice to avoid alcohol where it is contraindicated.

Unborn children

The foetus is most vulnerable to damage from high-risk drinking during the first few weeks after conception. Drinking above the low-risk guidelines can contribute to adverse outcomes for the baby (e.g. foetal death, growth retardation, behavioural deficits, congenital malformations). However, there is 'no discernible evidence' that one standard drink a day causes harm to an unborn child (NHMRC, 2001).

Women

Women are more susceptible to alcohol-related harms due to:

- their reduced ability to metabolise alcohol relative to males
- physical makeup (smaller body frame, liver, higher proportion of body fat, different biochemical processes relative to males (Litt et al., 1993)). Women are likely to develop complications earlier, and are more vulnerable to liver damage and cirrhosis at lower consumption levels. High consumption is related to breast cancer
- environmental influences which place women at greater risk of intoxication-related harms (e.g. assault and injury)

Risk factors for hazardous and harmful drinking patterns in women include:

- a positive family history

- childhood problem behaviours related to impulse control
- poor coping responses in the face of stressful life events
- depression, divorce or separation
- having a drinking partner and working in a male dominated environment

Although women enter treatment at about half the rate of men, treatment outcomes are similar. Attitudes towards women drinkers, depression, concerns about children, or fear of removal of children are potent barriers to seeking treatment (NHMRC, 2001).

Occupational groups

Workers in some occupational groups engage in risky or harmful drinking patterns more often than others due to a range of social and environmental factors.

These groups may include:

- trades (e.g. building, mining, construction, forestry, transport, fishing)
- hospitality industry
- women employed as specialist managers (finance, personnel, public policy, sales)
- transport, publishing, wholesale and service industries (e.g. entertainment) (NHMRC, 2001)

IDENTIFYING HARMS

As with other drugs, alcohol-related harms are not specific to the effects of the drug. Alcohol-related harms result from the interaction between:

The drug

- patterns of use (how much, when used, how often)
- and other drugs used

The individual

- age, weight, gender and general health
- tolerance and previous experience of alcohol use, intoxication, after effects and withdrawal
- expectations of use and effects
- current mood and psychological health

The environment

Factors that influence the drug's effects and patterns of use such as:

- social settings and company
- context of use
- patterns of drug use according to ritual or culture

Table 3-3
Classification of alcohol-related harms

	Intoxication	Regular Excessive Use	Dependence
Examples of problems	<ul style="list-style-type: none"> • hangovers • insomnia • reduced work performance • road and industrial accidents • unintended unsafe sexual practices • violence 	<ul style="list-style-type: none"> • irritability • depression • anxiety • altered sleep patterns • hypertension • weight gain • gastritis • impotence • fatty liver • memory loss • financial issues 	<ul style="list-style-type: none"> • cirrhosis • pancreatitis • oesophageal varices • peripheral neuritis • tolerance • withdrawal symptoms • anxiety • depression
Consumption patterns	In a single session: <ul style="list-style-type: none"> • > 6 standard drinks (male) • > 4 or more standard drinks (female) 	Standard drinks / day <ul style="list-style-type: none"> • see NHMRC guidelines for short- and long-term harms (Appendix A) 	Standard drinks / day <ul style="list-style-type: none"> • male > 10 • female > 8
Prevalence	Common <ul style="list-style-type: none"> • 10–15%, especially in adolescence and early 20s 	Risky / harmful <ul style="list-style-type: none"> • 10–20% population 	Relatively uncommon <ul style="list-style-type: none"> • < 5% males • < 2% females

Source: Thorley (1980) adapted by Litt et al. (1993, p. 5)

Thorley's model (see Table 3–3) is a useful guide to identifying specific harms related to 'Intoxication', 'Regular Excessive Use' and 'Dependence'. This model enables practitioners to:

- assess the type of problem
- assess severity of problems
- facilitate individually tailored responses

General points:

- intoxication-related problems have substantially greater impact on the community than dependence, however, dependence results in more severe problems for individuals
- regular use is not generally considered a problem unless it exceeds the 'at-risk' thresholds described by the NHMRC (2001)
- primary care practitioners are likely to have most success in their interventions with people experiencing problems related to intoxication and regular use
- patients experiencing problems related to dependence are best referred to specialist agencies

ALCOHOL ASSESSMENT

Early Recognition of Alcohol-related Problems

Alcohol-related problems are more likely to be identified early when the health professional:

- is aware that psychosocial problems occur before most physical problems
- is willing to follow up with detailed enquiry and appropriate investigations



www.health.gov.au/pubhlth/publicat/document/alcproblems.pdf

Four Key Assessment Steps

1. Establish patterns of use

Techniques for incorporating use of alcohol into history taking include:

- incorporating questions about general lifestyle issues, such as smoking, diet, exercise, recreational activities
- asking specific questions (type of drug/s, dose, frequency of use, duration of use, recency of use, how used)
- focusing on the current week's patterns
- use of a visual Standard Drinks Chart (e.g. www.dasc.sa.gov.au), (see Table 3–2)
- asking about concurrent use of other drugs, e.g. tobacco, amphetamines, benzodiazepines, heroin

These strategies help prevent patients from giving general responses such as 'I only drink socially'. Conversation starters might include:

'Now that we have dealt with...(presenting complaint)...let's have a look at other areas that may contribute to your health. Are you allergic to anything? Do you smoke? When did you last have a drink?'

'We often find that eating, smoking and drinking habits affect our health. I'd like to ask you a few questions about these things.'

Assume the patient consumes alcohol to some degree, so introduce drinking as a normal practice, for example:

'Most of us like to have a drink. How often would you have a drink during the week and at the weekends?'

'Did you have a drink yesterday? What did you have, where were you, and how long were you drinking?'

(Adapted from the APF, 2001 and Litt et al., 1993)

Additional aspects of a drinking history should include:

- pattern of consumption over past 7 days, commencing with today, and working backwards
- establishing pattern over a 'typical week', including alcohol use during special events (e.g. anniversaries, celebrations)
- determining whether alcohol consumption is related to cultural/religious practices/beliefs

2. Establish risk

Indicators of risk for alcohol-related harm include:

- health or social problems related to alcohol use
- concerns (self or family) about levels of consumption
- concerns about consequences related to intoxication or high risk use, such as accidents, assaults, injuries, driving offences, embarrassment related to behaviour whilst intoxicated
- use of other drugs (alcohol may interact with or enhance the effects of other drugs)
- physical trauma, possibly attributable to alcohol use
- signs of intoxication or hangover
- consumption regularly exceeding NHMRC guidelines for short-term high-risk use
- anxiety, depression, sleeping difficulties not otherwise explained

Signs and symptoms suggestive of alcohol dependence may include:

- current intoxication (positive BAC, smell of alcohol on the breath, slurred speech, ataxia)
- withdrawal symptoms (tremor, sweating, agitation, anxiety, increased blood pressure, pulse)
- signs of liver disease (hepatomegaly, spider naevi)

- signs indicative of poor functioning, e.g. poor general appearance, poor hygiene
- repeated admissions for possible alcohol-related conditions
- peripheral neuropathy
- cerebellar ataxia (broad based gait)
- cognitive dysfunction (impaired minimal examination)
- past history (withdrawal and treatment history, periods of abstinence)
- indicators from relevant pathology tests (NSW Health, 2000)



See Appendix B

Use Figure 3–2 with patients as a tool for opening discussion about indicators of high risk drinking patterns, and how alcohol use may be related to interpersonal, social, psychological and general health problems.

3. Identify problems associated with use

- medical
- financial
- legal
- employment
- relationships (social, work, family)
- violence
- psychological and psychiatric
- sexual

4. Match presentation to intervention

For appropriate matching of patient and intervention, consider:

- the patient's wants and needs
- 'stage of change'



See Chapter 13
Psychosocial Interventions

- type and severity of problems (physical, social, emotional), and links with problems related to intoxication, regular excessive use or dependence
- patient safety (including other health or social risks) (Alliance of NSW Divisions, 2000)

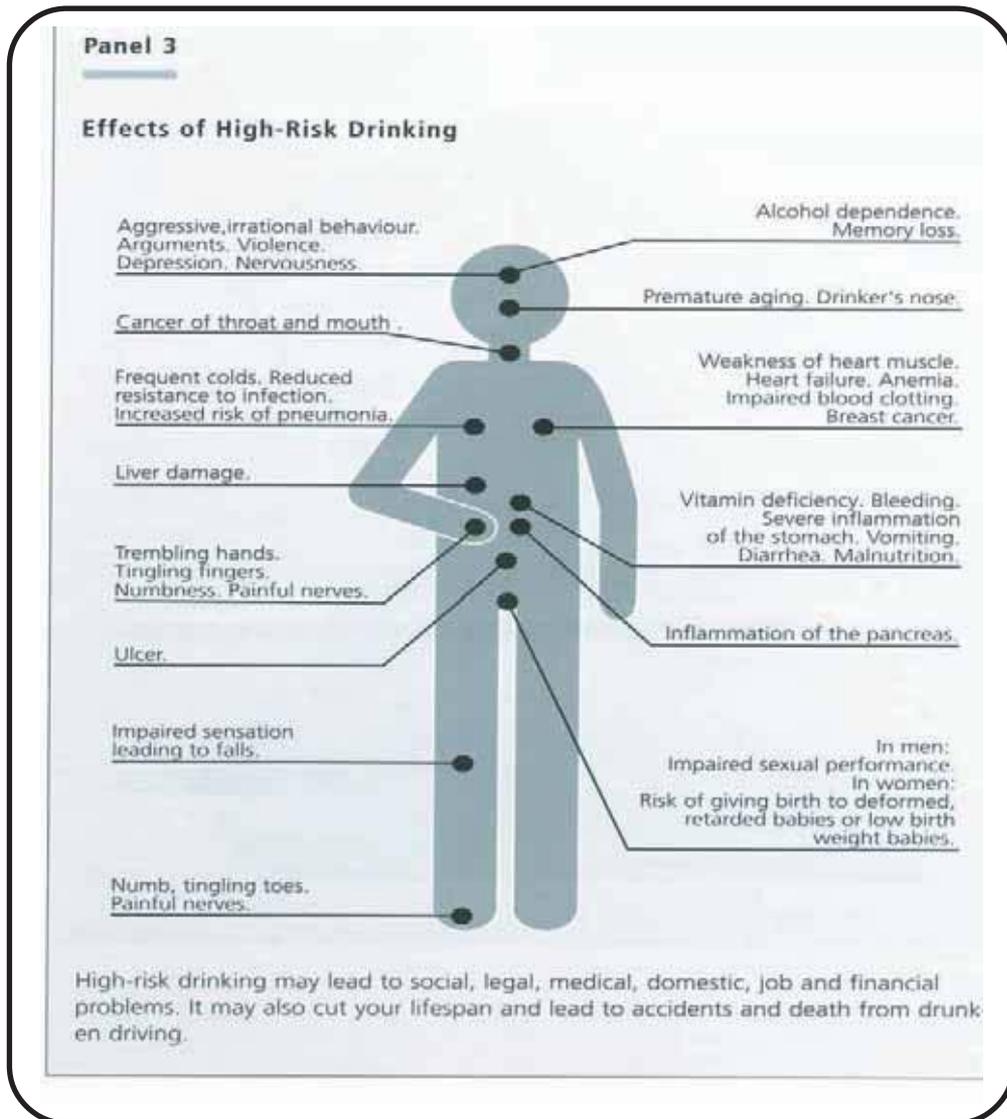


Figure 3–2
Common effects of high-risk drinking

Source: Babor, T., Higgins-Biddle, J.C. Saunders, J. & Monteiro, M.G. (2001)

SCREENING FOR ALCOHOL USE

Invasive Measures

Estimating BAC (Blood Alcohol Concentration)

A breathalyser is a reliable way to determine BAC. Breath analysis offers a good correlation between body burden of alcohol and concentration of alcohol in pulmonary blood circulation through measuring end-expiratory breath. Breath analysis:

- indicates recent consumption however, cannot identify patterns of high-risk or harmful patterns of use
- is accurate, but expensive (a breathalyser unit needs to be purchased and regularly calibrated)
- results are available immediately

Note: The term Blood Alcohol Concentration (BAC) may be used interchangeably with Blood Alcohol Level (BAL).

See www.dasc.sa.gov.au for the 'DRINKMETER Program', an interactive guide for determining BAC after a 'typical' drinking session (taking into consideration age, height, weight etc.).

Laboratory investigations

Use of laboratory tests may assist the practitioner to relate drinking consequences with physical sequelae, and encourage the patient to think about their drinking. Whilst elevated biochemical markers may be indicative of liver disease, most tests are neither sensitive or specific to both long-term or hazardous alcohol use. Liver function tests (LFTs) provide general information about the impact of alcohol on the body. Carbohydrate deficient transferrin (CDT) tests are more sensitive and specific indicators of long-term high-risk use.

Most laboratory measures are less sensitive than good clinical judgment and self-report measures, such as AUDIT and CAGE, in detecting alcohol dependence and related health care problems (Dawe et al., 2002).



See Appendix B

Non-invasive Measures

Detecting alcohol-related problems is more effective with the use of specific purpose screening tools. The screening tools most frequently used in Australia include:

- AUDIT
- CAGE
- T-ACE
- TWEAK

(For information on T-ACE and TWEAK refer to Dawe et al., 2002.)

CAGE

The CAGE is a four item screening questionnaire designed to identify problem or 'at-risk' drinking.

It can be administered as part of an interview, or as a self-report measure, and has been successfully used across health settings, and across cultures with minor modifications (Dawe et al., 2002). The items are:

1. Have you ever felt you ought to **C**ut down on your drinking?
2. Have people **A**nnoyed you by criticising your drinking?
3. Have you ever felt bad or **G**uilty about your drinking?
4. Have you ever had a drink first thing in the morning (**E**ye-opener) to steady your nerves or get rid of a hangover?

Scoring 'yes' to two or more questions is predictive of current hazardous or harmful drinking patterns, and indicates a need for further assessment. The CAGE has a sensitivity and specificity of 84% and 95% respectively, and a positive predictive value of 45% using a cut off of > 2 positive responses. But because CAGE is considered insensitive to detection of low levels of problematic drinking the AUDIT is considered the screening instrument of choice, given that little additional time is required to complete, score and interpret the AUDIT (Dawe et al., 2002).

The AUDIT

The AUDIT is a 10 item screening instrument designed to identify hazardous and harmful alcohol consumption as well as dependence. It is easy to use, short, and enables valuable patient feedback. The AUDIT is consistent with ICD-10 definitions of harmful alcohol use and dependence and focuses on recent use of alcohol. It has been validated across countries, cultures and languages (Babor & Higgins-Biddle, 2001; Dawe et al., 2002).

Administration of AUDIT

The AUDIT can be administered as an interview or as a self-report measure (see Babor et al., 2001).



See Appendices C & D

Interpreting the AUDIT

The 10 questions are each given a score of between 0 and 4, with a maximum overall score of 40. Whilst a single global score is considered representative of overall drinking behaviour, examination of individual responses to each question are important, as this will help:

- identify pattern of use (quantity and frequency of use, level of risk)

- assist in informing the type of intervention that would be appropriate (e.g. strategies for a young person drinking infrequently but at high-risk levels will be different to an intervention offered to an older person drinking less, but more frequently)
- indicate areas requiring further assessment (e.g. where the patient is showing signs of dependence) (refer to Dawe et al., 2002; Babor et al., 2001).

BRIEF INTERVENTIONS

Health professionals are well placed to:

- identify alcohol-related harms, and problems related to consumption or after effects of use
- assist patients to link patterns of consumption with current lifestyle, social or health-related problems
- provide specific, tailored interventions, that have demonstrated efficacy within a single, or short series of consultations.

A brief intervention consisting of a short five minute session may incorporate:

- identification of current patterns of use, linking consumption patterns with identified problems
- identification of 'stage of change'



See Chapter 13
Psychosocial Interventions

- assistance to assess pros and cons of current patterns of use
- motivational interviewing techniques
- advice on safe drinking guidelines and ways to cut down



See Appendix E

- provision and explanation of self-help materials e.g. drinking diaries
- relapse prevention strategies (if relevant)

Brief interventions conducted over a short series of 2–3 sessions tend to have an educational focus, and may include:

- comprehensive assessment
- specific advice
- counselling
- teaching goal setting strategies to assist moderating drinking patterns or reducing harms (Lopatko et al., 2002)



See Chapter 13
Psychosocial Interventions

Table 3–4 shows the FLAGS approach to alcohol intervention using the AUDIT.

ALCOHOL INTOXICATION

Acute Alcohol Intoxication

Intoxication may be recognised by:

- ataxia and slurred speech
- emotional lability and disinhibition
- smell of alcohol on the breath
- mood variations

Assessment of Alcohol Intoxication

- obtain alcohol and other drug use history (especially recency of use)
- observe vital signs
- physical examination
- mental health examination to establish:
 - level of consciousness
 - orientation
 - memory

- judgment
- comprehension
- mood
- speech
- perception (hallucinations)

Complications of acute alcohol intoxication or overdose

Possibly:

- respiratory paralysis, particularly if vomit is inhaled
- obstructive sleep apnoea
- fatal cardiac arrhythmia when blood alcohol is greater than 0.4 mg / ml

Alcohol intoxication will only resolve with time. Management of intoxication depends on the location of the affected person, type of service available, and skills of the worker to monitor and observe for complications. Management includes:

- managing behaviours of affected person (e.g. don't engage in discussion of emotive topics, diffuse aggressive behaviour)
- provide a non-threatening and non-stimulating environment
- encourage sleep
- observe for signs of other medical conditions that mimic intoxication (e.g. head injury, diabetes, infection, epilepsy, drug toxicity). Refer to NSW Health (2000) for further detail.



www.health.nsw.gov.au

Clinical signs of alcohol-related overdose may include:

- decreased consciousness, coma or stupor
- changing mental status
- cold and clammy skin, lowered body temperature

Table 3-4
Using the AUDIT score with the FLAGS approach for treatment interventions

AUDIT score + history + observations		
Low risk (M: < 8; F: 7)	Risky or harmful (M: 8-15; F: 7-15)	Problematic (16-19)
Feedback results	Feedback results	Feedback results
Listen to patient's concerns	Listen to patient's concerns	Listen to patient's concerns
<p>Provide Alcohol education and information</p>	<p>Simple Advice and information Creates awareness of low risk range Informs patient about consequences of continued drinking, brief counselling, ongoing monitoring</p>	<p>Advise patient re. need for further assessment/referral to specialist</p>
<p>Goals of treatment General awareness Reinforces/maintains low risk drinking Assists patients problems, patients who have cut down, or whose circumstances may change</p>	<p>Goals of treatment Assists those drinking at risky levels Encourage reduction of consumption to recommended limits e.g. 2 alcohol free days per week</p>	<p>Goals of treatment Likely dependent Discuss importance/ relevance of abstinence Provide information Establish treatment goals</p>
<p>Strategies discussed and implemented Gain greater understanding of 'trigger' situations Offer self-help booklet Offer follow up appointment to discuss progress, and use of booklet</p>	<p>Strategies discussed and implemented Possible: <ul style="list-style-type: none"> Withdrawal management (detoxification) Pharmacotherapies PLUS supportive therapy Weigh pros and cons of treatment. Negotiate goals. Encourage supportive therapies Monitor and follow up or refer to AOD worker or specialist if necessary</p>	<p>Strategies discussed and implemented Consider: <ul style="list-style-type: none"> Withdrawal management (detoxification) Pharmacotherapies PLUS supportive therapy Specialist help or primary care and community-based support Monitor and follow up</p>

Source: adapted from APF (2001, p. 14), Babor et al. (2001) and O'Connor & Simmons (2002)

- lowered blood pressure, tachycardia/bradycardia
- breathing difficulties, slow and noisy respirations

Managing overdose:

- maintain airway, breathing and circulation
- refer to hospital for further assessment

As polydrug use complicates any clinical picture, obtain drug use history where possible, in particular use of other central nervous system depressants such as methadone and heroin.

ALCOHOL DEPENDENCE

Alcohol dependence is a complex syndrome, with both physiological and psychological signs and symptoms.



See Chapter 1
Overview and Introduction

Key features of alcohol dependence include:

- tolerance or narrowing of drinking repertoire
- a perceived 'loss of control' over one's drinking behaviour/salience of alcohol over other issues
- withdrawal (physical and psychological) symptoms on cessation of use
- relief or avoidance of withdrawal symptoms by drinking
- rapid recommencement of pre-established, or high-risk drinking patterns after a period of abstinence

ALCOHOL WITHDRAWAL

General Guidelines for Alcohol Withdrawal Management

Withdrawal management is just one aspect of managing alcohol dependence and should never be considered 'the cure'. Changing established behaviours takes time; relapse is common. Well planned interventions and engagement in activities supporting behaviour change will assist long-term recovery.



See Chapter 13
Psychosocial Interventions



www.answd.com.au
Tip Sheets

Depending on drinking history, medical risk and level of social support, alcohol withdrawal can be effectively managed in a home or inpatient setting. To establish a withdrawal management plan:

1. Assess current consumption levels

- undertake AOD, medical, social history and mental health assessment

2. Predict likelihood and severity of withdrawal

Withdrawal is likely where:

- there is an alcohol-related reason for admission/assessment
- there is regular alcohol use of > 80 grams per day (males), > 60 grams per day (females)
- patient > 30 years (significant alcohol withdrawal is unlikely under the age of 30)
- < 10 days after last drink (withdrawal usually commences within 6–24 hours of last drink, and may last 2–12 days)
- there is a history of alcohol dependence/significant previous withdrawal history
- AUDIT Score > 12

- other depressant/sedative medications are currently used
- pathology results are unusual e.g. raised serum GGT or MCV
- there is presence of alcohol-related disease (e.g. alcohol-related liver or cardiac disease, pancreatitis, hepatomegaly)
- physical appearance is suggestive of harmful alcohol use (parotid swelling, abnormal skin vascularisation, conjunctival injection)
- there is serious intercurrent illness e.g. head injury, diabetes, epilepsy, psychosis, infection, poor nutrition, head injury, significant liver disease, pancreatitis, cardiac or respiratory disorders (NSW Health, 2000)

The severity of alcohol withdrawal can be predicted by:

- previous withdrawal history (past history of seizures, hallucinations, delirium)
- duration and amount of alcohol used (quantity > 150g per day predictive of severe withdrawal)
- presence of other illness or injury increases severity and likelihood of complications
- use of other psychotropic drugs may result in additive or synergistic effects

(Adapted from NSW Health, 2000; Hulse et al., 2002)

The progress of the alcohol withdrawal syndrome can be seen in Figure 3–3.

Home withdrawal management

Home withdrawal may be suitable where:

- GP is able and willing to provide home monitoring
- carer support is available at home
- the patient has organised responsibilities and commitments (e.g. work)
- the patient's physical and emotional condition is appropriate for home withdrawal

Ensure the patient and carer are actively involved in developing the treatment plan and are aware of:

- withdrawal commencement date
- possible symptoms and has discussed expectations of withdrawal process
- medication regimes
- support and emergency systems



See Chapter 2
General Principles

Psychosocial and Physical Support During Alcohol Withdrawal

- reorientate and provide reassurance
- use simple commands, brief explanations, repetition (if required), use calm but firm voice
- treat symptoms e.g. headache, diarrhoea, generalised aches and pains, nausea and vomiting etc. and monitor and observe for seizures or other medical complications
- encourage fluids and light meals e.g. vegemite on toast
- ensure calm, uncluttered and comfortable environment with dim lighting, comfortable clothing and clean bedclothes

Delirium Tremens (the 'DTs')

Delirium tremens is a medical emergency associated with untreated alcohol withdrawal, occurring 3–14 days after stopping drinking. It occurs in < 5% of patients (Lopatko et al., 2002) and may be fatal (Ryder et al., 2001).

Main features of the DTs include agitation, restlessness, gross tremor, disorientation, fluid and electrolyte imbalance, sweating and high fevers, visual hallucinations and paranoia (Lopatko et al., 2002; NSW Health, 2000).

Observation and ongoing assessment — alcohol withdrawal observation charts

Withdrawal charts provide a guide to the severity of withdrawal symptoms and use of pharmacotherapy, but are not diagnostic instruments in themselves. A chart and guidelines for clinical management incorporating the validated Clinical Institute Withdrawal Assessment for Alcohol — Revised Version (CIWA-AR), the most commonly used instrument in Australia, is provided at Appendix F.



See Appendix F

Pharmacological Management of Alcohol Withdrawal

Medications (see Table 3–5) (including benzodiazepines), combined with supportive care can assist in reducing severity of withdrawal symptoms in the home and inpatient environment.

Diazepam

Benzodiazepines (most commonly diazepam, or oxazepam in the case of impaired liver function) are the drugs of choice in managing alcohol withdrawal, as they:

- alleviate many withdrawal symptoms
- are effective in preventing development of complex withdrawal features when given early
- have a wide margin of safety, provided supervision is adequate
- have low likelihood of cross-dependence, when established regimes for withdrawal management are used

If essential prerequisites for home withdrawal management have been met, home withdrawal using benzodiazepines may be appropriate (see Table 3–6) (See also Saunders et al., 1996).



See Chapter 2
General Principles

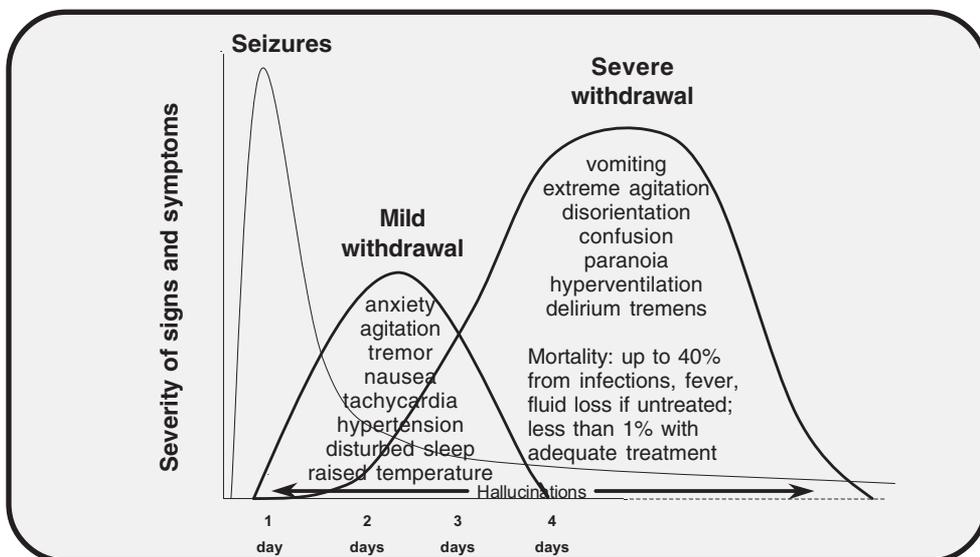


Figure 3–3
Progress of alcohol withdrawal syndrome
Source: NSW Health (2000, p. 41)



www.health.nsw.gov.au

Benzodiazepines for withdrawal management in inpatient/hospital setting

An inpatient setting is more appropriate if severe withdrawal or seizures are likely, or for those who are older, polydrug users, or physically or psychologically unwell. The Alcohol Withdrawal Observation Chart provides a diazepam regime for inpatient settings. Also see Palmer (2001) and NSW Health (2000).

Withdrawal may complicate any hospital presentation. Withdrawal management should occur several weeks prior to surgery, where possible.

There is NO place for the prescription of alcoholic beverages in the management of alcohol withdrawal.



See Appendix G

Table 3–5
Medications commonly used for withdrawal management

Drug	Main Indications
Thiamine 100 mg	IMI/O for at least 5 days (oral dose for two weeks, or until eating well). Treats or prevents Wernicke's, cerebellar ataxia and peripheral neuropathy, and assists cognitive recovery
Paracetamol	p.r.n. for management of headache and mild muscular pain (exclude prior liver disease)
Multivitamins	e.g. folic acid, Multi B forte for poor nutrition or poor initial appetite
Antiemetics	e.g. metoclopramide; for control of nausea and vomiting, p.r.n. Sedative effects assist sleeping
Antipsychotics	e.g. haloperidol may be indicated in small doses (2–5 mg if hallucinating, or if agitation is of concern and uncontrolled by diazepam). Avoid phenothiazines as they lower seizure threshold
Antidiarrhoeal agents p.r.n.	e.g. loperamide (as indicated)

Source: Lopatko et al. (2002); Palmer (2001); Wood & Pead (1995)

AFTER-CARE

Self-help Resources

Self-help resources can be useful tools to assist patients to reduce or cease use of alcohol. These resources work best when used in conjunction with a health check-up, follow-up or counselling session.



See p. 54, Resources

Self-help Groups

Alcoholics Anonymous, or AA, is a worldwide mutual help organisation with over 2 million members. It accepts no outside contributions and is run by and for 'recovering alcoholics'. The recovery program is based on universal spiritual principles. AA is a fellowship of support that encourages altruistic behaviour but makes no demands of its members apart from a desire to remain sober. The longevity of sobriety and positive attitude of many members can be exemplars for many patients. AA meets in most towns in Australia, many of

which also offer support groups for partners (Al-Anon) and children (Al-Ateen) of drinkers. Locate your local AA program in the telephone directory or the regional service via the Internet.

Pharmacotherapies to Reduce Relapse/Promote Abstinence

Controlled trials have shown that some medications effectively reduce relapse amongst dependent drinkers. These medications are best used as part of a comprehensive psychosocial treatment plan, such as counselling, motivational interviewing, relapse prevention, goal setting, risk and cue identification (APF, 2001). The most common medications used for promoting abstinence are described in Table 3–7.

ALCOHOL-RELATED BRAIN INJURY (ARBI)

Prolonged high risk use of alcohol may result in specific psychological and biochemical changes that may be described as alcohol-

Table 3–6
Diazepam regime for home/outpatient withdrawal

	8 a.m.	12 midday	5 p.m.	10 p.m.
Day 1	10 mg	10 mg	10 mg	10 mg
Day 2	10 mg	5 mg	10 mg	10 mg
Day 3	10 mg	5 mg	5 mg	10 mg
Day 4	5 mg	5 mg	5 mg	5 mg
Day 5	5 mg	–	5 mg	5 mg
Day 6	5 mg	–	–	5 mg
Day 7	–	–	–	5 mg
Day 8	–	–	–	–

Source: Palmer (2001, p. 12)

related brain injury (ARBI). This condition is often confused with dementia, or pre-morbid deficits associated with learning disorders, and manifests in various ways, including:

- disturbance in executive functions
 - poor attention, planning, organising, problem solving abilities, have difficulty in new environments or with new routines
 - 'concrete' thinking, difficulties with self-awareness and insight, appear poorly motivated
 - rigid repetitive behaviour patterns and inability to recognise consequences of behaviour
 - problems responding to changes in routine
- memory disturbances
 - poor short-term memory, may confuse dates/events
 - problems learning new information
- non-verbal disturbance
 - problems with hand-eye coordination and perception related tasks

With abstinence, proper nutrition, and psychological intervention, significant improvement is possible. Physical examination, CT scan, and blood tests (LFT, MCV etc.) will assist diagnosis.

Wernicke–Korsakoff's Syndrome

This 'syndrome' is the result of thiamine deficiency, essential for effective CNS function.

The acute phase, Wernicke's encephalopathy, is a life-threatening condition, manifesting in:

- global confusional state
- ocular disturbances: horizontal nystagmus, ophthalmoplegia or 6th nerve palsy, resulting in diplopia
- ataxia: wide-based and reeling steps, although may be obscured by polyneuropathies

One symptom is required for a diagnosis (DASC, 2000). When severe, main features include difficulty walking unaided, disinterest and lassitude. Following administration of parenteral thiamine, rapid recovery is possible.

Korsakoff's psychosis (the chronic form of the syndrome) manifests in short-term memory loss, confusion, confabulation, and Wernicke's-type symptoms. Total recovery is rare, 25% of cases are irreversible and constant supervision and care may be required (Luckman & Sorenson, 1982).

Table 3-7
Pharmacotherapies indicated for alcohol dependence

Acamprosate [Campral®]	Demonstrated efficacy in increasing abstinence, reducing relapse, and reducing amount and frequency of drinking
What it does	Does not prevent withdrawal symptoms but effective in dealing with post-withdrawal cravings. Restores activity levels of GABA (inhibitory transmitter) and glutamate (excitatory transmitter) to normal. Does not interact with alcohol, is not known to have dependence inducing potential, and cessation does not produce withdrawal syndrome. Available on PBS (Authority required)
Commence	Post-withdrawal (2–7 days after the last drink). Does not treat withdrawal
Treatment time	Estimated at 12 months PLUS supportive therapy. Treatment goal is abstinence.
Side effects	> 1% patients complain of nausea, diarrhoea, skin rash, which may last the first 1–2 weeks only
Contra-indications	Advanced hepatic failure, renal insufficiency (serum creatinine > 120 micromol / L, pregnancy, lactation (refer to prescribing information)
Disulfiram [Antabuse®]	Trials demonstrate modest and inconsistent efficacy in promoting abstinence
What it does	Produces an aversive response to the ingestion of alcohol. Inhibits production of acetaldehyde dehydrogenase, so when alcohol is consumed acetaldehyde accumulates resulting in an unpleasant flushing reaction, nausea and dizziness, vomiting, chest pain, palpitations. Large doses of alcohol may produce hypotension, arrhythmia, seizures, death
Commence	> 24 hours after last drink. Does not treat withdrawal
Treatment time	Long-term. Most effective under daily supervision
Side effects	Drowsiness, psychosis, peripheral neuropathy, hepatotoxicity, metallic taste, headache, visual disturbance
Contra-indications	Severe hepatic impairment, severe renal impairment, severe myocardial disease, hypersensitivity, thiuram derivatives, pregnancy
Precautions	Diabetes, hypothyroidism, epilepsy, impaired hepatic +/-renal function, cardiovascular system disease, asthma, contact eczema, contact dermatitis, lactation, prolonged used. Plan relapse (at least 7 days) to prevent adverse reactions (refer to prescribing information)

Table 3–7 (continued)
Pharmacotherapies indicated for alcohol dependence

Naltrexone [Revia®]	Demonstrated efficacy in increasing abstinence, reducing relapse, and reducing amount and frequency of drinking
What it does	Anti-craving agent, competitive opioid antagonist. Blocks euphoric effects of alcohol. Non-aversive i.e. does not interact with alcohol. Not known to have dependence inducing potential. Available on PBS (Authority required)
Commence	Post withdrawal, usually 3–4 days alcohol free. Does not treat withdrawal
Treatment time	Controlled trials suggest 3 months (in practice may need to consider extending). Patients should carry a warning card in case of need for opiate analgesia.
Side effects	About 1% patients complain of nausea, headache, dizziness, fatigue, nervousness, vomiting, insomnia, depression, anxiety lasting first 2–3 weeks
Contra-indications	Opioid dependency (will precipitate withdrawal) or concurrent opioid use, acute hepatitis, hepatic failure
Precautions	Pregnancy, lactation, hepatic or renal impairment. Opioid analgesics will not work (refer to prescribing information)

Source: adapted from APF (2001); Palmer (2001)

RESOURCES

Drinking Guidelines

NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: The Costs and Benefits of Alcohol Consumption*, Commonwealth of Australia, Canberra.



www.nhmrc.gov.au

AUDIT

Dawe, S., Loxton, N.J., Hides, L., Kavanagh, D.J. & Mattick, R.P. 2002, *Review of diagnostic screening instruments for alcohol and other drug use and other psychiatric disorders*, 2nd edn., Commonwealth Department of Health and Ageing, Canberra.

Babor, T. & Higgins-Biddle, J.C. 2001, *Brief Intervention for Hazardous and Harmful Drinking: A Manual for Use in Primary Care*, WHO, Department of Mental Health and Substance Dependence, Connecticut, USA.

Indigenous and Public Health Media Unit 1999, *National Recommendations for the Clinical Management of Alcohol-related Problems in Indigenous Primary Care Settings*, Department of Health and Aged Care, Canberra, www.health.gov.au/oatsih/pubs/pdf/rec.pdf



www.dasc.sa.gov.au
(pamphlets & posters)

Standard Drink Measures

ATODS (Alcohol Tobacco and Other Drugs Services) 1997, *The Standard Drink Guide*, ATODS, QLD, www.health.qld.gov.au/atods/resources/STDDRINK.pdf

Self-help Resources

Obtain resources from your state ADIS, such as:

DASC 1995, *The drinker's guide to cutting down or cutting out*, Drug and Alcohol Services Council, Adelaide.

DASC 2001, *The Women's Drinking Guide*, Drug and Alcohol Services Council, Adelaide.

NCETA/DASC 2000, *Partners of drinkers: A resource book*, Drug and Alcohol Services Council, Adelaide.

Turning Point 1996, *Getting through Alcohol Withdrawal*, Turning Point Alcohol and Drug Centre Inc., Fitzroy, Victoria.

IEMDGP 1996, *Simple strategies to help your patients balance the use of alcohol in their lives*, Inner East Melbourne Department of General Practice, Melbourne.

Other Resources and Websites

DASC 2000, *Alcohol Related Brain Injury*, available at the DASC website or ADIS.



www.dasc.sa.gov.au

MIMS Website:



www.mims.hcn.net.au

CDHA 2001, *Public Health for Educating Clinicians: Alcohol and Other Drugs*, Module.



www.cme.net.au/phec

Alliance of NSW Divisions of General Practice.



www.answd.com.au

Shand, F., Gates, J., Fawcett, J. & Mattick, R. 2003, 'The Treatment of Alcohol Problems: A Review of the Evidence', National Drug and Alcohol Research Centre, Department of Health and Ageing, Canberra.



www.health.gov.au/pubhlth/publicat/document/alcproblems.pdf

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.
- Alliance of NSW Divisions 2000, GP Liaison Project Alcohol and Other Drugs Tip Sheet Series, Central Coast Health Service Drug and Alcohol General Practitioner Project, 1994–2000, www.answd.com.au/html/drug_alcohol_resources/tip_sheets_for_gps/AOD%20Tip%20Sheets.doc
- APF (Alcohol Pharmacotherapy Forum) 2001, *Diagnosis and Management of Alcohol Misuse: A Guide for General Practice in Australia*, Intramed Pty. Ltd., North Sydney.
- ATODS (Alcohol Tobacco and Other Drugs Services) 1997, *The Standard Drink Guide*, ATODS, QLD, www.health.qld.gov.au/atods/resources/STDDRINK.pdf.
- Babor, T. & Higgins-Biddle, J.C. 2001, *Brief Intervention for Hazardous and Harmful Drinking: A Manual for Use in Primary Care*, WHO, Department of Mental Health and Substance Dependence, Connecticut, USA.
- Babor, T., Higgins-Biddle, J.C., Saunders, J. & Monteiro, M.G. 2001, *The Alcohol Use Disorders Identification Test (AUDIT): Guidelines for Use in Primary Care* (2nd edn.) WHO, Department of Mental Health and Substance Dependence, Geneva, www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS.
- CDHA (Commonwealth Department of Health and Ageing) 2002, *National Alcohol Research Agenda: A Supporting Paper to the National Alcohol Strategy, A Plan for Action 2001 to 2003–04*, CDHA, Canberra.
- CDHAC (Commonwealth Department of Health and Aged Care) 2001, *Alcohol in Australia: Issues and Strategies. A Background Paper to the National Alcohol Strategy: A Plan for Action 2001 to 2003/2004*, CDHAC, Canberra.
- CDHSH (Commonwealth Department of Human Services and Health) 1994, *National Drug Strategy Household Survey: Urban Aboriginal and Torres Strait Islander Peoples Supplement 1994*, AGPS, Canberra.
- Chikritzhs, T., Jonas, H., Heale, P., Dietze, P., Hanlin, K. & Stockwell, T. 1999, 'Alcohol-caused Deaths and Hospitalisations in Australia, 1990–1997', *National Alcohol Indicators Bulletin No. 1*, National Drug Research Institute, Perth, Western Australia.
- Collins, D.J. & Lapsley, H.M. 2002, *Counting the Cost: Estimates of the Social Costs of Drug Abuse in Australia 1998–9*, Commonwealth Department of Health and Ageing, Canberra.
- DASC (Drug and Alcohol Services Council) 2000, *Alcohol Related Brain Injury*, Drug and Alcohol Services Council, Adelaide.

- Dawe, S., Loxton, N.J., Hides, L., Kavanagh, D.J. & Mattick, R.P. 2002, *Review of Diagnostic Screening Instruments for Alcohol and Other Drug Use and Other Psychiatric Disorders*, 2nd edn., Commonwealth Department of Health and Ageing, Canberra.
- Hulse, G.K., White, J.M. & Cape, G. 2002, *Management of Alcohol and Drug Problems*, Oxford University Press.
- Litt, J., Ali, R. & White, J. 1993, *Dealing with Alcohol Problems in General Practice*, Commonwealth Department of Health Housing Local Government and Community Services, Canberra.
- Lopatko, O., McLean, S., Saunders, J., Young, R., Robinson, G. & Conigrave, K. 2002, 'Chapter 10: Alcohol' in Hulse, G.K., White, J.M. & Cape, G. (eds.) *Management of Alcohol and Drug Problems*, Oxford University Press, South Melbourne.
- Luckman, J. & Sorenson, K.C. 1982, *Medical–Surgical Nursing: a Psychophysiologic Approach*, 2nd edn., WB Saunders Company, Philadelphia.
- NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: Health Risks and Benefits*, Commonwealth of Australia, Canberra.
- NSW Health 2000, *Alcohol and Other Drugs Nursing Policy for Nursing Practice in NSW: Clinical Guidelines 2000–2003*, NSW Health Department, Gladesville, NSW, www.health.nsw.gov.au.
- O'Connor, M. & Simmons, M. 2002, *Alcohol Screening and Brief Intervention: A Training Program for Veteran Service Providers*, Commonwealth Department of Veterans Affairs, Canberra.
- Palmer, B. 2001, *Alcohol and Drug Withdrawal: A Practical Approach. A Manual for Doctors to Assist in the Treatment of Patients Withdrawing from Alcohol and Other Drugs*, 2nd edn., Next Step Specialist Drug and Alcohol Services, Perth, Western Australia, www.nextstep.health.wa.gov.au.
- Ryder, D., Salmon, A. & Walker, N. 2001, *Drug Use and Drug Related Harm : A Delicate Balance*, IP Communications, Melbourne.
- Saunders, J., Ward, H. & Novak, H. 1996, *Guide to Home Detoxification*, Drug and Alcohol Department, Central Sydney Area Health Service, Sydney.
- Sayer, G.P., Britt, H., Horn, F., Bhasale, A., McGeechan, K., Charles, J., Miller, G., Hull, B. & Scahill, S. 2000, *Measures of Health and Health Care Delivery in General Practice in Australia*. (General Practice Series No. 3), AIHW cat. no. GEP 3, Australian Institute of Health and Welfare, Canberra.
- Shand, F., Gates, J., Fawcett, J. & Mattick, R. 2003, 'The Treatment of Alcohol Problems: A Review of the Evidence', National Drug and Alcohol Research Centre, Department of Health & Ageing, Canberra.

Victoria Police 2001, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medical Unit, Victoria Police, Melbourne.

Wood, B. & Peard, J. 1995, *Practice Guidelines for Health Professionals III Alcohol Withdrawal in the Hospital Setting*, University of Melbourne, Parkville, Victoria.

Tobacco

TOBACCO smoking causes an estimated 19,000 deaths and up to 10% of hospital admissions in those aged 35 years and over, each year in Australia (Ridolfo & Stevenson, 2001). Lifelong smokers have a 50% chance of dying from a tobacco-related disease with half these deaths occurring prematurely. (Doll et al., 1994).

No other single avoidable factor accounts for such a high proportion of deaths, hospital admissions or GP consultations (US Department of Health & Human Services, 2000).

Smoking starts young with one in seven 12–15 year olds smoking.

- 21% of males and
- 18% of females currently smoke (AIHW, 2002)

PHARMACOLOGY

Tobacco contains about 4,000 chemicals including:

- nicotine
- a number of known carcinogens (e.g. nitrosamines, toluidine, nickel, benzopyrene, cadmium and polonium 210)
- 2–6% carbon monoxide
- hydrogen cyanide
- various nitrogen oxides
- tar

Nicotine is the agent responsible for physical dependence. It is a toxic alkaloid, with a half life of 1–2 hours, that rapidly crosses the blood brain barrier to stimulate both the dopaminergic and noradrenergic pathways in the brain.

Nicotine effects on the cardiovascular system are mediated by sympathetic neural stimulation together with an increase in levels of circulating catecholamines. It has the apparent paradoxical effect of being both a stimulant (at low doses) and a relaxant (at high doses). Nicotine produces a range of toxic effects.

AT-RISK GROUPS

All individuals are at-risk from tobacco smoking but some groups are at special risk:

- socially disadvantaged groups — people of non-English speaking background, Indigenous Australians, and those with mental illness have higher prevalence of smoking
- pregnant women and unborn babies exposed involuntarily to environmental tobacco smoke (ETS)
- children exposed environmentally to tobacco in 'smoking' households and subject to peer pressure to commence smoking

DETECTION AND ASSESSMENT

Self-report of smoking status is both reliable and valid. Some smokers are sensitive about enquiry. A non-judgmental approach that also signals that all patients are asked will help minimise stigma.

ADVERSE PHYSICAL AND PSYCHOLOGICAL EFFECTS

Acute System Effects

- central nervous system (CNS) — headache, insomnia, dreams
- gastrointestinal (GI) — nausea, vomiting, heartburn, diarrhoea
- musculoskeletal system (MSS) — myalgia, arthralgias

Local Toxic Effects

These effects are mainly associated with nicotine replacement therapy (NRT):

- sore mouth, mouth ulcers (nicotine gum)
- local itching, erythema, burning (nicotine patches)
- nasal irritation, sneezing, watery eyes (nicotine spray)

Chronic System Toxicity

Cardiovascular disease

Smoking is associated with an increased incidence of cardiovascular disease (CVD) including:

- coronary heart disease — angina, myocardial infarction, sudden death, congestive heart failure
- cerebrovascular disease — transient ischaemic attacks (TIAs), stroke
- peripheral vascular diseases — claudication, aortic aneurysm

Respiratory

Smoking:

- is the primary cause of chronic obstructive airways disease through mucous hypersecretion, interference with ciliary function and alveolar destruction
- exacerbates existing hay fever and asthma
- contributes to acute and chronic rhinitis

Cancer and malignancies

Smoking is a direct cause of:

- lung cancer
- oral cavity cancers (tongue, pharynx)
- esophageal and stomach cancer
- cancer of the larynx
- kidney and bladder cancer
- pancreatic cancer
- leukaemia
- cancer of the liver

The incidence of cancer is related to the amount and duration of smoking. Concomitant heavy alcohol consumption further increases risk, especially with oral, pharyngeal and laryngeal cancer.

Gastrointestinal

Smoking is a risk factor for both peptic ulcer disease and Crohn's disease and exacerbates gastroesophageal reflux.

Complications related to pregnancy and reproduction

Smoking contributes to placental insufficiency and is a cause of placental abruption, premature labour, spontaneous abortion, stillbirth, neonatal and sudden infant death syndrome (SIDS).

Babies of mothers who smoke are more likely to:

- be born with a cleft lip and palate
- have a lower than average birthweight
- have a higher incidence of asthma, chronic serous otitis media, behavioural problems, SIDS

Women who smoke have a higher incidence of amenorrhoea, early menopause and problems with ovulation.

Men who smoke are more likely to develop impotence and have a low sperm count.

Degenerative disease

Smoking accelerates the ageing process of skin, delays wound healing and contributes to osteoporosis.

Injuries and trauma

One in two household fires is related to smoking with an estimated 30 deaths per year.

Ingestion of tobacco butts is toxic to infants.

Environmental tobacco smoke (ETS)

Tobacco smoke in the environment is derived from two sources:

- downstream smoke — exhaled by smokers
- sidestream smoke — arising from the burning end of the cigarette

The adverse effects of environmental tobacco exposure are similar to direct smoking for many conditions including:

- ischaemic heart disease
- cancer — lung and sinuses
- asthma
- chronic obstructive airway disease
- acute respiratory disease in children
- sudden infant death syndrome

Nicotine Dependence and Withdrawal**Nicotine dependence**

Tolerance to nicotine develops rapidly with 2 in 3 smokers demonstrating nicotine dependence i.e. emergence of withdrawal symptoms when they attempt to stop smoking.

Nicotine dependence can be assessed by asking two questions:

- how many cigarettes do you smoke a day?
- how long after you wake up do you have your first cigarette?

Those who smoke more than 15–20 a day and have their first cigarette within 30 minutes of waking are likely to be nicotine dependent and are also more likely to benefit from pharmacotherapy to help manage nicotine withdrawal.

Nicotine withdrawal effects

Withdrawal effects start within several hours of the last cigarette, peak in the first 24–72 hours and resolve in 2–4 weeks. Withdrawal symptoms include:

- craving
- irritability, restlessness, mood swings
- increased appetite and hunger
- sleep disturbances with resulting insomnia and fatigue
- dizziness
- anxiety and depression
- difficulty concentrating

While withdrawal is relatively short lived, many smokers relapse in the first three days and over half relapse in the first three months. A range of effective pharmacotherapies are available to help smokers overcome nicotine withdrawal.



See Chapter 4
Pharmacotherapies, p. 66

Psychological effects

Most psychological complications of smoking are a result of withdrawal from nicotine.

SOCIAL COMPLICATIONS

Highlighting the rising cost of cigarettes can be an effective strategy to reduce smoking. The high cost contributes to financial hardship and the poverty cycle, especially in socially disadvantaged groups.

Smoking is also responsible for considerable absenteeism and loss of productivity through its contribution to both acute and chronic illnesses.



SMOKING CESSATION STRATEGIES

It is useful to highlight the benefits of quitting to patients who often only see the unpleasant effects e.g. nicotine withdrawal, worsening cough. Quitting is beneficial, even after many years of smoking as it contributes to both improved quality of life and slows progression of many smoking-related diseases.

The Benefits of Quitting

The benefits of quitting smoking start immediately with noticeable effects in the first 72 hours (improved sense of smell and blood flow to hands and feet). Benefits continue to accumulate, even in those who have smoked for 20–30 years. Table 4–1 lists the benefits to be expected after quitting.

A range of policy and legislative changes have contributed significantly to the decline in smoking prevalence. These include:

- increasing the price of cigarettes
- regulating access
- making a number of public areas and facilities smoke-free
- banning the advertising of tobacco
- penetrating media campaigns depicting the damage done by each cigarette

Health care providers (especially GPs) can also make a significant difference due to:

- opportunity — over 80% of the population visit a doctor at least once a year and most smokers have several visits
- credibility/expectation/acceptability — many smokers (up to 50%) are interested in quitting and see doctors as having a key and supportive role in smoking cessation
- feasibility — brief, clear non-judgmental advice can take less than a minute

- effectiveness and efficiency — brief interventions incorporating advice, follow-up and possibly pharmacotherapies are effective and achievable. Research has shown that, with such intervention, one of every 5–6 patients will be a long-term quitter

Barriers to Assisting Smokers

Practice setting

While motivation and confidence to quit are important, one of the major barriers to clinician effectiveness is the failure to identify most smokers in a practice or clinical setting or to offer advice to those interested in quitting.

Patients

Around two thirds of smokers are interested in quitting, and half try to quit each year. Despite the difficulty of quitting, 50% of people who have ever smoked eventually successfully quit smoking. The success rate of those who use some form of assistance is double that of those who try to quit on their own.

Smoking Cessation Guidelines

Various tools can be used by health care professionals in attempting to help smokers quit:

- The Five 'A's
- CREATE (see below)
- Decision Balance Worksheet

These tools should be used in the context of the concepts and techniques described in Chapter 13 of this Handbook, such as:

- Prochaska and DiClemente's (1986) Model of Change
- the principles of a patient-centred approach
- techniques of motivational interviewing and brief intervention



See Chapter 13
Psychosocial Interventions

Table 4-1
Benefits of quitting smoking

Time elapsed	Benefit
20 minutes	<ul style="list-style-type: none"> • blood pressure drops to normal • pulse rate drops to normal • temperature of hands and feet increase to normal.
8 hours	<ul style="list-style-type: none"> • carbon monoxide level in blood returns to normal • oxygen level in blood returns to normal.
24 hours	<ul style="list-style-type: none"> • the immediate risk of heart attack starts to fall.
48 hours	<ul style="list-style-type: none"> • nerve endings start to regrow • ability to taste and smell enhanced.
14 days	<ul style="list-style-type: none"> • circulation improves • walking becomes easier • lung function increases up to 30%.
1 month	<ul style="list-style-type: none"> • most nicotine withdrawal symptoms disappear.
3 months	<ul style="list-style-type: none"> • lung function improves • nagging cough disappears • cilia regrow in the lungs, increasing their ability to handle mucus, clean themselves and reduce infection.
9 months	<ul style="list-style-type: none"> • risk of pregnancy complications and foetal death reduced to level of non-smoker.
1 year	<ul style="list-style-type: none"> • excess risk of coronary heart disease half that of a smoker. There is no safe point beyond which relapse will not occur. It continues at a much slower rate beyond one year of abstinence.
5 years	<ul style="list-style-type: none"> • risk of lung cancer decreases by half • stroke risk same as non-smoker • risk of mouth, throat and oesophageal cancer half that of a smoker.
10 years	<ul style="list-style-type: none"> • lung cancer death rate same as non-smoker • pre-cancerous cells replaced.
15 years	<ul style="list-style-type: none"> • risk of coronary heart disease same as a non-smoker • if you smoked 20 day, you've saved \$49,275 (assuming \$9 per pack of 20).

Collated by GASP from various sources

The Five ‘A’s

The Five ‘A’s, developed by the US Department of Health and Human Services and widely adopted is an evidence-based, rigorously evaluated framework for smoking cessation in health care settings.

The Five ‘A’s stand for:

- Ask
- Assess
- Advise
- Assist
- Arrange

The main components of the Five ‘A’s framework are described in Appendix H. For each component there are brief, moderate and intensive strategies.

Brief strategies should take between 1–3 minutes. If time is very limited and it is likely that the smoker could be seen again soon, spend the time available highlighting the value of quitting; alternatively give the smoker a Quitline card.



See Appendix H

CREATE

CREATE is an acronym representing a tool which can assist health care professionals to identify smoking status in all patients and provide assistance to those who are interested in quitting.



See Appendix I

CREATE arises from the evidence-based implementation guidelines developed by the RACGP (RACGP, 1998).

Also downloadable from the RACGP website:



www.racgp.org.au/folder.asp?id=301

Decision Balance Worksheet

Not all smokers wish to quit. The model of change (developed by Prochaska & DiClemente) is a useful framework to understand the process involved in changing health-related behaviour.



See Chapter 13
Psychosocial Interventions



The Decision Balance Worksheet is a simple tool which assists the smoker to assess their readiness to change. The health professional can help the smoker to complete a decision balance, by working through the smoker's own thoughts, beliefs and feelings.



See Appendix J

If the Decision Balance Worksheet reveals concerns that the smoker has about their smoking, brief motivational intervention can assist the health professional to shift the smoker towards quitting. Chapter 13 describes the concepts and techniques of motivational interviewing.



See Chapter 13
Psychosocial Interventions

While the goal is to identify all smokers and offer advice, premature advice or a confrontational approach can generate patient resistance. Tailor your approach to the smoker's readiness to quit.

Pharmacotherapies

It is important to undertake a holistic approach to smoking cessation, so that pharmacotherapy is not considered as a standalone treatment. Relapse prevention incorporates a range of psychosocial strategies which may include pharmacotherapies.

Pharmacotherapies to assist with smoking cessation include:

- nicotine replacement therapy (NRT)
- other drugs such as bupropion, clonidine and nortriptyline

Pharmacotherapies can minimise withdrawal symptoms and double the likelihood of the smoker successfully quitting. Pharmacotherapy should be considered for all smokers who have evidence of nicotine dependence.

Nicotine replacement therapies (NRTs)

NRTs help the smoker to minimise the effects of nicotine withdrawal. They are available without prescription from pharmacies. A number of smokers have tried these agents; however many have had insufficient instruction and assistance to use them effectively.

Choice of agent depends upon:

- patient preference
- pattern of any previous withdrawal symptoms
- combinations of agents may be necessary if patients are experiencing breakthrough nicotine withdrawal
- regular review is essential to tailor the dose and monitor progress
- it is important to emphasise that the smoker should abstain completely from smoking while using NRT

Table 4–2 lists the types of NRTs available, their doses and duration, side effects and contraindications.

Bupropion (Zyban®)

Bupropion offers an alternative to NRT. Smokers start bupropion 7 days before the negotiated quit date and take it for 7 weeks.

Absolute contraindications are:

- previous seizures or significant risk of seizures
- history of bipolar disorders
- history of eating disorders (bulimia, anorexia)

Table 4–3 describes the doses and duration, side effects and contraindications associated with bupropion (Zyban®).

Clonidine and nortriptyline, while effective, should be considered as second line agents as they have more troubling side effects.

Table 4–2
Pharmacotherapy of nicotine replacement therapies

Type	Dose and Duration			Side Effects	Contra-indications
	Less than 10 cigs per day	10-20 cigs per day	More than 20 cigs per day		
Patches	None	Nicobate® 14 mg Nicorette® 10 mg	Nicobate® 21 mg Nicorette® 15 mg	Transient skin irritation, itching, dreams, sleep disturbance, indigestion, diarrhoea	Relative: <ul style="list-style-type: none"> • Ischaemic heart disease Absolute: <ul style="list-style-type: none"> • Recent MI • Serious arrhythmias • Unstable angina • Pregnancy
Gum	None	2 mg, 8–12 per day	4 mg, 8–12 per day	Jaw discomfort, nausea, indigestion, hiccups, excess saliva, sore throat	
Inhaler	None	Nicorette® 6–12 cartridges per day	Not recommended	Mouth and throat irritation, cough, nausea and indigestion	

Table 4–3
Pharmacotherapy of bupropion (Zyban®)

Type	Dose and Duration			Side Effects	Contra-indications
	Less than 10 cigs per day	10–20 cigs per day	More than 20 cigs per day		
Bupropion	150 mg for 3 days, then 150 mg b.d. for 7 weeks			Headaches, dry mouth, impaired sleep, seizures, nausea, constipation, anxiety, and dizziness	1. seizure disorders or significant risk of seizure 2. bulimia 3. anorexia nervosa, 4. bipolar disorders

Other Strategies

Acupuncture, hypnosis and relaxation therapy have not been found to be effective in randomised controlled trials.



See Chapter 14
Alternative Therapies

RESOURCES

Quit books

These useful and practical booklets should be given to all smokers who are interested in quitting.

Quitline 131 848

Quitline counsellors have extensive training in both behavioural and motivational interviewing techniques. Counselling services are available in most languages (with interpreter support) and provide the flexibility of call-back and follow-up calls.

Websites

Australian Government Department of Health and Ageing National Tobacco Strategy

The 'National Tobacco Strategy 1999 to 2002-03: a framework for action' was endorsed by the Ministerial Council on Drug Strategy (MCDS) in June 1999. It builds on the National Health Policy on Tobacco 1991. It emphasises a national collaborative approach to tobacco control issues, nominating a range of government, non government and community partnerships and links.



[www.health.gov.au/pubhlth/
strateg/drugs/tobacco/](http://www.health.gov.au/pubhlth/strateg/drugs/tobacco/)

Quit SA

An expanding list of information sheets and links to related sites. Also provides information about Quit SA programs and services, and links to publications.



www.cancersa.org.au

National Tobacco Campaign

A reference to all facets of the current national campaign, 'Every cigarette is doing you damage' including background information. Also includes the *Quit Because You Can* booklet online and other help with quitting.



www.quitnow.info.au

Other Reading

Bowman, J.A. & Walsh, R.A. 2003, 'Smoking intervention within alcohol and other drug treatment services: a selective review with suggestions for practical management', *Drug and Alcohol Review*, vol. 22, pp. 73–82.

Action on Smoking and Health (Australia)

Links to newsletters, press releases, advocacy opportunities and information about litigation. Also links to Australian tobacco control legislation and a graphic depiction of what could happen to a smoker's body.



www.ashaust.org.au

Tobacco Control Supersite

Maintained by Simon Chapman, a leading Australian and international tobacco control advocate. A great source of links to other Australian and international tobacco control sites.



www.health.usyd.edu.au/tobacco

Treatobacco.net

A unique source of evidence-based data and practical support for the treatment of tobacco dependence. It is aimed at a wide range of professional groups including: physicians, nurses, pharmacists, dentists, psychologists, researchers and policy makers.



www.treatobacco.net

Royal Australian College of General Practitioners (RACGP) 'Green Book'

View or download a copy of the evidence based guidelines on implementation 'Putting Prevention Into Practice' ('Green Book').



www.racgp.org.au/reports/greenbook/implementation.htm

Treating Tobacco Use and Dependence

Public Health Service, US Department of Health & Human Services (2000). *Treating Tobacco Use and Dependence*.



www.surgeongeneral.gov/tobacco/

Guidelines for Smoking Cessation

National Advisory Committee on Health and Disability (1999). *Guidelines for Smoking Cessation*. (Wellington, New Zealand)



www.nzgg.org.nz

Groups Supporting Smoking Cessation

ACT

Cancer Council ACT
PO Box 84
Jamison Centre ACT 2614
Ph: 02 6262 2222

New South Wales

NSW Health — Tobacco & Health Unit
Locked Mail Bag 961
North Sydney NSW 2059
Ph: 02 9391 9620

Northern Territory

Department of Health and Community
Services
Tobacco Action Project
PO Box 40596
Casuarina NT 0811
Ph: 08 8999 2690

Queensland

Queensland Health
PO Box 48
Brisbane QLD 4001
Ph: 07 3234 1709

South Australia

Quit SA
PO Box 929
Unley SA 5061
Ph: 08 8291 4173

Tasmania

Quit Tasmania
2 Midwood St
New Town TAS 7008
Ph: 03 6228 2921

Victoria

Quit Victoria
PO Box 888
Carlton VIC 3053
Ph: 03 9635 5522

Western Australia

Quit WA
Department of Health
PO Box 8172
Perth Business Centre
Perth WA 6849

GASP (GPs Assisting Smokers Program)

A coalition of clinical groups with an interest in smoking cessation:

- RACGP
- AMA
- Flinders University
- Adelaide University
- Quit SA
- Anti-Cancer Council
- Asthma Foundation
- National Heart Foundation, SA Divisions Inc.
- Rural Doctors Association
- DATIS

REFERENCES

- AIHW (Australian Institute of Health and Welfare) 2002, *2001 National Drug Strategy Household Survey: First Results*. Drug Statistics Series, AIHW Canberra.
- Doll, R., Peto, R., Wheatley, K., Gray, R. & Sutherland, I. 1994, 'Mortality in relation to smoking: 40 years' observations on male British doctors', *BMJ*, 309, 901–11.
- GASP (GP Assisting Smokers Program), cited in Litt, J. 2002, 'How to provide effective smoking cessation advice in less than a minute without offending the patient', *Australian Family Physician*, vol. 31, issue 12, pp. 1087–1094
- Prochaska, J.O., DiClemente, C.C. 1986, 'Toward a comprehensive model of change' In Miller, W.R., Heather, N. (eds) *Treating Addictive Behaviors: Processes of Change*, Plenum Press, New York, 3–27.
- Ridolfo B, & Stevenson, C. 2001, *The Quantification of Drug Caused Morbidity and Mortality in Australia*, 1998, Australian Institute of Health and Welfare, Canberra.
- U.S. Department of Health and Human Services 2000, *Reducing Tobacco Use: A Report of the Surgeon General*, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, Atlanta, Georgia, USA.

Cannabis

CANNABIS is the most commonly used illicit drug in Australia. Cannabis is the general name used for the products of the plant *Cannabis sativa*. While most people who use cannabis do not experience problems, it is the most common illicit drug dependency among adults, with approximately 300,000 Australians suffering from a current cannabis use disorder (Swift et al., 2001a).

PHARMACOLOGY

Cannabis sativa contains over 400 chemical substances — about 60 are responsible for its unique effects. The principal psychoactive ingredient is delta-9-tetrahydrocannabinol (THC). THC is largely responsible for the person feeling 'stoned' with changes in mood, thoughts, perceptions and motor skills when intoxicated. THC is lipophilic and rapidly taken up by fatty tissue. This results in a slow elimination of metabolites. THC content varies greatly (from 0.5–12%), depending on genetic and environmental factors and the method of preparation.

Common Names

- marijuana: lower potency dried flowering heads and leaves of the cannabis plant
- hash(ish): extracted resin. Also known as hash oil

Routes of Administration

- smoking is the most common method of ingestion — onset of effects is more rapid and predictable. Frequently smoked with tobacco in a water pipe (bong) or rolled as a cigarette (joint)
- by mouth e.g. in food products or drunk in a tea

PHYSICAL AND PSYCHOSOCIAL COMPLICATIONS

Acute Effects

In common with other psychoactive drugs, the effects of cannabis depend on the dose, individual and setting. Many of the following effects are perceived as positive by users. The most common effects include:

- relaxation
- sense of wellbeing (euphoria)
- disinhibition
- heightened visual and auditory perceptions
- increased appetite
- altered time perception
- concentration:
 - general difficulty
 - tendency to focus awareness on a particular activity

Negative Acute Effects

There can also be negative acute effects such as:

- anxiety and panic
- paranoia
- visual or auditory hallucinations
- impaired coordination

- short-term memory loss
- tachycardia and supraventricular arrhythmias

Cannabis is not associated with fatal overdose.

Harms Associated with Chronic Use

There are several probable harms associated with regular (daily or near daily), sustained use (over several years):

- cannabis dependence syndrome: characterised by a variety of cognitive, physical and behavioural symptoms, such as an inability to control use, continued use despite problems, withdrawal and tolerance
- subtle cognitive impairment: affecting attention, memory, and the organisation and integration of complex information. Evidence to date suggests that these impairments are not grossly debilitating, but their reversibility is unknown
- adverse respiratory effects: associated with the route of administration, such as chronic bronchitis and mutagenic and carcinogenic histopathological changes of the parenchyma and epithelial cells
- an increased likelihood of carcinoma e.g. carcinoma of the oropharynx and bronchus
- reduced sperm count
- negative effects on the developing foetus. Avoiding cannabis is advisable if pregnant or trying to get pregnant

High Risk Groups

Certain groups are at a higher risk of developing adverse acute and chronic effects. These include:

- adolescents
- pregnant women. Continued smoking throughout pregnancy may increase the risk of having a low birthweight baby

- those with respiratory or cardiovascular disease, whose conditions may be aggravated by use
- those with a comorbid psychological disorder. Cannabis use is strongly associated with other drug use disorders and psychosis (Degenhardt et al., 2001). Those with schizophrenia may be particularly susceptible to the negative effects of cannabis. There is evidence that use may exacerbate psychotic symptoms in those with the disorder, and long-term, heavy use may precipitate schizophrenia in vulnerable individuals (Hall & Degenhardt, 2001).

MANAGEMENT AND INTERVENTION STRATEGIES

While many people with a substance use disorder do not seek assistance from a health professional, there has been a substantial increase in the number of cannabis smokers seeking professional assistance to quit, or to manage cannabis-related problems.

There are no maintenance pharmacotherapies available for the management of cannabis withdrawal or relapse prevention.

Assessment

Assessment should focus on:

- level and patterns of cannabis use and dependence
- evidence of psychiatric sequelae
- withdrawal symptoms
- health complications of cannabis use
- psychosocial context of use

Respiratory Function

- examination of respiratory function may be useful
- spirometry may be considered to provide feedback to a user regarding the acute consequences of smoking cannabis (alone or mixed with tobacco)
- significant respiratory problems such as emphysema, chronic bronchitis or exacerbation of asthma may be evident

Cardiovascular

- acute cardiovascular signs may also be present, either related to:
 - panic (e.g. hypertension, tachycardia); or
 - an exacerbation of angina pectoris

Detection by Urine Analysis

Psychotropic effects of cannabis are maximal at 20 minutes and last for 2–4 hours; cannabinoid levels can, however, be detected in urine up to 28 days after use. Urinary cannabinoid levels are therefore *not* an appropriate measure of recent cannabis use, intoxication or impairment.

Psychosocial Interventions

Psychosocial interventions for cannabis use disorder are still in their infancy. Most interventions used for cannabis dependence have been adapted from alcohol interventions. Psychosocial interventions are of greater benefit than no therapy, and the general principles of psychosocial interventions outlined in Chapter 13 are recommended for application in relation to problematic cannabis use.

Even one session of cognitive behavioural therapy can produce clinically significant reductions in the frequency and amount of cannabis use and related problems among severely dependent users (Copeland et al., 2001). Studies show that 6–9 sessions of cognitive behavioural therapy produce more fa-

avourable outcomes than brief motivational interventions, especially with more severely dependent users.



See Chapter 13
Psychosocial Interventions

Tolerance, Dependence and Withdrawal

A dependence syndrome associated with cannabis use has been well described (Swift et al., 2001a, 2001b). While severe dependence clearly exists, the cannabis dependence syndrome is generally less pronounced than dependence associated with drugs such as opioids and alcohol. However, the evidence is conflicting and concerns are emerging that dependence on cannabis in some younger people may develop rapidly and be more severe than previously believed.

The most common symptoms of cannabis dependence are difficulties controlling use and withdrawal (Swift et al., 2001b).

The most common symptoms of cannabis withdrawal reportedly include:

- anxiety, restlessness and irritability
- anorexia
- disturbed sleep and increases in vivid dreams
- gastrointestinal disturbances
- night sweats
- tremor

The symptoms are usually relatively mild and last a week or two. They do not require more than short-term symptomatic management.

Management and Intervention

Health professionals can significantly improve the outcome for patients presenting with cannabis use disorders by:

- providing information on the harms associated with heavy long term cannabis use
- providing advice on reducing or ceasing use
- adopting brief motivational and cognitive behavioural techniques to manage withdrawal and craving

Some people at the severe end of the dependence spectrum or with comorbid disorders may be helped by referral to specialised addiction and/or psychiatric services.

REFERENCES

- Copeland, J., Swift, W., Roffman, R. & Stephens, R. 2001, 'A randomised controlled trial of brief interventions for cannabis use disorder', *Journal of Substance Abuse Treatment*, vol. 21, pp. 55–64.
- Degenhardt, L., Hall, W. & Lynskey, M. 2001, 'Alcohol, cannabis and tobacco use among Australians: a comparison of their associations with other drug use and use disorders, affective and anxiety disorders and psychosis', *Addiction*, vol. 96, pp. 1603–1614.
- Hall, W. & Degenhardt, L. 2001, 'Cannabis use and psychosis: A review of clinical and epidemiological evidence', *Australian & New Zealand Journal of Psychiatry*, vol. 31, no. 4, pp. 659–668.
- Swift, W., Hall, W. & Teesson, M. 2001a, 'Cannabis use and dependence among Australian adults: results from the National Survey of Mental Health and Wellbeing', *Addiction*, vol. 96, no. 5, pp. 737–748.
- Swift, W., Hall, W. & Teesson, M. 2001b, 'Characteristics of DSM–IV and ICD–10 cannabis dependence among Australian adults: results from the National Survey of Mental Health and Wellbeing' *Drug & Alcohol Dependence*, vol. 63, pp. 147–153.

Cannabis

Amphetamines

AMPHETAMINES 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 well-being, 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 illicit, 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: <ul style="list-style-type: none"> • 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 'chasing the dragon'	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. 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 around 6 hours).	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. 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	<ul style="list-style-type: none"> • 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 tasks • with increasing doses, may increase libido • headache • teeth grinding 	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • tachycardia (possibly brief bradycardia), hypertension • palpitations, arrhythmias 	<ul style="list-style-type: none"> • cardiac stimulation (tachycardia, angina, arrhythmia*, MI) • vasoconstriction / hypertension • cardiovascular collapse*
Respiratory	<ul style="list-style-type: none"> • increased respiration rate and depth 	<ul style="list-style-type: none"> • respiratory difficulty/failure*
Gastrointestinal	<ul style="list-style-type: none"> • nausea and vomiting • constipation, diarrhoea or abdominal cramps 	<ul style="list-style-type: none"> • dry mouth • nausea and vomiting • abdominal cramps
Skin	<ul style="list-style-type: none"> • pale, sweaty skin • hyperpyrexia 	<ul style="list-style-type: none"> • flushing or pallor • hyperpyrexia, diaphoresis
Skeletal	<ul style="list-style-type: none"> • increased deep tendon reflexes 	
<p>(Items marked with * indicate that deaths have been attributed to amphetamine overdose, however death is rare)</p>		

(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)
- 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).

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).

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 (post-seizure, 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),

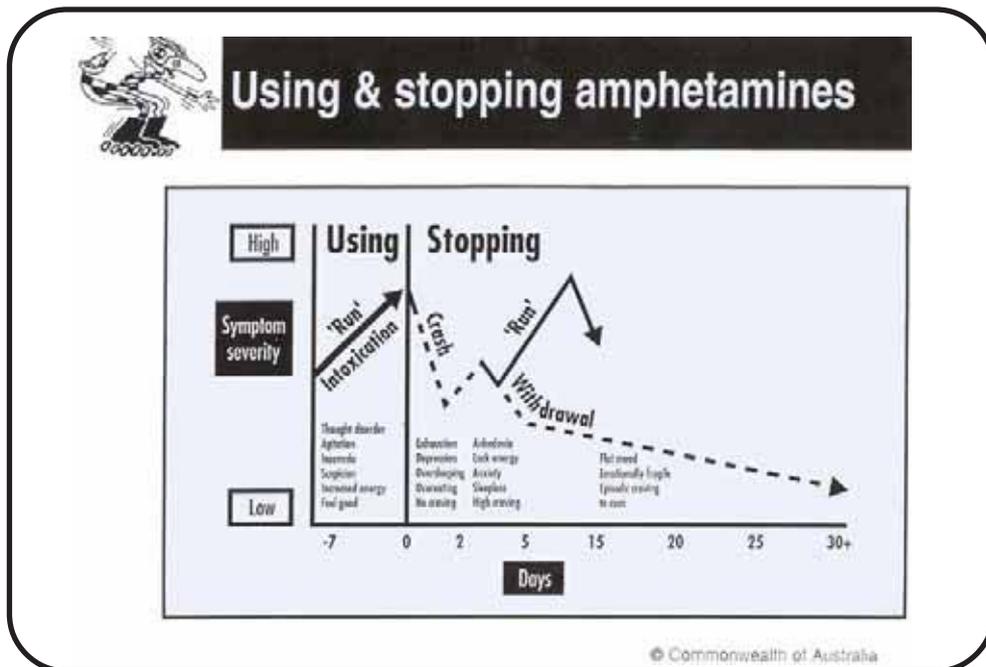


Figure 6–1
Using and stopping amphetamines (Peard et al., 1996, p. 30)

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



www.answd.com.au

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.



See Chapter 8
Cocaine

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.



See Chapter 13
Psychosocial Interventions

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



www.answd.com.au

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.