

# **Alcohol & Other Drugs:**

**A Handbook for  
Health Professionals**

# **Alcohol and Other Drugs:**

## **A Handbook for Health Professionals**

© Commonwealth of Australia 2004

ISBN 0 642 82312 X

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

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

Suggested Citation:

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

[www.nceta.flinders.edu.au](http://www.nceta.flinders.edu.au)

# About This Handbook

This Handbook is the third edition of a resource originally produced well over a decade ago. The last edition proved to be a valuable and much sought after document. In the intervening years since the last edition of the Handbook was produced the alcohol and other drugs field has changed, expanded and progressed in many ways. Hence, this new version covers expanded territory (such as polydrug use and coexisting mental health problems) and newer topic areas (such as gambling). Effort was directed at producing a resource document that was both user-friendly and also captured up-to-date, empirically sound health and medical advice. The information contained in the Handbook reflects evidence-based perspectives that are seen to be of practical value to the clinician in their day-to-day practice.

This Handbook has been produced with a very broad target audience in mind. Largely it will cater for the professional alcohol and other drugs needs of non-specialist medical practitioners and nurses. But it is also designed to be a useful practical tool for a wide range of other health and human services workers including psychologists, social workers and counsellors. We also envisage that it will be of value and interest to groups such as teachers, community workers and those with wider roles such as health promotion.

As a 'Handbook' this resource provides only a general summary overview of key issues pertinent to each of the topics covered. Readers are encouraged to seek more in-depth information from the various resources, links and contacts cited throughout the Handbook. Finally, it is important to note that the alcohol and other drugs field is particularly dynamic. While knowledge in any field is by necessity 'provisional', this may be considered to be especially the case in the changing and evolving area of alcohol and other drugs. The reader is therefore encouraged to use this resource as an entry point to the complex phenomenon of drug using behaviour and the challenging world of problem resolution.

Professor Ann M Roche  
Handbook Project Head  
Director  
National Centre for Education and Training on Addiction (NCETA)  
Flinders University



# Acknowledgments

The production of this Handbook on alcohol and other drugs has involved the input, support and collaboration of many players and partners. The proposal for the Handbook came from the consortium that formed the Editorial Group, and this was largely formed at the instigation of Professor James Rankin. Professor Rankin is thanked for his enthusiasm and drive in getting this project off the ground. The other consortium members are thanked for their sound advice and support throughout the duration of the production of the Handbook. The consortium's Editorial Group included:

- Professor Ann Roche (Project Head)
- Associate Professor Robert Ali
- Professor Richard Mattick
- Professor Brian McAvoy
- Professor James Rankin

The Handbook also involved the input of a range of individuals and groups that are not immediately apparent and this included our Reference Group and the Royal Australian College of General Practitioners (RACGP) who assisted in the piloting of the draft version of the document.

In addition, we would like to gratefully acknowledge the feedback received from many colleagues who contributed to various parts and stages of the process. Without the input of all of the above the production of this third edition of the Handbook would not have been possible.

We gratefully acknowledge the generosity of the Centre for Addiction and Mental Health (CAMH) in Canada for allowing us access to their publication:

*Managing Alcohol, Tobacco and Other Drug Problems:  
A Pocket Guide for Physicians and Nurses*

Illustrations by Simon Kneebone.

# List of Contributors

A wide range of contributors, each experts in specific areas, were involved in the writing of this Handbook. Some authors contributed whole chapters, others wrote smaller parts that were subsequently worked into larger sections or chapters. The authors who contributed to the Handbook in various ways are listed below.

Assoc. Professor Steve Allsop	Dr John Litt
Dr Michael Baigent	Professor Brian McAvoy
Dr Amanda Baker	Ms Annie Madden
Professor Bob Batey	Professor Andrea Mant
Dr Maggie Brady	Ms Heather Proudfoot
Dr Jan Copeland	Dr Alison Reid
Dr Kate Dolan	Professor Ann Roche
Professor Andrew Gilbert	Ms Jodie Shoobridge
Dr Tony Gill	Professor Tim Stockwell
Dr Linda Gowing	Dr Maree Teesson
Dr Paul Haber	Dr Libby Topp
Ms Jennifer Holmes	Professor Greg Whelan
Assoc. Professor Gary Hulse	Professor Jason White
Dr Nicole Lee	Dr Adam Winstock
Dr Patrick Lenehan	Dr Alex Wodak
Dr Nick Lintzeris	Dr Chris Wurm

In addition to the authors and the overseeing Consortium, an important role was played by a team of NCETA staff who worked extensively on the editing of the Handbook to ensure its accuracy and consistency in terms of style and presentation. The final editorial team mainly comprised Dr Carolyn Edmonds, Jodie Shoobridge and Professor Ann Roche.

In addition, a broader technical team also worked in the production of the Handbook and this group included Laura Jackson, Niola Curtis, Veronica Freund, Jane Malyschko, Joanne McDonald, Jodie Pearce, Sue Rayner, Chelsea Todd and Pamela Wright.

# Contents

## Overview

About This Handbook	i
Acknowledgments	iii
List of Contributors	iv
Contents – Overview	v
Contents – Detailed	vii
Preface	xxi
<b>PART 1: OVERVIEW AND INTRODUCTION</b>	<b>1</b>
1. Overview and Introduction	3
2. General Principles of Management and Intervention	17
<b>PART 2: THE DRUGS</b>	<b>29</b>
3. Alcohol	31
4. Tobacco	59
5. Cannabis	73
6. Amphetamines	79
7. Ecstasy	95
8. Cocaine	105
9. Heroin and Other Opioids	119
10. Volatile Substances	137
11. Benzodiazepines	147
12. Other Drugs	157
<b>PART 3: NON-MEDICAL INTERVENTIONS</b>	<b>165</b>
13. Psychosocial Interventions	167
14. Alternative Therapies	177



<b>PART 4: ISSUES FOR SPECIAL CONSIDERATION</b>	<b>183</b>
15. Pregnancy and Drug Use	185
16. Surgery and Substance Use	193
17. Managing Chronic Pain	199
18. Coexisting Mental Illness	207
19. Injecting and Communicable Diseases	215
20. Drug Issues in Correctional Services	223
21. Health Professionals As Patients	229
22. Gambling	233
<b>PART 5: APPENDICES AND GLOSSARY</b>	<b>239</b>
A. NHMRC Alcohol Guidelines – Short- and Long-term Risk	241
B. Laboratory Markers for Alcohol-related Damage	243
C. AUDIT – Interview Version	245
D. AUDIT – Self-report Version	247
E. Tip Sheet for Reducing Alcohol Consumption	249
F. Alcohol Withdrawal Assessment Scale (CIWA–AR)	251
G. Alcohol Withdrawal Observation Chart	253
H. The Five ‘A’s	255
I. CREATE	259
J. Proforma for Decision Balance Worksheet	261
K. Hepatitis C Referral Checklist	263
Glossary	265

# Contents

## Detailed

<b>PART 1: OVERVIEW AND INTRODUCTION</b>	<b>1</b>
<b>1. OVERVIEW AND INTRODUCTION</b>	<b>3</b>
<b>Types of Drugs and their Effects</b>	<b>4</b>
Identifying Harms	4
<i>The drug</i>	4
<i>The individual</i>	4
<i>The environment</i>	4
<b>Drug Usage</b>	<b>4</b>
Alcohol	5
Tobacco	5
Other Drugs	5
Polydrug Use	5
Routes of Administration	6
Terminology	7
<b>Definitions of Drug and Alcohol Problems</b>	<b>7</b>
Hazardous Use	7
Harmful Use	7
Substance ‘Abuse’	9
Substance Dependence	9
<b>Standards of Care</b>	<b>10</b>
Attitudes Towards Drug Users	10
Drug Users’ Rights	10
<b>Role of Health and Human Services Providers</b>	<b>10</b>
Medical Practitioners and Nurses	10
Other Frontline Workers	11
Health Professionals’ Role with Aboriginal and Torres Strait Islanders	12
Health Professionals’ Role with Culturally and Linguistically Diverse Groups	12
Prevention and Treatment Strategies	14
Secondary Prevention	14
Interpreting and Translation	14
Resources	14
<b>Other Special Needs Groups</b>	<b>14</b>
<b>References</b>	<b>15</b>

<b>2. GENERAL PRINCIPLES OF MANAGEMENT AND INTERVENTION</b>	<b>17</b>
<b>Harm Minimisation</b>	<b>17</b>
<b>Efficacy of Treatment</b>	<b>18</b>
<b>Early Recognition and Screening</b>	<b>18</b>
<b>Routine Enquiry About Alcohol and Drug Use</b>	<b>18</b>
Screening Questionnaires	18
Biological Screening	19
<b>Common Clinical Presentations</b>	<b>19</b>
Assessment	19
<b>Management of Low Level Problems</b>	<b>20</b>
Psychosocial Interventions	21
<b>Maintenance Pharmacotherapies</b>	<b>21</b>
<b>Pharmacotherapies for Alcohol Dependence</b>	<b>22</b>
Acamprosate (Campral®)	22
Naltrexone (Revia®)	22
Disulfiram (Antabuse®)	22
<b>Pharmacotherapies for Opioid Dependence</b>	<b>22</b>
Buprenorphine	22
Methadone	22
Levoalphaacetylmethadol (LAAM)	22
Naltrexone (Revia®)	23
<b>Withdrawal and Detoxification</b>	<b>23</b>
<b>Intoxication and Overdose</b>	<b>23</b>
<b>Coexisting Mental Health Problems</b>	<b>24</b>
<b>Drug Seeking</b>	<b>24</b>
Clinical Features	24
Management	25
<b>General Management Approaches</b>	<b>25</b>
Readiness to Change	26
<b>Resources</b>	<b>27</b>
<b>References</b>	<b>27</b>

## PART 2: THE DRUGS

29

### 3. ALCOHOL

31

#### Pharmacology

31

Absorption

31

Distribution

32

Metabolism

32

#### Patterns of Drinking

32

#### Benefits and Harms

32

#### Effects of Alcohol Consumption

33

#### Measuring Consumption: The 'Standard Drink'

35

Identifying 'At-risk' Drinking Levels

35

Groups at High Risk for Alcohol-related Harm

35

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

35

*People with mental health problems*

37

*Unborn children*

37

*Women*

37

*Occupational groups*

37

#### Identifying Harms

37

*The drug*

37

*The individual*

38

*The environment*

38

#### Alcohol Assessment

39

Early Recognition of Alcohol-related Problems

39

Four Key Assessment Steps

39

#### Screening for Alcohol Use

42

Invasive Measures

42

*Estimating BAC (Blood Alcohol Concentration)*

42

Non-invasive Measures

42

*CAGE*

42

*The AUDIT*

43

#### Brief Interventions

43

#### Alcohol Intoxication

44

Acute Alcohol Intoxication

44

Assessment of Alcohol Intoxication

44

#### Alcohol Dependence

46

#### Alcohol Withdrawal

46

General Guidelines for Alcohol Withdrawal Management

46

*Home withdrawal management*

47

Psychosocial and Physical Support During Alcohol Withdrawal

47

Pharmacological Management of Alcohol Withdrawal

48

#### After-care

50

Self-help Resources

50

Self-help Groups

50

Pharmacotherapies to Reduce Relapse/Promote Abstinence

50

<b>3. ALCOHOL (continued)</b>	
<b>Alcohol-related Brain Injury (ARBI)</b>	<b>50</b>
Wernicke–Korsakoff’s Syndrome	51
<b>Resources</b>	<b>54</b>
<b>References</b>	<b>56</b>
<b>4. TOBACCO</b>	<b>59</b>
<b>Pharmacology</b>	<b>59</b>
<b>At-risk Groups</b>	<b>60</b>
<b>Detection and Assessment</b>	<b>60</b>
<b>Adverse Physical and Psychological Effects</b>	<b>60</b>
Acute System Effects	60
Local Toxic Effects	60
Chronic System Toxicity	60
<i>Cardiovascular disease</i>	60
<i>Respiratory</i>	60
<i>Cancer and malignancies</i>	61
<i>Gastrointestinal</i>	61
<i>Complications related to pregnancy and reproduction</i>	61
<i>Degenerative disease</i>	61
<i>Injuries and trauma</i>	61
<i>Environmental tobacco smoke (ETS)</i>	61
Nicotine Dependence and Withdrawal	61
<i>Nicotine dependence</i>	61
<i>Nicotine withdrawal effects</i>	62
<i>Psychological effects</i>	62
<b>Social Complications</b>	<b>62</b>
<b>Smoking Cessation Strategies</b>	<b>63</b>
The Benefits of Quitting	63
Barriers to Assisting Smokers	63
Smoking Cessation Guidelines	63
<i>The Five ‘A’s</i>	65
<i>CREATE</i>	65
<i>Decision Balance Worksheet</i>	65
Pharmacotherapies	66
<i>Nicotine replacement therapies (NRTs)</i>	66
<i>Bupropion (Zyban®)</i>	66
<b>Other Strategies</b>	<b>68</b>
<b>Resources</b>	<b>69</b>
<b>References</b>	<b>72</b>

<b>5. CANNABIS</b>	<b>73</b>
<b>Pharmacology</b>	<b>73</b>
Common Names	73
Routes of Administration	74
<b>Physical and Psychosocial Complications</b>	<b>74</b>
Acute Effects	74
Negative Acute Effects	74
Harms Associated with Chronic Use	74
High Risk Groups	74
<b>Management and Intervention Strategies</b>	<b>75</b>
Assessment	75
Respiratory Function	75
Cardiovascular	75
Detection by Urine Analysis	75
Psychosocial Interventions	75
Tolerance, Dependence and Withdrawal	76
Management and Intervention	76
<b>References</b>	<b>77</b>
<b>6. AMPHETAMINES</b>	<b>79</b>
<b>Pharmacology</b>	<b>79</b>
Distribution	80
Metabolism	80
Availability and Quality	80
<b>Patterns of Use</b>	<b>81</b>
Routes of Administration	81
<b>Physical and Psychological Effects</b>	<b>81</b>
Acute Physical and Psychological Effects	81
Long-term Physical Effects	81
Long-term Psychological Effects	84
Amphetamine-related Harms	84
<b>Management and Intervention Strategies</b>	<b>85</b>
Acute Adverse Effects: Uncomplicated Intoxication	85
Acute Adverse Effects: Intoxication with Complications	86
<b>Using and Stopping Amphetamines</b>	<b>87</b>
Identification and Detection of Amphetamine Use and Related Problems	87
<b>Assessment</b>	<b>88</b>
<b>Withdrawal</b>	<b>89</b>
Non-pharmacological Management of Withdrawal	89
<i>Inpatient withdrawal management</i>	89
Pharmacotherapies for Managing Withdrawal and Relapse	90
Intervention Strategies Post-withdrawal	90
Harm Reduction Measures	91
<b>Resources</b>	<b>92</b>
<b>References</b>	<b>93</b>

<b>7. ECSTASY</b>	<b>95</b>
<b>Pharmacology</b>	<b>95</b>
<b>Patterns of Use</b>	<b>96</b>
<b>Physical and Psychosocial Complications</b>	<b>96</b>
Physical Effects of Ecstasy	96
<i>Hyperthermia</i>	96
<i>Hyponatraemia ('water intoxication')</i>	97
<i>Dose–response relationship</i>	98
<i>Liver damage</i>	98
Neurotoxicity	98
Psychological Effects and Complications	99
<b>Management and Intervention Strategies</b>	<b>99</b>
Strategies for Different Levels of Use	99
<i>Acute adverse effects</i>	99
Treatment for Ecstasy Use	99
<i>Pharmacological interventions</i>	99
<i>Non-pharmacological interventions</i>	99
<b>References</b>	<b>101</b>
<b>8. COCAINE</b>	<b>105</b>
<b>Pharmacology</b>	<b>105</b>
Australian Street Names	106
<b>Prevalence and Patterns of Use</b>	<b>106</b>
<b>Availability</b>	<b>106</b>
<b>Routes of Administration</b>	<b>106</b>
<b>Bingeing</b>	<b>107</b>
<b>Types of Users</b>	<b>107</b>
<b>Polydrug Use</b>	<b>107</b>
Snorters	107
Injectors	107
Cocaine and Alcohol	107
<b>Effects of Cocaine</b>	<b>108</b>
Factors Influencing the Effects	108
Desired Effects	108
Other Acute Effects of Low Doses	108
Acute Effects of High Doses ('Toxic Reactions')	108
Effects of Chronic Use	109
<b>Physical and Psychosocial Complications</b>	<b>109</b>
Physical Problems Relating to Route of Administration	109
<i>Intranasal users</i>	109
<i>Injecting users</i>	109
Other Physical Problems	109
Cocaine-related Death	109
Psychological Problems	109
Social Problems	109
Cocaine Dependence	110

<b>8. COCAINE (continued)</b>	
Cocaine Withdrawal Syndrome	110
Foetal Effects	110
<b>Management and Intervention Strategies</b>	<b>111</b>
Clinical Screening	111
<i>Acute toxic reactions</i>	111
Chronic Cocaine Use	111
Treatment of Toxic Reactions	111
Management of Withdrawal	112
Treatment of Cocaine Dependence	112
<i>Pharmacotherapy</i>	112
<i>Cognitive-behavioural therapy</i>	112
<i>Contingency management</i>	113
<i>Enhancement of psychosocial skills</i>	113
<i>Acupuncture</i>	113
<i>General approaches</i>	113
<i>Comorbid disorders</i>	113
<i>Readiness to change</i>	114
<b>References</b>	<b>115</b>
<b>9. HEROIN AND OTHER OPIOIDS</b>	<b>119</b>
<b>Patterns of Heroin Use</b>	<b>119</b>
<b>Opioid Drugs</b>	<b>119</b>
Heroin	119
Morphine	120
Methadone	120
Buprenorphine (Subutex®)	121
Naltrexone	122
<b>Tolerance</b>	<b>123</b>
<b>Withdrawal</b>	<b>123</b>
<b>Dependent Heroin Use</b>	<b>123</b>
The Dependence Syndrome	123
Natural History of Dependent Heroin Use	123
Features of Dependent Heroin Use in Australia	124
<b>Harms Associated with Heroin Use</b>	<b>124</b>
Overdose	124
Harms Related to Injecting	124
Psychological Harms	125
Social and Community Harms	125
<b>Management and Intervention Strategies</b>	<b>125</b>
<b>Withdrawal Services</b>	<b>125</b>
Objectives	125
Key Components	125
<i>Assessment</i>	125
<i>Setting</i>	125
<i>Supportive care</i>	125



<b>9. HEROIN AND OTHER OPIOIDS (continued)</b>	
<i>Frequent monitoring and review</i>	126
<i>Medication</i>	126
Medication Regimes for Opioid Withdrawal	127
Post-withdrawal Interventions	130
<i>Psychosocial interventions</i>	130
<i>Outpatient counselling</i>	130
<i>Therapeutic communities</i>	130
<i>Self-help groups</i>	131
Naltrexone Treatment	131
<i>Clinical aspects</i>	131
<i>Adverse events</i>	131
<i>Outcomes associated with naltrexone treatment</i>	131
<b>Substitution Maintenance Treatment</b>	
<b>with Methadone or Buprenorphine</b>	<b>131</b>
Rationale, Objectives and Outcomes	131
Delivering Substitution Treatment in Australia	132
Problems with Maintenance Substitution Treatment	133
Principles of Safe and Effective Methadone/Buprenorphine Treatment	133
<b>Resources</b>	<b>134</b>
<b>References</b>	<b>135</b>
<b>10. VOLATILE SUBSTANCES</b>	<b>137</b>
<b>Commonly Used Volatile Substances</b>	<b>137</b>
<b>Modes of Administration</b>	<b>138</b>
<b>Prevalence</b>	<b>138</b>
<b>Appeal</b>	<b>139</b>
<b>Physical Complications</b>	<b>139</b>
Acute Effects	139
Negative Acute Effects	139
Effects at Higher Doses	139
Specific Physical Effects	139
<i>Central nervous system</i>	139
<i>Maternal and neonatal</i>	140
<i>Heart</i>	140
<i>Lung</i>	140
<i>Kidneys, liver and bone marrow</i>	140
Other Morbidity and Mortality	140
<b>Psychosocial Complications</b>	<b>140</b>
<b>Management and Intervention Strategies</b>	<b>141</b>
Detection and Assessment	141
Intoxication	141
Experimental, Recreational and Chronic Use	141
Primary Prevention Strategies	143
<b>Resources</b>	<b>144</b>
<b>References</b>	<b>145</b>

<b>11. BENZODIAZEPINES</b>	<b>147</b>
<b>Pharmacology</b>	<b>147</b>
<b>Absorption</b>	<b>147</b>
<b>Distribution</b>	<b>148</b>
<b>Metabolism</b>	<b>148</b>
<b>Patterns of Use</b>	<b>148</b>
<b>Effects of Benzodiazepines</b>	<b>148</b>
Short-term Effects	148
Long-term Effects	149
<b>Uses and Problems</b>	<b>149</b>
<b>High Risk Groups</b>	<b>149</b>
<b>Prescribing Benzodiazepines</b>	<b>150</b>
Rational Use of Benzodiazepines	150
Precautions	150
Drug Interactions	150
Use in Management of Anxiety and Insomnia	150
<b>Management and Intervention Strategies</b>	<b>151</b>
Reviewing Benzodiazepines in Long-term Users: A Staged Approach	151
Dependence and Withdrawal	152
Aged Care Residential Facilities	153
<b>Benzodiazepine Misuse</b>	<b>153</b>
Habitual Drug Users ('Doctor Shoppers')	153
Drug Dependent Patients	153
<b>References</b>	<b>155</b>
<b>12. OTHER DRUGS</b>	<b>157</b>
<b>Hallucinogens</b>	<b>157</b>
Physical and Psychological Effects	158
Management and Intervention	158
<b>Party Drugs</b>	<b>158</b>
GHB	159
Ketamine	159
<b>Anabolic Steroids</b>	<b>160</b>
<b>Over The Counter Drugs</b>	<b>161</b>
Non-prescription Medication	161
<i>Analgesics</i>	161
<i>Antihistamines</i>	161
<i>Sympathomimetics</i>	161
<i>Cough suppressants</i>	162
Injecting Drug Users	162
<i>Other</i>	162
<b>References</b>	<b>163</b>

<b>PART 3: NON-MEDICAL INTERVENTIONS</b>	<b>165</b>
<b>13. PSYCHOSOCIAL INTERVENTIONS</b>	<b>167</b>
<b>Patient Readiness — A Model of the Process of Change</b>	<b>168</b>
Pre-contemplation Stage	168
Contemplation Stage	169
Preparation Stage	169
Action Stage	169
Maintenance Stage	169
<b>Clinical Strategies</b>	<b>169</b>
Patient-centred Approach	169
Decision Balance	169
Building A Therapeutic Alliance	169
<i>Empathy and reflective listening</i>	170
<i>Using open ended questions</i>	171
<i>Reflective listening</i>	171
<i>Summarising</i>	171
<i>Roadblocks to empathy</i>	171
Motivational Interviewing	172
Brief Motivational Interviewing	173
Problem Solving	174
Goal Setting	174
Relapse Prevention	175
Quality of Life	175
<b>References</b>	<b>176</b>
<b>14. ALTERNATIVE THERAPIES</b>	<b>177</b>
<b>Evidence of Effectiveness</b>	<b>177</b>
Acupuncture	178
Hypnosis	179
Conclusions	179
<b>References</b>	<b>180</b>

<b>PART 4: ISSUES FOR SPECIAL CONSIDERATION</b>	<b>183</b>
<b>15. PREGNANCY AND DRUG USE</b>	<b>185</b>
<b>Assessment</b>	<b>185</b>
<b>Information on Alcohol and Drug Effects</b>	<b>187</b>
Alcohol	187
Tobacco	188
Cannabis	189
Heroin	189
Psychostimulants — Amphetamines and Cocaine	190
Ecstasy	190
<b>Neonatal Abstinence Syndrome (NAS)</b>	<b>191</b>
<b>Child Protection</b>	<b>191</b>
<b>References</b>	<b>192</b>
<b>16. SURGERY AND SUBSTANCE USE</b>	<b>193</b>
<b>Tobacco</b>	<b>194</b>
<b>Alcohol</b>	<b>194</b>
Alcohol and Post-operative Morbidity	194
Peri-operative Management of Alcohol-related Complications	195
<b>Opioids</b>	<b>196</b>
Management Strategies	197
<b>Benzodiazepines</b>	<b>197</b>
<b>Stimulant Use</b>	<b>197</b>
<b>References</b>	<b>198</b>
<b>17. MANAGING CHRONIC PAIN</b>	<b>199</b>
<b>Acute and Chronic Pain</b>	<b>199</b>
<b>Assessment and Pain Management in Drug Users</b>	<b>200</b>
Screening for Substance Dependence	201
Alternative Options in Management	201
Dependence and Tolerance	202
Preventing Drug Dependence in Patients with Chronic Non-malignant Pain	202
Principles of Pain Management in Opioid Dependent Patients	203
<i>Methadone maintenance</i>	203
<i>Buprenorphine</i>	204
<i>Terminal illness</i>	204
<b>The Potential for Adverse Interactions</b>	<b>204</b>
<b>The Patient–Clinician Relationship</b>	<b>205</b>
<b>Resources</b>	<b>206</b>

<b>18. COEXISTING MENTAL ILLNESS</b>	<b>207</b>
<b>Epidemiology</b>	<b>207</b>
<b>Terminology</b>	<b>208</b>
<b>Some Specific Associations</b>	<b>208</b>
From a Drugs Perspective	209
From a Mental Health Perspective	209
<b>Management</b>	<b>210</b>
What Works?	210
Principles of Care	210
Assessment and Management	210
Harm Minimisation	211
<b>Resources</b>	<b>212</b>
<b>References</b>	<b>213</b>
<b>19. INJECTING AND COMMUNICABLE DISEASES</b>	<b>215</b>
<b>Infective Problems Associated with Injecting Drug Use</b>	<b>216</b>
<b>Blood Borne Communicable Diseases</b>	<b>216</b>
<b>Hepatitis C</b>	<b>216</b>
Testing for HCV	216
Natural History	217
Assessing Patients for Treatment	217
Antiviral Therapy	217
Managing Patients who have Failed Therapy or are Ineligible for Treatment	217
Liver Transplantation in HCV	217
Prevention of HCV	217
Alcohol and HCV	218
<b>Hepatitis B</b>	<b>218</b>
Testing for HBV	218
Natural History	218
Treatment of HBV	218
<b>Hepatitis D</b>	<b>219</b>
<b>Human Immunodeficiency Virus</b>	<b>219</b>
Testing for HIV	219
Treatment of HIV	219
Prevention	219
Effect of HIV on IDU Problems	219
<b>HTLV/III</b>	<b>220</b>
<b>Resources</b>	<b>221</b>

<b>20. DRUG ISSUES IN CORRECTIONAL SERVICES</b>	<b>223</b>
<b>Patterns of Drug Use Among Prisoners</b>	<b>223</b>
<b>History of Imprisonment and Prevalence of Injecting</b>	<b>224</b>
<b>Prevalence and Transmission of Blood Borne Viral Infections</b>	<b>224</b>
<b>Interventions to Reduce Risk Behaviour and Transmission</b>	<b>224</b>
Bleach Programs	225
Condoms	225
Methadone Maintenance Treatment	225
Syringe Exchange Schemes in Prison	225
<b>References</b>	<b>227</b>
<b>21. HEALTH PROFESSIONALS AS PATIENTS</b>	<b>229</b>
<b>Treating a Health Professional with a Drug or Alcohol Problem</b>	<b>230</b>
<b>Dealing with a Colleague with a Drug or Alcohol Problem</b>	<b>230</b>
Registering Authorities	230
The Steps to Take	231
<b>Being a Health Professional with a Drug or Alcohol Problem</b>	<b>231</b>
<b>22. GAMBLING</b>	<b>233</b>
<b>Problem Gambling</b>	<b>233</b>
Identifying Problem Gamblers	234
<b>Presentation of Problem Gamblers</b>	<b>234</b>
<b>Treatment/Referral Options</b>	<b>236</b>
<b>References</b>	<b>237</b>

## **PART 5: APPENDICES AND GLOSSARY** **239**

<b>A. NHMRC Alcohol Guidelines – Short- and Long-term Risk</b>	<b>241</b>
<b>B. Laboratory Markers for Alcohol-related Damage</b>	<b>243</b>
<b>C. AUDIT – Interview Version</b>	<b>245</b>
<b>D. AUDIT – Self-report Version</b>	<b>247</b>
<b>E. Tip Sheet for Reducing Alcohol Consumption</b>	<b>249</b>
<b>F. Alcohol Withdrawal Assessment Scale (CIWA–AR)</b>	<b>251</b>
<b>G. Alcohol Withdrawal Observation Chart</b>	<b>253</b>
<b>H. The Five ‘A’s</b>	<b>255</b>
<b>I. CREATE</b>	<b>259</b>
<b>J. Proforma for Decision Balance Worksheet</b>	<b>261</b>
<b>K. Hepatitis C Referral Checklist</b>	<b>263</b>
<b>Glossary</b>	<b>265</b>







**Part 1**

---

# **Overview and Introduction**



# Overview and Introduction

**P**ROBLEMS caused by the use of psychoactive drugs touch all areas of medicine and health care. Some problems are well known and highly visible such as the respiratory illnesses caused by smoking, liver disease from harmful alcohol consumption and overdose from heroin injection. Others are more subtle and often missed by health professionals as an underlying cause of a wide range of health and social harms.

Personal and social problems from drug use are substantial and cut across all domains of functioning including personal relationships, family life, employment and psychological health.

The health and economic costs associated with the use of drugs are high, with costs of legal drugs estimated to be substantially higher than those from illegal drugs. The annual cost of drug use in Australia is estimated to be \$34.4 billion (Collins & Lapsley, 2002) of which:

- \$21.1 billion was from tobacco
- \$7.6 billion from alcohol
- \$6.1 billion from illicit drugs

The Australian Institute of Health and Welfare estimated that in 1998 there were 23,313 drug-related deaths in Australia of which 19,019 were due to smoking tobacco, 3,271 to risky alcohol use and 1,023 to illicit drug use (Ridolfo & Stevenson, 2001). The bulk of the latter comprised deaths from heroin overdose.

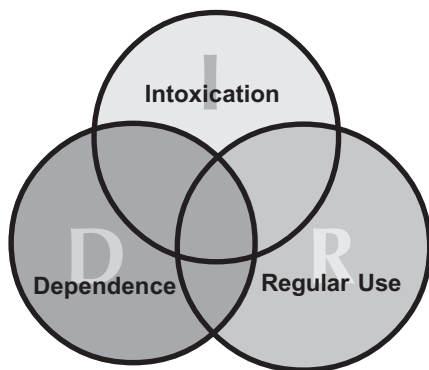
In addition to drug-related deaths, in 1997–1998 there were:

- 142,525 hospital separations attributable to tobacco smoking
- 71,422 attributable to risky alcohol use
- 14,471 to illicit drugs

There are three main patterns of risky drug use with corresponding patterns of problems. These are:

- intoxication (e.g. violence, falls, road trauma, overdose)
- regular use (e.g. liver disease, cancer)
- dependence (e.g. withdrawal symptoms, social problems)

These three distinct patterns of use can occur within the one person, or in different individuals.



There are also growing problems associated with injecting drug use including the spread of blood borne viruses. While the rate of HIV infection amongst Australian injecting drug users is still very low by world standards (1–2%), hepatitis C is an emerging concern and is likely to generate some thousands of cases of liver disease in future years (NCHECR, 1999).

## TYPES OF DRUGS AND THEIR EFFECTS

### Identifying Harms

Alcohol- and drug-related harms are not specific to the effects of the drug. 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 the substance including 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

## DRUG USAGE

Knowledge and understanding about patterns and correlates of drug use are derived from various sources including surveys. Surveys of drug use are usually (1) conservative estimates of prevalence and (2) do not give an indication of the number of people using drugs in *problematic* ways. In the clinical setting careful, individualised assessment is required to determine patterns and levels of use. The following provides brief highlights of key drug use patterns (see relevant chapters for more detail on specific drugs).

### Alcohol



See Chapter 3  
Alcohol

Most Australians drink alcohol – 80% of Australians aged 15 and over report drinking alcohol in the past year. The National Health and Medical Research Council's (NHMRC) new Alcohol Guidelines (NHMRC, 2001) define low risk regular use as no more than 4 standard drinks per day for men and 2 for women, and no more than 6 and 4 drinks for males and females respectively on occasion.



[www.nhmrc.gov.au/  
publications/pdf/ds9.pdf](http://www.nhmrc.gov.au/publications/pdf/ds9.pdf)

A higher level of intake is now considered to be low risk on an occasional basis, ie no more than 6 standard drinks for men and 4 for women, provided certain precautions and restrictions are observed (e.g. drinking less if at all, when pregnant, not drinking before driving). However, there are many drinkers who exceed these limits and 46% of males and 32% of females do so at least once a month (Heale et al., 2000). Heavier patterns of consumption are a concern for all health professionals as they are strongly associated with a wide range of acute and chronic harms.

### Tobacco



See Chapter 4  
Tobacco

The 2001 National Drug Strategy Household Survey found that 21% of males and 18% of females smoked daily (AIHW, 2002).

Tobacco use amongst Indigenous Australians is 2 to 3 times higher than the broader community.

Very few people smoke only occasionally and there is no established safe level of tobacco use. Early uptake of tobacco smoking by young people is of concern for several reasons including its highly addictive nature.

### Other Drugs



See Chapters 5–12

Cannabis is the most widely used illicit drug in Australia with 13% of all individuals aged 14 or over having used it during the previous 12 months (AIHW, 2002) and 33% at some time in their lives.

Amphetamine and ecstasy use has become increasingly prevalent: one in nine males aged 20–29 years reported using amphetamines in the last 12 months. Males are generally more likely to use, with the exception of teenagers where use by girls is more prevalent than by boys (AIHW, 2002).

Lifetime use of heroin is estimated to be 2% and of cocaine 3–4% of the population. It is estimated that in the year 2000 there were approximately 74,000 dependent heroin users or 0.7% of Australians aged 15 to 54 years of age (Hall et al., 2000).

### Polydrug Use

Until recently it was common to characterise illicit drug use by the drug, or class of drug, primarily used. For instance, heroin users were identified as a distinct category of user, as were stimulant users. These characterisations are no longer valid. Most illicit drug users are likely to use a variety of substances. Drug substitution also occurs. When there is a shortage of some drugs (e.g. heroin) other drugs (e.g. amphetamines, alcohol) may be used as an alternative. Increased ease of availability of drugs is likely to have contributed to diversity in patterns of use.

Certain associations are well recognised; for example:

- cigarettes and alcohol often go hand in hand, particularly where heavy use of either substance is involved
- cannabis smokers are almost invariably tobacco smokers (although obviously the reverse is not the case)
- heroin users often also take drugs such as cocaine and benzodiazepines, and nearly all heroin users are also cigarette smokers
- heavy drinkers also often use illicit drugs

Many illicit drug users possess sophisticated pharmacological knowledge. Users often exhibit considerable skill in the titration of various substances when used in concert with one another. For example, combinations of drugs such as heroin and cocaine (known as 'speedballs'\*) allow the sedative action of one drug (i.e. the heroin) to take the sharp edge off the stimulant (i.e. the cocaine). Similarly, some substances are less commonly taken when using another preferred drug e.g. some users avoid taking ecstasy and alcohol concurrently.

Multiple substance use complicates the assessment process. Signs and symptoms of intoxication for various drugs can be similar. Also concurrent use can complicate withdrawal. Polydrug use also confounds our understanding of dependence problems (Gossop, 2001). A person who uses a range of different psychoactive substances may not be dependent on all drugs that he/she uses. Comprehensive drug use histories are required, and no assumptions should be made about patterns of use or non-use. It is important to note that most available assessment tools assess dependence (and not usually

\* Note that the term 'speedball' sometimes also refers to a combination of heroin and amphetamine.

*problematic use*) and for a single drug only, or provide separate substance specific measures. Careful decisions regarding prioritisation for treatment are needed. This should be done in consultation with the client/patient.

## Routes of Administration

Drugs can be taken in various ways. The mode of administration is a significant mediating factor on the effect of a drug. Various routes of administration are preferred because they can enhance or facilitate drug effects. Different modes of administration have advantages and disadvantages. The most common routes of administration are:

- *oral ingestion*: probably the oldest and the most common form of taking drugs. Advantages are convenience, no special paraphernalia is required and degree of safety for some drugs. Disadvantages are the slow absorption of some substances
- *chewing*: used for coca leaf, tobacco, betel-nut and tea. Absorption occurs across the oral mucosa
- *nasal insufflation*: includes snuffing, nasal inhalation or snorting. Absorption is through the nasal mucosa. Snuffing can be used for cocaine, powdered opium, heroin and tobacco. Sniffing of amyl nitrite occurs, as does sniffing of petrol and other volatile substances
- *smoking*: is used for a wide variety of substances including tobacco, cannabis, opium, heroin, cocaine, amphetamines and phencyclidine (PCP)
- *rectal administration*: commonly used in medical treatment, it is also a method sometimes used by drug users. Disadvantages are the potential for irregular, unpredictable and incomplete absorption
- *parenterally (via injection)*: became possible in the late 19<sup>th</sup> century with the development of the hypodermic needle. Arguably this has irrevocably transformed hedonistic drug use. Administration can be intravenous (via a vein), intramuscu-

lar (via a muscle), or subcutaneous (under the skin). Each has advantages and disadvantages. Injection carries with it a range of important health risks including transmission of viral and bacterial diseases and tissue damage

Harm minimisation strategies provide opportunities to educate users about safer ways to administer drugs. Safe injecting techniques are especially important. Changing from one route of administration to another may also be a useful stepping stone to cutting down and quitting.

Table 1–1 lists the major psychoactive drugs and describes their intoxication effects and potential adverse health effects.

For more information regarding specific effects of particular drugs, including a discussion of acute effects, high dose effects and effects of chronic use, refer to the individual chapters in Part 2 of this Handbook.



See Chapters 3–12

The website of the National Institute of Drug Abuse (NIDA) located in the United States of America contains additional useful information about common names of drugs, routes of administration and references for further reading.



[www.nida.nih.gov](http://www.nida.nih.gov)

### Terminology

Throughout this Handbook, you will note some variations in the language used to describe drug and alcohol use and associated problems. In Australia, the preferred terminology is ‘problematic use’ as this is less pejorative than other terms. However, some of the international and official classifications

include terms such as ‘abuse’ or ‘misuse’. The preference is to avoid the use of negative or value-laden terms, labels or language.

As this Handbook is intended for a wide range of health and human services workers terms such as ‘patient’ and ‘client’ are used interchangeably.

### DEFINITIONS OF DRUG AND ALCOHOL PROBLEMS

Problematic drug use has been formally categorised in some systems as:

- hazardous use
- harmful use
- substance abuse
- substance dependence

#### Hazardous Use

Hazardous use refers to a pattern of substance use that increases the risk of harmful consequences for the user. These consequences can include physical and/or mental health problems; some would also include social consequences. Hazardous use refers to patterns of use that are of public health significance despite the absence of any current disorder in the individual user.

Refer to National Institute on Alcohol Abuse and Alcoholism (NIAAA) Thesaurus on their website.



[www.etoh.niaaa.nih.gov/AODVol1/Aodthome.htm](http://www.etoh.niaaa.nih.gov/AODVol1/Aodthome.htm)

#### Harmful Use

Harmful use (ICD–10) is defined as a pattern of psychoactive substance use that is causing damage to health. The damage may be physical (e.g. hepatitis following injection of drugs) or mental (e.g. depressive episodes



**Table 1-1**  
**Intoxication and potential adverse health effects**

	<b>Intoxication effects</b>	<b>Potential adverse health effects</b>
<b>Alcohol</b>	<ul style="list-style-type: none"> <li>reduced pain and anxiety</li> <li>feeling of wellbeing</li> <li>lowered inhibitions</li> </ul>	<ul style="list-style-type: none"> <li>trauma and a range of effects on cardiovascular, respiratory, gastrointestinal, haematological and neurological systems</li> <li>dependence</li> </ul>
<b>Opioids</b> <ul style="list-style-type: none"> <li>heroin</li> <li>codeine</li> <li>fentanyl</li> <li>morphine</li> <li>methadone</li> <li>buprenorphine</li> <li>pethidine</li> </ul>	<ul style="list-style-type: none"> <li>pain relief</li> <li>euphoria</li> <li>drowsiness</li> </ul>	<ul style="list-style-type: none"> <li>respiratory depression and arrest</li> <li>nausea</li> <li>confusion</li> <li>constipation</li> <li>sedation</li> <li>unconsciousness</li> <li>coma</li> <li>tolerance</li> <li>dependence</li> </ul>
<b>Stimulants</b> <ul style="list-style-type: none"> <li>amphetamines</li> <li>cocaine</li> <li>ecstasy/MDMA</li> <li>methylphenidate</li> <li>nicotine</li> <li>caffeine</li> </ul> <b>Depressants</b> <ul style="list-style-type: none"> <li>barbiturates</li> <li>benzodiazepines</li> </ul>	<ul style="list-style-type: none"> <li>increased heart rate, blood pressure, metabolism</li> <li>feelings of exhilaration, energy, increased mental alertness</li> </ul> <ul style="list-style-type: none"> <li>reduced pain and anxiety</li> <li>feelings of wellbeing</li> <li>lowered inhibitions</li> <li>slowed pulse and breathing</li> <li>lowered blood pressure</li> <li>poor concentration</li> </ul>	<ul style="list-style-type: none"> <li>rapid or irregular heartbeat</li> <li>reduced appetite</li> <li>weight loss</li> <li>heart failure</li> <li>dependence</li> </ul> <ul style="list-style-type: none"> <li>confusion</li> <li>fatigue</li> <li>impaired coordination, memory, judgement</li> <li>respiratory depression and arrest</li> <li>dependence</li> </ul>
<b>Cannabinoids</b> <ul style="list-style-type: none"> <li>cannabis</li> <li>hash</li> </ul>	<ul style="list-style-type: none"> <li>euphoria</li> <li>slowed thinking and reaction time</li> <li>confusion</li> <li>impaired balance and coordination</li> </ul>	<ul style="list-style-type: none"> <li>cough</li> <li>frequent respiratory infections</li> <li>impaired memory and learning</li> <li>increased heart rate</li> <li>anxiety</li> <li>panic attacks</li> <li>tolerance</li> <li>dependence</li> </ul>
<b>Other – includes:</b> <ul style="list-style-type: none"> <li>hallucinogens such as LSD; dissociative anaesthetics (ketamine, PCP); inhalants (solvents, nitrites and other gases); steroids</li> </ul>	<ul style="list-style-type: none"> <li>various effects</li> </ul>	<ul style="list-style-type: none"> <li>various effects</li> </ul>

Source: adapted from the US National Institute of Drug Abuse (NIDA) website. Information about alcohol has been added.

**Table 1–2**  
**DSM–IV–TR (APA, 2000) Criteria for substance dependence**

The maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12 month period.

1. Tolerance, as defined by either a need for markedly increased amounts of the substance to achieve intoxication or desired effect or a markedly diminished effect with continued use of the same amount of the substance
2. Withdrawal, as defined by either the characteristic withdrawal syndrome for the substance or where the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
3. The substance is often taken in larger amounts or over a longer period than was intended
4. There is a persistent desire or unsuccessful attempts to cut down or control substance use
5. A great deal of time is spent on activities necessary to obtain the substance or to recover from its effects
6. Social, occupational or recreational activities are given up or reduced
7. Substance use is continued despite awareness of recurrent problems associated with use

secondary to heavy alcohol intake). Harmful use commonly, but not invariably, has adverse social consequences. Social consequences, however, in themselves, are not sufficient to justify a diagnosis of harmful use.

### **Substance ‘Abuse’**

Substance ‘abuse’ is a term used by DSM–IV–TR (APA, 2000, p. 199). It is defined as a maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one or more of the following, occurring within a 12 month period:

- failure to fulfil major role obligations
- use in situations in which it is physically hazardous
- recurrent substance-related legal problems

- continued use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance

Unlike dependence, ‘abuse’ is not characterised by withdrawal, tolerance or a pattern of compulsive use, only the adverse consequences of repeated use.

### **Substance Dependence**

Substance dependence on the other hand, is defined (APA, 2000) as a characteristic set of cognitive, behavioural and physiological signs in which the individual will continue to use the substance despite considerable related problems. Tolerance has developed and withdrawal symptoms are present upon cessation of the drug. The actual criteria for dependence are

summarised in Table 1–2 DSM–IV–TR (2000) Criteria for Substance Dependence.

In addition to these criteria, the World Health Organization (WHO) International Classification of Diseases, 10th Edition (ICD–10) suggests that another essential characteristic of dependence is that the individual must possess a strong desire to take the substance and is indeed consuming it (Proudfoot & Teesson, 2000).

It is important to note that many problems associated with the use of alcohol or other psychoactive drugs do not involve dependence. That is, you do not need to be dependent on a drug to experience harms from its use.

## STANDARDS OF CARE

### Attitudes Towards Drug Users

Psychoactive drug users often experience discrimination and stigma when accessing health services. While not all health professionals discriminate against drug users, poor treatment and discriminatory practices have been identified as primary barriers to accessing health care.

Negative attitudes are often based on stereotypes and fears. Such stereotypes can result in discrimination, stigma and marginalisation. Like other groups in the community, drug users are a diverse group with differing needs and backgrounds. In the health care context, recognising the diverse needs of every individual is critical to professional and effective treatment and ensures appropriate standards of care are met.

### Drug Users' Rights

Treating all illicit drug users as 'drug seeking', unreliable and disruptive will not result in a positive outcome for either the person

who uses drugs or the health professional. Participation in an illegal behaviour does not mean that individuals surrender their basic health and human rights. Illicit drug users should be treated in the same way as other people, that is, as individuals with specific needs requiring information and communication on all options, professional diagnosis and where appropriate, treatment.

## ROLE OF HEALTH AND HUMAN SERVICES PROVIDERS

### Medical Practitioners and Nurses

Medical practitioners and nurses who are not specialists in drug and alcohol have a critically important role to play in the provision of drug and alcohol treatments.

General practitioners and other primary care health professionals are particularly well suited for this role because:

- 85% of the population visit a general practitioner at least once per year
- general practitioners and primary health professionals are usually the first point of contact with the health care system
- patients are often at a learning moment and expect to receive lifestyle advice from general practitioners
- general practitioners are in an ideal position to link prevention with comprehensive, continuing and holistic care
- general practitioners provide a range of services that span the health care continuum from prevention of illness to treatment and rehabilitation

(RACGP National Preventive & Community Medicine Committee, 1998)

Medical practitioners and nurses are ideally placed to:

- provide relevant information about drugs and alcohol to all patients
- identify drug- and alcohol-related problems
- provide interventions
- refer for specialist assessment and treatment when required; and
- coordinate care and follow up patients over time

There is a growing body of evidence about the effectiveness of interventions and benefits of treatment that medical practitioners and nurses can provide. These include:

- screening
- assessment
- information and advice
- brief interventions for tobacco, alcohol and to a lesser extent cannabis
- detoxification, including home detoxification
- pharmacotherapy for tobacco, alcohol and opioid dependence
- counselling, including motivational interviewing, and relapse prevention
- referral to clinicians with specialist skills in drug and alcohol
- follow-up monitoring and care coordination

These interventions have been shown to be effective in specialist and non-specialist settings.

For clinicians with specific drug and alcohol competencies, a more comprehensive role in the care of patients can be undertaken including:

- management of intoxication and withdrawal
- motivational interviewing
- management of detoxification
- pharmacotherapy treatments
- counselling
- treatment of medical comorbidities

- management of psychiatric comorbidities
- care of pregnant women with drug- and alcohol-related problems and their neonates; and
- follow-up monitoring and review

### Other Frontline Workers

The complexity and diversity of problems associated with alcohol and drug use has increased substantially over the past decade. The potential support and intervention roles for health and human services workers has increased accordingly. Evidence for the efficacy of early intervention has been well established and identifies an important role for any professional in a position to intervene for alcohol and drug problems.

Key professional groups identified as pivotal frontline workers include:

- alcohol and other drug specialist workers
- general health workers such as medical practitioners, nurses, Indigenous health workers and psychologists
- volunteer workers in a variety of community groups including parent and family groups, self-help groups, church groups and counselling support groups
- police and law enforcement personnel
- welfare professionals, including social workers, youth workers and other community-based workers
- teachers and education personnel

It is no longer assumed that support and intervention for alcohol and other drug (AOD) problems is the exclusive province of specialist professionals. While interventions and treatments have become more specific and technical in recent years (most notably in relation to pharmacological interventions) there is also an expanded role for generalist frontline workers especially from a prevention, harm minimisation and early intervention perspective.

## Health Professionals' Role with Aboriginal and Torres Strait Islanders

There is a range of special considerations in relation to the AOD use of Indigenous Australians. Patterns and correlates of use are often quite different and health care needs more complex than for the wider community.

Proportionately fewer Indigenous people drink than in the Australian community at large. However, amongst those who consume alcohol the majority do so at hazardous and harmful levels, often drinking heavily on a single occasion. There is often intense social pressure for Indigenous drinkers to continue to drink. Relatedness to others is deeply embedded within Aboriginal social life and sharing alcohol (and increasingly other drugs) naturally plays an important part in this. Public pressure to share and socialise around alcohol is very strong, and those who try to moderate or give up may be criticised. Health care workers can be valuable aids in supporting moderate use or cessation.

Prevalence of tobacco smoking is 2 to 3 times higher than the national average, and there are very high rates of cannabis (yarni, ganya) use. It has also been recognised recently that rates of injecting drug use amongst young Indigenous people have grown exponentially and are associated with very high levels of diseases such as hepatitis C. There are also increasing levels of use of other drugs such as heroin with high levels of needle sharing.

General practitioners and other health care workers have considerable potential to help motivate Indigenous patients to reconsider their drinking and/or drug use. Health professionals should not feel constrained (e.g. by fears of being culturally inappropriate), to provide a range of brief interventions to patients just because they are Indigenous. As is the case with any patient or client, such advice should be offered sensitively and in a non-judgmental manner, and avoid any

implication of criticism. Research into self-quitting amongst Indigenous people suggests that health care workers can be more influential than they think (see Table 1–3).

The Australian Drug Information Network (ADIN) contains useful links:



[www.adin.com.au/  
indigenous.html](http://www.adin.com.au/indigenous.html)

## Health Professionals' Role with Culturally and Linguistically Diverse (CALD) Groups

### *Cultural background and drug use*

Australia is ethnically a highly diverse country. A person's cultural background i.e. country of birth, language spoken at home, religion and ethnic background may have an impact on drug use and/or associated problems and their resolution. Different cultures vary in their attitudes to and use of alcohol and other drugs. Alcohol consumption, for example, varies greatly within and between countries. In Italy, for instance wine is commonly consumed with meals but intoxication is not accepted. Some cultures favour the use of drugs little known in Australia (e.g. khat, betel nut), while alcohol is much less widely used in many countries, including some which are significant sources of refugees and migrants to Australia. In many Asian countries, the traditional use of opioids once tended to be by smoking. However, this is rapidly changing with injecting becoming increasingly common among Asian populations.

Religious affiliation may also be relevant. Religious observance is often an important aspect of culture, and may play a part in the manner and extent of drug use. A person of Islamic background for instance may develop a problem with alcohol, but be less willing to discuss it and may fear community criticism.

**Table 1–3**  
**Why health care workers are influential amongst Indigenous people**

Reason	Explanation
Privacy of consultation	Avoids the potential stigma of attending an identified alcohol and other drug service, and provides the necessary confidentiality.
Expectations of the doctor's role	Indigenous patients expect doctors and health care workers to talk honestly about their health problems, to diagnose and give advice. It is particularly important to link the presenting problem with alcohol- or drug-related problems where possible, as patient's knowledge about these links may be minimal. It is important to stress the effect of the patient's drinking and/or drug use on their family responsibilities.
Respect for specialised knowledge	Medical practitioners in particular are known to have specialised knowledge of the body. This invests them with considerable authority amongst Indigenous people, and provides doctors with significant potential to motivate for change in drinking and other drug use behaviour.
Personalised advice and providing evidence of harm	Linking advice on alcohol consumption to the individual's presenting problem is more influential than a general talk about alcohol awareness. Indigenous patients seem to respond well to offers of biological tests, the results of which provide objective proof of the harmful effects of alcohol misuse. Such evidence can be particularly useful to the indigenous patient.
Neutral advice from an informed outsider	Professional advice on changing drinking behaviour can motivate the individual to consider change, partly because a doctor is usually an 'outsider', not of the patient's family or community. Community health workers known to the patient can sometimes find it intrusive to discuss alcohol- or drug-related matters with other indigenous people. In the face of intense social pressures to drink, authoritative advice from an outsider can be of particular value. Having an external reason can legitimise an individual's refusal to participate in drinking sessions with friends and family members, without causing offence.

The circumstances under which a person came to Australia may also be important. Those migrating under family reunion quotas may have more support than refugees who may have previously faced poverty, illness and war. Some individuals may have depression or post-traumatic stress disorder following trauma or torture in their country of origin.

## Prevention and Treatment Strategies

Prevention and treatment programs need to take into account the characteristics of individuals with Indigenous and non-English speaking backgrounds. Different cultural values as well as language issues need to be considered in any prevention strategy. There is untapped potential for ethnic newspapers and broadcasting services to be used in primary prevention strategies.

## Secondary Prevention

Many screening tools have not been validated with different ethnic or cultural groups and should be used and interpreted with care. The AUDIT alcohol screening tool has been validated with different cultural groups.



See Chapter 3  
Alcohol  
'The AUDIT', p. 45

## Interpreting and Translation

The use of skilled interpreters with the appropriate dialect and of the patient/client's preferred gender is crucial. It is inappropriate to use family members as interpreters. Even if the patient does not see the need, an interpreter may still be required to ensure an accurate assessment and appropriate management strategy.

## Resources

NSW Drug and Alcohol Multicultural Education Centre, DAMEC.



[www.damec.org.au](http://www.damec.org.au)

## OTHER SPECIAL NEEDS GROUPS

There are several other groups who have special needs in relation to AOD use. These groups may not engage well in treatment unless their special needs are met. These groups include:

- those located in rural and remote areas
- women
- those of different sexual orientations
- youth

The last group, youth, are particularly important to highlight. More young people are engaging in problematic AOD use at younger ages. They are especially vulnerable to AOD-related problems due to age and inexperience. Health and human services workers are increasingly called upon to provide youth-friendly and youth-appropriate services.

### 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.
- APA (American Psychiatric Association) 2000, *Diagnostic and Statistical Manual of Mental Disorders*, 4<sup>th</sup> edn., (DSM-IV), APA, Washington DC.
- 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.
- Gossop, M. 2001, 'A web of dependence', *Addiction*, vol. 96, pp. 677–678.
- Hall, W., Ross, J., Lynskey, M., Law, M. & Degenhardt, L. 2000, *How Many Dependent Opioid Users Are There in Australia?*, National Drug And Alcohol Research Centre Monograph no. 44., University of NSW, Sydney.
- Heale, P., Stockwell, T., Dietze, P., Chikritzhs, T. & Catalano, P. 2000, *Patterns of Alcohol Consumption in Australia, 1998*. National Alcohol Indicators Project, Bulletin No. 3., National Drug Research Institute, Curtin University of Technology, Perth, Western Australia.
- NCHECR (National Centre in HIV Epidemiology and Clinical Research) 1999, *HIV/AIDS, Hepatitis C and Sexually Transmissible Infections in Australia: Annual Surveillance Report*, NCHECR, Sydney.
- NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: Health Risks and Benefits*, Commonwealth of Australia, Canberra.
- Proudfoot & Teesson 2000, *Investing in Drug and Alcohol Treatment*, National Drug and Alcohol Research Centre, Sydney, [www.ndarc.med.unsw.edu.au](http://www.ndarc.med.unsw.edu.au).
- RACGP National Preventive & Community Medicine Committee 1998, *Putting Prevention into Practice: Guidelines for the Implementation of Prevention in the General Practice Setting*, RACGP, Melbourne.
- Ridolfo, B. & Stevenson, C. 2001, *The Quantification of Drug-caused Mortality and Morbidity in Australia, 1998*, Drug Statistics Series No. 7, AIHW, Canberra.
- Stockwell, T., Heale, P., Dietze, P., Chikritzhs, T. & Catalano, P. (in press), 'How much alcohol is consumed in Australia in excess of the new NHMRC national drinking guidelines?', *Medical Journal of Australia*.



# Overview

## Introduction

# General Principles of Management and Intervention

**T**HIS CHAPTER reviews key principles involved in identification, management and intervention of alcohol and other drug (AOD) problems. Issues covered here are expanded on in relevant chapters.

Problems associated with the use of alcohol or other drugs can be related to:

- intoxication
- regular use
- dependence

Not all problems are related to dependence or addiction. Many problems are related to non-dependent patterns of use that are risky for either the person or those around them. Interventions should be tailored to the type of problems experienced or the nature of the risks to which the individual is exposed.

## HARM MINIMISATION

Harm minimisation is an important principle in the management and intervention of AOD problems. Many intervention options are pragmatic in nature and based on an understanding that changing behaviour can be a lengthy, complex process. From a harm minimisation perspective abstinence may not be the highest or most immediate priority: Emphasis is placed on reducing as many prob-

lems as possible associated with alcohol and drug use, and not just focusing on the drug use per se. Harm minimisation strategies address the overall health and wellbeing of the individual and the community at large.

### EFFICACY OF TREATMENT

It is important to stress the efficacy of treatments specific to problematic AOD use. In contrast to common perceptions, there is a range of effective, empirically evaluated treatment options available. Treatment can be successful. It is as successful as many general medical treatments that are held in high regard.

Intervention earlier rather than later is strongly recommended. Treatment for long-term chronic problems can also be very effective.

**Table 2-1**  
Treatment success for dependence

Drug of Dependence	Success Rate (%)
Alcohol	50 (40-70)
Opioids	60 (50-80)
Cocaine	55 (50-60)
Nicotine	30 (20-40)

Source: O'Brien and McLellan (1996)

### EARLY RECOGNITION AND SCREENING

Early recognition is important as it can enable intervention to occur before dependence or irreversible damage has developed. However, alcohol and drug problems can be difficult to detect, especially in the early stages.

Reasons include:

- not knowing what to look for
- lack of vigilance
- embarrassment about asking questions
- not knowing what to do if a problem is uncovered
- the person's denial or evasion

(Edwards, Marshall and Cook, 1997)

Detection rates can be improved by:

- routine enquiry about alcohol and drug use
- screening questionnaires
- biological screening (pathology tests)
- knowledge of common clinical presentations

### ROUTINE ENQUIRY ABOUT ALCOHOL AND DRUG USE

General practice and primary health care offer a variety of opportunities to enquire about alcohol and drug use; for example, in the context of:

- new patients — as part of initial information gathering
- management of chronic problems — alcohol for example, is a risk factor in cardiovascular disease, diabetes, depression
- management of acute problems, especially trauma, gastrointestinal disorders, anxiety/stress, psychological problems
- preoperative assessment
- pre-conception and antenatal care
- enhanced Primary Care Medicare Benefit Schedule items — health assessment, care plans and case conferences

### Screening Questionnaires

Use of general questionnaires covering lifestyle issues such as smoking, diet, exercise, alcohol and drug use may be less threaten-

ing and stigmatising for patients.

There is also a number of short, well validated questionnaires which can be used to screen for alcohol problems. Screening and brief interventions can readily be combined in a single general practice consultation.



See Chapter 13  
Psychosocial Interventions

## Biological Screening

A number of blood tests can be used to screen for alcohol problems. However, they can be less sensitive and specific than questionnaires. These screening tests include:

- full blood count, including MCV
- liver function tests, including gamma GT
- triglycerides

Urine testing can detect alcohol, other drugs (e.g. cocaine, opioids, cannabis, benzodiazepines and barbiturates) and/or their metabolites.

Screening tests for drug use include:

- full blood count, including white cell count
- liver function tests
- hepatitis B and C and HIV serology

## COMMON CLINICAL PRESENTATIONS

Indicators of alcohol- or drug-related problems are wide-ranging and can involve:

- cardiovascular
- gastrointestinal
- musculoskeletal
- neurological

- dermatological
- genito-urinary systems
- accidents/trauma, social and legal incidents

Indicators of problematic drug use can include:

- infections (injecting users)
- accidents/trauma
- psychiatric problems
- behavioural, social and legal incidents



See Chapters 3–12 for more detail about individual drugs

## Assessment

Assessment is critical and has several purposes:

- to identify substance use behaviour early
- to discover the extent of use and its health effects
- to examine the social context of substance use in both the patient and significant others
- to determine a care plan and appropriate interventions

(Rassool, 1998)

The assessment phase should fulfil four important functions:

1. developing a therapeutic relationship based on trust, empathy and a non-judgmental attitude
2. helping the client to accurately reappraise their drug use, which may in turn facilitate the desire to change
3. facilitating a review of the client's past and present circumstances and linking these to current drug use
4. encouraging the client to reflect on the

choices and consequences of drug using behaviour  
(Helfgott, 1997)

Traditionally treatment success was measured by abstinence. Today there is more emphasis on the client's:

- wellbeing
- beliefs about drinking and drug use
- readiness to change
- alcohol- and drug-related expectancies
- social functioning and social support

These are all important predictors of success.

Current approaches to treatment of alcohol- and drug-related problems reflect a continuum of treatment.

## MANAGEMENT OF LOW LEVEL PROBLEMS

Low level drug and alcohol problems are much more common than dependence and are major causes of morbidity and mortality. Individuals with low level problems are better suited to brief and early interventions whereas individuals experiencing more severe problems need more specialised treatment (National Expert Advisory Committee on Alcohol, 2001).

A 'brief intervention' is considered to be:

*'any intervention that involves a minimum of professional time in an attempt to change drug use... Any intervention requiring a total of between five minutes and two hours'*

(Heather, 1990)

Brief interventions are particularly suitable for primary care but can also be used in emergency departments, hospital wards or outpatient clinics and a range of non-medical settings.

They are recommended for individuals with:

- hazardous/harmful alcohol use without dependence
  - a low to moderate dependence on alcohol
  - a dependence on nicotine
  - a low to moderate dependence on cannabis
- (Best Practice in Alcohol and Other Drug Interventions Working Group, 2000).

There is compelling evidence for the effectiveness of brief interventions to reduce hazardous and harmful alcohol consumption by 30–40% (WHO Brief Intervention Study Group, 1996).

Brief interventions are not considered suitable for:

- more complex patients with additional psychological/psychiatric issues
- patients with severe dependence
- patients with poor literacy skills
- patients with difficulties related to cognitive impairment

In these instances, more in-depth intervention is recommended (Heather, 1995).

Brief intervention can take a variety of forms but often includes:

- brief assessment
- self-help materials
- information on safe levels of consumption
- advice on reducing consumption
- harm reduction
- relapse prevention
- assessment of readiness to change, in-

cluding motivational interviewing

- brief counselling, including problem solving and goal setting
- follow-up

Six therapeutic elements are common to successful brief interventions (FRAMES):

- F.** Feedback — provide feedback from your clinical assessment
- R.** Responsibility — emphasise the person's personal responsibility for their drug use and associated behaviour
- A.** Advice — provide clear, practical advice and self-help material
- M.** Menu — offer a range of behaviour change and intervention options
- E.** Empathy — express non-judgmental empathy and support
- S.** Self-efficiency — stress belief in the person's capacity for change

(Miller and Sanchez, 1993)



See Chapter 13  
Psychosocial Interventions

## Psychosocial Interventions

Psychological interventions are a key component of a comprehensive treatment program and can involve group therapy or individual counselling.

*'...counselling alone is not usually sufficient to change the drug taking behaviour of most clients'*

(Jarvis et al., 1995)

Counselling is a joint approach between the counsellor and the client with treatment plans negotiated and agreed upon by both parties. No single psychological approach is superior, and the treatment program should be tai-

lored to the individual patient/client, taking into consideration such factors as culture, age, gender and presence of comorbidity.

General counselling should include:

- linking patients with the appropriate services while the patient is still engaged
- anticipating and developing strategies with the patient to cope with difficulties before they arise
- specific evidence-based interventions where appropriate (e.g. goal setting, cognitive behavioural therapy, motivational enhancement therapy, problem solving)
- focusing on positive internal and external resources and successes as well as problems and disabilities
- consideration of the wider picture and helping the patient on a practical level (e.g. with food, finances, housing)
- where appropriate, involving key supportive others to improve the possibility of behavioural change outside the therapeutic environment

(Best Practice in Alcohol and Other Drug Interventions Working Group, 2000)

Mutual aid groups such as Alcoholics Anonymous, Narcotics Anonymous, Al-Anon (for relatives of alcohol dependent individuals) and Alateen (for adolescent relatives) are also available. Their approaches are based on the 12 Steps, a set of principles that emphasise personal responsibility and honesty.

## MAINTENANCE PHARMACOTHERAPIES

A number of effective therapeutic drugs are now available for the treatment of dependence, primarily for alcohol, nicotine and opioid de-

pendence. It is likely that the use of pharmacotherapies will increase in the future.

Pharmacotherapies should not be considered as stand alone treatments but should be used as part of a comprehensive treatment program, including supportive counselling, other relevant therapies and social support.

## PHARMACOTHERAPIES FOR ALCOHOL DEPENDENCE

### Acamprosate (Campral®)

This is an anticraving agent which acts as a GABA-receptor agonist. Randomised controlled trials have shown:

- reduced quantity and frequency of drinking in patients who do not achieve complete abstinence
- reduced rates of relapse (where relapse is defined as consumption of any alcohol)
- increased percentages of abstinent days during treatment
- increased rates of abstinence

### Naltrexone (Revia®)

This anticraving agent is a competitive opioid antagonist which blocks the euphoric and reinforcing effects of alcohol. Randomised controlled trials have shown:

- reductions in the amount and frequency of drinking overall
- reductions in the rate and relapse into heavy drinking (where relapse is defined as a return to > 5 drinks per day)
- increased rates of alcohol abstinence

Both naltrexone and acamprosate are available on the Pharmaceutical Benefits Scheme (PBS) for use within a comprehensive treat-

ment program.

### Disulfiram (Antabuse®)

An alcohol-sensitising agent which inhibits aldehyde dehydrogenase causing a toxic build-up of acetaldehyde if alcohol is consumed. This results in unpleasant symptoms such as facial flushing, nausea, vomiting, sweating and palpitations.

Randomised controlled trials have shown variable results and only a modest effect in promoting abstinence. Disulfiram is not available on the PBS.

## PHARMACOTHERAPIES FOR OPIOID DEPENDENCE

### Buprenorphine

A strong opioid analgesic with both partial agonist and partial antagonist properties. It is an alternative to methadone for withdrawal and maintenance treatment, and has a much lower risk of death from overdose than methadone. Buprenorphine is listed on the PBS as S100 under Section 100 of the National Health Act 1953 and is approved by the Therapeutic Goods Administration (TGA).

### Methadone

This is a long-acting synthetic opioid which can be used for both withdrawal and maintenance treatment. It decreases the need for heroin-dependent individuals to regularly use intravenous opioids. Methadone maintenance programs monitor drug use and should provide ongoing counselling and support. Methadone is listed on the PBS as S100 under Section 100 of the National Health Act 1953 and is approved by the Therapeutic Goods Administration (TGA).

## Levoalphacetylmethadol (LAAM)

LAAM is a synthetic opioid analgesic which acts similarly to methadone. It is long-acting and only needs to be taken three times per week. Overseas trials suggest that LAAM is as safe as methadone and has similar treatment outcomes and patient retention. This drug is not available for use in Australia.

## Naltrexone (Revia®)

This is a competitive opioid antagonist, which completely blocks the effects of opioids for 24 to 72 hours. Maintenance therapy is suitable for highly motivated patients who wish to remain abstinent, are socially and psychologically stable and have good social support. Naltrexone is not listed on the PBS for opioid dependence but is approved by the TGA as an adjunctive therapy. Clinical trials are also underway using naltrexone for rapid detoxification.

## WITHDRAWAL AND DETOXIFICATION

Detoxification is withdrawal from a drug in a supervised way in order to minimise withdrawal symptoms and risks related to withdrawal.

Details of withdrawal management are covered in the management and intervention sections of relevant chapters on specific drugs.

Effective withdrawal management may be performed in the home supported by the GP, other health workers and non-using supportive relatives or friends. This form of withdrawal management depends on:

- the drug of dependence
- the severity of the dependency
- the wishes of the patient

Home-based withdrawal management should be considered:

- when there is no evidence of severe withdrawal, e.g. tremor, hallucinations, disorientation\*
- where there is no past history of delirium tremens or of fits\*
- in the presence of supportive relatives who elect to stay with the patient during the period of detoxification
- when there is no evidence of a medical illness such as pneumonia or pancreatitis\*
- when no previous history or evidence of suicide is contemplated
- where the patient does not have access to the drug from which they are being withdrawn

(\* in the case of alcohol)

Withdrawal can be medicated (assisted by the use of controlled sedatives) or non-medicated. The latter is appropriate for patients who have no co-existing medical disorders and when only a mild withdrawal can be anticipated.

In cases of multiple drug use, patients may not wish to withdraw from all substances at the same time. Withdrawal management should be part of an ongoing treatment program linked to coping and relapse prevention strategies.

## INTOXICATION AND OVERDOSE

Intoxication is defined as the intake of a quantity of a substance which exceeds the individual's tolerance.



Further reading:  
Ellenhorn & Barceloux (1988)



Overdose is defined as the state that occurs when a person has ingested a quantity of a drug that exceeds tolerance and produces behavioural and physical abnormalities.

Details of intoxication and overdose management are covered under the management and intervention sections in each Chapter.

When presented with an intoxicated or overdose patient the priority is ABC First Aid procedures:

- A — Airway
- B — Breathing
- C — Circulation/cardiac

In acute overdose it is recommended that patients are closely observed, monitored and referred to an acute hospital.

Do not assume that alcohol or drugs are the sole cause of the patient's coma. Other possible causes include:

- trauma
- epilepsy
- metabolic abnormalities – diabetes, hepatic failure, hypercalcaemia, renal failure
- cerebrovascular events – cerebral haemorrhage/thrombosis, abscess, tumour
- cardiovascular events – arrhythmias, myocardial infarction
- respiratory failure
- infection – meningitis, encephalitis

Once intoxication or overdose has been treated it is important to:

- ask about depression, suicidal ideation (may need a referral to a psychiatrist)
- explore withdrawal management and treatment options

## COEXISTING MENTAL HEALTH PROBLEMS

In patients using alcohol and other drugs coexisting mental health problems, such as anxiety and depression, are not uncommon. It is important to distinguish symptoms which are:

- part of a primary psychiatric disorder
- secondary to problems such as marital conflict, homelessness or legal problems
- drug or alcohol induced



See Chapter 18  
Coexisting Mental Illness

## DRUG SEEKING

### Clinical Features

Patients seeking prescribed drugs for non-medical use often approach emergency departments of major hospitals or general practitioners' surgeries at busy times or shortly before closing. Patients may choose a general practitioner who does not know them or who are known to prescribe drugs very readily.

Drug seeking patients are usually:

- polydrug using
- males
- aged in their twenties or thirties

The drugs sought are usually:

- opioid analgesics; or
- benzodiazepines

The presentations for analgesics usually involve painful conditions where there are few physical signs, such as headache, renal colic

or backache. The names of analgesics which have proved effective on previous occasions are often referred to with familiarity.

The most frequently requested opioid is pethidine by injection.

Commonly, patients will claim that analgesics other than the preferred type:

- have previously proved ineffective and/or
- resulted in severe side effects including an allergic reaction.

A careful history plus physical examination contributes substantially to the diagnosis and management plan. Suspicion of illicit drug use is supported by a history of common complications of drug use (such as hepatitis C, endocarditis or previous incarceration). Physical examination should include inspection for track marks. It is often helpful to have a patient discretely observed by an experienced nurse for signs of variability in the severity of signs of pain.

## Management

As special tests usually cannot prove if the patient's symptoms result from organic disease, the final clinical decision depends on the doctor's judgment and experience. In most cases, decisions are relatively easy. When uncertain, the choice has to be made between possibly withholding analgesia from a patient in severe pain or possibly prescribing an opioid analgesic to a malingering patient. The former is a far more serious error.

Injections of ketorolac (Toradol®), a non-steroidal anti-inflammatory drug (NSAID), is a safe way of providing a potent analgesic without prescribing an opioid.

Buprenorphine is a potent analgesic which provides minimal euphoria but this can precipitate opioid withdrawal if the patient has withheld a history of recent opioid use.

Presentations for benzodiazepines often involve a history of anxiety or insomnia due to a recent bereavement. Short-acting benzodiazepines should only be prescribed in very special circumstances and then only in small quantities. If benzodiazepines have to be prescribed, it is better to select long-acting forms but in small quantities.

Doctors who consider that a patient is seeking drugs should gently advise the patient that they are concerned about this possibility and offer relevant assistance or referral.

## GENERAL MANAGEMENT APPROACHES

Support and treatment for drug users should follow the general supportive and common-sense approaches described below.

Clinicians should:

- *not* judge the user and should not insist on abstinence
- seek to engage and retain the user in treatment for as long as possible, as retention is associated with better outcomes (Simpson et al., 1999)
- ensure understanding of the client/patient's treatment goals:
  - to make it through an acute crisis?
  - to reduce frequency and/or quantity of drug use?
  - to achieve long-term abstinence?
- tailor the treatment where possible to meet those goals, including referral when appropriate to:
  - treatment programs
  - individual counsellors
  - family counsellors
  - self-help groups such as NA

- remember the need for flexibility of service delivery; as goals and outcomes change throughout the course of treatment, the treatment program should be adjusted to reflect these changes
- provide as multifaceted and intensive a program as possible, as more intensive psychosocial treatment programs are associated with better outcomes (Crits-Cristoph, 1999).

## Readiness to Change

In patients who do not wish to become abstinent despite significant impairment related to drug use, the clinician should attempt to:

- establish an empathetic, respectful relationship
- retain contact with the client
- maximise physical and mental health, as clients will find it difficult to achieve long-term abstinence if chronic medical problems have not been adequately treated
- enhance motivation toward abstinence by educating clients and their significant others about the usual course of drug dependence and the relationship between drug use and current and/or future problems
- emphasise the client's responsibility for their own actions
- help clients rebuild a life without drugs through:
  - vocational counselling
  - family counselling
  - helping them build a network of non-drug using peers
  - showing them how to use free time appropriately

### RESOURCES

Further reading: A basic modern text on toxicology is Ellenhorn, M. & Barceloux, D.G. (1988), *Medical Toxicology: Diagnosis and Treatment of Human Poisonings*, Elsevier, New York.

### REFERENCES

- Best Practice in Alcohol and Other Drug Interventions Working Group 2000, *Evidence Based Practice Indicators for Alcohol and Other Drug Interventions*, [www.wa.gov.au/drugwestaus](http://www.wa.gov.au/drugwestaus)
- Crits-Cristoph, P. 1999, 'Psychosocial treatments for cocaine dependence: National Institute on Drug Abuse Collaborative Cocaine Treatment Study', *Archives of General Psychiatry*, vol. 56, pp. 493–502.
- Edwards, G., Marshall, E.J. & Cook, C.C.H. 1997, *The Treatment of Drinking Problems: A Guide for the Helping Professions*, Cambridge University Press, New York.
- Heather, N. 1990, cited in Ali, R., Miller, M. & Cormack, S. 1992, *Future Directions for Alcohol and Other Drug Treatment in Australia*, AGPS, Canberra.
- Heather, N. 1995, 'Psychology and brief interventions', *British Journal of Addiction*, vol. 84, pp. 357–370
- Helfgott, S. 1997, 'Assessment' in Helfgott, S., (ed.), *Helping Change: The Addiction Counsellor's Training Program*. Western Australian Alcohol and Drug Authority, Perth.
- Rassool, G. Hussein (ed.) 1998, *Substance Use and Misuse: Nature, Context and Clinical Interventions*, Blackwell Science, Oxford.
- 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.
- Miller, W.R. & Sanchez M.C. 1993, 'Motivating young adults for treatment and lifestyle change' cited in Howard, G. (ed.), *Issues in Alcohol Misuse by Young Adults* University of Notre Dame Press, Notre Dame, pp. 55–79.
- National Expert Advisory Committee on Alcohol 2001, *National Alcohol Strategy. A Plan for Action 2001 to 2003–04*, Commonwealth Department of Health and Aged Care, Canberra.
- O'Brien, C.P. & McLellan, A.T. 1996, 'Myths about the treatment of addiction', *Lancet*, vol. 347, pp. 237–240.

Simpson, D.D., Joe, G.W., Fletcher, B.W., Hubbard, R.L. & Anglin, M.D. 1999, 'A national evaluation of treatment outcomes for cocaine dependence', *Archives of General Psychiatry*, vol. 56, pp. 507–514.

WHO Brief Intervention Study Group 1996, 'A Cross-national trial of Brief Interventions with Heavy Drinkers', *American Journal of Public Health*, vol. 86, pp. 948–955.

## Part 2

---

# The Drugs



# Alcohol

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

\*Blood Alcohol Concentration

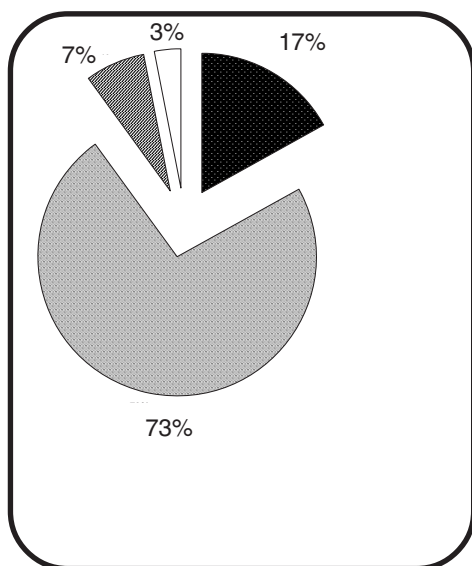
Source: adapted from Victoria Police (2001, p. 1.8) and Ryder et al. (2001, p. 162).

## 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
<b>BEER</b>			
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
<b>WINE</b>			
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
<b>FORTIFIED WINES</b> E.g. port, sherry			
	60 ml glass	21%	1.0
	750 ml bottle	18%	11.0
	2 litre (cask/flagon)	18%	28.0
<b>PREMIX COOLERS AND SODAS</b>			
	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
<b>SPIRITS</b>			
	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 contra-indicated.

### **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**

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

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](http://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](http://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

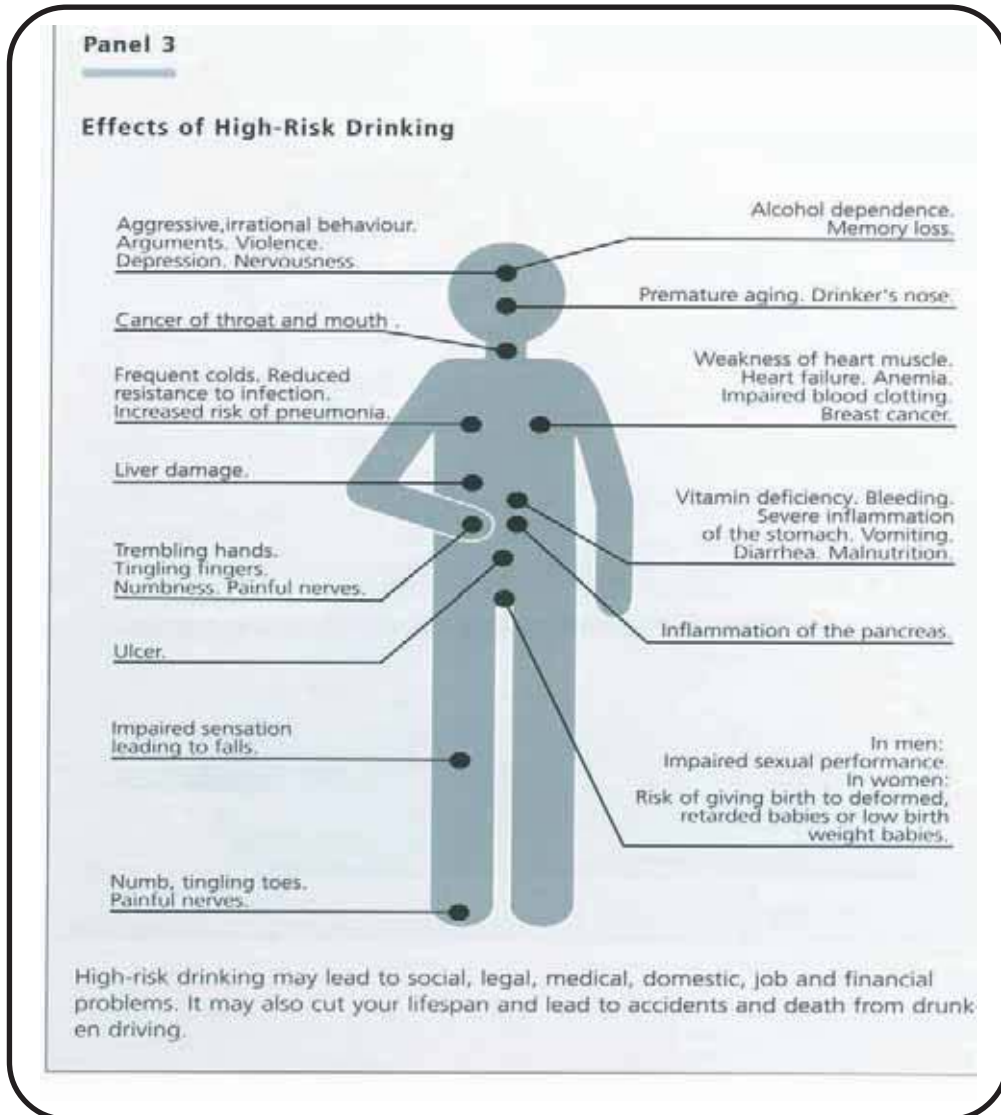
For appropriate matching of patient and intervention, consider:

- 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](http://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](http://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)
Alcohol Dependent (> 20)		
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><b>Goals of treatment</b> General awareness Reinforces/maintains low risk drinking Assists patients with problems, patients who have cut down, or whose circumstances may change</p>	<p><b>Goals of treatment</b> Assists those drinking at risky levels Encourage reduction of consumption to recommended limits e.g. 2 alcohol free days per week</p>	<p><b>Goals of treatment</b> Likely dependent Discuss importance/ relevance of abstinence Provide information Establish treatment goals</p>
<p><b>Strategies discussed and implemented</b> Gain greater understanding of 'trigger' situations Offer self-help booklet Offer follow up appointment to discuss progress, and use of booklet</p>	<p><b>Strategies discussed and implemented</b> Possible:  <ul style="list-style-type: none"> <li>Withdrawal management (detoxification)</li> <li>Pharmacotherapies PLUS supportive therapy</li> </ul>                     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><b>Strategies discussed and implemented</b> Consider:  <ul style="list-style-type: none"> <li>Withdrawal management (detoxification)</li> <li>Pharmacotherapies PLUS supportive therapy</li> </ul>                     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](http://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

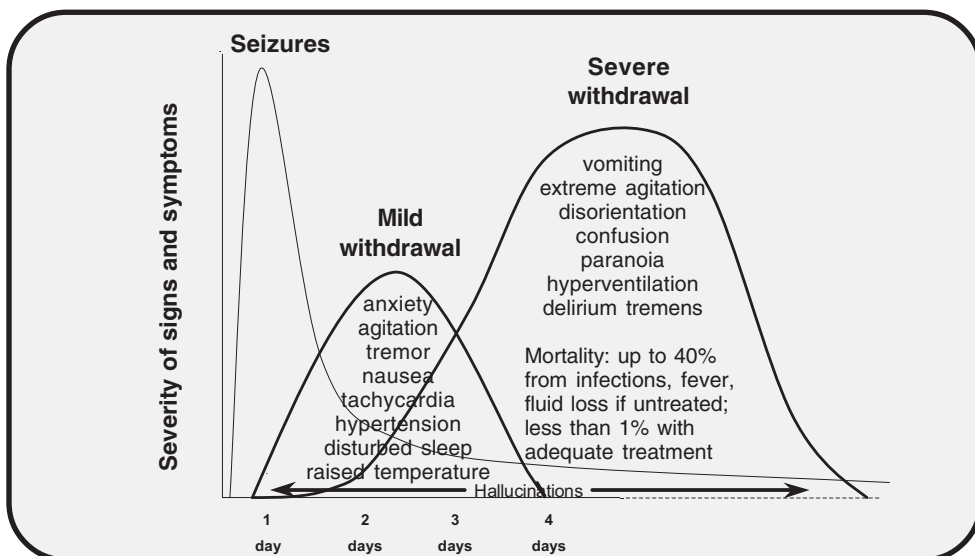


Figure 3–3  
Progress of alcohol withdrawal syndrome  
Source: NSW Health (2000, p. 41)



[www.health.nsw.gov.au](http://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**

<b>Acamprosate</b> [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)
<b>Disulfiram</b> [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, thiamine 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**

<b>Naltrexone [Revia®]</b>	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](http://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*, 2<sup>nd</sup> 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](http://www.health.gov.au/oatsih/pubs/pdf/rec.pdf)



[www.dasc.sa.gov.au](http://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](http://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](http://www.dasc.sa.gov.au)

MIMS Website:



[www.mims.hcn.net.au](http://www.mims.hcn.net.au)

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



[www.cme.net.au/phec](http://www.cme.net.au/phec)

Alliance of NSW Divisions of General Practice.



[www.answd.com.au](http://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](http://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](http://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](http://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* (2<sup>nd</sup> edn.) WHO, Department of Mental Health and Substance Dependence, Geneva, [www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS](http://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*, 2<sup>nd</sup> 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*, 2<sup>nd</sup> 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](http://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*, 2<sup>nd</sup> edn., Next Step Specialist Drug and Alcohol Services, Perth, Western Australia, [www.nextstep.health.wa.gov.au](http://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. & Pead, J. 1995, *Practice Guidelines for Health Professionals III Alcohol Withdrawal in the Hospital Setting*, University of Melbourne, Parkville, Victoria.

# Tobacco

**T**OBACCO 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

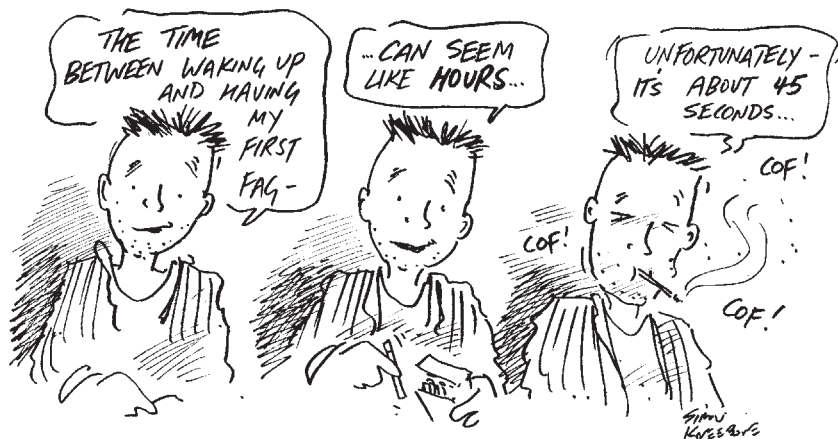
### **Psychological effects**

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**

<b>Time elapsed</b>	<b>Benefit</b>
20 minutes	<ul style="list-style-type: none"> <li>• blood pressure drops to normal</li> <li>• pulse rate drops to normal</li> <li>• temperature of hands and feet increase to normal.</li> </ul>
8 hours	<ul style="list-style-type: none"> <li>• carbon monoxide level in blood returns to normal</li> <li>• oxygen level in blood returns to normal.</li> </ul>
24 hours	<ul style="list-style-type: none"> <li>• the immediate risk of heart attack starts to fall.</li> </ul>
48 hours	<ul style="list-style-type: none"> <li>• nerve endings start to regrow</li> <li>• ability to taste and smell enhanced.</li> </ul>
14 days	<ul style="list-style-type: none"> <li>• circulation improves</li> <li>• walking becomes easier</li> <li>• lung function increases up to 30%.</li> </ul>
1 month	<ul style="list-style-type: none"> <li>• most nicotine withdrawal symptoms disappear.</li> </ul>
3 months	<ul style="list-style-type: none"> <li>• lung function improves</li> <li>• nagging cough disappears</li> <li>• cilia regrow in the lungs, increasing their ability to handle mucus, clean themselves and reduce infection.</li> </ul>
9 months	<ul style="list-style-type: none"> <li>• risk of pregnancy complications and foetal death reduced to level of non-smoker.</li> </ul>
1 year	<ul style="list-style-type: none"> <li>• 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.</li> </ul>
5 years	<ul style="list-style-type: none"> <li>• risk of lung cancer decreases by half</li> <li>• stroke risk same as non-smoker</li> <li>• risk of mouth, throat and oesophageal cancer half that of a smoker.</li> </ul>
10 years	<ul style="list-style-type: none"> <li>• lung cancer death rate same as non-smoker</li> <li>• pre-cancerous cells replaced.</li> </ul>
15 years	<ul style="list-style-type: none"> <li>• risk of coronary heart disease same as a non-smoker</li> <li>• if you smoked 20 day, you've saved \$49,275 (assuming \$9 per pack of 20).</li> </ul>

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](http://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"> <li>• Ischaemic heart disease</li> </ul> Absolute: <ul style="list-style-type: none"> <li>• Recent MI</li> <li>• Serious arrhythmias</li> <li>• Unstable angina</li> <li>• Pregnancy</li> </ul>
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](http://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](http://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](http://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](http://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](http://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](http://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/](http://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](http://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

**C**ANNABIS 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

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

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

## PHARMACOLOGY

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

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



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

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

- dopamine
- noradrenaline
- serotonin

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

- *CNS effects*: euphoria; increased 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"> <li>• inflammation, infection, scarring, or abscess at IV site</li> <li>• introduction of contaminants, which may result in thrombosis</li> <li>• increased risk of developing tolerance and dependence</li> <li>• acute intoxication risks from IV use such as psychosis, seizures, cardiovascular complications (incl. arrhythmias, cerebrovascular accident), hallucinations, accidents and injury.</li> </ul>
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"> <li>• overstimulation, insomnia dizziness, mild tremor</li> <li>• euphoria/dysphoria, restless, talkative, excited with need to speak</li> <li>• increased confidence, self-awareness</li> <li>• mild confusion, panic (rarely psychotic episodes)</li> <li>• appetite suppression</li> <li>• pupillary dilatation</li> <li>• increased energy, stamina and reduction in fatigue</li> <li>• heightened alertness and psychomotor activity with improved performance or concentration on simple fatigue impaired tasks</li> <li>• with increasing doses, may increase libido</li> <li>• headache</li> <li>• teeth grinding</li> </ul>	<ul style="list-style-type: none"> <li>• stereotypic or unpredictable behaviour</li> <li>• violent or irrational behaviour, mood swings, including hostility and aggression</li> <li>• pressured or slurred speech</li> <li>• paranoid thinking, confusion and perceptual disorders</li> <li>• headache, blurred vision, dizziness</li> <li>• psychosis (hallucinations, delusions, paranoia)</li> <li>• cerebrovascular accident*</li> <li>• seizures</li> <li>• coma</li> <li>• teeth grinding</li> <li>• gross body image distortions</li> </ul>
Cardiovascular	<ul style="list-style-type: none"> <li>• tachycardia (possibly brief bradycardia), hypertension</li> <li>• palpitations, arrhythmias</li> </ul>	<ul style="list-style-type: none"> <li>• cardiac stimulation (tachycardia, angina, arrhythmia*, MI)</li> <li>• vasoconstriction / hypertension</li> <li>• cardiovascular collapse*</li> </ul>
Respiratory	<ul style="list-style-type: none"> <li>• increased respiration rate and depth</li> </ul>	<ul style="list-style-type: none"> <li>• respiratory difficulty/failure*</li> </ul>
Gastrointestinal	<ul style="list-style-type: none"> <li>• nausea and vomiting</li> <li>• constipation, diarrhoea or abdominal cramps</li> </ul>	<ul style="list-style-type: none"> <li>• dry mouth</li> <li>• nausea and vomiting</li> <li>• abdominal cramps</li> </ul>
Skin	<ul style="list-style-type: none"> <li>• pale sweaty skin</li> <li>• hyperpyrexia</li> </ul>	<ul style="list-style-type: none"> <li>• flushing or pallor</li> <li>• hyperpyrexia, diaphoresis</li> </ul>
Skeletal	<ul style="list-style-type: none"> <li>• increased deep tendon reflexes</li> </ul>	
<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 (Peard 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),

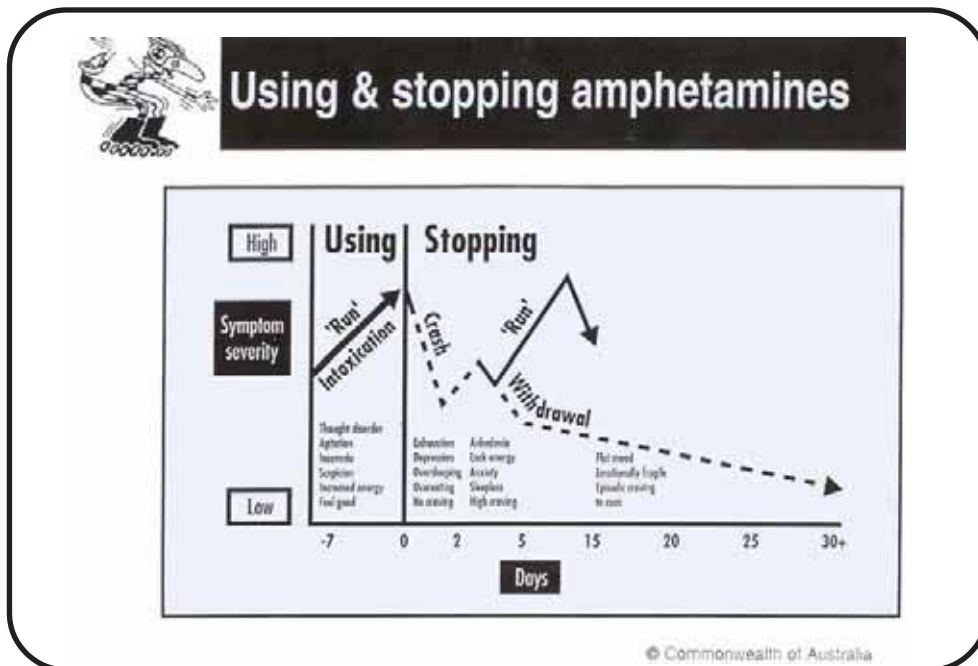


Figure 6–1  
Using and stopping amphetamines (Peard et al., 1996, p. 30)



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](http://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](http://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](http://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](http://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*, 2<sup>nd</sup> edn., Custodial Medical Unit, Mornington, Victoria.

Wickes, W. 1993, *Amphetamines and Other Psychostimulants: A Guide to the Management of Users*, AGPS, Canberra.

# Ecstasy

**E**CSTASY is the street name generally applied to 3,4-methylenedioxymethamphetamine or MDMA. However, other drugs are sold as ecstasy, and ecstasy tablets often contain a range of drugs (including amphetamine, various amphetamine derivatives, caffeine, aspirin, paracetamol, or ketamine) in addition to, or in place of MDMA (Wolff et al., 1995).

Ecstasy is usually sold as a tablet or capsule. The tablets are typically identified by a symbol impressed on the surface. This leads users to refer to them as 'white doves', 'love hearts', etc. Other common street names are 'E', 'Eccy', 'Adam' and 'XTC'.

## PHARMACOLOGY

MDMA initially enhances the extracellular brain concentrations of serotonin but eventually serotonin becomes depleted. MDMA also induces a rapid and substantial elevation of dopamine. Serotonin has a role in regulation of aggression, mood, sexual activity, sleep, sensitivity to pain, memory and body temperature (Schloss & Williams, 1998). Dopamine plays a role in the control of movement, cognition, motivation and reward (Rawson, 1999). It is probably the mechanism underlying the stimulant properties of MDMA (Daws et al., 2000).



MDMA is well absorbed from the gastrointestinal tract (Mas et al., 1999). Effects become apparent about 20 minutes after administration and last about 4 hours. Dose and blood concentration relationship may not be linear (de la Torre et al., 2000), and small increases in dose may produce disproportionate increases in effect, possibly contributing to toxicity. Some of the metabolic products of MDMA are themselves bioactive and may also contribute to toxicity (Mas et al., 1999).

MDMA is metabolised in the liver. Some people have low activity of CYP2D6, one of the enzymes involved (Tucker et al., 1994). It has been suggested (but not validated) that, due to reduced metabolism, these individuals are at greater risk of MDMA toxicity (O'Donohoe et al., 1998; Schwab et al., 1999).

Drug interactions may influence MDMA toxicity by altering elimination of MDMA from the body, or through an additive effect. Reported cases of adverse reactions involving ecstasy in combination with fluoxetine (Bingham et al., 1998; Coore, 1996) and ritonavir (Henry & Hill, 1998) support this as a possibility.

## PATTERNS OF USE

In the 2001 Australian National Drug Strategy Household Survey, lifetime use of ecstasy or other designer drugs was reported by 6.1% of people aged 14 and over, while 2.9% reported using ecstasy in the previous 12 months (AIHW, 2002). The current trend is one of increasing prevalence of use.

Ecstasy is almost exclusively taken in a social setting (McKetin et al., 1999; Topp et al., 1997b) usually as part of youth culture centred on dance music. Use of ecstasy by friends is a significant factor in initiation and continuation of ecstasy use.

The quantity of active ingredient in one tablet is usually in the range 75–100 mg. Normally one or two tablets are taken at a time but there are reports of greater doses being used, especially by experienced users (Topp et al., 1997b).

Ecstasy is mainly taken orally, but there may be a trend of increasing use by injection (Humeniuk, 2000; Topp et al., 1997b). Most users appear able to regulate their use of ecstasy but some progress to problematic use (Topp et al., 1997b). Whether such problematic use constitutes dependence is an area of debate (Jansen, 1999; Topp et al., 1997a).

## PHYSICAL AND PSYCHOSOCIAL COMPLICATIONS

MDMA produces immediate positive psychological effects of euphoria, increased energy, and a feeling of closeness to others, and (less commonly) negative psychological effects of paranoia, anxiety and depression.

### Physical Effects of Ecstasy

The incidence of serious acute adverse events arising from ecstasy use is low. It is the unpredictable nature of those adverse events and the risk of mortality and substantial morbidity in young people that make the health consequences of ecstasy significant.

Table 7–1 lists the short- and long-term physical effects of ecstasy.

#### *Hyperthermia*

The most significant adverse effect of ecstasy use is hyperthermia. It can quickly become life threatening. The degree of hyperthermia is predictive of mortality.

It is typically accompanied by a number of clinical problems, including:

- seizures
- disseminated intravascular coagulation
- rhabdomyolysis
- renal and liver impairment which may be induced or exacerbated by the hyperthermia (Green et al., 1995)

Clinical signs and symptoms are consistent with malfunction of normal temperature control and water balance. MDMA can produce hyperthermia in quiet surroundings, but in the setting of 'raves' or dance parties, toxicity appears to be enhanced. It is probably a combination of:

- the direct effects of MDMA
- high ambient temperature
- sustained physical activity; and
- inadequate fluid replacement

All impair temperature regulation (Green et al., 1995; Henry et al., 1992).

### **Hyponatraemia ('water intoxication')**

Ecstasy use has also been associated with hyponatraemia. Cases are marked by:

- features of confusion
- reduced consciousness; and
- in some cases, seizures or convulsions

In general symptoms resolve as sodium levels are normalised, with full recovery achieved within a few days. However, fatalities have been reported, apparently due to cerebral oedema associated with excess fluid.

In most cases of hyponatraemia, copious amounts of water were consumed. This may be a response to a sensation of thirst induced by MDMA. Alternatively, behavioural disturbance, including stereotyped repetitive actions such as water consumption, may arise from MDMA ingestion (White et al., 1997). The administration of MDMA is associated with inappropriate release of anti-diuretic hormone, arginine vasopressin (Henry et al., 1998). This would reduce

**Table 7-1**  
Physical effects of ecstasy

Short-term effects	Long-term effects
<ul style="list-style-type: none"> <li>• pupil dilation</li> <li>• increased jaw tension and grinding of teeth</li> <li>• loss of appetite</li> <li>• dry mouth</li> <li>• tachycardia</li> <li>• hot and cold flushes</li> <li>• sweaty palms</li> <li>• hyperthermia</li> <li>• hyponatraemia or 'water intoxication'</li> </ul>	<ul style="list-style-type: none"> <li>• insomnia</li> <li>• depression</li> <li>• headaches</li> <li>• muscle stiffness</li> </ul>

urine formation and the body's capacity to excrete excess fluid.

First reports of hyponatraemia occurred after dance club owners encouraged users to take dance breaks in a cool room and drink water. This advice is still sound for prevention of hyperthermia, but:

- an upper limit of 500 ml per hour is considered the amount able to be handled by the body

Drug screening undertaken in cases of acute adverse effects commonly indicate the presence of a range of drugs in addition to MDMA. However, the reporting of cases of hyperthermia or disturbances of salt or water balance where MDMA was the only drug detected, demonstrate that MDMA alone can produce adverse effects. Given that hyperthermia and disturbances of salt or water balance generally occur when MDMA is used in nightclub or dance party settings, these data also suggest that the acute adverse effects of MDMA arise primarily from the way it is used.

### ***Dose–response relationship***

The dose of MDMA is not predictive of severity of outcome (Gowing et al., 2002). In the absence of a dose–response relationship, it has been suggested that some form of metabolic myopathy or individual variability in metabolism of MDMA may underlie adverse effects. However, instances of muscle abnormality or impaired MDMA metabolism have not been identified in any cases of severe reactions and there appears to be a mix of first time and experienced MDMA users affected, making this explanation unlikely, or at least uncommon.

Severe reactions might be due to contaminants in the preparation taken. However, reports of affected persons taking from the same supply as others, who did not experience severe reactions, means that contaminants are an unlikely explanation (Hall, 1997). The combination of

dose, setting and individual behaviour most likely determines outcome.

### ***Liver damage***

Severe liver damage can occur shortly after ingestion of ecstasy, typically in conjunction with hyperthermia. However, liver damage, apparently unrelated to hyperthermia, can also occur days or weeks after single or multiple episodes of ecstasy use (Jones & Simpson, 1999). Most reported cases resolved spontaneously over weeks to months, but a minority progressed to full liver failure requiring transplantation, with some cases being fatal.

It appears that those who resume ecstasy use after recovery are at risk of recurrence of liver damage and development of chronic hepatitis (Andreu et al., 1998). The mechanism of ecstasy-related liver damage is uncertain and, relative to other causes, ecstasy use remains a minor contributor to the incidence of liver failure (Andreu et al., 1998; Jones & Simpson, 1999).

### ***Neurotoxicity***

Animal studies show administration of MDMA produces damage to serotonin axons in the brain (McCann et al., 2000). Brain imaging techniques have found persisting abnormalities in brain morphology in ex-users of ecstasy, even with moderate use (Gamma et al., 2000; Kish et al., 2000; Reneman et al., 2000). Psychological tests in current and former ecstasy users compared to non-using controls have consistently found impairment in short-term memory function in ecstasy users (Gouzoulis-Mayfrank et al., 2000; Parrott et al., 2000; Rodgers 2000; Wareing et al., 2000).

These studies constitute mounting evidence of ecstasy having a neurotoxic effect.

## Psychological Effects and Complications

Depression, or low mood, and concentration and/or memory problems are commonly reported in the week following ecstasy use (Curran, 2000). Cases of persistent depression, panic disorders, 'flashbacks' and delusions have been related to ecstasy use (Benazzi & Mazzoli, 1991; Cohen & Cocores, 1997).

The risk of psychiatric sequelae is probably greater when:

- other drugs, particularly cannabis, are used in addition to ecstasy
- ecstasy is used repeatedly and at high doses over a period of months
- there is a family or personal history of psychiatric disorders (Schifano et al., 1998)

## MANAGEMENT AND INTERVENTION STRATEGIES

### Strategies for Different Levels of Use

#### *Acute adverse effects*

Reassurance, observation and monitoring for several hours in a subdued environment until symptoms subside, is appropriate in most ecstasy intoxication cases (Williams et al., 1998).

Hyperthermia and hyponatraemia are the most significant complications necessitating intervention. In both conditions the treatment response needs to be rapid and intense to avert significant morbidity and mortality. In the case of hyperthermia, the patient may deteriorate rapidly towards multiple organ failure, requiring intensive support of cardiovascular, respiratory and renal systems (Hall, 1997). This requires admission to an intensive care unit.

Many cases of ecstasy-induced liver damage will resolve without intervention, and simply require monitoring. However, patients developing jaundice, or with evidence of hepatic failure, require specialist care.

It is also important to educate users about the importance of controlling body temperature and fluid intake, early signs of adverse effects, and the need to seek medical assistance promptly.

### Treatment for Ecstasy Use

Those who use ecstasy more frequently (monthly to weekly) and/or use larger amounts, and those who use by injection are likely to be at increased risk of harm and hence constitute targets for intervention.

#### *Pharmacological interventions*

There is currently very little information on pharmacological interventions for ecstasy users. Selective serotonin reuptake inhibitors (SSRIs), if taken concurrently with MDMA, have been shown to block usual subjective effects of MDMA (Stein & Rink, 1999). However, administration of SSRIs (e.g. fluoxetine, citalopram) subsequent to MDMA may potentiate the effects of released serotonin, worsening any adverse effects (Green et al., 1995) and limiting their value as a treatment agent.

#### *Non-pharmacological interventions*



See Chapter 13  
Psychosocial Interventions

Non-pharmacological interventions (also see Chapter 13) which have demonstrated most efficacy in treating psychostimulant users are:

- relapse prevention
- cue exposure/response prevention
- multifaceted behavioural therapy

Contingency management approaches may also be of value.

Attracting users into treatment and intervening prior to development of problematic use is a priority. An approach well suited to these purposes is early and brief intervention (Barry, 1999), administered opportunistically when possible ecstasy use is identified.

## 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.
- Andreu, V., Mas, A., Bruguera, M. et al. 1998, 'Ecstasy: a common cause of severe acute hepatotoxicity', *Journal of Hepatology*, vol. 29, no. 3, pp. 394–397.
- Barry, K.L. 1999, *Brief Interventions and Brief Therapies for Substance Abuse*, Treatment Improvement Protocol (TIP) Series No. 34, US Department of Health and Human Services, Rockville, Maryland.
- Benazzi, F. & Mazzoli, M. 1991, 'Psychiatric illness associated with "ecstasy"', *Lancet*, vol. 338, no. 1520.
- Bingham, C., Beaman, M., Nicholls, A.J. & Anthony, P.P. 1998, 'Necrotizing renal vasculopathy resulting in chronic renal failure after ingestion of methamphetamine and 3,4-methylenedioxymethamphetamine ('ecstasy')', *Nephrology Dialysis Transplant*, vol. 13, no. 10, pp. 2654–2655.
- Cohen, R.S. & Cocores, J. 1997, 'Neuropsychiatric manifestations following the use of 3,4-methylenedioxymethamphetamine (MDMA: "Ecstasy")', *Progress in Neuropsychopharmacol Biological Psychiatry*, vol. 21, no. 4, pp. 727–734.
- Coore, J.R. 1996, 'A fatal trip with ecstasy: a case of 3,4-methylenedioxymethamphetamine/3,4-methylenedioxymphetamine toxicity', *J R Soc Med*, vol. 89, no. 1, 51P–52P.
- Curran, H.V. 2000, 'Is MDMA ('Ecstasy') neurotoxic in humans? An overview of evidence and of methodological problems in research', *Neuropsychobiology*, vol. 42, no.1, pp. 34–41.
- Daws, L., Irvine, R.J., Callaghan, P.D., Toop, P.N., White, J.M. & Bochner, F. 2000, 'Differential behavioural and neurochemical effects of para-methoxyamphetamine and 3,4-methylenedioxymethamphetamine in the rat', *Progress in Neuropsychopharmacol Biological Psychiatry*, vol. 24, pp. 955–977.
- de la Torre, R., Farre, M., Ortuno, J. et al. 2000, 'Non-linear pharmacokinetics of MDMA ('ecstasy') in humans', *British Journal of Clinical Pharmacology*, vol. 49, no. 2, pp. 104–109.
- Gamma, A., Frei, E., Lehmann, D., Pascual-Marqui, R.D., Hell, D. & Vollenweider, F.X. 2000, 'Mood state and brain electric activity in ecstasy users', *Neuroreport*, vol. 11, no. 1, pp. 157–162.
- Gouzoulis-Mayfrank, E., Daumann, J., Tuchtenhagen, F., et al. 2000, 'Impaired cognitive performance in drug free users of recreational ecstasy (MDMA)', *Journal of Neurosurgery & Psychiatry*, vol. 68, no. 6, pp. 719–725.

- Gowing, L.R., Henry-Edwards, S.M., Irvine, R.J. & Ali, R.L. 2002, 'The health effects of "ecstasy": a literature review', *Drug & Alcohol Review*, vol. 21, no. 1, pp. 53–63.
- Green, A.R., Cross, A.J. & Goodwin, G.M. 1995, 'Review of the pharmacology and clinical pharmacology of 3,4-methylenedioxymethamphetamine (MDMA or 'Ecstasy')', *Psychopharmacology*, vol. 119, pp. 247–260.
- Hall, A.P. 1997, '“Ecstasy” and the anaesthetist', *British Journal of Anaesthesia*, vol. 79, no. 6, pp. 697–698.
- Henry, J.A., Fallon, J.K., Kicman, A.T., Hutt, A.J., Cowan, D.A. & Forsling, M. 1998, 'Low-dose MDMA ("ecstasy") induces vasopressin secretion', *Lancet*, vol. 351, no. 9118, p. 1784.
- Henry, J.A. & Hill, I.R. 1998, 'Fatal interaction between ritonavir and MDMA', *Lancet*, vol. 352, no. 9142, pp. 1751–1752.
- Henry, J.A., Jeffreys, K.J. & Dawling, S. 1992, 'Toxicity and deaths from 3,4-methylenedioxymethamphetamine ('ecstasy')', *Lancet*, vol. 340, pp. 384–387.
- Humeniuk, R. 2000, *South Australian Drug Trends 1999. Findings from the Illicit Drug Reporting System*, NDARC Technical Report No. 88, National Drug and Alcohol Research Centre, Sydney.
- Jansen, K.L. 1999, 'Ecstasy (MDMA) dependence', *Drug & Alcohol Dependence*, vol. 53, no. 2, pp. 121–124.
- Jones, A.L. & Simpson, K.J. 1999, 'Review article: mechanisms and management of hepatotoxicity in ecstasy (MDMA) and amphetamine intoxications', *Alimentary Pharmacology & Therapeutics*, vol. 13, no. 2, pp. 129–133.
- Kish, S.J., Furukawa, Y., Ang, L., Vorce, S.P. & Kalasinsky, K.S. 2000, 'Striatal serotonin is depleted in brain of a human MDMA (Ecstasy) user', *Neurology*, vol. 55, no. 2, pp. 294–296.
- McCann, U.D., Eligulashvili, V. & Ricaurte, G.A. 2000, '(+/-)3,4-Methylenedioxymethamphetamine ('Ecstasy')-induced serotonin neurotoxicity: clinical studies', *Neuropsychobiology*, vol. 42, no. 1, pp. 11–16.
- McKetin, R., Darke, S., Hayes, A. & Rumbold, G. 1999, *Drug Trends 1998. A Comparison of Drug Use and Trends in Three Australian States: Findings from the Illicit Drug Reporting System (IDRS)*, NDARC Monograph No. 41, National Drug and Alcohol Research Centre, Sydney.
- Mas, M., Farre, M., de la Torre, R. et al. 1999, 'Cardiovascular and neuroendocrine effects and pharmacokinetics of 3,4-methylenedioxymethamphetamine in humans', *Journal of Pharmacology & Experimental Therapeutics*, vol. 290, no. 1, pp. 136–145.
- O'Donohoe, A., O'Flynn, K., Shields, K., Hawi, Z. & Gill, M. 1998, 'MDMA toxicity: no evidence for a major influence of metabolic genotype at CYP2D6', *Addiction Biology*, vol. 3, pp. 309–314.

- Parrott, A.C., Sisk, E. & Turner, J.J. 2000, 'Psychobiological problems in heavy 'ecstasy' (MDMA) polydrug users', *Drug & Alcohol Dependence*, vol. 60, no. 1, pp. 105–110.
- Rawson, R.A. 1999, *Treatment for Stimulant Use Disorders*, Treatment Improvement Protocol (TIP) Series No. 33, Department of Health and Human Services, Rockville, Maryland, USA.
- Reneman, L., Booij, J., Schmand, B., van den Brink, W. & Gunning, B. 2000, 'Memory disturbances in "Ecstasy" users are correlated with an altered brain serotonin neurotransmission', *Psychopharmacology (Berl)*, vol. 148, no. 3, pp. 322–324.
- Rodgers, J. 2000, 'Cognitive performance amongst recreational users of "ecstasy" ', *Psychopharmacology (Berl)*, vol.151, no. 1, pp. 19–24.
- Schifano, F., Di Furia, L., Forza, G., Minicuci, N. & Bricolo, R. 1998 'MDMA ('ecstasy') consumption in the context of polydrug abuse: a report on 150 patients', *Drug & Alcohol Dependence*, vol. 52, no. 1, pp. 85–90.
- Schloss, P. & Williams, D.C. 1998, 'The serotonin transporter: a primary target for antidepressant drugs', *Journal of Psychopharmacology*, vol. 12, no.2, pp. 115–121.
- Schwab, M., Seyringer, E., Brauer, R.B., Hellinger, A. & Griese, E.U. 1999, 'Fatal MDMA intoxication', *Lancet*, vol. 353, no. 9152, pp. 593–594.
- Stein, D. J. & Rink, J. 1999, 'Effects of "Ecstasy" blocked by serotonin reuptake inhibitors', *Journal of Clinical Psychiatry*, vol. 60, no. 7, p. 485.
- Topp, L., Hall, W. & Hando, J. 1997a, *Is There a Dependence Syndrome for Ecstasy?* NDARC Technical Report No. 51, National Drug and Alcohol Research Centre, Sydney.
- Topp, L., Hando, J., Degenhardt, L., Dillon, P., Roche, A. & Solowij, N. 1997b, *Ecstasy Use in Australia*, NDARC Monograph No. 39, National Drug and Alcohol Research Centre, Sydney.
- Tucker, G.T., Lennard, M.S., Ellis, S.W. et al. 1994, 'The demethylation of methylenedioxymethamphetamine ("ecstasy") by debrisoquine hydroxylase (CYP2D6)', *Biochemical Pharmacology*, vol.47, no. 7, pp. 1151–1156.
- Wareing, M., Fisk, J.E. & Murphy, P.N. 2000, 'Working memory deficits in current and previous users of MDMA ('ecstasy')', *British Journal of Psychology*, vol. 91, no. 2, pp. 181–188.
- White, J. M., Bochner, F. & Irvine, R. J. 1997, 'The agony of "ecstasy" ', *Medical Journal of Australia*, vol. 166, no. 3, pp. 117–118.
- Williams, H., Dratcu, L., Taylor, R., Roberts, M. & Oyefeso, A. 1998, ' "Saturday night fever": ecstasy related problems in a London accident and emergency department', *Journal of Accident & Emergency Medicine*, vol. 15, no. 5, pp. 322–326.
- Wolff, K., Hay, A.W., Sherlock, K. & Conner, M. 1995, 'Contents of "ecstasy" ', *Lancet*, vol. 346, no. 8982, pp. 1100–1101.



# Ecstasy

# Cocaine

**C**OCAINE is a stimulant derived from the South American coca plant. It is imported in the form of a salt, cocaine hydrochloride, a white odourless crystalline powder with a bitter taste. Cocaine base can be extracted from the powder to form rocks or crystals known as 'freebase' or 'crack' that are smoked and produce strong subjective effects almost immediately.

Although it is relatively easy to make crack from cocaine hydrochloride, and some Australian cocaine users report doing this, there is little evidence of widespread or problematic crack cocaine smoking in Australia to date.

## PHARMACOLOGY

Cocaine blocks the reuptake of dopamine (DA), noradrenaline and serotonin at presynaptic locations, thus increasing the concentration of these transmitters at postsynaptic receptor sites (Chesher, 1993). DA concentration is particularly increased, and is thought to be the basis for cocaine's abuse potential. Cocaine also stimulates the sympathetic nervous system, which accounts for its activating effects.

Tolerance to the acute effects develops extremely rapidly, before the depletion of plasma levels. Most of the active drug is metabolised in the liver, but some is acted on by plasma esterases, and a small amount is excreted unchanged in the urine (Schuckit, 1995). Cocaine metabolites may be detected in urine for three days or longer following use.

## Australian Street Names

Most common:

- cocaine
- coke
- charlie

Less common:

- okey doke
- nose candy
- toot
- blow
- snow
- white lady

## PREVALENCE AND PATTERNS OF USE



Darke et al., 2000

### Lifetime

The proportion of the Australian population who reported having used cocaine at some time increased from 2.5% in 1993 to 4.4% in 2001 (AIHW, 2002).

### Past Year

The proportion who had used cocaine in the past year increased from 0.5% in 1993 to 1.3% in 2001 (AIHW, 2002).

### Gender

In 1998, males were more likely than females to report lifetime (5.3% versus 3.3%) and past year (1.9% versus 0.9%) cocaine use.

### Age

Cocaine use is most common amongst young people. In 1998, 8% of Australians aged

20–29 years reported having used cocaine in their lifetime, and 3% in the past year.

## AVAILABILITY

Since the late 1990s there has been a marked increase in cocaine availability and use, especially in Sydney (Darke et al., 2002a). Although cocaine is available in other jurisdictions it is harder to get and more expensive than in Sydney (Topp et al., 2002).

The late 1990s saw an increase in use in Sydney. It was most apparent amongst committed heroin injectors, who administered the two drugs simultaneously in a 'speedball' or 'CC' (cocaine cocktail) or sequentially. In 2001, when the availability and use of heroin decreased substantially, the frequency of injection of cocaine amongst former primary heroin users in Sydney increased markedly (Darke et al., 2002b).

## ROUTES OF ADMINISTRATION

In Australia, cocaine is generally administered intranasally (snorted) or intravenously (injected). Onset of action is rapid via either route of administration: within eight minutes when snorted and within two minutes when injected. Peak blood levels develop within five to 30 minutes. Duration of action is relatively brief: the half-life of cocaine's active metabolites is typically 15 to 30 minutes when the drug is injected and 60 minutes when snorted (Chesher, 1993; Platt, 1997).

Those who inject cocaine tend to have a higher quantity and frequency of use, and experience more associated harm, than those who snort it (Kaye et al., 2000).

## BINGEING

A substantial proportion of those who use cocaine heavily do so in 'binges', i.e. where it is administered at short intervals repeatedly until either the supply or the user is exhausted. This destructive pattern of use appears to arise because tolerance to the rewarding effects of cocaine develops extremely quickly, as a result of rapid neuroadaptation (i.e., where the neurons on which cocaine exerts its effects attempt to restore normal function) (Chesher, 1993). Thus, the intense pleasure experienced after cocaine injection is of only short duration and is followed by either an absence of euphoria or even a dysphoria. Such rapid mood changes seem to stimulate the need for more cocaine.

## TYPES OF USERS

Most Australians who use cocaine snort small amounts infrequently and with few problems.

People who use cocaine more heavily tend to fall into two groups:

- middle-class, well-educated professionals who generally snort the drug; and
- injecting drug users who inject cocaine, often (but not always) in association with heroin

## POLYDRUG USE

Cocaine users tend to be extensive polydrug users; other drugs are used both in conjunction with cocaine as well as to medicate the 'come down' (aversive recovery period following use).

## Snorters

Those who snort cocaine tend to do so in a social context such as at dance parties, and tend to use other party drugs such as ecstasy, methamphetamine, ketamine, and/or GHB, as well as alcohol and cannabis, and may use benzodiazepines to come down (Topp et al., 2000). A small proportion of this group progress to problematic cocaine use, in which large amounts of the drug are snorted frequently.

## Injectors

Those who inject cocaine tend to be either:

- former heavy cocaine snorters who have developed nasal problems and/or a high level of tolerance and so make the transition to injecting; or
- committed injecting drug users who have added cocaine to their injection repertoire, and will usually inject other drugs including heroin and methamphetamine, and use a wide range of other drugs including alcohol, cannabis, benzodiazepines and methadone (Kaye et al., 2001)

It is estimated that the latter group constitutes a higher proportion of cocaine injectors than the former.

## Cocaine and Alcohol

- a common pattern of polydrug use
- with repeated administration, alcohol sensitises the body's reaction to cocaine, and cocaine attenuates the development of tolerance to alcohol
- the combination produces a third active substance, cocaethylene (Schuckit, 1995), which has a half-life of two hours (as opposed to about 30 minutes for cocaine alone)
- concurrent use of cocaine and alcohol leads to a significantly elevated risk for sudden cardiac deaths

## EFFECTS OF COCAINE

### Factors Influencing the Effects

- form (powder versus crack)
- dose (influenced by purity as well as quantity)
- route of administration
- intensity and duration of use
- concurrent polydrug use

### Desired Effects

- euphoria
- sociability, gregariousness and talkativeness
- increased confidence and feelings of control
- energy
- decreased need for sleep
- temporary increase in functional activity or efficiency
- suppressed appetite

### Other Acute Effects of Low Doses

- local anaesthesia
- pupillary dilation
- vasoconstriction
- increased respiration
- increased heart rate
- increased blood pressure
- increased body temperature

## Acute Effects of High Doses ('Toxic Reactions')



See p. 111  
'Treatment of Toxic Reactions'

Toxic reactions are cocaine 'overdoses'. They occur after excessive doses. Any of the following signs and symptoms may be expected as part of a toxic reaction to cocaine:

- stereotyped, repetitive behaviour
- anxiety/severe agitation/panic
- aggression/hostility
- muscle twitches/tremors/loss of coordination
- heightened reflexes
- respiratory failure
- markedly elevated blood pressure
- chest pain/angina
- pulmonary oedema
- acute renal failure
- convulsions
- blurred vision
- acute stroke
- pallor
- confusion/delirium
- hallucinations, most often auditory or tactile, e.g. formication (the feeling of bugs crawling under the skin)
- dizziness
- muscle rigidity
- weak, rapid pulse
- cardiac arrhythmias including malignant arrhythmias
- myocardial ischaemia and infarction
- sweating/very high body temperature (up to 41°C rectally)
- headache
- stomach pain/nausea/vomiting

## Effects of Chronic Use

- insomnia
- depression
- aggression or violence
- loss of appetite and concomitant weight loss
- muscle twitching
- anxiety
- psychosis — paranoid delusions, hallucinations
- loss of libido and/or impotence
- heightened reflexes
- increased pulse rate

## PHYSICAL AND PSYCHOSOCIAL COMPLICATIONS

### Physical Problems Relating to Route of Administration

#### *Intranasal users*

Intranasal users may suffer from:

- runny nose
- blood nose
- nasal ulcers
- sinusitis
- epistaxis
- perforated nasal septum
- slight risk of hepatitis C transmission due to sharing of straws or other equipment used to snort cocaine which may contain traces of blood

#### *Injecting users*

Injecting users may suffer from:

- systemic and local infections which may be viral, bacterial, fungal or parasitic
- local inflammatory and infection complications can be more common than with

heroin due to the vasoconstrictive and anaesthetic properties of cocaine

- injection-related abscesses, cellulitis, phlebitis
- bacterial endocarditis
- transmission of blood borne viral infection such as hepatitis C, hepatitis B and HIV

### Other Physical Problems

Other physical problems may be experienced regardless of route of administration, particularly cardiovascular complications.

### Cocaine-related Death

Death is relatively rare, but is associated with:

- muscle rigidity
- delirium
- agitation
- a stroke-like CNS vascular picture
- cardiac arrhythmias
- elevated body temperature

### Psychological Problems

Psychological complications are the most common cocaine-related problems. See 'Acute Effects of High Doses' and 'Effects of Chronic Use' above.

### Social Problems

Intensity of cocaine use may incur:

- interpersonal problems
  - heightened discord in significant relationships
  - paranoia leading to irrational jealousy
  - alienation from social support networks
- occupational problems
  - impaired productivity
  - absenteeism
  - job loss

- financial problems
  - cocaine is expensive and use can escalate rapidly
  - debt to dealers and/or others may grow
  - dealing or other criminal activity may appear a viable financial option
  - financial problems may be compounded by job loss
- legal problems
  - may be directly drug-related
  - may be the result of criminal activity designed to support use

### Cocaine Dependence

Key criteria for diagnosing drug dependence are:

- continued use of a drug despite knowing that it causes significant harm
- loss of control over use manifest by using more or for longer than intended
- repeated relapse despite resolving to reduce or eliminate use

(APA, 1994)

Some cocaine users clearly develop such symptoms of dependence, along with others including tolerance.



### Cocaine Withdrawal Syndrome

The existence of cocaine withdrawal is contentious because the syndrome is dominated by symptoms rather than clinical signs. The aversive nature of the experience for users, and the strong motivation to resume use to alleviate withdrawal, however, is well documented.

DSM-IV (APA, 1994) describes withdrawal after several days of heavy cocaine use as consisting of:

- dysphoric mood (anhedonia or sadness rather than depression) and at least two of the following symptoms:
  - fatigue
  - insomnia or hypersomnia
  - psychomotor agitation or retardation
  - craving
  - increased appetite
  - vivid, unpleasant dreams
- withdrawal reaches its peak in 2–4 days
- dysphoric symptoms persist for up to 10 weeks (Lago & Kosten, 1994). Some suggest that cocaine craving and a desire to resume use may persist indefinitely, even after withdrawal is complete and normal mood and the ability to enjoy experiences have returned (Gawin & Kleber, 1986)

### Foetal Effects

Exposure to cocaine during pregnancy has been associated with:

- shorter gestation
- premature delivery
- abruption of placenta
- retardation of growth
- behavioural problems

However, many of the perinatal and postnatal adverse effects commonly attributed to cocaine may be caused by multiple confounders

that can occur in a cocaine using mother, rather than by cocaine itself (Addis et al., 2001), such as:

- polydrug use, including alcohol and cigarettes
- poor prenatal care
- single motherhood
- poverty
- poor quality postnatal environment

## MANAGEMENT AND INTERVENTION STRATEGIES

### Clinical Screening

The most common clinical problems associated with cocaine use are anxiety conditions, temporary psychosis and cardiovascular problems.

#### *Acute toxic reactions*

Possible cocaine use should be considered in individuals who manifest:

- dilated pupils
- dry mouth
- increased reflexes
- elevated temperature
- sweating
- increased heart rate
- a restless, hyperalert state
- an anxiety-like attack (usually nervousness plus rapid pulse)
- emotional lability or irritability
- aggressive or violent outbursts
- paranoia or suspiciousness
- hallucinations, especially auditory or tactile
- confusion or an organic brain syndrome
- behavioural abnormalities
- acute ischaemic events

If the clinician suspects cocaine use, blood or urine toxicological analysis will confirm the diagnosis.

### Chronic Cocaine Use

Chronic cocaine users who do not disclose their use may manifest:

- depression/anxiety
- suicidal ideation
- paranoia/hallucinations
- lethargy
- insomnia
- loss of libido
- social problems (as outlined above)
- evidence of IV drug use (track marks, abscesses)
- abnormalities in the nasal lining or mucosa
- worn teeth (from tooth grinding during intoxication)
- missed appointments and other signs of chaotic lifestyle
- seeking of medications such as benzodiazepines, antidepressants or opioids to relieve withdrawal, medicate side effects or to sell for profit

### Treatment of Toxic Reactions

The treatment chosen will depend on the condition of the patient at the time of presentation. Priorities are:

- emergency care to ensure a clear airway, circulatory stability and treatment of shock
- control of elevated body temperature with hydration, sedation, cold water, ice packs or in extreme cases, a hypothermic blanket
- control of seizures with doses of IV diazepam of 5 to 20 mg injected very slowly and repeated as required
- diazepam will also reduce agitation
- vigorous treatment of a sustained elevation in blood pressure with pentalomine (5–10 mg IV) to prevent CNS haemorrhage



- CT scans and lumbar puncture in the confused or unconscious patient will rule out the possibility of cerebral haemorrhage
- excretion of cocaine can be hastened through acidification of the urine with 500 mg ammonium chloride orally every 3–4 hours. The goal is a urinary pH under 6.6
- low doses of an antipsychotic such as haloperidol may be required to manage psychotic patients when benzodiazepines are insufficient. Such patients should be closely monitored as haloperidol can reduce the seizure threshold and may increase the risk of seizures (Nathan et al., 1998)
- once the patients start to recover, they should be reassured and comforted, preferably by supportive friends or relatives, and placed in a quiet room with minimal stimulation to be closely monitored
- if the patient is markedly despondent, (temporary) suicide precautions may be necessary
- severe and persistent depression may require antidepressants. Antidepressants are not effective in reducing cocaine use itself, but can be effective in the management of major depressive episodes associated with cocaine use
- care should be taken in prescribing SSRIs if cocaine use is continued, as toxic interactions have been described (Barrett et al., 1996), and, in mice, SSRIs have facilitated cocaine-induced convulsions (O'Dell et al., 2000)

## Treatment of Cocaine Dependence

### Pharmacotherapy

There is no widely effective pharmacotherapy for cocaine dependence:

- disulfiram as an adjunct to buprenorphine or methadone maintenance may reduce cocaine use in opioid-dependent clients (George et al., 2000; Petrakis et al., 2000)
- however, there is a potential interaction between disulfiram and cocaine that increases cocaine associated cardiovascular responses and consequently may increase cocaine toxicity (McCance-Katz et al., 1998)

Behavioural and psychosocial therapies have produced better results.

### Cognitive-behavioural therapy

- aims to reduce cocaine use by helping the client master an individualised set of coping strategies as effective alternatives to cocaine use (Carroll, 2000)
- typical skills taught include:
  - identifying high-risk situations for relapse
  - identifying the functions of cocaine use
  - developing skills for coping with craving

## Management of Withdrawal

There is, as yet, no generally accepted, effective pharmacotherapy for cocaine withdrawal. Management of withdrawal is largely supportive.

Issues to be considered include:

### Assessment

- careful neurological and physical examination
- detailed psychiatric history
- detailed drug use history
- concomitant use of other drugs, licit and illicit
- reasons for withdrawal

### Management

- the patient should be placed in quiet surroundings for several days and allowed to sleep and eat as much as is needed
- benzodiazepines may be prescribed on a short-term basis for agitation

- has been shown to be more effective than control treatments for more severely dependent cocaine users and those with comorbid mental disorders
- is more effective than less intensive approaches
- effects are durable, with clients continuing to reduce their cocaine use even after they leave treatment (Carroll, 2000)

### **Contingency management**

- has shown promise in increasing cocaine abstinence and treatment retention in research-based treatment programs
- uses an escalating reward system in which violations are punished both by denying the immediate reward and taking away the benefits of an escalated payment (Sindelar & Fiellin, 2001)
- different types of rewards have been used, including money and vouchers which can be exchanged for retail goods

### **Enhancement of psychosocial skills**

- an adjunct to conventional therapy associated with better treatment outcome is the enhancement of social skills through training programs (Volpicelli et al., 2000)

### **Acupuncture**

- may be useful for some cocaine dependent clients, particularly those maintained on methadone (Avants et al., 2000)

### **General approaches**

Given the lack of generally accepted, effective treatments for cocaine dependence in Australia, efforts aimed at rehabilitation of cocaine users should follow the same general supportive and commonsense approaches used for those dependent on other drugs.

Clinicians should:

- *not* judge the user and should not insist on abstinence
- seek to engage and retain the user in treatment for as long as possible, as retention is associated with better outcomes (Simpson et al., 1999)
- ensure understanding of the client/patient's treatment goals (e.g., to make it through an acute crisis; to reduce frequency and/or quantity of cocaine use; to achieve long-term abstinence)
- tailor the treatment where possible to meet those goals, including referral when appropriate to:
  - treatment programs
  - individual counsellors
  - family counsellors
  - self-help groups such as NA
- remember the need for flexibility of service delivery; as goals and outcomes change throughout the course of treatment, the treatment program should be adjusted to reflect these changes
- provide as multifaceted and intensive a program as possible, as more intensive psychosocial treatment programs are associated with better outcome (Crits-Cristoph et al., 1999)

NIDA has produced a manual for an individual counselling approach for cocaine dependence that consists of 36 sessions designed to take place over six months (Merder & Woody, 1999).

### **Comorbid disorders**

Cocaine users often have multiple psychiatric and psychosocial problems. It is estimated that 30% of cocaine treatment presentations suffer with anxiety disorders, 20% with bipolar disorders, and 5% with attention deficit disorders (Tutton & Crayton, 1993). Programs that assess and address these issues

have better outcomes than those which do not (McLellan et al., 1997, 1998).

### ***Readiness to change***

In patients who do not wish to become abstinent despite significant impairment related to cocaine use, the clinician should attempt to:

- establish an empathetic, respectful relationship
- retain contact with the client
- maximise physical and mental health, as clients will find it difficult to achieve long-term abstinence if chronic medical problems have not been adequately treated
- enhance motivation toward abstinence by educating clients and their significant others about the usual course of cocaine dependence and the relationship between cocaine use and current and/or future problems
- emphasise the client's responsibility for their own actions
- help clients rebuild a life without cocaine through:
  - vocational counselling
  - family counselling
  - helping them develop a network of non-drug using peers
  - showing them how to use free time appropriately

## REFERENCES

- Addis, A., Moretti, M.E., Syed, F.A., Einarson, T.R. & Koren, G. 2001, 'Fetal effects of cocaine: An updated meta-analysis', *Reproductive Toxicology*, vol. 15, pp. 341–369.
- 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.
- APA (American Psychiatric Association) 1994, *Diagnostic and Statistical Manual of Mental Disorders*, 4<sup>th</sup> edn., (DSM–IV), APA, Washington DC.
- Avants, S.K., Margolin, A., Holford, T.R. & Kosten, T.R. 2000, 'A randomized controlled trial of auricular acupuncture for cocaine dependence', *Archives of Internal Medicine*, vol. 160, no. 15, pp. 2305–2312.
- Barrett, J., Meehan, O. & Fahy, T. 1996, 'SSRI and sympathomimetic interaction'. *British Journal of Psychiatry*, vol. 168, p. 253.
- Carroll, K.M. 2000, 'Implications of recent research for program quality in cocaine dependence treatment', *Substance Use Misuse*, vol. 35, pp. 2011–2030.
- Chesher, G.B. 1993, 'Pharmacology of the sympathomimetic psychostimulants' in *Illicit Psychostimulant Use in Australia*, (eds.) Burrows, D., Flaherty B. & MacAvoy M., AGPS, Canberra, pp. 9–30.
- Crits-Cristoph, P. 1999, 'Psychosocial treatments for cocaine dependence: National Institute on Drug Abuse Collaborative Cocaine Treatment Study', *Archives of General Psychiatry*, vol. 56, pp. 493–502.
- Darke, S., Kaye, S., & Topp, L. 2002a, 'Cocaine use in New South Wales, Australia, 1996–2000: 5 year monitoring of trends in price, purity, availability and use from the Illicit Drug Reporting System', *Drug & Alcohol Dependence*, vol. 67, pp. 81–88.
- Darke, S., Kaye, S. & Topp, L. 2002b, *NSW Drug Trends 2001: Findings from the Illicit Drug Reporting System (IDRS)*. NDARC Technical Report No. 125, National Drug and Alcohol Research Centre, Sydney.
- Darke, S., Ross, J., Hando, J., Hall, W. & Degenhardt, L. 2000, *Illicit Drug Use in Australia: Epidemiology, Use Patterns and Associated Harm*, Commonwealth Department of Health and Aged Care, Canberra.
- Gawin, F.H. & Kleber, H.D. 1986, 'Abstinence symptomatology and psychiatric diagnosis in cocaine abusers', *Archives of General Psychiatry*, vol. 43, pp. 107–133.
- George, T.P., Chawarski, M.C., Pakes, J., Carroll, K.M., Kosten, T.R. & Schottenfeld, R.S. 2000, 'Disulfurim versus placebo for cocaine dependence in buprenorphine-maintained subjects: A preliminary trial', *Biological Psychiatry*, vol. 47, pp. 1080–1086.

- Kaye, S., Darke, S. & McKetin, R. 2000, *The Prevalence, Patterns and Harms of Cocaine Use Among Injecting and Non-injecting Drug users in Sydney*. NDARC Technical Report Number 99, National Drug and Alcohol Research Centre, Sydney.
- Kaye, S., Darke, S. & Topp, L. 2001, *An Examination of Cocaine Dependence Among Injecting and Non-Injecting Cocaine Users in Sydney*. NDARC Technical Report Number 116, National Drug and Alcohol Research Centre, Sydney.
- Lago, J.A. & Kosten, T.R. 1994, 'Stimulant withdrawal' *Addiction*, vol. 89, pp. 1477–1481.
- McCance-Katz, E.F., Kosten, T.R. & Jatlow, P. 1998, 'Disulfurim effects on acute cocaine administration', *Drug & Alcohol Dependence*, vol. 52, pp. 27–39.
- McLellan, A.T., Grissom, G.R., Zanis, D., Randall, M., Brill, P. & O'Brien, C.P. 1997, 'Problem-service 'matching' in addiction treatment', *Archives of General Psychiatry*, vol. 54, pp. 730–735.
- McLellan, A.T., Hagan, T.A., Levine, M., Gould, F., Meyes, K., Bencivengo, M. & Durrell, J. 1998, 'Supplemental social services may improve outcomes in public addiction treatment', *Addiction*, vol. 93, pp. 1489–1499.
- Merder, D.E. & Woody, G.E. 1999, *An Individual Drug Counseling Approach to Treat Cocaine Dependence: The Collaborative Cocaine Treatment Study Model*. National Institute on Drug Abuse Therapy Manuals for Drug Addiction, Manual No. 3, National Institutes of Health, Maryland, USA.
- Nathan, K.I., Bresnick, W.H. & Batki, S.L. 1998, 'Cocaine abuse and dependence: Approaches to management' *Central Nervous System Drugs*, vol. 10, pp. 43–59.
- O'Dell, L.E., George, F.R. & Ritz, M.C. 2000, 'Anti-depressant drugs appear to enhance cocaine-induced toxicity', *Experimental and Clinical Psychopharmacology*, vol. 8, pp. 133–141.
- Petrakis, L.L., Carroll, K.M., Nich, C., Gordon, L.T., McCance-Katz, E.F., Frankforter, T. & Rounsaville, B.J. 2000, 'Disulfurim treatment for cocaine dependence in methadone-maintained opioid addicts'. *Addiction*, vol. 95, pp. 219–228.
- Platt, J.J. 1997, *Cocaine Addiction: Theory, Research and Treatment*. Harvard University Press, Cambridge, Massachusetts.
- Schuckit, M.A. 1995, *Drug and Alcohol Abuse: A Clinical Guide to Diagnosis and Treatment* (4<sup>th</sup> edn.), Plenum Medical Book Company, New York.
- Simpson, D.D., Joe, G.W., Fletcher, B.W., Hubbard, R.L. & Anglin, M.D. 1999, 'A national evaluation of treatment outcomes for cocaine dependence', *Archives of General Psychiatry*, vol. 56, pp. 507–514.
- Sindelar, J.L. & Fiellin, D.A. 2001, 'Innovations in treatment for drug abuse: Solutions to a public health problem', *Annual Review of Public Health*, vol. 22, pp. 249–272.

Topp, L., Hando, J., Dillon, P., Roche, A. & Solowij, N. 2000, 'Ecstasy use in Australia: Patterns of use and associated harms', *Drug & Alcohol Dependence*, vol. 55, pp. 105–115.

Topp, L., et al. 2002, *Australian Drug Trends 2001: Findings of the Illicit Drug Reporting System (IDRS)*, NDARC Monograph, National Drug and Alcohol Research Centre, Sydney.

Tutton, C.S. & Crayton, J.W. 1993, 'Current pharmacotherapies for cocaine abuse: A review', *Journal of Addictive Diseases*, vol. 12, pp. 109–127.

Volpicelli, J.R., Markman, I., Monterosso, J., Filing, J. & O'Brien, C.P. 2000, 'Psychosocially enhanced treatment for cocaine-dependent mothers: Evidence of efficacy', *Journal of Substance Abuse Treatment*, vol. 18, pp. 41–49.

# Cocaine

# Heroin and Other Opioids

**O**PIOID drugs mainly act on the opioid receptor system to produce a range of effects which may be considered therapeutic or adverse (side effects). Opioids affect the nervous, gastrointestinal, endocrine and other physical systems as listed in Table 9–1.

## PATTERNS OF HEROIN USE

The use of prescribed opioids and illicit heroin has been steadily increasing in Australia since the 1980s. Most people who use illicit psychoactive drugs such as heroin do so on an irregular basis. However, it is estimated that about one in three heroin users develop dependence.

## OPIOID DRUGS

### Heroin

Heroin is a potent opioid derived from morphine. Heroin is an illegal drug in Australia, and is usually available in the form of a water-soluble, white crystalline powder:

- heroin is usually injected intravenously, although increasing numbers of Australians smoke or snort the drug. Oral heroin is largely metabolised by the liver before exerting



its main effects (high first-pass metabolism), so that few users take heroin orally

- heroin is a short acting drug with rapid onset of effects. Onset of effects occur within minutes of smoking or injection, with significant effects lasting up to 3–6 hours in regular users

- it has a similar duration of action to heroin, with significant effects for 3–6 hours

- slow-release oral morphine preparations are available allowing once or twice a day dosing for chronic pain

## Morphine

Morphine is prescribed for a range of medical conditions, most notably as an analgesic.

- it has variable but significant first-pass liver metabolism (the bioavailability of oral morphine is about 25% of injected doses)

## Methadone

Methadone is a long acting opioid used in the management of chronic pain (methadone tablets) and for the treatment of opioid dependence (methadone syrup or solution).



See Tables 9–2 & 9–8

**Table 9–1**  
Opioid effects

Physical system	Effect
Nervous system	<ul style="list-style-type: none"> <li>• analgesia (pain relief)</li> <li>• euphoria</li> <li>• sedation, drowsiness, respiratory depression</li> <li>• reduced cough reflex</li> <li>• pupillary constriction</li> </ul>
Gastrointestinal actions	<ul style="list-style-type: none"> <li>• nausea and vomiting</li> <li>• constipation</li> <li>• biliary spasm (elevated tone of Sphincter of Oddi)</li> </ul>
Endocrine actions	<ul style="list-style-type: none"> <li>• changes in sex hormones in women (low follicle-stimulating hormone (FSH) and leutenising hormone (LH); raised prolactin) resulting in menstrual changes, reduced libido, galactorrhoea</li> <li>• reduced testosterone in men with reduced libido</li> <li>• elevated anti-diuretic hormone (ADH), reduced ACTH</li> </ul>
Other	<ul style="list-style-type: none"> <li>• itching, sweating, flushed skin (histaminic reaction)</li> <li>• dry mouth, skin and eyes</li> <li>• difficulty passing urine</li> <li>• low blood pressure</li> </ul>

## Buprenorphine (Subutex®)

Buprenorphine is registered as an analgesic (low dose sublingual tablets), and for the management of opioid dependence (high dose sublingual tablets). Buprenorphine is a partial opioid agonist (i.e. low activity) and binds tightly (high affinity) at the mu opioid receptors. This means that:

- buprenorphine generally produces typical opioid effects
- buprenorphine binds in preference and more tightly to receptors than other opioids such as heroin or methadone. It can therefore precipitate opioid withdrawal if taken by individuals who have recently used other opioids (e.g. within 6–8 hours of heroin use or within 24 hours of even low methadone doses)
- it reduces (blocks) the effects of other opioids. This can complicate efforts to

achieve additional opioid analgesia in clients on buprenorphine

- onset of effects approximately 30 to 60 minutes after a dose. Peak effects occur 3 to 8 hours after a dose
- duration of effects are dose related:
  - low doses (e.g. 0.2 to 0.8 mg used for analgesia): 4–12 hours
  - medium doses (e.g. 4 to 8 mg): 12–24 hours
  - high doses (e.g. 12 mg or more): 24–72 hours (this allows some clients to be dosed every 2 or 3 days)
- buprenorphine appears to have a milder withdrawal syndrome than withdrawal from equivalent amounts of morphine or methadone



See Table 9–7

**Table 9–2**  
Methadone effects and duration

Effect	Timing
Onset of action	30–90 minutes after oral dose
Peak effects	3–8 hours after dose
Duration of effects in substitution treatment of opioid dependence	20–30 hours, allowing once a day dosing
Duration of analgesic effects in pain management	8–12 hours, and is usually taken two or three times a day for pain management
Half-life	Approximately 15–30 hours. It takes approximately 5 half lives to achieve steady-state equilibrium after a dose change. This is important when starting methadone — during the first few days of treatment the client will experience increasing opioids effects after each dose. Hence caution is required when starting methadone treatment.

## Naltrexone

Naltrexone is an opioid antagonist. It binds to opioid receptors but produces no opioid effects, and importantly prevents other opioids from binding to receptors. This blocks the effects of other opioids (e.g. heroin) and relapse to heroin use is prevented as long as

naltrexone is taken. There is no withdrawal on ceasing naltrexone. Naltrexone is registered in Australia for the prevention of relapse in heroin and alcohol dependence.



See Chapter 3  
Alcohol

**Table 9-3**  
Common symptoms and time frames of opioid withdrawal

Time since last heroin use	Common symptoms
6 to 12 hours	<ul style="list-style-type: none"> <li>runny eyes and nose, sneezing, yawning</li> <li>sweating</li> </ul>
12 to 24 hours	<ul style="list-style-type: none"> <li>agitation and irritability</li> <li>goosebumps</li> <li>sweating, hot and cold flushes</li> <li>loss of appetite</li> </ul>
more than 24 hours	<ul style="list-style-type: none"> <li>strong urges (cravings) to use heroin</li> <li>stomach cramps, diarrhoea</li> <li>poor appetite, nausea, vomiting</li> <li>back pain, pain in joints, legs or arms, headache</li> <li>poor sleep</li> <li>lethargy, fatigue</li> <li>restlessness, irritability, agitation</li> <li>poor concentration</li> <li>hot and cold flushes, increased sweating</li> </ul>
2nd to 4th days	<ul style="list-style-type: none"> <li>symptoms reach their peak</li> </ul>
5th to 7th days	<ul style="list-style-type: none"> <li>most physical symptoms begin to settle down. Appetite returns</li> </ul>
second week	<ul style="list-style-type: none"> <li>'physical' discomfort subsiding. May have ongoing problems with poor sleep, tiredness, irritability, cravings</li> </ul>
weeks to months	<ul style="list-style-type: none"> <li>return of normal sleep, levels of activity and mood. Improvement in general health, and cravings reduce</li> </ul>

## TOLERANCE

The body adapts to the repeated use of opioid drugs so that a higher dose is required to produce the same effect that was once obtained at a lower dose. The process is called neuro-adaptation and results in the phenomenon of tolerance. Tolerance results in a reduction in response to opioids after regular use.

## WITHDRAWAL

A withdrawal syndrome is the emergence of characteristic signs and symptoms upon the reduction or cessation of heavy and prolonged opioid use. Opioid withdrawal is very unpleasant, but not life-threatening. Table 9–3 shows the characteristic features and time frames for short acting opioids such as heroin and morphine.

**Table 9–4**  
Factors impacting upon severity of withdrawal

Factor	Impact
Opioid type	Withdrawal from longer acting opioids (e.g. methadone) is typically slower in onset and lasts longer (weeks to months). Withdrawal from partial agonists (e.g. buprenorphine) appears to be less severe.
Opioid dose	Higher doses are generally associated with greater withdrawal severity.
Duration of regular opioid use	A short history of regular use (e.g. < 6 months) is generally associated with a milder withdrawal syndrome.
Prior experience of withdrawal and expectancy	Individuals who are particularly anxious about withdrawal often experience greater discomfort.
Concomitant medical or psychiatric conditions	May increase the severity of withdrawal, or decrease the individual's capacity to cope with symptoms.
Setting	The environment in which withdrawal is undertaken impacts upon the experience.

## DEPENDENT HEROIN USE

### The Dependence Syndrome

- dependence has *grades* of severity — it is not an 'all or nothing' phenomenon
- the most salient feature of the syndrome is the loss of control over the use of a drug, with persistent use despite significant harms
- physical dependence is not a requisite of drug dependence, nor is physical dependence alone sufficient for a diagnosis of dependence

### Natural History of Dependent Heroin Use

Most dependent heroin users describe first using heroin in their late teens to early twenties, with regular use usually commencing several years later.

Heroin dependence is a chronic, relapsing–remitting condition. Long-term follow-up of those entering treatment suggests:

- 10% of heroin users will become and remain abstinent in the first year after treatment
- approximately 2–3% of users will achieve and remain abstinent in each subsequent year

## Features of Dependent Heroin Use in Australia

- dependent heroin use is difficult to sustain for most people
- heroin is a *short acting* drug: 2 to 4 injections a day is common
- illicit heroin has variable concentration and adulterants, and is expensive (costing \$50 to \$200 per day in 2001)
- stigma associated with heroin use can deter people from seeking treatment or disclosing their drug use to family, friends, work colleagues and health workers
- polydrug use is common: over half of dependent heroin users use cannabis regularly, and approximately one third used benzodiazepines within last month

## HARMS ASSOCIATED WITH HEROIN USE

Side effects associated with opioids are described above. Other harms include:

### Overdose

- the estimated mortality rate (from all causes) for heroin users is approximately 1–2% per annum (10 to 20 times greater than age and gender matched controls). More common in male heroin users over age 25

- most opioid-related deaths occur following use of opioids with other drugs (alcohol, benzodiazepines)

## Harms Related to Injecting

- trauma and/or infection of injection sites: scarring, thrombosis, thrombophlebitis, cellulitis
- systemic bacterial or fungal infections: septicaemia, infective endocarditis, pneumonia, osteomyelitis, and renal complications (glomerulonephritis)
- blood borne viruses (HIV, HCV and HBV)
  - HIV: has been contained in Australian IDUs thus far (prevalence rates of 0.8%)
  - HCV: Prevalence (HCV Ab +ve) 64% of IDUs; incidence rate approximately 5 to 15% — an area of increasing concern
  - HBV: Estimated 17% from self-report

(National Seroprevalence Study of Users of Needle Exchange Programs, 2001)



## Psychological Harms

Dependent heroin users have a greater incidence of depression, anxiety, suicidal ideation and poor self-esteem. This relationship is often complex and causality difficult to establish. Psychological problems can subside following management of heroin dependence (e.g. methadone maintenance) without the need for psychiatric treatment; however, psychiatric assessment should be sought for clients with severe or persistent problems.

## Social and Community Harms

- financial, legal problems
- impaired functioning/retraction from other activities (work, parenting, friendships)
- stigma for individuals, families, friends
- economic cost to the community

## MANAGEMENT AND INTERVENTION STRATEGIES

There are two treatment pathways available for dependent heroin users as illustrated in Figure 9–1 overleaf.

## WITHDRAWAL SERVICES

### Objectives

Withdrawal can temporarily alleviate much of the stress of heroin dependence; however, withdrawal alone rarely results in long-term changes in drug use. Heroin dependence is a chronic condition, requiring long-term interventions for lasting benefits.

Withdrawal services are short-term interventions with the following objectives:

- alleviate the discomfort of heroin withdrawal
- prevent the development of complications (e.g. overdose)
- interrupt a pattern of heavy and regular drug use
- facilitate linkages to post-withdrawal services

## Key Components

### Assessment

Referral to or consultation with a specialist is recommended for patients with complex presentations (e.g. polydrug dependence, psychiatric or complex medical presentations).

### Setting

Withdrawal can usually be attempted at home (outpatient or home based withdrawal services). Clients with unsuitable home environments or repeated failure at outpatient withdrawal may require residential support (e.g. community withdrawal unit). Clients with significant medical or psychiatric comorbidity require more intensive residential withdrawal settings (e.g. inpatient detoxification units, general or psychiatric hospital).

### Supportive care

Supportive care can significantly reduce anxiety which, in turn, may reduce the severity of somatic complaints. Provide:

- information regarding the nature and duration of withdrawal symptoms, strategies for coping with symptoms, and the role of medication
- supportive counselling aimed at helping patient cope with symptoms, cravings and to maintain motivation. Defer addressing complex personal, emotional or relationship issues until after withdrawal

- crisis intervention addressing accommodation, personal safety, or other urgent welfare issues may be required

### **Frequent monitoring and review**

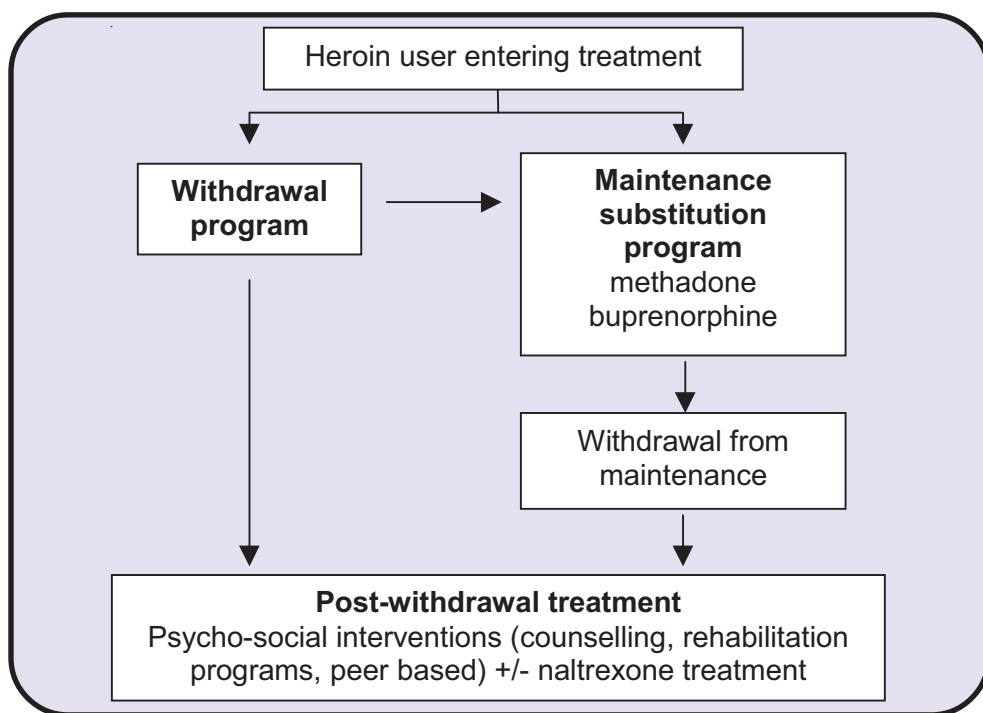
Patients should be reviewed by a health worker at least daily during the first few days, monitoring:

- general progress, ongoing motivation, complications or difficulties encountered
- severity of withdrawal (can be facilitated by the use of withdrawal scales)
- use of heroin and other drugs, and reasons identified by the patient for drug use
- use of, and response to, medication(s) including side effects

### **Medication**

Medication can be a useful adjunct to reduce withdrawal symptoms and cravings. Contemporary approaches for management of heroin withdrawal include:

- medications to manage somatic complaints such as analgesics, anti-emetics, anti-anxiolytics, benzodiazepines, clonidine
- partial opioid agonists — buprenorphine
- reducing doses of an opioid agonist, usually methadone
- opioid antagonists, such as naltrexone which aim to accelerate the withdrawal period



**Figure 9-1**  
Treatment pathways for dependent heroin users

## Medication Regimes for Opioid Withdrawal



See Tables 9–5 & 9–6  
Medications offering symptomatic relief



See Table 9–8  
Methadone for heroin withdrawal



See Table 9–7  
Buprenorphine for heroin withdrawal



See Table 9–9  
Accelerated withdrawal using opioid antagonists

**Table 9–5**  
Symptomatic medications for opioid withdrawal

Other symptomatic medications	
Benzodiazepines	<p>For sleep, anxiety. Concerns regarding abuse (overdose, intoxication, intravenous use, dependence) and/or delayed return of normal sleep pattern. Recommend:</p> <ul style="list-style-type: none"> <li>• limit access to medication (regular dispensing/responsible carer)</li> <li>• beware of multiple sedatives (e.g. clonidine, heroin, alcohol)</li> <li>• do not continue beyond 7 to 10 days.</li> </ul> <p>Diazepam 10 mg to 30 mg per day (to maximum 40 mg / day), in 2 or 3 divided doses; or temazepam 10 to 30 mg (tablets not capsules) nocte. Higher doses may be used in inpatient settings with experienced staff.</p>
Antiemetics	Use reducing doses over 3 to 7 days (e.g. metoclopramide 10 mg t.d.s.)
Antidiarrhoeal agents	<p>These may be useful during the first two to three days.</p> <p>Diphenoxylate i-ii b.d. p.r.n. for up to 5 days.</p>
Antispasmodic agents	May help severe abdominal cramps. Hyoscine butylbromide 10 mg t.d.s. p.r.n. (up to 7 days).
Quinine	For skeletal muscle cramps. Potentially toxic in high doses (blindness, severe liver disease). 300 mg i-ii nocte p.r.n.
NSAIDs	For muscle and joint pains. Ibuprofen 200-400 mg t.d.s. with food p.r.n.



**Table 9–6**  
**Symptomatic medications for opioid withdrawal: clonidine**

<b>Clonidine</b>		
Clonidine ( $\alpha$ -adrenergic agonist) effective in reducing ‘autonomic’ features (diarrhoea, nausea, abdominal cramps, sweating, rhinorrhoea); but less effective for sleep disturbances, aches, cravings. Limit access to large amounts of medication (risks of overdose/abuse).		
Precautions, contraindications	Use only if patient closely monitored (e.g. daily) — unsupervised use not recommended. Use with caution in depression, cerebrovascular disease, renal disease, or with other CNS sedatives. Safety in pregnancy and lactation not established. Contraindications: severe bradyarrhythmia, hypersensitivity.	
Side effects	Hypotension (experienced as dizziness, fainting, light-headedness), fatigue, lethargy, sedation, dry mouth. Severe arrhythmias (bradycardia) following clonidine overdose.	
Dosing regimes	Treatment requires upward dose titration according to the patient’s withdrawal severity and adverse events. A recommended outpatient regime is shown below. Higher doses (to maximum 15 mcg / kg / day) can be used in inpatient settings. Omit/reduce dose if patient is experiencing hypotension.	
	The maximum daily dose of clonidine = 12 mcg / kg / day, given in 3 or 4 divided doses.	
	Days 1–3:	Titrate dose of clonidine according to clinical response: <ul style="list-style-type: none"> <li>• for patients &lt; 60 kg (body weight): 1 x 100 mcg 3 or 4 times / day (300–400 mcg / day)</li> <li>• for patients &gt; 60 kg (body weight): 1 x 150 mcg 3 or 4 times / day (450–600 mcg / day).</li> </ul>
	Day 4:	The total dose is reduced to 75% of the day 3 dose and given in 3 or 4 divided doses.
	Day 5:	The total dose is reduced to 50% of the day 3 dose and given in 2 or 3 divided doses.
	Day 6:	The total dose is reduced to 25% of the day 3 dose and given in 1 or 2 doses.
	The maximum daily dose of clonidine = 12 mcg / kg / day, given in 3 or 4 divided doses	

**Table 9–7**  
**Buprenorphine for heroin withdrawal**

Buprenorphine is a partial opioid agonist useful in managing heroin withdrawal, either in short-term regimes (e.g. 3 to 10 days); or in gradual reduction regimes over several weeks (similar to methadone reduction programs). Issues include:

- opioid-like side effects (usually mild and tolerable) are common during first few days. The initial buprenorphine dose can precipitate opioid withdrawal in a person who has recently used heroin — first dose should be delayed until patient has features of opioid withdrawal (or at least 6–8 hours after last heroin use).
- an authorised medical practitioner requires a permit to prescribe buprenorphine, and it is dispensed under supervision at an authorised pharmacy.
- other medications for opioid withdrawal are not routinely required.
- some patients will experience ‘rebound’ withdrawal on stopping buprenorphine — this is generally greater with longer durations of buprenorphine treatment.
- a range of post-withdrawal treatment options available, including (a) transfer to maintenance buprenorphine, (b) naltrexone treatment or (c) ‘drug-free’ rehabilitation.

#### Dosing regimes

Short outpatient regimes	Proposed regime	Recommended lower and upper limits
Day 1	6 mg	4 to 8 mg
Day 2	8 mg	4 to 12 mg
Day 3	10 mg	4 to 16 mg
Day 4	8 mg	2 to 12 mg
Day 5	4 mg	0 to 8 mg
Day 6		0 to 4 mg
Day 7		0 to 2 mg
Day 8		0 to 1 mg
Short inpatient regimes	Proposed regime	Recommended lower and upper limits
Day 1	4 mg at onset of withdrawal, & additional 2 to 4 mg evening dose p.r.n.	4 to 8 mg
Day 2	4 mg mane, with additional 2 to 4 mg evening dose p.r.n.	4 to 8 mg
Day 3	4 mg mane, with additional 2 mg evening dose p.r.n.	4 to 6 mg
Day 4	2 mg mane p.r.n.; 2 mg evening p.r.n.	0 to 4 mg
Day 5	2 mg p.r.n.	0 to 2 mg
Day 6	no dose	
Day 7	no dose	

## Post-withdrawal Interventions

### *Psychosocial interventions*

These aim to prevent relapse back to dependent heroin use following withdrawal from heroin or maintenance substitution treatment. The evidence regarding their efficacy suggests:

- outpatient counselling programs are more effective for those individuals with good social support systems (e.g. relationships, employment), good cognitive functioning and higher levels of education
- those without good community supports or cognitive functioning may benefit from more structured interventions such as long-term residential rehabilitation (including therapeutic communities), self-help groups

### *Outpatient counselling*

Counselling can be individual, group or family based, and may be structured upon principles of supportive counselling; cognitive-behavioural or psychodynamic theory. Relapse prevention is a common approach used by drug treatment agencies aiming to:

- enhance commitment to abstinence
- identify environmental and psychological factors associated with relapse to drug use
- develop cognitive-behavioural coping skills to deal with these risk factors

As in most forms of counselling, the quality of the relationship between client and counsellor is critical.

### *Therapeutic communities*

These are usually long-term (e.g. 6 month to 3 year) residential programs in which drug

**Table 9–8**  
**Methadone for heroin withdrawal**

<p>Methadone can be used in short regimes (e.g. 7 to 14 days), or longer regimes with gradual reduction over weeks. Issues include:</p> <ul style="list-style-type: none"> <li>• opioid-like side effects (usually mild and tolerable) are common during first few days.</li> <li>• an authorised medical practitioner requires a permit to prescribe methadone, and it is dispensed under supervision at an authorised pharmacy.</li> <li>• withdrawal discomfort generally increases as the patient reduces their methadone dose, with greatest discomfort experienced when/soon after the patient ceases their methadone, and can continue for several days. This limits the use of methadone in inpatient settings.</li> <li>• other medications for opioid withdrawal are not routinely required until the patient has reduced their methadone dose, when short courses of symptomatic medication may be of value. Caution about using other sedating drugs (benzodiazepines, alcohol).</li> </ul>	
<p>Dosing regimes</p>	<p>Commence with doses between 20 and 30 mg (depending upon level of physical dependence and concomitant medical conditions). Dose is reduced according to proposed duration of regime (e.g. start 25 mg, reduce by 2.5 mg per day for 10 day regime).</p>

users move into a highly structured, isolated environment, with varying approaches to counselling and rehabilitation. Therapeutic communities tend to attract individuals with highly entrenched drug using lifestyles, few community supports or resources, often with legal conditions. This is an effective approach for those prepared to remain in treatment long-term; although rates of relapse on community re-entry are high.

### **Self-help groups**

Narcotics Anonymous (NA) is the main self-help group for opioid dependent individuals in Australia. It operates on a 12-step abstinence philosophy and is attractive for some drug users as it provides a support structure and 'community', but does not appeal to all.

## **Naltrexone Treatment**

Naltrexone is registered in Australia for the prevention of relapse for heroin or alcohol dependent patients. Naltrexone is an opioid antagonist that blocks the actions of other opioids.

### **Clinical aspects**

- clients should complete opioid withdrawal (at least 5 to 7 days without heroin use, 10 days without methadone) prior to starting naltrexone. Premature naltrexone treatment results in severe withdrawal — a naloxone challenge test is recommended prior to starting naltrexone
- recommended dose is 50 mg daily. Blockade effects wear off within 2 to 3 days of ceasing such a dose
- treatment outcomes can be optimised by:
  - participation in a comprehensive psychosocial treatment program
  - supervision of doses (e.g. by a family member) and close monitoring by treatment team

### **Adverse events**

- mood and sleep disturbances, abdominal cramps, nausea are common during the commencement of treatment, but usually subside with time
- overdose: overdose on opioids cannot occur whilst a client is taking naltrexone, however, there appears to be an increased risk of overdose upon stopping naltrexone, due to loss of opioid tolerance and possible increased opioid sensitivity following naltrexone treatment
- barriers to analgesia: naltrexone blocks the effects of opioids for analgesia

### **Outcomes associated with naltrexone treatment**

Naltrexone treatment generally has poor retention rates - trials of 'street heroin users' suggest:

- 40% are retained in naltrexone treatment beyond one month
- 10–20% are retained in naltrexone treatment at 6 months

Higher success rates can be achieved by carefully selecting clients for treatment — those who are employed, have good social support networks, are in relationships with non-drug users, and have strong motivation for abstinence (e.g. professionals, court-ordered individuals).

## **SUBSTITUTION MAINTENANCE TREATMENT WITH METHADONE OR BUPRENORPHINE**

### **Rationale, Objectives and Outcomes**

The rationale is to supply dependent heroin users with a long-acting opioid (methadone or buprenorphine) that substitutes for heroin thereby preventing withdrawal, reducing cravings and reducing the euphoric effects of

**Table 9–9**  
**Accelerated withdrawal using opioid antagonists**

Naloxone and naltrexone accelerate the onset and reduce the duration of opioid withdrawal. Rapid detoxification involves the use of naltrexone and symptomatic medications (clonidine, benzodiazepines, anti-emetics). Ultra-rapid detoxification involves inducing the patient onto antagonists using general anaesthesia or heavy sedation in highly supervised (hospital) setting. It is postulated that more rapid withdrawal and early induction onto naltrexone enhances long-term outcomes, although research is yet to establish such benefits. Naltrexone is not registered in Australia for this indication, and specialist consultation is recommended.

continued heroin use. This enables clients to stop using heroin, and to disassociate themselves from a drug-using lifestyle. The specific objectives are:

- to reduce heroin and other drug use by clients
- to improve the general health and well-being of clients
- to reduce mortality
- to reduce the transmission of blood borne viruses
- to improve the social functioning of clients, including a reduction in criminal activity

This is an effective treatment modality for most heroin users in reducing mortality, heroin use and criminality, and in improving psychosocial functioning. Australian research suggests that approximately 50–70% of heroin users commencing methadone treatment are retained in treatment at 1 year. Of these:

- approximately half will no longer be using heroin
- approximately one third will continue to use heroin at a markedly reduced frequency (e.g. weekly)
- approximately one sixth will continue to use heroin regularly (e.g. daily)

Treatment outcomes are enhanced in maintenance programs when:

- clients remain in treatment for long periods of time (> 12 months). Clients should be encouraged to remain in treatment until they have distanced themselves from heroin use and associated lifestyles
- clients are on adequate doses of methadone (e.g. 60–120 mg daily) or buprenorphine (e.g. 12–24 mg daily)
- there is a good therapeutic relationship between the client and service providers; and
- where the client participates in counselling and other psychosocial interventions

Methadone and buprenorphine are of comparable efficacy when used in equivalent doses.

### **Delivering Substitution Treatment in Australia**

- maintenance substitution treatment is restricted to medical practitioners, pharmacies or clinics specially authorised by state or territory governments. This generally requires a training program for medical practitioners
- doctors must receive a permit to treat individual clients with methadone or buprenorphine

- the administration of methadone and buprenorphine is usually supervised at a clinic or pharmacy. Stable clients can receive a number of take-away doses after a period of time (policies vary in each jurisdiction)
- treatment is increasingly being delivered by general practitioners and community pharmacies, thereby normalising treatment and increasing its accessibility. Public clinic programs are being oriented towards managing clients with more complex presentations

### Problems with Maintenance Substitution Treatment

- the inconvenience of daily/regular dispensing
- the cost to clients of supervised dispensing
- withdrawal syndrome and high relapse rates upon the cessation of treatment
- a minority have persistent side effects
- stigma associated with methadone treatment

Buprenorphine can be better tailored to the needs of the individual client.

### Principles of Safe and Effective Methadone/Buprenorphine Treatment

- comprehensive assessment and informed consent is required prior to commencing treatment. Methadone/buprenorphine treatment are only suitable for opioid dependent people
- commence with low doses, review client frequently during induction and titrate dose accordingly:
  - starting doses of methadone are usually between 20 to 30 mg (and never > 40 mg). Beware of accumulation of methadone during induction — in general only increase dose after every 3–4 days
  - starting doses of buprenorphine are usually between 4 and 8 mg (never greater than 8 mg), and delay first buprenorphine dose until client is in opioid withdrawal. Beware of precipitated withdrawal on starting buprenorphine, especially in clients transferring from methadone
  - only increase dose after review by an experienced treatment provider
- beware of combined use of other sedatives (e.g. alcohol, benzodiazepines), due to an increased overdose risk
- an experienced treatment provider should review clients who have missed multiple (> 3) consecutive methadone/buprenorphine doses prior to recommencing treatment — risk of overdose from drop in tolerance
- clients who are not responding to treatment (e.g. continued high risk drug use) require increased monitoring, psychosocial interventions and referral to specialist services

## RESOURCES

Alliance of NSW Divisions of General Practice:



[www.answd.com.au](http://www.answd.com.au)

CDHA 2002, *Illicit Drug Training for Pharmacists*, Commonwealth Department of Health and Ageing, Canberra.

Dale, A. & Marsh, A. 2000, *A Summary of the Evidence Based Practice Indicators for Alcohol and Other Drug Interventions*, Best Practice in Alcohol and Other Drug Interventions Working Group, Perth.

Dunlop, A., Thornton, D. Lintzeris, N., Muhleisen, P., Khoo, K. & Lew, R. 1996, *Coming Off Methadone*, Turning Point Alcohol and Drug Centre Inc., Fitzroy, Victoria.

Gill, T. 1997, *Heroin Addiction*, GP Drug and Alcohol Supplement No.8, [www.health.nsw.gov.au/public-health/dpb/supplements/supp8.pdf](http://www.health.nsw.gov.au/public-health/dpb/supplements/supp8.pdf)

Gill, T. & Evans, M. 1996, *Methadone in the Treatment of Opioid Dependence*, GP Drug and Alcohol Supplement No.2, [www.health.nsw.gov.au/public-health/dpb/supplements/supp2.pdf](http://www.health.nsw.gov.au/public-health/dpb/supplements/supp2.pdf)

Gowing, L., Ali, R. & White, J. 2000, 'The management of opioid withdrawal' *Drug and Alcohol Review*, vol. 19, pp. 309–318.

Lintzeris, N., Dunlop, A. & Thornton, D. 1996, *Getting Through Heroin Withdrawal*, Turning Point Alcohol and Drug Centre Inc., Fitzroy, Victoria.

NDARC (no date), *Heroin*, Reachout Fact Sheet – 'Drugs', National Drug and Alcohol Research Centre, Sydney.

NDARC (no date), *What You Need to Know about Methadone*, National Drug and Alcohol Research Centre, Sydney.

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*, Next Step Specialist Drug and Alcohol Services, Mt. Lawley, Western Australia.

Young, R., Saunders, J. Hulse, G., McLean, S, Martine, J. & Robinson, G. 2002, cited in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, 'Opioids', ch. 6, Oxford University Press, South Melbourne, Victoria, pp.79–123.

## REFERENCES

National Seroprevalence Study of Users of Needle Exchange Programs, 2001.



# Opioids

# Volatile Substances

**V**OLATILE substances are central nervous system (CNS) depressants and are chemical compounds that rapidly change from a liquid, or semisolid state to vapour when exposed to air.

Volatile substance use is the deliberate inhalation of vapour given off from a substance at ambient temperature to alter consciousness or cause intoxication.

Volatile substance use may be:

- experimental — using out of curiosity
- recreational — frequently a group practice
- chronic — an habitual and dominant activity

## COMMONLY USED VOLATILE SUBSTANCES

The most commonly used volatile substances include petrol, lacquers and varnishes containing benzene and adhesives, spray paints, glues and paint thinners containing toluene. Table 10-1 lists the major volatile substances and their sources. Also used are amyl nitrite and nitrous oxide found in canisters.

**Table 10-1**  
Major volatile substances and their sources

Major volatile substances	Sources
• benzene	• petrol, varnish, resins, lacquer
• toluene	• adhesives, spray paints, glues, paint thinners
• xylene	• lacquer, thinner, wood glues
• propane	• bottled fuel
• butane	• cigarette lighter fluid
• acetone	• nail polish remover
• n-hexane	• model glues, rubber cement
• trichloroethane	• dry cleaning agents, degreasing agents
• trichloroethylene	• dry cleaning agents, stain removers, degreasing agents
• trichlorofluoromethane	• aerosol propellant
• dichloromethane	• paint stripper
• butyl nitrite	• room air freshener

## MODES OF ADMINISTRATION

There are three major modes of volatile substance administration:

- *sniffing*: Vapours inhaled directly from a container. Lowest vapour concentration, with a significant quantity dissipated into the surrounding air
- *huffing*: A piece of saturated material (commonly a piece of clothing) held against the mouth or nose. In extreme cases may be held *in* the mouth. Spraying aerosol vapours directly into the mouth
- *bagging*: Vapours inhaled from a plastic or paper bag held firmly over the mouth and nose

Chronic users generally begin with sniffing and progress to huffing and bagging to increase vapour concentration availability and achieve and maintain euphoria (Henretig, 1996; Linden, 1990).

## PREVALENCE

There is a lack of good epidemiological data on the prevalence of use amongst general community groups and subgroups. However some trends in using volatile substances are emerging:

- there is a higher prevalence amongst the 14–17 year old age group than older adults (White, 2001)
- the trend for use is greatest amongst younger teenagers, aged 12 and over (White, 2001)
- male adolescents use more than female adolescents (AIHW, 1999)
- amongst recreational drug users volatile substances and cannabis were most commonly combined with ecstasy, amphetamine or LSD at rave scenes (Boys et al., 1997)

- compared with non-Indigenous counterparts, young Indigenous users
  - show greater habitual use
  - use more frequently
  - use over a longer period(Carroll et al., 1998)

### APPEAL

Volatile substances:

- are relatively inexpensive
- are readily available from supermarkets, hardware stores, homes, building sites, cars and offices, despite legislation in a number of Australian States to preclude their sale to minors
- can be packaged in small and discrete containers (e.g. cans or bottles) and easily concealed
- create rapid intoxication and rapid disappearance of intoxication. The user can indulge and then go home or to other venues in a sober state

### PHYSICAL COMPLICATIONS

#### Acute Effects

Following use, blood levels peak within minutes then rapidly decrease as the drug is absorbed into fat, including the central nervous system (CNS).

Common initial effects are:

- euphoria
- excitation
- exhilaration
- a sense of invulnerability

#### Negative Acute Effects

Users may also experience:

- nausea
- vomiting
- headaches
- diarrhoea
- abdominal pain

These effects commonly resolve within two hours (Liira et al., 1988).

#### Effects at Higher Doses

Central nervous system depression leads to:

- slurred speech, disorientation, confusion, delusions, weakness, tremor, headaches, visual distortions and visual hallucinations; then
- ataxia; followed by
- stupor
- final stages associated with seizures, coma, cardiopulmonary arrest and death (Linden, 1990)

In the novice, or infrequent user, desired effects are achieved after a few breaths. However, tolerance develops rapidly and within several days of repeated use the user requires a significant increase in dose to achieve the desired effect. Withdrawal symptoms commonly include headache, nausea and muscle and abdominal cramps.

#### Specific Physical Effects

##### *Central Nervous System*

The majority of solvents are fat soluble and readily absorbed from the blood into high fat tissue including nerve cells. This results in generalised reduction of nerve membrane functioning, which causes CNS depression (Lolin, 1989).

Of the commonly abused substances, toluene causes the most CNS damage. White matter damage, cortical atrophy and cerebellar damage are observed in long-term chronic users. Destruction of nerve cells also results in peripheral neuropathy (Lolin, 1989), optic atrophy and hearing loss (Fornazzari et al., 1983; Williams, 1988).

### **Maternal and neonatal**

Given their fat solubility, volatile substances readily cross the placenta. Neonatal toluene exposure is associated with malformations including:

- oral clefts and microcephaly
- spontaneous abortion
- foetal growth retardation
- low birthweight
- prematurity
- developmental delays

(Arnold et al., 1994; Jones & Balster, 1998)

### **Heart**

Sudden death associated with ventricular fibrillation and cardiac arrhythmia is a major concern. Hydrocarbons contained, for example, in aerosols, petrol and substances containing benzene and toluene, sensitise the myocardium to adrenaline. When the user is 'startled', the resulting sudden surge of adrenaline causes ventricular fibrillation (Shepherd, 1989). Approximately 20% of those who die from 'sudden death' in these circumstances have no prior history of volatile substance use (Ramsey, Anderson et al., 1989).

### **Lung**

Volatile substances displace oxygen and can directly damage lung tissue resulting in loss of consciousness.

It is not uncommon for asphyxiation or suffocation to occur from aspiration of vomit in plastic bags used for bagging. Asphyxiation can also be caused by material placed in the

mouth during huffing (Linden, 1990).

### **Kidneys, liver and bone marrow**

Compounds containing toluene cause renal tubular acidosis that interferes with functioning of the distal tubule and collecting ducts (Marjot & McLeod, 1989). Complete kidney and liver failure have been associated with toluene use (Dinwiddie, 1994). Chloroform and chlorinated hydrocarbon vapours result in toxic hepatitis with liver dysfunction associated with trichloroethane and trichloroethylene use (Hutchens & Kung, 1985).

Chronic use of benzene is associated with suppressed functioning of bone marrow production, aplastic anaemia and related morbidity and cancers such as myeloma, leukaemia and lymphoma (Rosner & Grunwald, 1980).

### **Other Morbidity and Mortality**

The sense of invulnerability associated with volatile substance use results in impulsive high risk behaviours that can cause accidents, injury, brain damage, trauma and death. Morbidity and mortality are also associated with fires resulting from combustion of inhalants.

## **PSYCHOSOCIAL COMPLICATIONS**

Adolescents who chronically use volatile substances are more likely than non-users to report poor family relations, family history of alcohol and other drug problems and unstable living environments, school absenteeism and academic problems, criminal activity, low self-esteem and associated depression and suicidal thoughts (Howard & Jenson, 1999). There are reports of violence amongst adolescent chronic volatile substance users, both towards other users and non-users (Dukarm et al., 1996).

## MANAGEMENT AND INTERVENTION STRATEGIES

### Detection and Assessment

To detect and assess volatile substance use:

- look for indicators of recent or chronic use
- obtain a comprehensive history
- conduct a thorough physical examination by a medical practitioner

#### *Clinical signs and symptoms*

Recent use may be indicated by the identification of solvent containers, or bags, bottles and cans with solvent residue or residual odour on breath, skin or contaminated clothing.

Mucous membrane irritation may result in increased sputum production, cough, wheeze, salivation, sneezing, or conjunctival injection (McHugh, 1987). Chronic use can lead to drying of mucous membranes and facial skin which causes irritation and may permit bacterial infections to become established. The resulting patchy redness of skin (erythematous spots) or pus producing skin lesions around the mouth and nose are commonly referred to as 'sniffer's or huffer's rash'. Recent use may also be associated with decreased reflexes and oscillatory movement of the eyes.

Excessive mood swings, disinhibition or inappropriate aggression coupled with one or more of the above physical symptoms may also indicate use.

Symptoms associated with polysubstance use may mask those of volatile substances.

Urine drug screening is not designed to detect volatile substances or metabolites.

Toluene use is confirmed by elevated levels of hippuric and benzoic acid in urine.

### Intoxication

In most instances, acute volatile substance intoxication resolves spontaneously. Clothing and skin should be decontaminated, and the user placed in a well ventilated, safe environment and observed. Cardiopulmonary function should be monitored until intoxication resolves.

There is no significant way to enhance the rate of volatile substance elimination. Cardiopulmonary assessment, stabilisation and monitoring is required. Electrocardiography is indicated in the presence of cardiac abnormalities. Hydration with saline may be required. Laboratory tests include complete blood count and oxygen saturation. Urine or blood screening may be indicated where polysubstance use is suspected.

### Experimental, Recreational and Chronic Use

#### *Experimental*

Volatile substance use is usually a transitory event amongst a kaleidoscope of experimental activities. It commonly resolves without intervention or major incident. Patterns of use in this group preclude the development of tolerance and associated excessive toxicity, and morbidity is low. Mortality from 'sudden death' is of concern. Education initiatives provided in early school years (late primary) as part of an integrated general curriculum may reduce the prevalence and frequency of use. However, any educational programs should be undertaken with care as they can inadvertently increase use.

#### *Recreational*

Similar to experimental use, volatile substance use is commonly an optional activity amongst recreational users. Social status, for example, in those attending rave dances, is more likely to be associated with body image and rave activities, than volatile substance use per se. Information on the relationship

between use, tolerance and morbidity and mortality will likely decrease the frequency and quantity of use in this group. Sudden death is a real possibility and information on the avoidance of use and co-activities that raise adrenaline levels (e.g. exertion and co-stimulant use), both popular activities amongst 'rave' users, is necessary. For both experimental and recreational users, the portrayal of volatile substances as a low class of drugs may reduce the prevalence and frequency of use.

### **Long-term management — the chronic abuser**

Management is difficult and lacks rigorous empirical evaluation. The objectives here are to:

- assess and care for medical complications
- minimise harm associated with use
- move the user away from chronic use patterns and associated lifestyles

### **Assess and care for medical complications**

Mental state, organ and neurological examination are necessary. Chest x-rays are required where there is evidence of pulmonary morbidity. Laboratory tests include complete blood count, oxygen saturation, serum electrolytes, and blood urea, nitrogen and creatinine. Additional tests may be required to assess the pres-

ence of metabolic disturbances and morbidity to other organs (e.g. kidneys).

### **Minimise harm associated with use**

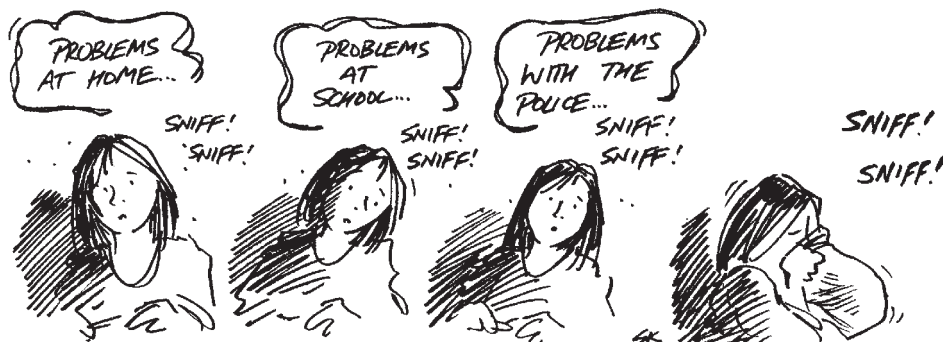
Although total abstinence may be the optimal objective, this may be initially impractical in high risk communities, short of removal of the user. Here harm minimisation to reduce morbidity and mortality is required. For example, non-use of plastic bags that cause asphyxia, removal of lead-based petrol and education about the development of tolerance to reduce overall acute and chronic tissue toxicity and damage.

### **Move the user away from habitual and chronic use patterns**

Exposure to those with significant morbidity, resulting from volatile substance use has been identified by chronic users as a significant factor in reducing use (Carroll, Houghton et al., 1998). Overall, initiatives should focus on the community and environment in which volatile substance use takes place (e.g. urban or Indigenous communities).

Use in these environments is often closely associated with:

- a lack of alternative social activities and poor economic prospects
- the conferring of greater status within the group upon those who consume the greatest quantities of volatile substances



Providing both desirable social activities and social status outside the user group are essential. Initiatives should be acceptable to and developed in collaboration with local communities. Community and family counselling and support may be required.

### Primary Prevention Strategies

Many volatile substance users commence use in early or pre-teen years, so early prevention initiatives at primary schools should be considered. Limited available information suggests that mass media, fear tactics and factual education programs are *not* hugely effective. Some modest success has been reported with programs utilising peer support initiatives and targeting social skills and healthy lifestyle acquisition (CDH, 1985).





## RESOURCES

Youth Substance Advice Service (2000) *Chroming (Inhalant Use) The Facts*. Fitzroy, Vic.



[www.ysas.org.au/drug/chroming](http://www.ysas.org.au/drug/chroming)

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.

CDHSH (Commonwealth, Department of Human Services and Health) 1994, *Secondary School Students' Drug Use: Comparison of Patterns in Victoria and New South Wales, 1992*, Canberra, ACT.

## REFERENCES

- AIHW (Australian Institute of Health & Welfare) 1999, *1998 National Drug Strategy Household Survey: First results*, AIHW cat. no. PHE 15, AIHW, Canberra.
- Arnold, G., Kirby, R., Langendoerfer, S. & Wilkins-Haug, L. 1994. 'Toluene ambryopathy: clinical delineation and developmental follow-up', *Pediatrics*, vol. 93, pp. 216–220.
- Boys, A., Lenton, S. & Norcross, K. 1997, 'Polydrug use at raves by a Western Australian sample', *Drug and Alcohol Review*, vol. 16, pp. 227–234.
- Carroll, A., Houghton, S. & Odgers, P. 1998, 'Volatile solvent use among Western Australian adolescents', *Adolescence*, vol. 33, no. 132, pp. 877–888.
- CDH, (Commonwealth Department of Health) 1995, *National Drug Strategy Household Survey*, Canberra.
- Dinwiddie, S. 1994, 'Abuse of inhalants: a review', *Addiction*, vol. 89, pp. 925–939.
- Dukarm, C., Byrd, R., Auinger, P. & Weitzman, M. 1996, 'Illicit substance use, gender, and the risk of violence behavior among adolescents', *Archives of Pediatrics & Adolescent Medicine*, vol. 150, pp. 797–801.
- Fornazzari, L., Wilkinson, D.A., Kapur, B.M. & Carlen, P.L. 1983, 'Cerebellar, cortical and functional impairment in toluene abusers', *Acta Neurologica Scandinavica*, vol. 67, pp. 319–329.
- Henretig, F. 1996, 'Inhalant abuse in children and adolescents', *Pediatr Ann*, vol. 25, pp. 47–52.
- Howard, M. & Jenson, J. 1999. 'Inhalant use among antisocial youth: prevalence and correlates', *Addiction Behaviour* vol. 24, pp. 59–74.
- Hutchens, K. & Kung, M. 1985, '“Experimentation” with chloroform', *American Journal of Medicine*, vol. 78, pp. 715–718.
- Jones, H. & Balster, R. 1998, 'Inhalant abuse in pregnancy', *Obstetrics & Gynecology Clinics of North America*, vol. 25, pp. 153–167.
- Liira, J., Riihimaki, V. & Pfaffli, P. 1988, 'Kinetics of methyl ethyl ketone in man: absorption, distribution and elimination in inhalation exposure', *International Archives of Occupational & Environmental Health*, vol. 60, pp. 195–200.
- Linden, C. 1990, 'Volatile substances of abuse', *Emergency Medicine Clinics of North America*, vol. 8, pp. 559–578.
- Lolin, Y. 1989, 'Chronic neurological toxicity associated with exposure to volatile substances', *Human Toxicology*, vol. 8, pp. 293–300.

- McHugh, M. 1987, 'The abuse of volatile substances', *Pediatric Clinics of North America*, vol. 34, pp. 333–340.
- Marjot, R. & McLeod, A. 1989, 'Chronic non-neurological toxicity from volatile substance abuse', *Human Toxicology*, vol. 8, pp. 301–306.
- Ramsey, J., Anderson, H., Bloor, K. & Flannagan, R. 1989, 'An introduction to the practice, prevalence and chemical toxicology of volatile substance abuse', *Human Toxicology*, vol. 8, pp. 261–269.
- Rosner, F. & Grunwald, H. 1980, 'Cytotoxic drugs and leukaemogenesis', *Clinical Haematology*, vol. 9, pp. 663–681.
- Shepherd, R. 1989, 'Mechanism of sudden death associated with volatile substance abuse', *Human Toxicology*, vol. 8, pp. 287–291.
- White, W. 'Australian secondary students' use of over-the-counter and illicit substances in 1999', cited in Miller, M & Dapper, G. 2001, *Statistics on Drug Use in Australia 2000*, no. 8, Australian Institute of Health and Welfare.
- Williams, D. 1988, 'Hearing loss in a glue sniffer', *Journal of Otolaryngology*, vol. 17, pp. 321–324.

# Benzodiazepines

**B**ENZODIAZEPINES are minor tranquillisers widely used in clinical practice for sedation and relief of anxiety. They are also widely used for illicit or non-prescribed purposes. The first benzodiazepine (chlordiazepoxide) was marketed in 1959 as a safe alternative to barbiturates. This was followed by the introduction of many related compounds that achieved immense popularity to become the most commonly prescribed class of drugs in the 1970s and 1980s.

Benzodiazepines are CNS depressants. They have antianxiety, anticonvulsant, hypnotic and muscle relaxant properties. Their use results in performance deficits (including memory impairment, motor incoordination, decreased reaction time and ataxia).

## PHARMACOLOGY

Benzodiazepines enhance the effects of gamma-aminobutyric acid (GABA) which is the main inhibitory neurotransmitter in the CNS. Benzodiazepines bind to receptors on the GABA-A receptor complex (Cape et al., 2002).

## ABSORPTION

Benzodiazepines are relatively lipophilic (fat soluble) and most are poorly water soluble with the exception of midazolam (used in anaesthetic practice but also as a 'date rape' drug). They are

generally rapidly and fully absorbed orally with peak plasma concentrations from ½ to 2 hours after ingestion. The more lipophilic agents e.g. diazepam are absorbed faster than the relatively more hydrophilic agents e.g. oxazepam (Cape et al., 2002).

## DISTRIBUTION

Benzodiazepines rapidly enter the CNS and are then distributed to less vascular adipose tissue. They all cross the placental barrier and can result in neonatal drowsiness, respiratory depression, hypotonicity and withdrawal (Therapeutic Guidelines Ltd., 2000).

## METABOLISM

Benzodiazepines must be converted to water soluble compounds before renal excretion. They are metabolised by both oxidation, which may produce active compounds, and by glucuronidation which inactivates them. Active metabolites may have longer half-lives than the parent drug resulting in prolonged effects (especially with chronic dosing).

## PATTERNS OF USE

- in 2001, 1.1% of Australians aged 14 years or over reported use of tranquillisers or sleeping pills in the previous 12 months for non-medical purposes, with peak use reported amongst those aged 20–29 years
- while there is little overall difference in prevalence of tranquilliser or sleeping pill use between men and women (M: 1.2%; F: 1.0%), men aged 20–29 were more likely than women of the same age to use these drugs (3.0% and 2.2% respectively)
- across age groups, people aged 40 years or over had the highest proportion of people

who reported use of prescription drugs for non-medical purposes every day, or every week (AIHW, 2001)

- according to the Australian Statistics on Medicines (PBS, 1998), a total of 8.89 million prescriptions for benzodiazepines were dispensed through Australian pharmacies in 1998 (including PBS/RPBS, private and under co-payment prescriptions)
- the use of night-time sedation with benzodiazepines increases markedly with age
- long-term use of benzodiazepines remains common

## EFFECTS OF BENZODIAZEPINES

All are sedating, anxiolytic and anti-convulsant.

### Short-term Effects

- drowsiness, lethargy, fatigue
- motor incoordination, decreased reaction time and ataxia
- impaired cognition and memory (especially anterograde amnesia)
- confusion
- muscle weakness or hypotonia
- depression
- nystagmus, vertigo
- dysarthria, slurred speech
- blurred vision, dry mouth
- headaches
- paradoxical euphoria, excitement, restlessness, hypomania and extreme disinhibited behaviour (especially high dose, users may feel 'invulnerable, invincible and invisible')
- potentiation of other CNS depressants e.g. alcohol and opioids increasing likelihood of respiratory depression

(Victoria Police, 2002; Cape et al., 2002)

## Long-term Effects

Similar to short-term effects (with no known organ toxicity) plus:

- tolerance to sedative/hypnotic and psychomotor effects (conflicting evidence whether tolerance develops to anxiolytic actions and effects on memory)
- emotional blunting (inability to feel normal highs or grief due to inhibition of arousal)
- menstrual irregularities, breast engorgement
- dependence (may develop after 3–6 weeks at therapeutic doses)

(Cape et al., 2002)

pared with normal subjects. Rebound insomnia frequently occurs on cessation of benzodiazepines

- polydrug use, or concurrent use of benzodiazepines, alcohol or opioids, increases the risk of overdose
- dependence and withdrawal can occur even when recommended doses are used (Busto et al., 1986) (i.e., within 3–6 weeks)
  - withdrawal symptoms may be apparent while the patient is still taking medication, possibly because the patient avoids increasing the dose to cover increased tolerance (Ashton, 1991) or due to the short half-life of some drugs
- adverse mood effects with inability to experience emotions or unwanted stimulation or aggression

## USES AND PROBLEMS

### Uses

Clinically useful in the treatment of anxiety and insomnia because of their efficacy, at least in the short-term, and relative safety compared to the barbiturates or tricyclic antidepressants.

Other uses in clinical practice include the treatment of alcohol withdrawal (to prevent delirium tremens), epilepsy, tremor and agitation in psychiatric disorders.

### Problems Associated with Benzodiazepines

- short- or long-term patterns of benzodiazepine use is associated with excess sedation, cognitive impairment, and increased risk of accidents (Oster et al., 1990). Advise patients of risks when driving or operating machinery
- adverse sleep effects. Studies amongst people with sleeping disorders have demonstrated that insomnia sufferers who use benzodiazepines have a similar quantity but poorer quality of night-time sleep com-

### HIGH RISK GROUPS

- elderly:
  - higher risk of falls and fractures amongst the elderly (Leipzig et al., 1999). Through long-term use, many elderly patients have become dependent on benzodiazepines as a sleep aid and therefore find cessation very difficult. Accumulation of doses can readily cause oversedation and increase the risk of accidents
  - are higher users of prescribed medications for the management of chronic disease. Use of benzodiazepines in combination with some medications places the patient at increased risk of negative side effects and/or dependence
- polydrug and injecting users:
  - high-dose use, particularly amongst polydrug users, may result in extremely disinhibited, or uncharacter-

istic behaviour. Described as the 'Rambo syndrome', a person may engage in assaults, shoplifting or other activities, in full view of witnesses, and be unable to recall any events related to the offence

- benzodiazepine use appears to be increasing amongst injecting drug users, and is associated with a higher rate of HIV risk-taking behaviour (Darke et al., 1992)
- risk of overdose in people using heroin is increased when other CNS depressants, such as alcohol and benzodiazepines, are used (Zador et al., 1996)

## PRESCRIBING BENZODIAZEPINES

### Rational Use of Benzodiazepines

The following guidelines (RACGP, 2000) are recommended when prescribing benzodiazepines:

- avoid prescribing benzodiazepines to people suspected of using other psychoactive drugs
- advise all patients prescribed benzodiazepines of the risk of dependence
- to prevent inadvertent dependence, encourage patients to see the same doctor for repeat prescriptions
- prescribe benzodiazepines in the lowest possible dose for the shortest possible time
- reduce benzodiazepine dose only with the patient's consent and cooperation
- rely on non-pharmacological approaches to manage anxiety and insomnia
- before writing a repeat prescription for benzodiazepines, undertake a review of all medications (and ask about visits to other general practitioners)

To reduce access and harm resulting from the prescribing of multiple, single, high-dose prescriptions, write short-term prescriptions and encourage regular review. There is a high risk associated with prescribing large quantities of benzodiazepines (and other drugs of dependence).

### Precautions

Benzodiazepines should be used with caution in patients:

- with renal failure
- with liver disease
- with respiratory disease
- in late pregnancy
- who are breastfeeding

### Drug Interactions

Refer to Table 11-1.

### Use in Management of Anxiety and Insomnia

Benzodiazepines are effective for relief of anxiety symptoms and will induce sleep if given in sufficient doses (Therapeutic Guidelines Ltd., 2000). Research has questioned the efficacy of prescribing benzodiazepines for symptom reduction in anxiety management, with studies demonstrating that counselling alone has similar, if not greater efficacy (Catalan et al., 1984).

Benzodiazepines have demonstrated efficacy in the short-term management of insomnia, however similar results have not been demonstrated for periods longer than two weeks (NHMRC, 1991). Insomnia should be regarded as a symptom requiring assessment and evaluation, rather than a diagnosis per se. A sleep-wake history may reveal the patient to:

- be functioning normally on the amount of sleep obtained

- have unrealistic expectations of the requirements for sleep
- have a disorder of the sleep–wake schedule (including problems associated with shift work) which is not improved with hypnotics
- have a specific sleep disorder such as sleep apnoea or narcolepsy, in which case hypnotics are contraindicated

Finally, many patients who have taken benzodiazepines for periods in excess of 4–6 months have unwittingly become, dependent and experience withdrawal insomnia (Busto et al., 1986; Therapeutic Guidelines Ltd., 2000).

## MANAGEMENT AND INTERVENTION STRATEGIES

### Reviewing Benzodiazepines in Long-term Users: A Staged Approach

- advise the patient you want to review their benzodiazepine medication with them
- assess dosage and pattern of use
- assess use of alcohol and other psychotropics
- assess withdrawal symptoms
- assess reported and observed side effects

**Table 11–1**  
**Drug interactions**

Interacting drug	Mechanism of interaction	Clinical effect
Alcohol or other CNS depressants	additive effect	increased sedation
Antacids, anticholinergics	decreased absorption	delayed onset of acute clinical effects of benzodiazepines
Oral contraceptives, isoniazid	reduction in metabolism	prolongation of elimination half-life and effect of benzodiazepine
Cimetidine	inhibition of metabolism	increased toxic effects due to elevated plasma concentrations of diazepam
Rifampicin	increased metabolism	elimination half-life of benzodiazepine shortened
Digoxin	protein binding diazepam altered	increased digoxin levels
L-dopa	unknown	exacerbation of parkinsonian symptoms
Disulfiram	decreased metabolism	increased effects of benzodiazepine

Source: Norman et al. (no date, p. 37)



- assess history of depression
- assess other medical problems (e.g. pain)
- discuss long-term use with the patient
- discuss withdrawal symptoms with the patient
- finalise the management plan

(Mant & Walsh, 1997; NPS News, 1999)

## Dependence and Withdrawal

- tolerance and withdrawal from benzodiazepines can occur in individuals who have been taking therapeutic doses of benzodiazepines for two or more weeks
- it is estimated that symptoms may occur in up to 45% of patients discontinuing low therapeutic doses and up to 100% of patients for high doses
- there is a significant risk of withdrawal if benzodiazepines are discontinued abruptly, particularly in the sick and elderly.

### Symptoms of withdrawal

Commonly include:

- insomnia
- anxiety
- irritability
- restlessness
- agitation
- depression
- tremor
- dizziness

Less common but medically serious:

- seizures (high dose ± alcohol)
- delirium

Other symptoms include:

- muscle twitching and pains
- anorexia, nausea
- metallic taste

- fatigue
- tinnitus
- hyperacusis, photophobia, perceptual disturbances
- depersonalisation, derealisation
- blurred vision

### Principles when helping the patient withdraw from benzodiazepines

- withdrawal must be gradual (e.g. 10–20% per week, slowing reduction at lower doses e.g. < 15 mg diazepam)
- a reducing regime will generally take 6–8 weeks (or longer especially with higher doses)
- make a contract with the patient
- gradually reduce the patient's dose using a set reducing dosage over a set time period (e.g. reduce the most important dose of the day by ¼ of the tablet)
- consider converting the patient to a benzodiazepine with a long half life e.g. diazepam, to reduce the severity of withdrawal symptoms (see benzodiazepine equivalence table below)

**Table 11–2**  
Benzodiazepine equivalence table

5 mg diazepam	= 0.5–1 mg alprazolam
	= 3–6 mg bromazepam
	= 10 mg clobazepam
	= 0.5 mg clonazepam
	= 1–2 mg flunitrazepam
	= 1 mg lorazepam
	= 5–10 mg nitrazepam
	= 15–30 mg oxazepam
	= 10–20 mg temazepam
= 0.25 mg triazolam	

- titrate the dosage reduction according to patient symptoms
- discuss sleep and stress management, diet and exercise
- review regimen weekly
- provide support, reassurance and explanation

(NPS News, 1999)

Dose equivalents are approximate, some drugs at higher doses may be more potent.

### Aged Care Residential Facilities

Prescribing for residents in aged care facilities (and other residential facilities) presents special difficulties. Accreditation of aged care facilities has heightened awareness of responsibilities of the facility for quality use of medicines (Australian Pharmaceutical Advisory Committee, 1997). This responsibility includes drug utilisation review by an accredited pharmacist. The use of benzodiazepines is lower where staff have received education in geriatric care and where the organisational culture is supportive (Roberts et al., 1998).

Benefits for the elderly in aged care accommodation following successful reduction in rates of benzodiazepine use include:

- increased mobility
- increased alertness
- reduced incontinence
- improved wellbeing (Gilbert et al., 1993)

## BENZODIAZEPINE MISUSE

### Habitual Drug Users ('Doctor Shoppers')

Almost all GPs come across patients who may be obtaining prescriptions from several doctors.

The following may help in responding effectively (Mant et al., 1997):

- do not prescribe a benzodiazepine on the first visit
- there is rarely a valid indication for benzodiazepines in young people
- say 'no' from the start to the patient's requests for the prescription, whilst offering your help as a doctor
- take the opportunity to discuss risks associated with drug use and consider referral to a specialist agency

### Drug Dependent Patients

The RACGP (2000) has endorsed the following protocol for prescribing benzodiazepines in high doses on a regular basis the definition of which is 'more than three occasions per month for more than two months in any one year.' There are high risks with patients seeking large quantities of benzodiazepines (and other drugs of dependence) from one prescriber or from multiple prescribers. Most high dose users cannot be managed with an ordinary script.

The protocol aims include the support of quality medical practice, reducing overdose deaths and indiscriminate prescribing to polydrug users while reducing barriers to doctors seeing drug-dependent patients.

A protocol for prescribing benzodiazepines in high doses on a regular basis (RACGP, 2000) follows.

Where relevant and appropriate the following should be undertaken and adequately documented in the medical record:

- a full history, including use of alcohol and other drugs and psychiatric comorbidity
- adequate physical examination
- problem/diagnosis list
- management plan, which should include the following:
  - consultation with another medical practitioner with experience in management of drug dependence
  - communication with other prescribers, notably methadone prescriber
  - supply of specified small quantities (e.g. daily), whether at the surgery or, if applicable, at a community pharmacy.
  - communication with the Health Insurance Commission to clarify whether the patient is seeing multiple doctors for prescriptions for benzodiazepines and/or narcotic analgesics
  - monitoring of consumption where applicable by the Health Insurance Commission, with agreement by the patient to attend only one doctor and one pharmacy and signed consent to the doctor receiving feedback on actual consumption for the period of the contract

## REFERENCES

- AIHW (Australian Institute of Health and Welfare) 2001, *2001 National Drug Strategy Household Survey: Detailed Findings*, Drug Statistics Series no.11, AIHW Cat. no. PHE 41, AIHW, Canberra.
- Ashton, H. 1991, 'Protracted withdrawal syndromes from benzodiazepines', *Journal of Substance Abuse Treatment*, 8, pp. 19–28.
- Australian Pharmaceutical Advisory Committee 1997. *Integrated Best Practice for Medication Management in Residential Aged Care Facilities*, AGPS, Canberra.
- Busto, U., Sellers, E.M., Naranjo, C.A., Cappell, H., Sanchez-Craig, M. & Sykora, K. 1986, 'Withdrawal reaction after long-term therapeutic use of benzodiazepines', *New England Journal of Medicine*, 315, pp. 854–859.
- Cape, G., Hulse, G., Robinson, G., Mclean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-hypnotics' in Hulse, G.K. (ed.), White, J.J. & Cape, G., *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.
- Catalan, J., Gath, D., Edmonds, G. & Ennis, J. 1984, 'The effects of non-prescribing of anxiolytics in general practice — 1: controlled evaluation of psychiatric and social outcome', *British Journal of Psychiatry*, 144, pp. 593–602.
- Darke, S., Hall, W., Ross, M. & Wodak, A. 1992, 'Benzodiazepine use and HIV risk-taking behaviour amongst injecting drug users', *Drug & Alcohol Dependence*, 31, pp. 314–36.
- Gilbert, A., Owen, N., Innes, J.M. & Sansom, L. 1993 'Trial of an intervention to reduce chronic benzodiazepine use amongst residents of aged-care accommodation', *Australian & New Zealand Journal of Medicine*, 23, pp. 343–7.
- Leipzig, R.M., Cumming, R.G. & Tinetti, M.E. 1999, 'Drugs and falls in older people: a systematic review and meta-analysis. 1. Psychotropic drugs', *Journal of the American Geriatric Society*, 47, pp. 30–39.
- Mant, A., de Burgh, S., Yeo, G., Letton, T. & Shaw, J. 1997, *Anxiety and Insomnia — Think Twice Before Prescribing*, 3<sup>rd</sup> edn., RACGP (The Royal Australian College of General Practitioners), Melbourne.
- Mant, A. & Walsh, R.A. 1997, 'Reducing benzodiazepine use', *Drug and Alcohol Review*, 16, pp. 77–84.
- NHMRC (National Health and Medical Research Council) 1991, *Guidelines for the Prevention and Management of Benzodiazepine Dependence*, vol. 14, AGPS, Canberra.
- Norman, T.R., Ellen, S.R. & Burrows, G. (no date), 'Benzodiazepines in anxiety disorders: managing therapeutics and dependence' *MJA Practice Essentials*, p.37, [www.mja.com.au/public/mentalhealth/articles/norman/norman.html](http://www.mja.com.au/public/mentalhealth/articles/norman/norman.html).

- NPS News 1999, *National Prescribing Service Newsletter and Prescribing Practice Review*, No 4 (July), National Prescribing Service Limited, Sydney.
- Oster, G., Huse, D.M., Adams, S.F., Imbimbo, J. & Russell, M.W. 1990, 'Benzodiazepine tranquilizers and the risk of accidental injury'. *American Journal of Public Health*, 80, pp. 1467–70.
- PBS (Pharmaceutical Benefits Scheme) 1998, *Australian Statistics on Medicines*, Dept. of Health and Ageing, Canberra.
- RACGP (Royal Australian College of General Practitioners) 2000, *RACGP Guidelines for Rational Use of Benzodiazepines*, Update October, RACGP, Melbourne, [www.racgp.org.au](http://www.racgp.org.au).
- Roberts, M.S., King, M., Stokes, J.A., Lynne T.A., Bonner, C.J., McCarthy, S., Wilson, A., Glasziou, P. & Pugh, W.J. 1998 'Medication prescribing and administration in nursing homes', *Age and Ageing*, May, 27, pp. 385–392.
- Therapeutic Guidelines Ltd. 2000, *Therapeutic Guidelines: Psychotropic 2000*, Version 4, Therapeutic Guidelines Ltd., North Melbourne.
- Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, 2<sup>nd</sup> edn., Custodial Medical Unit, Mornington, Victoria.
- Zador, D., Sunjic, S. & Darke, S. 1996, 'Heroin-related deaths in New South Wales, 1992: toxicological findings and circumstances', *Medical Journal of Australia*, 164, pp. 204–7.

# Other Drugs

**T**HERE are some substances not covered in earlier chapters that are used in non-medical contexts. These drugs do not share a common pharmacology or pattern of effects and are used for different purposes in a variety of contexts. They include:

- hallucinogens
- 'party drugs'
- anabolic steroids
- over the counter drugs

It is important that use of non-prescription and complementary medicines is included in any drug use screening program.

## HALLUCINOGENS

While a range of drugs have potential to produce hallucinations (e.g. cannabis, amphetamines), the label 'hallucinogenic' indicates that this is the major purpose for which the drug is consumed. These drugs are normally administered orally, on an irregular basis. Harms are likely to arise from acute drug effects (especially behavioural and psychiatric sequelae) rather than regular or dependent use.

The most common hallucinogens include:

- lysergic acid diethylamide (LSD or acid)
- magic mushrooms (containing psilocybin and other active compounds)
- anticholinergics (pharmaceuticals and plant sources e.g. datura, angels' trumpet)

### Physical and Psychological Effects

The irregular use of hallucinogens means that most problems that arise are due to acute toxicity. While the exact symptoms vary, a common presentation is a person who is actively hallucinating, and is disturbed by the experience.

This can result from:

- an overdose (the strength of effect is greater than the person is accustomed to); or
- a 'bad trip' (the experience has become dysphoric rather than euphoric)

### Management and Intervention

Agitation and feelings of panic and loss of control may be prominent. The best response is to try and calm and reassure the person. A quiet, non-threatening environment is important. If this is not successful, administration of a benzodiazepine (e.g. diazepam) may be required.

In isolated cases the symptoms do not completely subside when the drug effect has ceased. Some patients report daily recurrence of the unpleasant episode and may need psychiatric referral.

Other signs and symptoms depend on the drug consumed. LSD and psilocybin produce sympathomimetic effects including tachycardia, tremor, hyperreflexia. These are not usually problematic, but can be if the person has overdosed. Anticholinergic overdose is life-threat-

ening and the effects may persist for many hours and even days. Physostigmine has been used, but treatment is usually conservative.

## PARTY DRUGS

*Party drugs* is a term used to describe substances taken in the context of 'raves', night-clubs or similar situations. Two of the main party drugs, amphetamines and ecstasy, have been discussed in earlier chapters in this Handbook.



See Chapters 6 & 7  
Amphetamines; Ecstasy

Other drugs used as party drugs include:

- LSD (see above)
- GHB (gamma hydroxybutyrate)
- ketamine

Party drugs (sometimes known as '*club drugs*' or '*dance drugs*') are frequently taken in combination with other drugs, including alcohol. This practice increases the risk of intoxication, overdose and other harms.



See Chapter 1  
Overview and Introduction  
'Polydrug Use', p. 5



## GHB

Gamma hydroxybutyrate (GHB) is a clear, odourless and fairly tasteless powder usually taken in the form of a solution.

Street names include:

- liquid ecstasy
- fantasy
- GBH (grievous bodily harm)

GHB occurs naturally in some mammalian cells and is structurally similar to gamma aminobutyric acid (GABA). A synthetic form was initially developed as a hypnotic agent and is easy to manufacture.

### **Physical effects**

GHB is absorbed rapidly and reaches peak plasma concentrations in 20–60 minutes.

Common effects include:

- placidity
- mild euphoria
- pleasant disinhibition

Unpleasant side effects may include:

- drowsiness
- dizziness
- nausea
- vomiting

GHB has a steep dose response curve and consequent narrow therapeutic index. There is wide interpersonal variation in tolerance and metabolism. It is easy to overdose. Adverse effects usually subside within 12 hours.

### **Detection and assessment**

GHB is very difficult to detect or measure in body fluids. Taking an oral history is the best method for assessing GHB use.

### **Management and intervention**

In milder cases of intoxication, supportive treatment ensuring adequate respiratory function should be provided.

GHB overdose is a real danger, usually occurring within 15–20 minutes of ingestion. Most fatalities associated with GHB occur when it is taken with other substances, most notably alcohol. It may present as:

- nausea and vomiting
- seizures
- aggressive outbursts
- respiratory depression
- coma

Table 12–1 outlines the features of the management of GHB intoxication, as described by McDowell (1999).

## Ketamine

Ketamine is a dissociative anaesthetic and n-methyl-d aspartate (NMDA) receptor antagonist. It has recently become popular amongst party drug users. It may be sold as ketamine or as a constituent of pills sold as 'ecstasy'.

Its street names include:

- K
- super K
- vitamin K
- special K

Ketamine is usually snorted but may also be injected or taken orally.

### **Physical effects**

Ketamine has a rapid onset but short duration (1–2 hours) of action. Dosage titration is difficult and the effects are highly sensitive to setting.



**Table 12-1**  
**Management of GHB intoxication**

For spontaneously breathing patients:

1. Maintain oxygen supplementation and intravenous access
2. Maintain comprehensive physiological and cardiac monitoring
3. Attempt to keep the patient stimulated
4. Use atropine for persistent symptomatic bradycardia
5. Admit the patient if he or she is still intoxicated after 6 hours
6. Discharge the patient if he or she is clinically well in 6 hours

Patients whose breathing is laboured should be managed in an intensive care unit.

- 'bad trips' (known as the 'K hole')
- nausea and vomiting (especially if taken with alcohol)
- tachycardia
- chest pain
- hypertension
- temporary paralysis
- analgesia and sensory dissociation thereby creating a high risk of accidental injury
- coma

Ketamine can create dependency in some individuals (McDowell, 1999, p. 301).

### **Management and intervention**

Most clinical presentations are short lived and require symptomatic relief and observation. An environment with low lighting and stimulation should be provided. Levels of patient anxiety should be closely monitored.

Ketamine can cause:

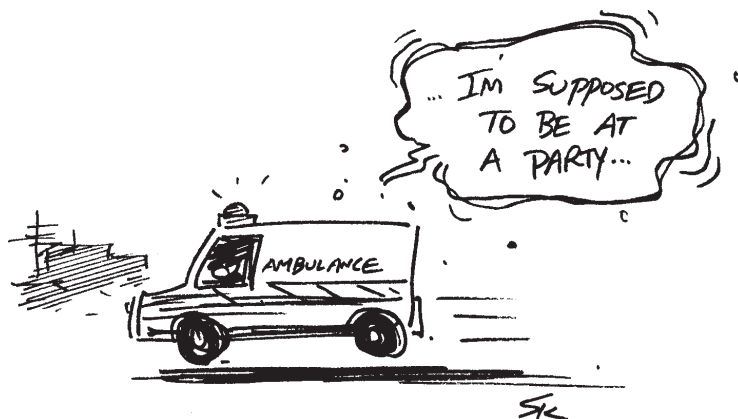
- thought disorders, out of body experiences, aphrodisiac effects, hallucinations and other perceptual distortion (psychedelic effects)
- stimulant effects

Adverse effects can include:

- anxiety
- agitation

## **ANABOLIC STEROIDS**

Anabolic steroids are synthetic variations of the male sex hormone testosterone. Traditionally, they have been associated with enhancing sporting performance but are now widely used for cosmetic reasons to modify body shape. Anabolic steroid use is currently dominated by males but a growing number of women use these drugs.



They are primarily taken orally or by intramuscular injection, generally in cycles with a period of use followed by a period of abstinence. The length of a cycle can vary widely but an average would be 6 to 8 weeks with a small number of people using continuously.

Anabolic steroids are commonly used in 'stacks', that is, a number of anabolic steroids or other drugs are taken at the same time.

A vast array of side effects have been associated with anabolic steroids. These range from relatively minor cosmetic changes such as acne, lowering of the voice and baldness to potentially life threatening complications involving the cardiovascular system, liver and kidneys.

## OVER THE COUNTER DRUGS

A number of over the counter drugs have psychoactive effects. Drugs in the following medication groups can cause concern:

- analgesics
- antihistamines
- sympathomimetics
- cough suppressants

## Non-prescription Medication

Few data are available on the extent of intentional misuse of non-prescription pharmaceuticals. However anecdotal accounts (and some monitoring by State authorities) indicate cause for concern in the following medication groups:

### **Analgesics**

*Paracetamol, codeine*

Concern for codeine-containing products:

- codeine is an opioid and can be used to make home-bake heroin
- need to be aware of the potential for abuse and sale as a street drug

- abuse of paracetamol/codeine/doxylamine succinate combinations, i.e. analgesic plus antihistamine
- combination products (e.g. codeine plus paracetamol) increase the likelihood of hepatic damage from high dose paracetamol

### **Antihistamines**

*Chlorpheniramine, dexchlorpheniramine, diphenhydramine, pheniramine, promethazine hydrochloride, trimепразине*

- used alone or in combination with analgesics or sympathomimetics. There is little therapeutic justification for these combination products and recommendation should be avoided
- use of older style antihistamines for sedative effects
- the combination with alcohol increases sedation
- paradoxical stimulation, including hallucinations, can occur, particularly at higher doses

### **Sympathomimetics**

*Pseudoephedrine, phenylpropanolamine, phenylephrine*

- potential for misuse by people dependent on stimulants
- high potential for diversion into manufacture of amphetamines
- concern for use in pregnancy
- overdose causes tachycardia, palpitations, and more rarely arrhythmias and seizures

### **Cough suppressants**

*Codeine, dihydrocodeine, dextromethorphan, pholcodeine*

- often available in combinations with antihistamines and many other drugs

## **Injecting Drug Users**

Dose form is cause for extreme caution; in particular requests for liquid preparations (e.g. cough and cold mixtures) or preparations in soft gelatin caps (e.g. diphenhydramine gel caps, ibuprofen/codeine gel caps).

### ***Other***

Anecdotal reports of experimentation with use of a wide range of products including eye drops and complementary medicines are not uncommon.

### REFERENCES

McDowell, D.M. 1999, 'MDMA, Ketamine, GHB and the "Club Drug" Scene', cited in Galanter, M. & Kleber, H.D. (Eds.) 1999, *Textbook of Substance Abuse Treatment*, 2<sup>nd</sup> Edn., American Psychiatric Press, Washington D.C., p. 301.

# Other Drugs

# Non-medical Interventions



# Psychosocial Interventions

**T**HERE is a strong evidence base for the efficacy of psychosocial interventions in addressing problematic alcohol and drug use. A number of different models and approaches are outlined below. However, there are key features of psychosocial interventions that are consistent across all of these approaches.

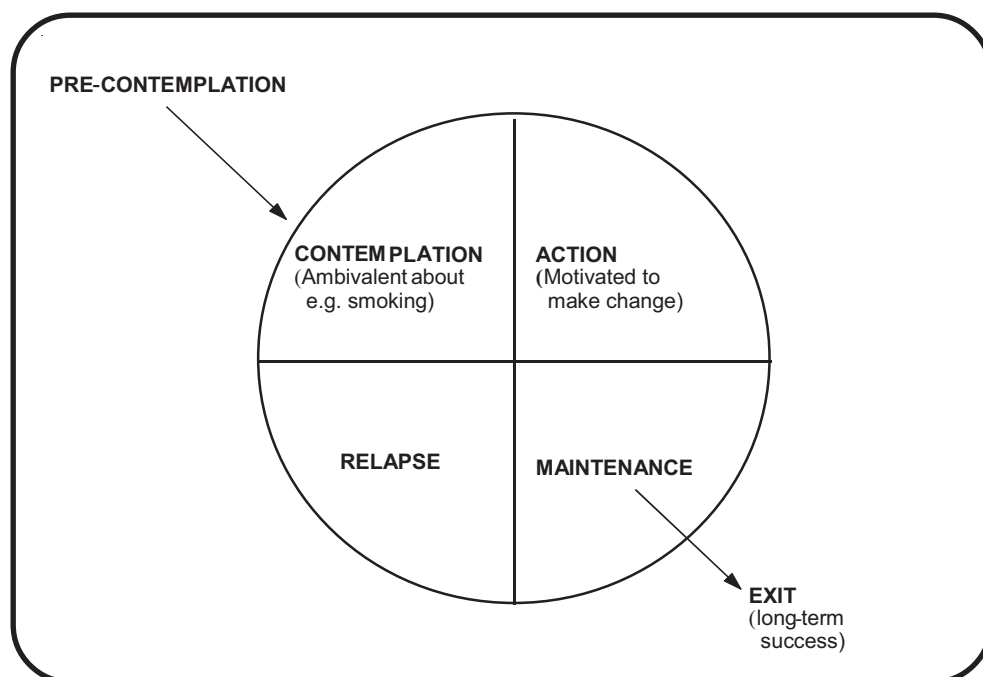
This Chapter briefly covers the following clinical strategies:

- patient centred approaches
- decision balance
- building a therapeutic alliance
- motivational interviewing
- problem solving
- goal setting
- relapse prevention
- quality of life



## PATIENT READINESS — A MODEL OF THE PROCESS OF CHANGE

People do give up harmful drug use, sometimes assisted by formal interventions, other times without such help. Having some understanding of the process of change can guide clinical effort. Prochaska and DiClemente (1986) developed a useful five-stage cyclical model of the process. While empirical support for this model is limited the model can still help structure the focus of interventions. Clinicians should use the model with caution, avoiding simplistic and rigid categorisations of patients.



**Figure 13-1**  
**Model of the process of change** (progress usually occurs in a clockwise direction)  
Source: Prochaska and DiClemente (1986)

### Pre-contemplation Stage

During Pre-contemplation the pros of continuing use outweigh the cons of continued drug use. Disadvantages of change outweigh advantages. You may be concerned about some consequence of your patient's drug use, but the patient may accept this as collateral damage.

Commonly, there is resistance to 'action oriented interventions' and explanations about how to 'give up', but relevant information about risks, and how to avoid or minimise them, may be well received. For example, a person injecting amphetamines might welcome information about how to avoid blood borne viruses or how to manage sleep disorders. A heroin user may be keen to get advice on how to avoid overdose.

Use motivational interviewing (see p. 172) to help the patient explore the advantages and disadvantages of current patterns of drug use.

### Contemplation Stage

The balance of costs and benefits begin to shift, although there is still ambivalence about change.

*'I should give up because of all the problems. But what am I going to do instead? — I'll miss it and my friends.'*

Explore this ambivalence using motivational interviewing.

### Preparation Stage

The balance has shifted. The patient is preparing to take action and has confidence in their capacity to change. Change is seen as worthwhile. This is often a planning stage. Goal setting, identifying internal and external supports/resources and identifying strategies to support change can help.

### Action Stage

The patient is taking steps to change. Support and specific skill training can be provided. Review initial reasons that led to the decision to change.

### Maintenance Stage

Changes in behaviour maintained for six months or more are usually associated with substantial improvements in the quality of life (e.g. housing, employment, relationships, physical and mental wellbeing). Without such changes, the effort to change may not seem worth it and relapse is more likely. Encourage patients to articulate the positive reasons for maintaining change to reinforce their decisions.

## CLINICAL STRATEGIES

The following set of empirically tested clinical strategies can facilitate a good therapeutic relationship, enhance quality of support provided and maximise probability of behaviour change.

### Patient-centred Approach

The patient-centred approach is outlined in Table 13–1.

### Decision Balance

The Decision Balance can help you to better understand the first two ingredients of change. It can help people re-assess the pros and cons of substance use. It can be used either during the consultation or completed by the patient at home.

**Exercise:** The patient is asked to first consider advantages and disadvantages of their current situation as a substance user. Then to think ahead to an imagined scenario when they no longer use. Consider the advantages and disadvantages of change. The health professional's role is to listen and reflect back the patient's self re-evaluation without judging or hurrying the process to a premature conclusion.



See Appendix J

## Building A Therapeutic Alliance

Even when a treatment is delivered as specified, clinicians can significantly influence outcome. It is important to build a relationship, based on trust, where the patient can communicate concerns without fear of being judged.

**Table 13–1**  
**Patient-centred approach**

<p>Regard the person's behaviour as their personal choice</p>	<ul style="list-style-type: none"> <li>• acknowledge benefits as well as costs to behaviour</li> <li>• understanding and acknowledging the patient's choices enhances their autonomy and responsibility</li> </ul>
<p>Let the person decide how much of a problem they have, i.e. how important it is for them to quit</p>	<ul style="list-style-type: none"> <li>• systematically explore benefits (likes) and costs (dislikes) as perceived by the patient</li> <li>• use examples and issues raised by the patient</li> <li>• encourage the patient to rate their motivation and confidence out of 10. If score is low, explore what would increase this score. If the score is high, why?</li> </ul>
<p>Avoid argumentation and confrontation</p>	<ul style="list-style-type: none"> <li>• confrontation <i>within</i> the patient is the goal</li> <li>• separate <i>information</i> from '<i>persuasive imperative</i>'</li> </ul>
<p>Encourage discrepancy</p>	<ul style="list-style-type: none"> <li>• change is likely to occur when behaviour is seen to be in conflict with personal goals</li> <li>• use the Decision Balance to identify discrepancies</li> </ul>
<p>Help patients re-evaluate their substance use</p>	<p>Three ingredients are necessary for any behaviour change:</p> <ul style="list-style-type: none"> <li>• concern with current behaviour</li> <li>• belief that change will lead to improvement</li> <li>• belief that change is possible (self-efficacy)</li> </ul>

Improved treatment adherence, treatment retention and treatment outcome result from clinical practice that:

- is empathetic
- communicates confidence and optimism
- facilitates informed choice of treatment method and treatment goal
- is non-judgmental and genuine

### ***Empathy and reflective listening***

Empathy involves:

- listening to the patient
- understanding the patient and their concerns
- communicating this understanding to the patient — formulate a brief response that captures the essence of what the patient is trying to communicate

This is to help both the patient and the clinician better understand, and to help devise action

based on this understanding (Egan, 1994). You are seeking to understand the patient's unique experience of drug use and related harm.

Listening and communicating understanding involves several micro-skills including:

### **Using open ended questions**

Closed questions may be useful for getting factual information, but discourage exploration by the patient.

Avoid questions such as:

*'Do you inject more often now?'*

Instead, ask open questions such as :

*'How has your injecting changed over time?'*

*'What can you tell me about your injecting?'*

### **Reflective listening**

Reflective listening is a way to check whether what you 'heard' is what the patient intended. It allows the patient to correct any wrong understanding and add more information. It is also a way to feed back the patient's concerns.

Some reflective listening is made as a statement rather than a question. However, sometimes a patient (e.g. a young person) could be reluctant to correct a statement by an authority figure. In this case it might be useful to use simple reflective questions (i.e. invite correction). Because many communications (verbal or otherwise) can have several levels of meaning, it is important to be tentative rather than too definitive.

Reflective listening can include:

- simple reflection back to the patient:

*'You don't see why your amphetamine use is a problem when your friends use more than you and they don't seem to have any problems.'*

- amplified reflection attempts to elicit the other side of the patient's ambivalence by amplifying or exaggerating (not in a sarcastic way):

*'So if your friends don't have any problems, there's nothing for you to worry about.'*

Clearly you need to be cautious using such a strategy.

- double sided reflection:

*'I can see how this might be confusing. On the one hand you're here because you have some concerns about your amphetamine use. On the other hand, you don't seem to be using more than your friends.'*

### **Summarising**

An effective clinician can attend to, understand and summarise information. This skill can involve the ability to:

- highlight main discoveries
- encourage exploration of more detail
- give patients the opportunity to hear their own concerns or reasons for change
- highlight ambivalence (to change or stay the same)

### **Roadblocks to empathy**

There are a number of common 'roadblocks' that can prevent empathy (Jarvis et al., 1995). These include:

- ordering or commanding
- warning or threatening
- arguing or persuading
- moralising
- ridiculing or labelling
- giving advice or providing solutions

It is also important to avoid:

- insincerity
- repetition
- clichés
- using jargon
- collusion



See Jarvis et al. (1995) for more detail of these skills.

## Motivational Interviewing

Many patients are strongly attached to drug use. This attachment to behaviours that cause harm perplexes many clinicians. As a result, some confront patients, only to find it unsuccessful, often generating more resistance. Motivational interviewing has been proposed as a method to work with ambivalence and help patients explore *their* reasons to change drug use.

### Elements of motivational interviewing

Motivational interviewing involves the following (Miller & Rollnick, 1991):

- express empathy  
Motivational interviewing consists of more listening and less ‘telling’.
- develop discrepancy  
Focus the patient’s attention on discrepancy:

*‘I like using heroin, **but** I hate the hassles with my family and the police.’*

This can include raising awareness:

*‘How do you see the connection between your smoking and your poor respiratory health?’*

- avoid argumentation

The *patient*, and not the clinician, is encouraged to argue for change.

- roll with resistance

Try *not* to provide solutions. Provide opportunity for the patient to identify solutions (sometimes with the clinician’s help). If the patient resists, this may be an indication that you are taking a wrong approach. Help the patient consider issues from other perspectives. For example:

- ask the patient their view of your clinical findings
- ask the patient what they think the view of a significant other might be etc.

- support self-efficacy

The patient’s confidence in their ability to implement and sustain changed behaviour will influence whether or not they attempt and persist with efforts to change.

### Guidelines for motivational interviewing

In motivational interviewing you:

- explore positive and negative consequences of drug use
- provide opportunity to explore the patient’s specific concerns
- use reflective listening and summaries to understand and communicate understanding
- elicit self-motivational statements:

*‘What are the things you like and don’t like about your cannabis use?’*

*‘What have other people said about your drinking?’*

*‘What makes you think you might need to change?’*

- help the patient decide whether to change:

*'Where does this leave you now?'*

*'What does this mean for your drug use?'*

Avoid:

- arguing with a patient
- imposing a diagnostic label on them
- telling them what they must do
- trying to break down denial with confrontation

It should never feel as though you are confronting the patient. It should feel you and the patient are confronting the problem(s) together.

## Brief Motivational Interviewing

Brief motivational interviewing and opportunistic interventions are well researched (Rollnick et al., 1999). Two factors are central and clinically useful:

- importance — e.g. some think it is important to quit smoking but are not clear how they can do it
- confidence — e.g. some are confident they can change, but it is not important to them

Brief motivational interviewing consists of the following seven components:

### Scaling questions

Ask questions such as:

*'On a scale of 0–5 how important is it for you to give up smoking?'*

*'On a scale of 0–5 how confident are you about giving up?'*

You can use scaling to help quickly identify the most important areas to work on. You can then use this information:

*'Why is it so high?'* (Even if a '1' 'Why isn't it a zero?')

*'What will help keep you at this level?'*

*'What will help you move higher?'*

*'How high does it have to be before you make an attempt to change?'*

*'What can I do to help?'*

### Exploring importance

*'What are the benefits of your cannabis use?'*

*'What are some of the less good things?'*

### Summarise

*'Where does that leave you now?'*

### Building confidence

*'In the past, what has been helpful when you have tried to change your drug use?'*

*'Is there anything you can learn from these past attempts?'*

*'Is there anything you can learn from other people's attempts to change?'*

### Exchanging information

How you share information and your expertise is important.

*'How much do you already know about the risks of injecting?'*

*'Some people find that ...how about you?'*

*'How do you see the connection between your amphetamine use and your sleeping problems?'*

*'Is there anything more you'd like to know about injecting?'*

## **Reducing resistance**

Express empathy, especially about the difficulty of changing.

Emphasise personal choice and control.

Don't try to provide solutions — invite the patient to collaborate in providing a solution. The onus is then on the patient, *not you*, to make a decision to change.

## **Summarise and invite action**

*'What do you think you should do about your cannabis use?'*

(Based on Mason, 1997).

## **Problem Solving**

The ability to respond effectively to problems is associated with improved treatment outcome. Supporting development of problem solving skills can be clinically useful and is best achieved through:

- a combination of verbal and written information
- demonstration (when possible)
- learning through practice and feedback

Developing problem solving skills can consist of identifying occasions when the patient has solved other problems and noting the steps they took.

Effective problem solving consists of five steps that can be learned:

### **1. Orientation**

Stand back from the problem; view it as a challenge, not a catastrophe. How might someone else solve this?

### **2. Define the problem — it is important to be specific**

Patient: *'My wife and I do not get on'*

Clinician: *'Give me an example of what you mean'*

Patient: *'She doesn't like me being out on Friday nights'*

### **3. Brainstorm solutions**

At this stage, anything goes. Identify as many solutions as possible — discourage evaluation and a search for quality.

### **4. Decision making**

The patient (with the help, but not direction, of the clinician) reviews the positives and negatives of each of the options, and their ability to implement them, and makes an informed choice of the best option(s) to embrace.

### **5. Implementation**

A plan of action is developed and the option is implemented. Sometimes it is useful to rehearse the option (where possible) to test out the viability of the strategy and to increase self-efficacy (confidence).

It is not your responsibility to solve the patient's problems, but to teach a skill that he or she can use in a variety of circumstances.

## **Goal Setting**

Effective goal setting is:

- consistent with the patient's 'stage of change' (e.g. a 'pre-contemplator' may resist a goal of total abstinence, but may embrace reducing the risk of infection)
- negotiated. Negotiation is not bestowed on a patient. It is a strategy to influence behaviour. Negotiated goals are more likely to generate patient commitment and adherence
- realistic
- specific and achievable. A broad therapeutic goal may be broken down into several component parts

- short-term; so that progress can be monitored and success quickly realised
- solution-focused, or defined in positive terms. Changing behaviour will be more successful if couched in positive terms of acquisition, rather than reduction; presence, not absence (e.g. increasing the number of days without heavy alcohol use as opposed to decreasing the number of drinking days)

### Relapse Prevention

Relapse is a common experience when changing drug using behaviour. Research evidence indicates that major predictors of relapse risk are belief systems consistent with disease models (*'I'm an addict and can't stop'*), and the absence of coping skills.

The following strategies are useful in preventing and managing relapse:

- enhance commitment to change (e.g. use motivational interviewing)
- identify high-risk situations (e.g. When does the patient use heavily? What situations have been associated with relapse in the past?)
- teach coping skills (e.g. problem solving; social skills; self-management skills; self-monitoring of drug use and drug-related harm)
- develop strategies that can be part of a relapse drill
  - what should the patient do in the event of a lapse occurring?
  - where can they get support?
  - what role can friends/family provide?
  - How soon should the patient make an appointment to come back to your practice?

### Quality of Life

Successful maintenance of change is associated with factors such as employment, the quality of relationships, financial security, housing and spiritual support (variously defined). You cannot be expected to address all these factors, but you may be able to facilitate access to a range of advice and support services.

These might include, but are not limited to:

- housing services
- financial support services
- legal advice
- employment, education and training



## REFERENCES

- Egan, G. 1994, *The Skilled Helper: a Systematic Approach for Effective Helping*, Brooks/Cole, California.
- 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.
- Mason, P. 1997, *Training People to Use Motivational Interviewing Skills*, National Centre for Education and Training on Addiction, Flinders University of South Australia, Adelaide.
- Miller, W.R. & Rollnick, S. 1991, *Motivational Interviewing: Preparing People to Change Addictive Behavior*, Guilford Press, New York.
- 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, pp. 3–27.
- Rollnick, S. Mason, P. & Butler, C. 1999, *Health Behaviour Change: a Guide for Practitioners*, Churchill Livingstone, Edinburgh.

# Alternative Therapies

**T**HE TERM 'alternative therapies' or 'complementary and alternative medicine' is applied to a diverse collection of non-orthodox therapeutic practices, including:

- acupuncture
- herbs
- homeopathy
- therapeutic massage
- traditional oriental medicine
- faith healing
- hypnosis
- chiropractic
- music therapy

The alternative therapies that have been most commonly applied to the treatment of substance use are acupuncture and hypnotherapy.

## EVIDENCE OF EFFECTIVENESS

There are variable levels of evidence in regard to alternative therapies. Assessing effectiveness is made difficult by the:

- small number of studies (particularly controlled studies)
- small sizes of studies
- considerable heterogeneity in participants
- interventions and outcome measures

Perhaps in part because of these factors, results of studies are frequently contradictory (Linde et al., 2001).

## Acupuncture

Acupuncture is a family of procedures involving stimulation of anatomical locations on the skin by a variety of techniques (Smith et al., 1997). The most studied form employs penetration of the skin by thin, solid, metallic needles, which are manipulated manually or by electrical stimulation. Acupuncture points may also be stimulated by pressure, heat, and lasers (NIH, 1997).

For the treatment of substance abuse, five points on the ear are most commonly used (McLellan et al., 1993).

Most research into the effectiveness of acupuncture relates to smoking cessation. In a systematic review of 18 studies of acupuncture for smoking cessation White et al. (2000) concluded that there is no clear evidence that acupuncture is effective for smoking cessation. This review looked at abstinence from smoking both early (up to 6 weeks) and late (6 to 12 months) after acupuncture treatment.

They found that:

- acupuncture was not superior to sham acupuncture, or any other anti-smoking intervention, at any time point after treatment
- acupuncture did appear to be superior to no intervention at the early follow-up, but this difference was not sustained

The US National Institute on Drug Abuse also concluded that there is no clear evidence that acupuncture is effective compared to placebo

or to existing treatments in:

- the detoxification
- primary rehabilitation
- relapse prevention of opioid or cocaine dependence

Conversely there is very little evidence that acupuncture is not effective in the treatment of these conditions (McLellan et al., 1993).

Two subsequent randomised controlled trials have also failed to identify either benefits or harms from acupuncture as an adjunct treatment for cocaine users (Bullock et al., 1999; Otto et al., 1998) or alcohol dependent out-patients (Sapir-Weise et al., 1999).

However, in a randomised controlled trial Avants et al. (2000) compared acupuncture to a relaxation control and a needle-insertion control for the treatment of cocaine-dependent methadone-maintained patients. The trial found that the acupuncture group was more likely to provide cocaine-negative urine samples than either of the two control groups. These researchers went to some effort to identify sham acupuncture sites with sufficiently low level of activity to be used as a control, supporting the view that selection of sham sites may be a factor influencing outcomes (NIH, 1997); however, the benefit of reduced cocaine use in the acupuncture group was countered by significantly lower rates of retention in treatment — the mean survival time was 5.2±3.0 weeks for the acupuncture group, 6.7±2.5 weeks for the needle-insertion control and 7.0±2.3 weeks for the relaxation control group.

### ***Adverse Effects of Acupuncture***

Recent surveys of acupuncture practitioners identified bleeding and pain at the needle site as the most common adverse effects. Aggravation of symptoms, fainting, nausea and vomiting, psychological and emotional reactions also occurred but much less frequently (MacPherson et al., 2001; White et al., 2001).

### **Hypnosis**

The exact definition of hypnosis is a matter of debate, but a generally accepted description would be 'a state of awareness that permits the patient to accept suggestions without censoring them' (Temes, 1999). It appears that hypnosis is generally seen as an aid in the treatment of substance use problems, and not a treatment in itself. A role it might play is in the reduction of anxiety, relaxation training, and in helping patients to learn to manage negative emotional states (Hall, 1999). Hypnosis may also help patients become more responsive to a treatment approach (Stoil, 1989).

Again most of the research into the effectiveness of hypnotherapy relates to smoking cessation. This research was the subject of a systematic review by Abbot et al. (2000) who were unable to show that hypnotherapy has a greater effect on six month quit rates than any other interventions or no treatment.

### **Conclusions**

Research evidence is scarce and confounded, but suggests that acupuncture and hypnotherapy are no more effective than placebo or existing approaches in the treatment of problematic substance use. However the risks of adverse effects are low. Consequently these approaches may be useful for some patients as part of a comprehensive management program (NIH, 1997). Indeed, it may be that any adjunct treatment will be beneficial for some patients (Richard et al., 1995).

## REFERENCES

- Abbot, N.C., Stead, L.F., White, A.R., Barnes, J. & Ernst, E. 2000, *Hypnotherapy for Smoking Cessation*, Cochrane Library, Issue 2, Update Software, Oxford.
- Avants, S.K., Margolin, A., Holford, T.R. & Kosten, T.R. 2000, 'A randomized controlled trial of auricular acupuncture for cocaine dependence', *Archives of Internal Medicine*, vol. 160, no. 15, pp. 2305–2312.
- Bullock, M.L., Kiresuk, T.J., Pheley, A.M., Culliton, P.D. & Lenz, S.K. 1999, 'Auricular acupuncture in the treatment of cocaine abuse. A study of efficacy and dosing', *Journal of Substance Abuse Treatment*, vol. 16, no. 1, pp. 31–38.
- Hall, H. 1999, 'Hypnosis and pediatrics', in *Medical Hypnosis: An Introduction and Clinical Guide*, Churchill Livingstone, Philadelphia, Pennsylvania, USA, pp. 79–93.
- Linde, K., Vickers, A., Hondras, M., ter Riet, G., Thormahlen J., Berman, B. & Melchart, D. 2001, 'Systematic reviews of complementary therapies—an annotated bibliography. Part I: Acupuncture', *BMC Complementary and Alternative Medicine*, vol. 1, no. 3, [www.biomedcentral.com/1472-6882/1/3](http://www.biomedcentral.com/1472-6882/1/3)
- McLellan, A.T., Grossman, D.S., Blaine, J.D. & Haverkos, H.W. 1993, 'Acupuncture treatment for drug abuse: a technical review', *Journal of Substance Abuse Treatment*, vol. 10, no. 6, pp. 569–576.
- MacPherson, H., Thomas, K., Walters, S. & Fitter, M. 2001, 'The York acupuncture safety study: prospective survey of 34,000 treatments by traditional acupuncturists', *British Medical Journal*, vol. 323, pp. 486–487.
- NIH (National Institutes of Health) 1997, 'Acupuncture', *NIH Consensus Statement*, vol. 15, no. 5, [www.odp.od.nih.gov/consensus/cons/107/107\\_intro.htm](http://www.odp.od.nih.gov/consensus/cons/107/107_intro.htm)
- Otto, K.C., Quinn, C. & Sung, Y.F. 1998, 'Auricular acupuncture as an adjunctive treatment for cocaine addiction. A pilot study', *American Journal on Addictions*, vol. 7, no. 2, pp. 164–170.
- Richard, A.J., Montoya, I.D., Nelson, R. and Spence, R.T. 1995, 'Effectiveness of adjunct therapies in crack cocaine treatment', *Journal of Substance Abuse Treatment*, vol. 12, no. 6, pp. 401–413.
- Sapir-Weise, R., Berglund, M., Frank A. & Kristenson, H. 1999, 'Acupuncture in alcoholism treatment: a randomized out-patient study', *Alcohol & Alcoholism*, vol. 34, no. 4, pp. 629–635.
- Smith, M.O., Brewington, V., Culliton, P.D., Ng, L.K., Wen, H.L. & Lowinson, J.H. 1997, 'Acupuncture', in Lowinson, J.H., Ruiz, P., Millman, R.B. & Langrod, J.G. Williams, and Wilkins, *Substance Abuse: A Comprehensive Textbook*, Baltimore. pp. 484–492.
- Stoil, M.J. 1989, 'Problems in the evaluation of hypnosis in the treatment of alcoholism', *Journal of Substance Abuse Treatment*, vol. 6, no. 1, pp. 31–35.

Temes, R. 1999, 'Welcome to Hypnosis', in *Medical Hypnosis: An Introduction and Clinical Guide* Churchill Livingstone: Philadelphia, Pennsylvania, USA, pp. 3–5.

White, A., Hayhoe, S., Hart, A. & Ernst, E. 2001, 'Adverse events following acupuncture: prospective survey of 32,000 consultations with doctors and physiotherapists', *British Medical Journal*, vol. 323, pp. 485–486.

White, A.R., Rampes, H. & Ernst, E. 2000, *Acupuncture for Smoking Cessation*, Cochrane Library, Issue 2., Update Software, Oxford.

# Alternative Therapies

# Issues for Special Consideration





# Pregnancy and Drug Use

**D**RUG and alcohol use during pregnancy is known to cause a range of adverse effects. Early detection and intervention with women who are pregnant and using drugs is an effective way of improving pregnancy outcome.

As with all other aspects of health care an empathic, non-judgemental approach will build rapport and foster a therapeutic relationship. This will allow a comprehensive assessment to be undertaken and ongoing care to be initiated.

## ASSESSMENT

The goals of the assessment process are to:

- determine the quantity and frequency of alcohol and other drug use from the date of the woman's last menstrual period
- establish whether substance use is continuing
- assess the possible impact of the substance use on the pregnancy
- provide factual information about the effects of alcohol and drug use during pregnancy
- explore a range of choices for action

**Determine the quantity and frequency of alcohol and other drug use from the date of the woman's last menstrual period**

Obtaining this information requires the same skills as for any other history. Detailed information on how to obtain a drug and alcohol history is available in Chapter 2.



See Chapter 2  
General Principles

**Establish whether substance use is continuing**

For most women confirmation of pregnancy is a powerful incentive to cease all alcohol and other drug use. However for a small percentage of women this is not possible because:

- they may be unaware of the possible risks of continued use
- they may not be able to stop by themselves
- it may be inadvisable for them to stop abruptly e.g. women dependent on heroin or benzodiazepines

An exploration of each woman's readiness to change and her resources to achieve change is necessary when undertaking an assessment.

Using a Motivational Interviewing style as described in Chapter 13 may be helpful.



See Chapter 13  
Psychosocial Interventions

**Assess the impact of substance use on the pregnancy**

Most women are very concerned about the effects of the drug use on their pregnancy and developing foetus. As many pregnancies are unexpected, women are concerned about damage they may have done prior to finding out about the pregnancy. Some feel pressure or are pressured into considering terminations because of their drug or alcohol use.



**Provide factual information about the effects of substance use during pregnancy**

Factual information about drug effects will assist women in deciding what is the best choice of action for their individual circumstances.

**Explore a range of choices for action**

A range of choices for pregnant women is desirable, whether they decide to continue with the pregnancy or to continue or modify their drug use. If a decision to continue the pregnancy is made, pregnancy (antenatal) care is a priority.

Adequate pregnancy care, even in the face of continued substance use will improve outcomes for the mother and baby.

The range of substance use treatment choices available will depend upon each individual's resources and the treatment options available. Consultation with and/or referral to specialist drug and alcohol treatment services is recommended.

The goal of intervention is to facilitate cessation or reduction of drug use or transfer to a safer alternative such as methadone maintenance or nicotine replacement therapy.

## INFORMATION ON ALCOHOL AND DRUG EFFECTS

The effects of substance use during pregnancy vary and are dependent on multiple factors:

- type and amount of substances used
- route of administration
- timing and duration of use
- concurrent drug use, particularly tobacco
- maternal nutrition and health status
- amount and quality of pregnancy care

Each woman's individual experience needs to be carefully assessed and possible risks explained. The art is to provide balanced information without scaring women unnecessarily and without minimising the possible risks of continued use.

Research on the extent and effects of prenatal exposure to alcohol and other drugs is complex and sometimes contradictory. There are multiple methodological challenges with this research. These include:

- finding appropriate samples of pregnant women
- difficulty in isolating effects of a particular substance
- determining the relationship between the effects and both the amount of substance used and the timing of use during pregnancy

Finally there is the issue of bias which may influence what studies are published (Brady et al., 1994).

Despite the attention given to illicit substances such as heroin and cocaine it is the licit substances, alcohol and tobacco that are the

more commonly used by women during pregnancy and whose known adverse consequences are more significant.

### Alcohol

Alcohol is the drug most commonly used by women in Australia and it can be toxic to the developing foetus. Research implicates alcohol in a wide range of prenatal (during pregnancy), foetal and infant effects.

Women are advised to think carefully about drinking alcohol during pregnancy however the infrequent consumption of one standard drink is thought to be unlikely to have any adverse consequences.

#### *Prenatal effects*

- increased risk of spontaneous abortion (miscarriage) and stillbirth
- premature birth
- reduced birth size and weight

#### *Foetal effects*

The effects of alcohol exposure on the foetus are related to:

- the amount of alcohol ingested
- the stage of pregnancy
- the general health of the woman

These effects occur along a continuum from a small decrease in cognitive functioning to Foetal Alcohol Syndrome (Day & Richardson, 1994).



[www.nhmrc.gov.au](http://www.nhmrc.gov.au)

### **Foetal Alcohol Syndrome (FAS)**

The cardinal features of FAS include:

- slow growth before and after birth involving height, weight and head circumference
- anomalies of brain structure and function resulting in development delay and disability
- a consistent pattern of birth defects including minor structural anomalies of the face together with heart and limb deformities in some instances

Severe effects of alcohol use during pregnancy such as foetal death, severe developmental disability and the cranio-facial deformities are associated with chronically high alcohol intake throughout pregnancy i.e. 42 standard drinks per week or more (Jacobson & Jacobson, 1994).

Infants born to women who are dependent on alcohol are at risk of developing withdrawal.

### **Alcohol and breastfeeding**

The level of alcohol in breast milk is the same as the woman's blood alcohol level. The infant's brain is sensitive to alcohol therefore alcohol use while breastfeeding is not recommended.

For women who do not wish to stop using alcohol during lactation the following harm reduction strategy is suggested:

- consume alcohol at times when it will have minimal effect on breast milk
- drink no more than one standard drink after the infant has been fed and settled

This level of consumption is not considered harmful as long as another feed is not undertaken for 2 to 4 hours since the alcohol will be metabolised during this time (Capus & Holmes, 1997).

### **Tobacco**

Nicotine, carbon monoxide and other constituents of tobacco smoke restrict the oxygen supply to the foetus. Women who smoke during pregnancy have infants that are significantly smaller and of shorter gestation compared with women who do not smoke. However based on findings in the available literature, smoking during pregnancy is unlikely to cause an increase in the congenital malformation rate (Woods & Raju, 2001).

#### **Prenatal effects**

- increased risk of miscarriage
- increased risk of premature labour

#### **Foetal effects**

- reduced foetal growth. Birthweight decreases in direct proportion to the number of cigarettes smoked.
- premature birth

#### **Infant effects**

There is an increased risk of Sudden Infant Death Syndrome (SIDS) associated with maternal smoking during pregnancy. There is also evidence that household exposure to tobacco smoke after birth has an independent additive effect. Parental drug misuse has an additional small but significant effect on the risk of SIDS (Blair et al., 1996) and increased incidence of respiratory infections, asthma and middle ear infections.

#### **Smoking and breastfeeding**

Tobacco use reduces the breast milk supply. Due to the risk of passive smoking exposure to the infant smoking is best avoided prior to, during or within the vicinity of a feeding infant. As a harm reduction measure, smoke after baby's feed only and not within their general vicinity.

## Cannabis

The impact of cannabis use on pregnancy is similar to that of tobacco but the evidence is less compelling, and compounded by the concurrent use of tobacco, for women who use cannabis (Hall & Solowij, 1998).

### **Foetal effects**

- reduced birthweight
- possible increase in premature birth rate

### **Infant effects**

Although a number of infant neurobiological and developmental abnormalities have been reported in some studies the clinical significance of these findings are unclear at this time (Hall & Solowij, 1998).

### **Cannabis and breastfeeding**

Breastfeeding whilst using cannabis is not recommended.



## Heroin

Babies born to women dependent on heroin tend to be of lower birthweight than those born to women maintained on methadone or non-drug using women.

Heroin using women are also at increased risk of:

- premature delivery
- antepartum haemorrhage
- intra-uterine foetal death

It is unclear whether these effects are specific to heroin use or to the poor health and nutritional status of women dependent on an illicit substance (Ward et al., 1998, Kaltenbach et al., 1998).

Adequate pregnancy care for heroin dependent women can improve pregnancy outcome (Keen & Alison, 2001).

### **Prenatal effects**

- increased risk of miscarriage
- increased risk of placental insufficiency
- premature labour
- increased rate of breech presentation
- increased risk of intrauterine foetal death

### **Foetal effects**

- reduced birthweight, head circumference
- foetal distress (meconium staining)
- premature birth
- increased risk of bloodborne virus infection: hepatitis B & C, HIV

### **Infant effects**

- Neonatal Abstinence Syndrome (NAS)
- increased incidence of SIDS

The long-term developmental outcomes are uncertain, particularly behavioural problems,

as these are related to environmental, social and parenting factors after birth; not just prenatal heroin exposure (Brady et al., 1994).

### **Methadone maintenance treatment**

The treatment of choice for pregnant women who are heroin dependent is methadone maintenance. Pregnant women have priority access to treatment services. Slow reductions in methadone dose are possible during the second trimester of pregnancy under medical supervision.

### **Methadone and breastfeeding**

The advantages of breastfeeding outweigh any potential disadvantages of women on methadone breastfeeding. Only low levels of methadone are present in breast milk (Ward et al., 1998). Women on higher doses of methadone (80 mg or more) are advised to wean their infants slowly to reduce the risk of withdrawal symptoms.

Women who are hepatitis C positive are advised to stop breast feeding if they develop bleeding nipples.

Women who are using illicit drugs while breast feeding should be advised to express and discard their breast milk until they stop using or are stabilised on methadone treatment (Capus & Holmes, 1997).



For more detailed information see Ward et al. (1998)

## **Psychostimulants — Amphetamines and Cocaine**

The impact of psychostimulants on pregnancy varies considerably due to a number of factors:

- gestational period in which the drug exposure occurs
- amount of and pattern of drug use
- differences in metabolism

Malformations and long-term behavioural effects are not all or nothing phenomena. Whether damage occurs depends on interacting factors such as nutritional status, genetic differences, polydrug use, and environmental and social status. The continued use of psychostimulants in pregnancy will increase the risk of adverse pregnancy outcomes (Plessinger, 1998).

### **Prenatal effects**

- maternal hypertension
- placental abruption and haemorrhage
- premature labour

### **Foetal effects**

- premature birth
- foetal distress (meconium staining)
- reduced birthweight, head circumference
- possible increased risk of congenital malformations

However a large, prospective, systematic evaluation for congenital anomalies did not identify an increased number or consistent pattern of malformation associated with psychostimulant use during pregnancy (Behnke et al., 2001).

### **Infant effects**

- possible increased risk of behavioural problems but inconclusive evidence at this time

## **Ecstasy**

No conclusive information on the impact of ecstasy use on pregnancy was available at the time of writing.

## NEONATAL ABSTINENCE SYNDROME (NAS)

Infants prenatally exposed to heroin or methadone have a high incidence of Neonatal Abstinence Syndrome (NAS).

NAS is a generalised disorder characterised by signs and symptoms of:

- Central nervous system hyperirritability — increased muscle tone, disturbed sleep pattern, irritability and tremor
- gastrointestinal dysfunction — excessive yet uncoordinated sucking, poor feeding, vomiting and diarrhoea
- respiratory distress — nasal flaring, tachypnoea, chest recession
- vague autonomic symptoms — yawning, sneezing, mottling and fever

The majority of symptoms appear within 72 hours of birth. Many factors impact on the severity of NAS including:

- the nature and dosage of drugs used; and
- time of last use

Heavy benzodiazepine use during pregnancy may exacerbate and prolong the course of NAS. The type of labour and the use of anaesthetics and analgesia can also impact on severity of NAS as does the size and gestational age of the infant. Full term infants require treatment for NAS more often than premature infants. The relationship between maternal methadone dose and the severity of NAS has been difficult to establish (Kaltenbach et al., 1998).

### Management of Neonatal Abstinence Syndrome

Infants at risk of NAS are monitored using a modified score chart developed originally by Dr Loretta Finnegan. Pharmacotherapy treatment is instigated when scores reach a

predetermined level and the infant is at risk of serious health consequences if not treated. An aqueous solution of morphine administered orally is the most common medication used to manage NAS in Australia.

Mothercraft techniques can provide significant symptom relief to the infant experiencing mild to moderate NAS. Swaddling in cotton sheets and the use of swing cradles or hammocks for sleeping has a calming effect. Dummies provide an opportunity to suck, which also has a settling effect (Capus & Holmes, 1997).

Neonatal Abstinence Syndrome can have a negative impact on mother–infant bonding if not effectively managed. Separation of mother and infant during the treatment of NAS should be minimised. The ongoing treatment of NAS can be successfully managed via a specialist outpatient clinic once the infant is stable on medication thus reducing separation and inpatient length of stay (Oei et al., 2001).

## CHILD PROTECTION

Studies of drug using parents indicate that many were victims of child abuse and/or had poor parenting. These parents are at increased risk of neglecting or abusing their children (Keen & Alison, 2001).

It is vital that the wellbeing of infants born to drug using parents is assessed prior to their discharge from hospital. A discharge-planning meeting including the family and all health and welfare professionals involved with the family is required and management plans agreed to and documented. Where risk of neglect or abuse is identified the statutory child protection services must be involved in the care planning and ongoing monitoring of the family.



## REFERENCES

- Behnke, M., Eyler, F., Garvan, C. & Wobie, K. 2001, 'The search for congenital malformations in newborns with fetal cocaine exposure', *Pediatrics*, vol. 107, no. 5.
- Blair, P., Fleming, P., Bensley, D., Smith, I., Bacon, C., Taylor, E., Berry, J., Golding, J. & Tripp, J. 1996, 'Smoking and the sudden infant death syndrome: Results from 1993–5 case-control study for confidential inquiry into stillbirths and deaths in infancy', *British Medical Journal*, vol. 313, no. 7015, pp. 195–198.
- Brady, J., Posner, M., Lang, C. & Rosati, M. 1994, 'Risk and reality: The implications of prenatal exposure to alcohol and other drugs' [Online], <http://aspe.os.dhhs.gov/hsp/cyp/drugkids.htm>.
- Capus, C. & Holmes, J. 1997, 'Drugs in pregnancy', in *Nursing Care of Drug & Alcohol Problems*, ed. Novak, H., Drug & Alcohol Department, CSAHS, Camperdown, Sydney.
- Day, N. & Richardson, G. 1994, 'Comparative teratogenicity of alcohol and other drugs', *Alcohol Health and Research World*, vol. 18, no. 1, pp. 42–48.
- Hall, W. & Solowij, N. 1998, 'Adverse effects of cannabis', *Lancet*, vol. 352, pp. 1611–1616.
- Jacobson, J. & Jacobson, S. 1994, 'Prenatal alcohol exposure and neurobehavioural development: Where is the threshold?', *Alcohol Health and Research World*, vol. 18, no. 1, pp. 30–36.
- Kaltenbach, K., Berghella, V. & Finnegan, L. 1998, 'Opioid dependence during pregnancy: Effects and management.', *Obstetrics and Gynecology Clinics of North America*, vol. 25, no. 1, pp. 139–151.
- Keen, J. & Alison, L. 2001, 'Drug misusing parents: Key points for health professionals.', *Archives of Disease in Childhood*, vol. 85, no. 4, pp. 296–299.
- Oei, J., Feller, J. & Lui, K. 2001, 'Coordinated outpatient care of the narcotic-dependent infant.', *Journal Paediatric Child Health*, vol. 37, pp. 266–270.
- Plessinger, M. 1998, 'Prenatal exposure to amphetamines: Risks and adverse outcomes in pregnancy.', *Obstetrics and Gynecology Clinics of North America*, vol. 25, no. 1, pp. 119–138.
- Ward, J., Mattick, R. & Hall, W. 1998, *Methadone Maintenance Treatment and other Opioid Replacement Therapies*, Harwood Academic Publishers, Netherlands.
- Woods, S. & Raju, U. 2001, 'Maternal smoking and the risk of congenital birth defects: A cohort study', *The Journal of the American Board of Family Practice*, vol. 14, no. 5, pp. 330–334.

# Surgery and Substance Use

**P**ATIENTS with substance use problems are common on surgical wards. People may suffer trauma while intoxicated, or vascular injury and infection from injecting drugs. In other cases, the substance use is unrelated to the indication for surgery and is easily missed.

Assessment for any form of surgery should involve brief assessment for any substance use issues including drug dependence. If problems are identified, intervention should commence at the earliest possible opportunity ideally by the treating team or via referral to drug and alcohol services. Delaying the treatment until development of avoidable post-operative withdrawal increases the costs of hospitalisation and leads to poorer outcomes.

Drug dependence is a chronic condition and consultation with a drug and alcohol specialist is advised coupled to continuing care after discharge from hospital.

Problems related to surgery in people with substance use problems include:

- pre-operative recognition and intervention
- anaesthetic problems
- post-operative withdrawal
- peri-operative morbidity and mortality
- management of drug seeking and drug use on the ward
- management of post-operative pain

Psychoactive substance use to consider in a patient about to undergo surgery includes:

- tobacco
- alcohol
- opioids
- benzodiazepines
- stimulants

Clinical concerns relate to both intoxication and dependence, and especially withdrawal in the case of the latter.

## TOBACCO

The association between smoking and airways disease and post-operative chest infections is widely recognised.

Patients often accept the need to quit smoking pre-operatively and may be more receptive to consideration of the long-term benefits of quitting. Preparation for surgery should include advice to quit smoking and the offer of appropriate intervention or referral.

Post-operative nicotine withdrawal should be considered if:

- the patient smoked until the time of surgery; and
- complains of withdrawal symptoms such as craving for cigarettes and irritability

Post-operative symptoms may be multifactorial and other factors should be considered. Nicotine replacement therapy (NRT) should be offered where indicated and provided where no contra-indications (such as active ischaemic heart disease) are present. This often does not occur despite the high prevalence of nicotine dependence. Motivational interviewing to quit smoking long-term and referral for further treatment should be offered.

Attempts by in-patients to obtain cigarettes can be interpreted as drug seeking behaviour. Many hospitals do not permit smoking inside or even on hospital grounds. Staff should not help patients to obtain cigarettes or access smoking areas.

An exception should be made for patients in a palliative care setting or those with severe psychiatric comorbidity.

## ALCOHOL

### Alcohol and Post-operative Morbidity

Alcohol consumption exceeding 60 g per day adversely affects post-operative outcomes in several respects:

- increased total morbidity
- increased post-operative mortality
- significantly more care required
- longer duration of hospitalisation
- increased need for repeat surgery
- higher hospital costs

A broad range of morbidities may occur, including:

- alcohol withdrawal syndromes
- infections
- bleeding; and
- cardiopulmonary insufficiency

Follow-up after surgery has confirmed poorer outcomes.

The presence of alcoholic liver disease is associated with major increases in post-operative complications and assessment by a gastroenterologist or hepatologist is advised.

Adverse outcomes have been demonstrated in a variety of clinical settings including:

- colorectal surgery
- hysterectomy
- evacuation of subdural haematoma
- osteosynthesis of malleolar fractures

### Peri-operative Management of Alcohol-related Complications

The pre-operative setting provides an opportunity time for intervention; however, clinical staff tend to focus on the surgical problem and alcohol problems are often overlooked.

It is crucial to obtain an accurate drug and alcohol history prior to surgery. Key questions must consider:

- drugs used (licit & illicit)
- patterns of use
- recency of use
- likelihood of tolerance and possibility of cross-tolerance with other drugs e.g. alcohol and benzodiazepines
- likelihood and severity of withdrawal

Pre-operative assessment should include:

- alcohol and drug history
- psychosocial history and available supports
- physical examination; e.g. monitor:
  - BP (blood pressure)
  - HR (heart rate)
- examine for signs of:
  - cardiomyopathy (rare)
  - respiratory disease
- laboratory tests:
  - liver disease (abnormal LFTs, signs of decompensation such as jaundice, ascites or encephalopathy)

- haematology (platelet count and coagulation studies)
- metabolic (BSL, electrolytes, magnesium)

If abnormalities are found, pre-operative specialist referral may be required.

An appropriate intervention should be initiated when disorders of alcohol use are recognised. Two weeks of abstinence from alcohol improves depressed cellular immunity, but two months of sobriety is necessary to normalise it. A randomised controlled trial has shown that intervention to reduce alcohol consumption prior to elective surgery reduces post-operative morbidity (Tonnesen et al., 1999). The nature of pre-operative treatment does not differ from alcohol interventions offered in other contexts and described elsewhere. Thiamine is given.



See Chapter 3  
Alcohol

Surgery should be avoided in alcohol dependent patients until the course of withdrawal is complete. Surgery during withdrawal may increase withdrawal severity, likelihood of complications and risk of developing delirium tremens. Delirium tremens is a medical emergency associated with untreated alcohol withdrawal, occurring 3–14 days after stopping drinking. If surgery is unavoidable, withdrawal symptoms should be anticipated and managed as part of the post-operative management plan.

Post-operative confusion is often multifactorial with chest infection, hypoxia and delirium tremens often coexisting. In such settings, over-sedation must be carefully avoided.

## OPIOIDS

Opioid dependence should be stabilised pre-operatively using methadone, commencing at 20–40 mg daily and increasing as required every 3 days.

More rapid increases may be used in hospital provided drowsy patients are not dosed. In this setting, twice daily dosing is effective. Many patients who will not accept methadone maintenance often accept in-hospital treatment. This increases retention in hospital and allows surgical treatment to be completed.

Maintenance after discharge can be encouraged and if taken up, the patient is switched to single daily dosing by simply combining the two doses 1-2 days before discharge.

Detoxification pre-operatively has been recommended, but patients usually require post-operative opioid analgesia so this approach is unlikely to succeed.

Post-operative analgesia is an issue. Patients may be conceptualised as suffering two disorders and should be prescribed appropriate treatment for both. It is important to explain to the patient that adequate analgesia will be provided and that opioid analgesia will be withdrawn when no longer indicated.

Decide an appropriate duration of parenteral treatment early in the management plan and advise the patient when parenteral medication will be switched to oral.

Non-opioid analgesia should be used as appropriate.

A higher dose of opioids will be required due to the presence of opioid tolerance.

Pethidine:

- is rarely an appropriate drug in this setting
- has a short half-life
- has marked euphoric effects and hence high abuse potential
- has the toxic metabolite norpethidine which commonly precipitates seizures after high doses

Continue or commence methadone and add longer acting opioids such as morphine for analgesia. Prescribe fixed doses of analgesia rather than p.r.n. dosing to minimise conflicts between staff and patients about when the next dose is due. Otherwise, requests for analgesia may be interpreted as drug seeking, or may evolve into drug seeking.

A trial of patient controlled analgesia (PCA) may be considered after surgery, but the patient should be instructed that PCA will stop if abused. In such cases, switch to a regimen of regular fixed dose morphine in adequate doses.

Extend the parenteral treatment if the clinical circumstances change but avoid this otherwise.

Consult the hospital drug and alcohol service. Set an appropriate discharge goal.

Opioid dependent people with a continuing need for analgesia are likely to return to heroin use after discharge from hospital. Methadone maintenance is strongly indicated in such cases, in addition to other analgesia.

Patients with previous opioid dependence are at risk of relapsing. Set treatment goals of providing analgesia that will not lead to ongoing dependence and explain these to the patient. Where possible, discontinue opioids before discharge from hospital or write the time for discontinuation on the discharge letter and communicate this to the GP.

Drug seeking behaviour should be recognised and managed as per the guidelines in this Handbook.

Drug use on the wards causes ethical and practical problems.

Three common reasons for ongoing drug use should be considered:

- unrelieved pain
- anxiety
- continuing dependence

### Management Strategies

- analgesia may be increased
- anxiety causes and interventions explored
- methadone dose increased
- drug and alcohol consultation should be obtained
- motivational interviewing offered

If drug use continues, discharge from the hospital may be required.



See Chapter 2  
General Principles  
'Drug Seeking', p. 24

## BENZODIAZEPINES

Benzodiazepine dependence may be missed if a careful drug history is not taken or the patient does not disclose recent use. This often presents with withdrawal during the post-operative period. In such cases, the patient is managed as for benzodiazepine dependence in other clinical settings.

A single long-acting benzodiazepine, usually diazepam, is substituted at the minimum dose required to suppress withdrawal symptoms. This is slowly withdrawn over ensuing weeks. It is important to collaborate with the patient's GP who may not be aware of the extent of the problem or the number of doctors being seen by the patient.



See Chapter 11  
Benzodiazepines

## STIMULANT USE

Cocaine and amphetamines are not often major problems in hospital (with the exception of the Emergency Department). Psychological problems and drug-induced psychosis may require psychiatric consultation. Cardiovascular changes may lead to haemodynamic instability until the effects abate.

## REFERENCES

Tonnesen, H., Rosenberg, J., Nielsen, H.J., Rasmussen, V., Hauge, C., Pedersen I.K. & Kehlet, H. 1999, *British Medical Journal*, vol. 318, pp.1311–6.

# Managing Chronic Pain

**M**ANAGING chronic pain is increasingly challenging. Provision of effective analgesia is only part of the medical management of acute and chronic painful conditions. A careful explanation of the clinical problem is crucial, as is development of a trusting therapeutic relationship.

## ACUTE AND CHRONIC PAIN

The medical management of pain is far more successful in acute than chronic conditions. A basic principle of therapeutics, founded on the cardinal notion of 'first do no harm', is to use the lowest possible dose of a drug for the shortest possible duration and minimise the risk of side effects.

Aim:

- keep the patient as comfortable as possible and minimise or avoid serious analgesic side effects

Clinical objective:

- reduce pain to levels that are bearable and reasonably constant
- avoid peaks of pain during a nadir in the analgesic's plasma concentration
- avoid temporary sedation or euphoria coinciding with peak plasma analgesic concentrations





[www.ebandolier.com](http://www.ebandolier.com)

While opioids have an important role in treatment of chronic, non-malignant pain, many patients with chronic pain do *not* require opioids. Although opioids are commonly used in management of painful, chronic, non-malignant conditions, evidence of significant benefit is equivocal.

Chronic pain patients without pre-existing risk factors are at low risk of opioid dependence. But opioids should be used with caution, if at all, for patients with a current or past history of substance abuse.

A more even concentration of analgesics over time is achieved if longer acting agents are administered by mouth, suppository, infusions or skin patch rather than by injection. Injections of short acting opioids provide excellent pain relief in acute conditions. But management of chronic pain with injections of these agents often achieves a poor long-term clinical outcome, as plasma concentrations fluctuate widely.

Pethidine injections should be avoided for chronic pain management because other analgesics provide more effective pain relief with fewer side effects. Long-term prescription of pethidine injections is more likely to be complicated by severe problems of drug seeking behaviour and dependence than with morphine or other opioids. Also, active pethidine metabolites can accumulate causing complications, especially in the presence of high doses or renal impairment.

Paracetamol or aspirin provide excellent relief in cases involving mild pain.

It is generally agreed that orally, well-absorbed, long acting opioids, sustained release forms of oral morphine or oral oxycodone and metha-

done, are first line agents for the management of moderate to severe chronic non-malignant pain. These options:

- provide relatively even relief over time with low peaks and shallow troughs
- avoid the need for injections
- allow the size and frequency of doses to be readily modified according to need
- provide great flexibility

Adjuvant drugs, such as some antidepressants or anticonvulsants, have no significant analgesic effects of their own but augment the analgesia provided by opioids.

## ASSESSMENT AND PAIN MANAGEMENT IN DRUG USERS

There is no formally recognised treatment protocol to guide standards of practice in pain management specifically for drug-dependent patients, but there are excellent general guidelines and protocols for pain management (e.g. the NHMRC guidelines).



[www.nhmrc.gov.au/publications](http://www.nhmrc.gov.au/publications)

A comprehensive initial assessment is pivotal.

Assessment of the chronic pain patient entails:

- previous records
- full history
- physical examination
- investigations to document organic pathology
- current, past or family history of alcohol and/or other substance abuse
- psychiatric history to identify mood and anxiety disorders

- social history including supports at home and stressors
- current coping strategies
- regular use of illicit drugs
- previous long-term, heavy prescription opioid or benzodiazepine use

### Screening for Substance Dependence

All patients should be screened for a current, past or family history of substance abuse or dependence.

See Chapter 3 Alcohol 'CAGE', p. 42

Opioids should be used with caution if:

- positive CAGE
- alcohol :
  - > 6 standard drinks/day (men),
  - > 4 standard drinks/day (women)

### Alternative Options in Management

There are many options to explore for the management of chronic pain before opioids are considered:

- lifestyle adjustment; e.g. exercise, change in work tasks
- supportive counselling, cognitive-behavioural therapy
- physiotherapy
- other analgesics e.g. non-steroidal anti-inflammatory agents (NSAIDs), salicylates — follow the WHO analgesic ladder (see Figure 17–1)

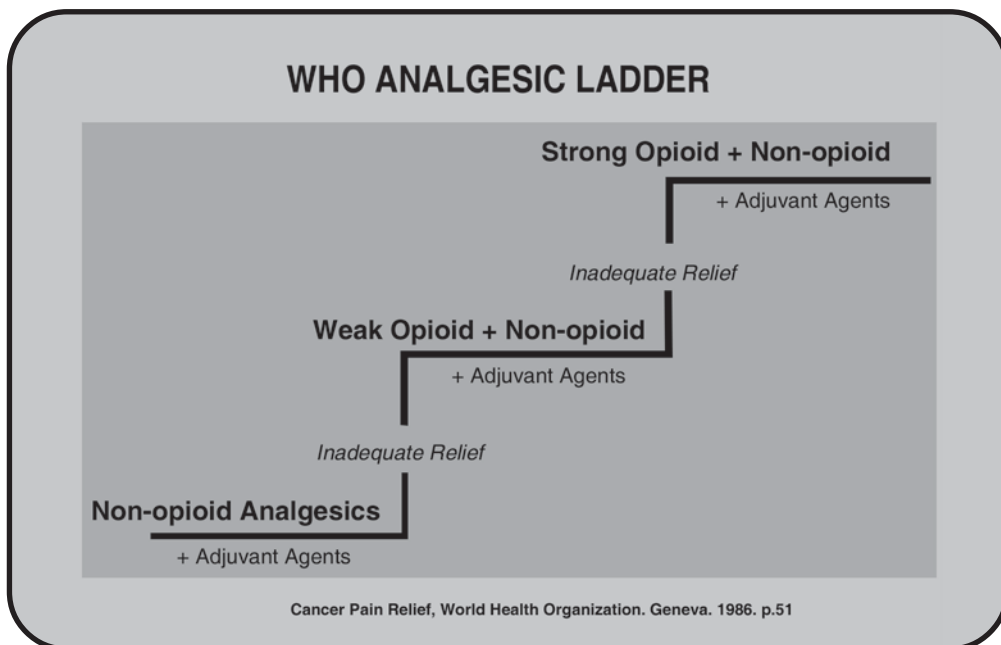


Figure 17–1 WHO Analgesic Ladder Source: World Health Organization

Chronic Pain

- antidepressants
- anticonvulsants
- anxiolytics, tranquillisers and hypnotics
- muscle relaxants
- antispasmodics
- antihistamines
- corticosteroids
- local anaesthetics
- Transcutaneous Electrical Nerve Stimulation (TENS)
- radiotherapy

A team based, holistic approach to chronic pain management is often helpful, involving nurses, psychologists, psychiatrists, physiotherapists and pain specialists.

Issues to be considered to improve pain management for injecting opioid users include:

- tolerance to analgesics
- potential adverse interactions with other sedative drugs
- difficulties and misunderstandings which arise in communication between clinicians and all patients
- real and perceived legal constraints for prescribers

Better outcomes require a thoughtful and considered response.

## Dependence and Tolerance

Consumption of central nervous system (CNS) depressant drugs, including opioids (e.g. heroin, morphine or methadone) and other psychoactive substances (e.g. alcohol or benzodiazepines), can result in dependence (including tolerance):

- higher doses and longer duration of consumption increase severity of drug dependence

- dependence and tolerance decrease when consumption declines or ceases

Tolerance means that a drug diminishes its effect over time (or higher doses of a drug are required to maintain usual effect). Tolerance is a relative rather than an all-or-none phenomenon and can only be estimated approximately.

People who are opioid tolerant and develop a painful condition require larger doses of opioid analgesia more frequently and for longer periods in order to achieve satisfactory analgesia compared to an opioid-naive patient with an identical physical condition.

For example, patients with a long history of heroin use presenting with a fractured humerus, will require larger doses of pain relief administered more frequently and for a longer duration than a patient of the same age, sex and body weight with a similar fracture but no history of previous opioid use.

Some recent evidence suggests that chronic exposure to opioids may also reduce tolerance to pain.

Prescription of an analgesic with sedative properties in the presence of other central nervous system sedatives, including alcohol or benzodiazepines, may result in excessive sedation.

## Preventing Drug Dependence in Patients with Chronic Non-malignant Pain

Preventing drug dependence from developing should always be a high priority, but balanced against the critical obligation to relieve pain and suffering. These obligations are often in competition to some extent.

Minimise the risk of drug dependence by:

- keeping patients informed. Patients who fully understand their condition and have a strong therapeutic relationship with their doctor are less likely to become dependent on analgesic medication
- maximising the benefits of non-pharmacological pain management measures. A growing list of aids is now available including relaxation tapes, discussion groups and explanatory booklets for patients with chronic pain
- referring patients to one of a small but growing number of allied health professionals interested in management of chronic non-malignant pain. They are more familiar with the range of materials available, are probably better equipped and have more time to respond to these patients.

The following pharmacological principles also help reduce the risk of drug dependence:

- use lower doses of a drug for a shorter duration
- use non-injectable long acting opioids rather than injectable short acting opioids
- avoid pethidine injections and other obsolete preparations.

### Principles of Pain Management in Opioid Dependent Patients

Use of methadone in the management of chronic painful conditions is controversial. Some experts argue that excellent relief can be achieved using methadone provided that clinicians are familiar with the unusual pharmacological properties of the drug and the substantial variability in handling the drug between patients. Others argue that alternative drugs are easier to use than methadone. The long half-life of methadone means that there is an inevitable delay of up to several days between the initiation of treatment and onset of maximal effect. Orally well-absorbed opioids with a shorter half-life, such as

oxycodone, are usually more effective, especially if the painful condition is likely to only last for a short period.

Some clinicians and patients prefer to separate pain management from the management of heroin dependence. This makes good clinical sense because the time scale of these problems is usually quite different. If different agents are being used to control these problems, the dose of the medication can be modified separately according to the fluctuating severity of each one.

Explaining these issues to patients is particularly helpful as many injecting drug users feel, often with good reason, that medical staff may provide inadequate analgesia to patients with a history of drug dependence. Likewise, some medical staff may consider that some injecting drug users deceitfully attempt to obtain analgesics from doctors by complaining of spurious symptoms of a condition known to be unaccompanied by physical signs or findings from special investigations.

If different doctors are responsible for management of pain and management of drug dependence, close liaison is imperative to avoid mishap and ensure that the total opioid dose remains reasonable.

#### *Methadone maintenance*

It is generally accepted that methadone should not be used as the primary form of analgesia for acute or chronic pain in patients enrolled in methadone maintenance treatment. Increasing the dose of methadone does provide additional analgesia when tolerance to methadone has developed but this often takes several days to achieve and further complicates an already complicated treatment system.

Improved pain relief can often be achieved through the addition of another opioid analgesic, such as oxycodone, or a sustained release oral morphine preparation. A further

alternative is prescribing a long acting NSAID such as diclofenac. Ketorolac is a NSAID which can be administered by injection in cases of severe acute pain. Adjuvant drugs in combination with opioids augment the effect of the primary analgesic.

### ***Buprenorphine***

Patients receiving moderate or high doses of methadone or any other opioid should not be prescribed buprenorphine. In the presence of moderate to high doses of opioids, e.g. > 40 mg of methadone, the antagonist (i.e. naltrexone like) properties of buprenorphine can trigger unpleasant opioid withdrawal symptoms. Buprenorphine is a very potent analgesic effective in managing severe pain in patients tolerant to opioids — provided that large doses of any opioid have not been consumed recently. The decision will require:

- consideration of the dose of the other opioid
- the half-life of the opioid
- the duration since the last dose was taken

N.B. Information regarding the most recent use of heroin is often unreliable, especially if the drug dependent patient fears repercussions from disclosure.

### ***Terminal illness***

If drug dependent patients develop a severely painful condition as part of a terminal illness, then efforts to alleviate pain and discomfort become paramount. Fear of exacerbating drug dependence becomes a secondary concern. When life expectancy becomes very short, alleviation of pain becomes the only consideration.

## **THE POTENTIAL FOR ADVERSE INTERACTIONS**

The liver has a central role in the metabolism and elimination of many drugs, including most opioid analgesics. An impaired liver is less able to metabolise drugs, potentially increasing or decreasing the observed effect.

Avoid long-term use of large doses of paracetamol i.e. > 4 grams/day in patients with liver disease as further liver damage can ensue. Drugs containing small quantities of paracetamol can be used quite safely for pain relief in people with liver disease.

The use of alcohol to relieve pain should be avoided as other agents are more effective and safer. People with hepatitis C should be encouraged to reduce alcohol consumption to low levels or abstain as alcohol increases progression of liver disease.

The likelihood of adverse effects of analgesics in patients with liver disease depends on the severity of the hepatic impairment and the particular drugs prescribed. Severe liver impairment increases the effects of many opioids. The dose of opioid analgesics prescribed for these patients should be reduced and the frequency increased while sedative effects of the drug should be carefully monitored.

Some benzodiazepines, such as diazepam and temazepam, may also produce greater than expected effects in some patients with impaired hepatic function due to active metabolites.

Health professionals should always err on the side of caution. In practice, the large reserve capacity of the liver enables most patients with even quite severe hepatic impairment to manage with usual doses of opioids or benzodiazepines until almost terminal impairment has developed.

Methadone does not cause liver damage in injecting drug users, notwithstanding urban mythology to the contrary.

### THE PATIENT–CLINICIAN RELATIONSHIP

Fear of poor treatment and discrimination undoubtedly prevents some drug dependent individuals from seeking assistance from health care providers.

People who use illicit drugs are as entitled as any other patient with severe pain to proper professional management including effective pain relief. Professional concerns about the risk of exacerbating dependence cannot be ignored. Providing comfort and relieving suffering should always be the paramount objective.

Doctors who are uncertain about the proper course of action in a particular case should seek advice from more experienced colleagues.

Placebo drugs should never be provided in the pretence that effective analgesia has been offered.

NSAIDs or buprenorphine may be useful in some cases where the doctor remains uncertain about the organic or drug-seeking nature of the pain.

A common concern amongst doctors is that state regulatory authorities will criticise their prescribing habits for drug dependent patients. In some jurisdictions, legislation covering the prescription of 'drugs of addiction for persons known to be addicts' ensures that doctors and health authorities have to carefully follow statutory requirements.

When treating drug dependent patients, clinicians should discuss pain relief openly and frequently and resolve any conflict or unresolved issues as soon as possible. Discussing these matters in an open manner will:

- increase the chance of developing a productive patient–clinician relationship
- increase patient compliance
- increase the likelihood of a good treatment outcome for treatment providers and consumers alike; and
- decrease the likelihood of the patient feeling the need to 'self-medicate' with additional illicit and licit drugs to achieve adequate pain relief

## RESOURCES

Bell, J. 1997, 'Australian trends in opioid prescribing for chronic non-cancer pain 1986–1996', *Medical Journal of Australia*, vol. 167, pp. 26–29.

Bamingbade, T.A. & Langford, R.M. 1998, 'The clinical use of tramadol hydrochloride', *Pain Reviews*, vol. 5, pp. 155–182.

Centre for Mental Health 2001, *Mental Health for Emergency Departments – A Reference Guide*, (Pocket Version), NSW Health Department, August.

Graziotti, P.J. & Goucke, C.R. 1997 'The use of oral opioids in patients with chronic non-cancer pain: management strategies', *Medical Journal of Australia*, vol. 167, pp. 30–34.

DASC (Drug and Alcohol Services Council) 1996, *Guidelines for the Management of Patients Complaining of Severe, Recurrent or Chronic Pain*, DASC, Adelaide.



[www.ebandolier.com](http://www.ebandolier.com)

Haddox, J.D., Joranson, D., Angarola, R.T., Brady, A., Carr, D.B., Bronsky, E.R., Burchiel, K., Gitlin, M., Midcap, M., Payne, R., Simon, D., Vaswleuan, S., Wilson, P. & Portney, R.K. 1997, 'The use of opioids for the treatment of chronic pain. A consensus statement from the American Academy of Pain Medicine and the American Pain Society'. *Clinical Journal of Pain*, vol. 13, no. 1, pp. 6–8.

Hagen, N., Flynn, P., Hays, H. & MacDonald, N. 1995, 'Guidelines for managing chronic non-malignant pain: opioids and other agents', *Canadian Family Physician*, vol. 41, pp. 49–53.

Harris, N., Hugh, G. & Greenway, S. (unpublished), 'Emergency Department Mental Health Manual (Draft)', NSW Health Department. November 2000 .

Jadad, A.R., Carroll, D., Glynn, C.J., Moore, R.A. & McQuay, H.J. 1992, 'Morphine responsiveness of chronic pain: double-blind randomised crossover study with patient-controlled analgesia', *Lancet*, vol. 339, pp. 1367–1371.

Moulin, D.E., Lezzi, A., Amireh, R., Sharpe, W.K.J., Boyd, D. & Merskey, H. 1996, 'Randomised



[www.nhmrc.gov.au/  
publications](http://www.nhmrc.gov.au/publications)

trial of oral morphine for chronic non-cancer pain', *Lancet*, vol. 347, pp. 143–147.

Portenoy, R.K. 1996, 'Opioid therapy for chronic non-malignant pain: a review of critical issues'. *Journal of Pain & Symptom Management*, vol. 11, pp. 203–217.

# Coexisting Mental Illness

**T**HERE is a high level of coexisting AOD problems and various forms of mental health problems. The characteristic features of such problems are that:

- they are common
- they are heterogeneous and variable in nature
- they are associated with poor outcomes and individuals experience higher drop out rates when standard treatment approaches are used
- treatment often involves a range of different services; but
- best outcomes are achieved through integrated and comprehensive treatment

## EPIDEMIOLOGY

Prevalence rates vary greatly across different populations. Overall, the 12 month prevalence of a substance use disorder together with a mental disorder is estimated to be 10% of the general population (ABS, 1998; Jablenski et al., 2000), and lifetime prevalence 20–30% (Todd, 2002). The evidence also suggests that for most people with mental health disorders, between 30 and 50% experience a substance use disorder, with highest rates reported in people with antisocial personality disorder, and to a lesser extent bipolar disorder and schizophrenia (Todd, 2002).



Definitions applied also greatly impact on reported prevalence levels. Consumption and patterns of drug use that do not meet diagnostic criteria (e.g. for dependence) may still impact on mental disorders.

No term is consistently used or favoured. The different terms also have slightly different meanings. For example, ‘*dual diagnosis*’ implies the existence of an alcohol or other drug problem and *one* other problem. But all terms mean the co-existence of an AOD problem and at least one mental health problem.

## TERMINOLOGY

Various terms are used, often interchangeably, including:

- dual diagnosis
- coexisting disorders
- coexisting mental illness
- comorbidity

## SOME SPECIFIC ASSOCIATIONS

Coexisting disorders encompass a wide range of combinations of problems. Because of the heterogeneous nature of coexisting disorders appropriate treatment requires details of the specific combination of problems. Some common combinations are shown in Table 18–1 and detailed below.

**Table 18–1**  
Mental health problems commonly associated with psychoactive drug use

Psychoactive Drugs	Amnesic Disorder	Anxiety Disorder	Delirium	Mood Disorders	Psychotic Disorders	Sexual Dysfunction	Sleep Disorders
<b>CNS Depressants</b>							
Opioids			☐	☐		☐	☐
Sedative/Hypnotics	☐	☐	☐	☐		☐	☐
Solvents and Inhalants	☐	☐	☐	☐			☐
<b>CNS Stimulants</b>							
Amphetamines		☐	☐	☐	☐	☐	☐
Caffeine		☐					☐
Cocaine		☐	☐	☐	☐	☐	☐
Nicotine		☐					☐
Psychedelics	☐	☐	☐	☐			

Source: adapted from “Table of disorders commonly associated with psychoactive drug use”, Pagliaro, A. & Pagliaro, L. (2000)

## From a Drugs Perspective

### ***Alcohol and mood disorders***

Commonly associated with a depressive disorder. Depression is generally resolved following several weeks abstinence. Bipolar disorder is also common and alcohol may also confound treatment, increasing the rate of the cycle and likelihood of relapse during a manic phase.

### ***Alcohol and psychosis***

Problematic alcohol use has been associated with increased risk of hallucinations and delusions in those with psychotic disorders. Amongst those with schizophrenia alcohol use contributes to non-adherence to medication use, increased symptoms, more medical problems and higher rates of disruptive behaviour when unwell.

### ***Cannabis and psychosis***

A common experience with cannabis use is mild psychotic symptoms e.g. paranoia. Cannabis may infrequently induce a psychotic episode that can last several days after the intoxication subsides. Cannabis may also precipitate onset of schizophrenic psychosis in those with a predisposition to the disorder.

### ***Opioids and mental health disorders***

A very high rate of mental health conditions accompanies opioid dependence. These include depression, social phobia and other anxiety disorders.

### ***Stimulants and mental health disorders***

Intoxication from stimulants (e.g. amphetamines) can result in a number of psychiatric symptoms. An amphetamine-induced psychosis can exist beyond the period of intoxication, usually for several days but sometimes up to several weeks. Occasionally, a chronic schizophrenia-like condition may occur after chronic heavy amphetamine use (it is unclear whether this occurs only in those with a pre-

existing disposition). Heavy prolonged use sensitises the user to unwanted psychotic symptoms when the drug is used again.

## From a Mental Health Perspective

### ***Anxiety disorders and drug use***

People with anxiety disorders also experience high rates of alcohol and drug problems. However, anxiety disorders also can occur as part of an alcohol or drug syndrome. Panic disorder and social phobia are common in alcohol dependent people. Ceasing drug use before treating anxiety disorders is optimal.

Also consider misuse of benzodiazepines in a patient with an anxiety disorder when:

- symptoms persist despite taking them
- the patient is reluctant to consider other treatment approaches
- there is extensive use of benzodiazepines to overcome nearly all potentially anxiety-provoking situations

### ***Personality disorders and substance use disorders***

Antisocial personality is a common association. Other personality disorders prone to substance misuse include the histrionic, borderline, narcissistic, avoidant and obsessive-compulsive.

### ***Psychotic illnesses and substance use problems***

- people with psychotic illnesses have increased rates of violence, homelessness, worse psychosis, poor compliance and slower recovery from substance use disorders
- users of alcohol and cannabis with psychotic illnesses are five times and three times respectively more likely to be dependent than study controls (Degenhardt & Hall, 2001)

- cannabis may produce acute psychotic symptoms if used in large doses but is unlikely to cause a chronic psychosis. It can precipitate psychotic illnesses in vulnerable individuals and will exacerbate symptoms in those with pre-existing illnesses
- stimulants and hallucinogens are preferred by patients with psychotic illnesses but they exacerbate psychosis

### ***Suicide and substance misuse***

- post-mortem studies find alcohol or other drugs at measurable levels in 30–50% of suicides
- substance misuse predisposes to suicide by:
  - disinhibiting or providing ‘courage’ to overcome resistance in carrying through the act
  - clouding of one’s ability to see alternatives
  - worsening mood or psychosis

## **MANAGEMENT**

There is growing consensus regarding the nature of treatment for coexisting disorders.

### **What Works?**

- integrated approaches within services and by therapists with skills and knowledge in both areas are the most effective and acceptable
- non-confrontational approaches to substance use
  - assertive care and involuntary management if needed

## **Principles of Care**

1. Safety — above all else ensure the safety of the patient and others
2. Stabilisation — address acute intoxication or withdrawal, psychotic symptoms, psychosocial crisis, severe anxiety or depressive symptoms etc.
3. Comprehensive assessment — is essential and is an ongoing process
4. Clinical case management — often initiated by mental health team but requires coordination and continuity of care
5. Treatment integration — involves treatment for both the drug use and mental health condition

## **Assessment and Management**

### ***1. Screen for both disorders***

#### ***2. Assess***

- undertake a thorough assessment
- manage withdrawal and reassess if needed
- multiple reviews over time may be needed
- ask:
  - which came first?
  - were the psychiatric symptoms there during periods of prolonged abstinence?
  - observe mental state after intoxication effects have dissipated and the patient has withdrawn from the substance and substance induced symptoms have had time to resolve (see below).

### ***3. Engage for long-term treatment***

- vital but difficult. Relapse for AOD and some mental health problems is common.

### ***4. Treat***

- establish motivation to change, goals and realistic outcomes e.g. patients with poorly controlled schizophrenia are unlikely to change unless their lives are going to be better when drug-free

- motivational enhancement (modified approach for psychiatrically ill and refer to a specialist agency where appropriate)
- apply harm minimisation strategies (frequently overlooked for patients with coexisting mental illnesses)
- adopt a long-term perspective.

### 5. *Attend to both disorders*

- often drug use decreases when psychosis is well controlled, and depression often goes once the patient is abstinent from alcohol
- prescribe medication to substance using patients, but cautiously; depending on the substance used and the medication prescribed, for example, tricyclic antidepressants and alcohol are best avoided. The problems of interaction between alcohol and modern antidepressants are small relative to the possible benefits. Consider providing limited quantities at a time e.g. daily collection with methadone, or twice weekly pick up from a pharmacist/clinic
- generally psychiatric treatments are less effective (but not totally ineffective) when substance misuse continues e.g. antidepressants are less effective for depression and anxiety when taken with alcohol, graded exposure for agoraphobia is ineffective if taking more than 5–10 mg of diazepam per day.

### Harm Minimisation

Apply a harm minimisation approach, for example, ensure prescription of thiamine for alcohol dependence, or access to clean injecting equipment if injecting drug use continues. A harm minimisation approach may involve abstinence from alcohol or other drugs, but it also acknowledges incremental change can be an improvement or valid end point.

## RESOURCES



[www.som.flinders.edu.au/  
FUSA/PARC/Publications](http://www.som.flinders.edu.au/FUSA/PARC/Publications)

Holmwood C. 2003, 'Comorbidity with mental disorders and substance use: A brief guide for the primary care clinician'. *Commonwealth Department of Health and Ageing*. Accessed from: [www.health.gov.au/pubhlth/publicat/document/comorbid\\_brief.pdf](http://www.health.gov.au/pubhlth/publicat/document/comorbid_brief.pdf)

McCabe D. & Holmwood C. 2003, 'Comorbidity in general practice: The provision of care for people working with coexisting mental health problems and substance use by general practitioners'. *Commonwealth Department of Health and Ageing*. Accessed from: [www.health.gov.au/pubhlth/publicat/document/comorbid\\_gp.pdf](http://www.health.gov.au/pubhlth/publicat/document/comorbid_gp.pdf)



[www.mentalhealth.org](http://www.mentalhealth.org)

### REFERENCES

- ABS (Australian Bureau of Statistics) 1998, *National Survey of Mental Health and Wellbeing of Adults: User's Guide*, ABS, Canberra.
- Degenhardt L. & Hall W. 2001, 'The association between psychosis and problematical drug use among Australian adults: findings from the National Survey of Mental Health and Well-Being'. *Psychological Medicine*, vol. 31, pp. 659–668.
- Jablenski, A., McGrath, J., Herrman, H., Castle, D., Gureje, O., Evans, M., Carr, V., Morgan, V., Korten, A. & Harvey, C. 2000, 'Psychotic disorders in urban areas: an overview of the Study on Low Prevalence Disorders', *Australian & New Zealand Journal of Psychiatry*, vol. 34, pp. 221–236.
- Pagliari, A. & Pagliaro, L. 2000, *Substance Use Among Women*, Brunner/Mazel, Philadelphia, p. 183.
- Todd, F. 2002, 'Chapter 20: Coexisting alcohol and drug use and mental health disorders' in (ed.) Hulse G., White, J. & Cape G. 2002, *Management of Alcohol and Drug Problems*, Oxford University Press, South Melbourne, Victoria, pp. 359–373.

# Mental Illness

# Injecting and Communicable Diseases

**T**HE REALITY of the human immunodeficiency virus (HIV) and hepatitis C (HCV) epidemics has served to clarify thinking about the risks of acquiring communicable diseases through injecting drug use. Hepatitis B (HBV), which poses a significant risk to the injecting user population, did not have such an impact and only now is its importance in this population being recognised.

With reliable estimates predicting an increasing use of illicit drugs, there is an urgent need to provide clear and unequivocal messages about the risks of infection associated with these practices.

Recent studies have demonstrated that *all* of the paraphernalia linked to injecting use have the potential to transmit blood borne infectious agents. Even if sharing of equipment does not occur, poor cleaning of personal equipment and contaminated drug supplies can still expose the individual user to infections from bacteria and fungi.



## INFECTIVE PROBLEMS ASSOCIATED WITH INJECTING DRUG USE

- endocarditis. While infection of the tricuspid valve is more common, left sided subacute bacterial endocarditis (SBE) regularly occurs in injecting drug users (IDU). Staphylococcal and fungal infections (left sided) are well recognised
- ophthalmitis
- systemic candida infections
- bacteraemia
- tuberculosis
- tetanus

## BLOOD BORNE COMMUNICABLE DISEASES

Numerically these infections are more significant. They include:

- hepatitis C
- hepatitis B
- hepatitis D
- HIV
- HTLV-I/II
- malaria

There is a real possibility that other, as yet unidentified, viruses exist.

Hepatitis G (HGV) antibodies are found in approximately 10% of injecting users but the significance of this virus remains unclear. Transfusion-transmitted virus (TTV) antibodies are also common but neither HGV nor TTV co-infection alter the course of HCV or HBV infection.

## HEPATITIS C

This is the most commonly transmitted pathogenic virus in Australian IDU populations. The annual risk of hepatitis C (HCV) seroconversion in regular users is 15% and for those who have used for more than 10 years rates of HCV positivity reach > 90%.

Some facts about HCV:

- approximately 15,000 new cases of HCV occur in Australia per annum
- most of these will occur in IDU settings
- transmission occurs through blood contamination of equipment
- sexual transmission occurs uncommonly
- vertical transmission occurs in 5–8% of HCV RNA positive mothers (recent data suggests caesarian section may reduce the risk)
- no evidence of transmission from breast feeding

## Testing for HCV

- HCV antibody (HCV Ab) remains the first line test but it does not distinguish between acute, chronic or resolved infection.
- HCV Ab is passively transferred to neonates who will remain Ab positive for up to 12 months
- HCV Ab becomes positive 15–30 weeks post-exposure
- HCV RNA detectable within 2–3 weeks of exposure to the virus
- HCV RNA now funded by Medicare in suspected acute HCV infection and in patients with persistently normal liver tests (to confirm viral clearance). Testing also approved in pregnant patients
- HCV genotype and viral load now funded by Medicare in relation to commencing treatment. These tests may be ordered by a GP who is linked to a treatment centre

## Natural History

- up to 30% of infected individuals clear the virus within 12 months
- 10–15% of chronically infected individuals progress to cirrhosis over 20–30 years. Risk of cirrhosis is increased by:
  - older age at infection
  - male
  - alcohol > 40 g / day
  - possibly increasing age
- risk of liver (hepatocellular) carcinoma (HCC) is approximately 4% per annum in cirrhotic patients
- most patients with HCV will not die of HCV related complications

## Assessing Patients for Treatment

S100 guidelines for antiviral therapy specify a subset of patients that may access treatment. The following tests are required before treatment may be initiated and they can be ordered by GPs evaluating patients. A Referral Checklist is at Appendix K of this Handbook.



See Appendix K

## Antiviral Therapy

- interferon monotherapy is no longer regarded as appropriate unless patient is unable to tolerate ribavirin
- if interferon monotherapy is to be used it should be with pegylated forms of the molecule as they provide significantly improved sustained response (SR) rates (approx 40%)
- interferon-ribavirin therapy gives an overall SR in 40–50% of patients with non 1 genotypes achieving up to 80% SR

- combination therapy is contraindicated in the presence of overt or suspected coronary artery disease. Interferon may exacerbate autoimmune disease. Thyroiditis, which can progress to thyroid failure, occurs in approximately 20% of interferon treated patients

## Managing Patients who have Failed Therapy or are Ineligible for Treatment

- monitor liver function 6 monthly
- repeated HCV RNA tests are only indicated if liver tests change significantly
- in cirrhotic patients monitor a fetoprotein 6 monthly and perform abdominal ultrasounds 6 monthly to detect HCC development
- consider HBV and HAV vaccination although cost-effectiveness studies do not favour universal recommendation

## Liver Transplantation in HCV

- HCV end stage liver disease is the most common indication for liver transplantation in most Western countries
- reinfection of the graft occurs in all patients and cirrhosis evolves in approximately 10 years

## Prevention of HCV

- do not share any injecting equipment or paraphernalia
- reduce injecting drug use, encourage routes of non-injecting administration and encourage users to quit use through treatment programs
- fast track pregnant patients with HCV onto a methadone program
- in household settings: do not share razors, toothbrushes or other items that may be contaminated by blood
- wear gloves when cleaning blood spills
- avoid at-risk sexual exposure

## Alcohol and HCV

- high daily intake will markedly worsen liver tests
- > 40 g / day will increase rate of progression to cirrhosis
- do not consider treatment in those drinking > 40 g / day as response is reduced and ALT may normalise in some if alcohol is ceased

## HEPATITIS B

Exposure to this virus is common in certain countries and in certain groups within the Australian community. Unlike HCV infection, only 5% become chronically infected. Children infected at birth have a much higher chronicity rate (approximately 80%). Evidence of exposure to HBV is found serologically in 40–50% of IDUs in Australia.

- HBV now exists in our community in a wild and a mutant form. Co- or re-infection can thus occur
- HBV may be transmitted from blood exposure but also sexually and by exposure to other infected bodily secretions
- prevention of HBV will be achieved by applying guidelines included in the HCV section
- an effective HBV vaccine exists and it should be recommended to all at risk of this infection
- currently in Australia HBV vaccine is made available free to:
  - all newborn babies
  - adolescents who missed vaccination at birth
  - health care workers through their place of employment
  - attendees at sexual health clinics

## Testing for HBV

- interpreting HBV tests should always be undertaken carefully
- serological tests include HbsAg, HbsAb, HbcAb, HbeAg and HbeAb
- with mutant strains now more common it is possible to have mutants that produce no HBeAg and even no HBsAg
- HBV DNA measurement is becoming more useful and even necessary

## Natural History

- hepatitis B will produce a fulminant hepatitis in < 1% and a clinical illness in < 50% of infected individuals
- immunity to HBV is life-long but wild strain immunity may not protect against mutant strain infection
- neonatal infection usually results in the 'asymptomatic' carrier state with HbsAg positivity and normal liver tests. Some individuals develop 'flares' of abnormal ALT and may progress to cirrhosis
- the asymptomatic carrier may still progress to hepatocellular carcinoma without cirrhosis

## Treatment of HBV

- the release of lamivudine on S100 has modified treatment significantly
- available for 12 months therapy to those whose biopsy shows chronic active hepatitis
- therapy does induce the development of resistant (YMDD) mutants
- if transplantation is being considered then lamivudine monotherapy should not be commenced without discussion with a transplant unit
- interferon monotherapy can be used and 20–30% may be expected to lose HBeAg and become HBeAb positive. Only 5–7% will clear HBsAg

## HEPATITIS D

Fortunately this infection is becoming less frequent in the Australian community. HDV is an incomplete viral particle that requires the HBsAg coat to allow it to infect hepatocytes.

- HDV can only infect HBsAg positive individuals
- infection may be a co- or super- infection
- in either instance the disease in the dually infected individual is more severe than if only HBV has been contracted
- prevention and treatment of HDV infection is that of HBV
- vaccination against HBV will prevent HDV susceptibility in the HBV immune individual

## HUMAN IMMUNODEFICIENCY VIRUS

Fortunately in Australia the human immunodeficiency virus (HIV) positivity rate in IDUs remains low, unlike the situation in many other countries (including the USA, Scotland and Canada). The potential for increased rates of infection continues as does the need for support of Needle Syringe Programs (NSPs) at all levels of our community. The Australian experience suggests that NSPs have been of great value in reducing the spread of HIV in the IDU community.

In Australia the HIV seroprevalence rate in IDUs is 1–2% compared to a rate of 5–10% in homosexual/bisexual men.

Consider HIV exposure and acute infection in IDUs complaining of:

- Epstein-Barr virus (EBV) seronegative 'glandular fever' type symptoms
- flu-like symptoms out of season
- fever > 3 days

- maculo-papular rash
- recent evidence of sexually transmitted infections
- recent high-risk exposure

### Testing for HIV

- HIV serological tests may be positive 3 weeks after the start of a primary HIV illness
- HIV DNA may be detectable within a few days of symptoms and negative at 1 month
- seek advice from an HIV expert in determining a testing sequence depending on the clinical setting
- it is appropriate to screen all past IDUs for HIV exposure, providing pre- and post-test counselling is provided

### Treatment of HIV

- as HIV infection is uncommon in the IDU population in Australia it is recommended that if a positive test is obtained, advice be sought from a specialist clinician
- treatment is available for prophylaxis following a positive exposure, for acute infection, for chronic HIV infection and for those who present with an AIDS defining illness
- the course of HIV and AIDS has been radically improved with the advent of multiple anti-HIV agents

### Prevention

- advise of the risks of sexual and IDU-related transmission
- advise methadone maintenance as a means of reducing IDU

### Effect of HIV on IDU Problems

- an active IDU should not be denied resources for the investigation and treatment of HIV, HBV or HCV infections and their complications

- the risk of IDU linked bacterial infections will be increased in HIV positive IDUs. Consciously consider pneumonias, tuberculosis (TB), subacute bacterial endocarditis (increased mortality), and the well recognised pneumocystis, cryptococcal, candida and cryptosporidial infections
- HIV co-infection with either HBV or HCV worsens the prognosis of the liver disease
- always ensure adequate support if diagnosis of HIV is made. Suicidal depression may follow diagnosis

## HTLV/III

Infection with these retroviruses is less frequent in IDUs than is infection with HCV, HBV or HDV.

- transmission occurs parenterally and sexually
- incidence of clinical disease is low with these infections
- no specific therapies are recommended

## RESOURCES

Hepatitis C Council of New South Wales



[www.hepatitisc.org.au/  
other\\_resources/  
other\\_resources.htm](http://www.hepatitisc.org.au/other_resources/other_resources.htm)



# Drug Issues in Correctional Services

**E**ACH Australian state and territory has its own prison system. There are no federal prisons and no national policy (nor mandate) for prisons in Australia.

According to the 1999 National Prison Census, there were 21,538 prisoners, representing an imprisonment rate of 120 per 100,000 adult population. For Indigenous Australians, however, this rate climbs to 1,690 per 100,000 (Australian Institute of Criminology, 2000).

## PATTERNS OF DRUG USE AMONG PRISONERS

Approximately 800 inmates in NSW were assessed in the Inmate Health Survey in 1996 (Butler, 1997). The study found:

- 72% smoked tobacco
- 50% reported drinking alcohol at 'harmful' levels prior to entering prison
- 64% of females and 40% of males had a history of injecting drug use
- half of the injectors had injected in prison

Reported levels of a history of injecting drug use among prison populations in other states are:

- 36% in South Australia (Gaughwin et al., 1991)
- 46% in Victoria (Crofts et al., 1995)



## HISTORY OF IMPRISONMENT AND PREVALENCE OF INJECTING

Table 20–1 shows the number of imprisoned Needle Syringe Program (NSP) clients and among those clients the level of injecting drug use whilst in prison.

- NSW prisons have recorded 5 cases of hepatitis C transmission (Haber et al., 1999; Post et al., 2001)
- Australian prisons have been recorded 4 cases of HIV transmission (Dolan et al., 1999)

## PREVALENCE AND TRANSMISSION OF BLOOD BORNE VIRAL INFECTIONS

- HIV infection remains low among Australian prisoners at less than half a percent (NCHECR, 2001)
- hepatitis C prevalence is 33% among male and 66% among female inmates in NSW (Butler et al., 1997)

## INTERVENTIONS TO REDUCE RISK BEHAVIOUR AND TRANSMISSION

Three interventions to prevent the transmission of HIV and hepatitis C are bleach, condoms and methadone programs. Table 20–2 shows which states have these programs.

**Table 20–1**  
Needle Syringe Program male and female clients who were imprisoned and injected in prison in 1996

Location	Males and Females Imprisoned (N)	% Imprisoned	Injected % (of those in prison)
NSW	M = 335	25	35
	F = 216	14	55
QLD	M = 356	7	64
	F = 160	6	78
VIC	M = 307	18	30
	F = 147	7	40
ACT	M = 59	22	46
	F = 27	7	0
NT	M = 76	12	33
	F = 24	4	0

## Bleach Programs

Two studies of the NSW bleach program found inmates had easy access to bleach and were using bleach to clean their syringes (Dolan et al., 1998, 1999). While bleach can decontaminate HIV from syringes it is unclear how effective it is against hepatitis C.

## Condoms

An evaluation of the condom program in NSW prisons found:

- most inmates were in favour of the program
- the location of the vending machines was appropriate
- 28% reported obtaining condoms
- 52% of those having anal sex always used condoms (Lowe, 1996)

## Methadone Maintenance Treatment

The NSW prison methadone program started in 1986. In 2001, there were about 1,000 inmates in methadone treatment. A randomised controlled trial of the program found that treated inmates had significantly lower levels of heroin use when measured by hair analysis and self-report (Dolan et al., submitted). There was a non-significant trend to reduced hepatitis C incidence among the treated group (24.3 vs. 31.7 per 100 person years).

## Syringe Exchange Schemes in Prison

Twenty prisons operate syringe exchange programs in Switzerland, Germany and Spain (Rutter et al., 2001). Research into these programs found that they were accepted by staff

**Table 20-2**  
Implementation of prevention measures in Australian prisons (2002)

Jurisdiction	Bleach	Condoms	Methadone (MMT)
NSW	Yes via dispensers	Yes	Yes
Victoria	Yes for general cleaning	Yes	No *
Queensland	Yes for general cleaning	No	Yes if on MMT at entry
Western Australia	No	Yes	No *
South Australia	No	No	Yes
Tasmania	Yes for general cleaning	No	Yes if on MMT at entry
Northern Territory	No	No	No
ACT	Yes on request	Yes	No

\*Methadone is available to remandees and inmates who are pregnant.

and inmates. No inmate started injecting in prison and reports of syringe sharing decreased. No assaults occurred and no new cases of HIV or hepatitis C infection were recorded. No Australian prison operates a needle and syringe program.

## REFERENCES

- Australian Institute of Criminology 2000, *Australian Crime Facts and Figures*, Canberra.
- Butler, T. 1997, *Preliminary Findings from the NSW Inmate Health Survey of the Inmate Population in the NSW Correctional System*, Corrections Health Service.
- Butler, T., Dolan, K., Ferson, M., McGuinness, L., Brown, P. & Robertson P. 1997, 'Hepatitis B and C in New South Wales prisons. Prevalence and risk factors', *Medical Journal of Australia*, vol. 166, pp. 127–130.
- Crofts, N., Stewart, T., Hearne, P., Ping X.Y., Breskin, A.M. & Locarnini, S.A. 1995, 'Spread of bloodborne viruses among Australian prison entrants'. *British Medical Journal*, vol. 310, no. 6975, pp. 285–88.
- Crofts, N., Webb-Pullman, J. & Dolan, K. 1996, 'An analysis of trends over time in social and behavioural factors related to the transmission of HIV among IDUs and prison inmates', *Evaluation of the National HIV/AIDS Strategy*, Technical Appendix 4, AGPS, Canberra.
- Dolan, K., Mattick, R.P., Shearer, J., MacDonald, M., Hall, W. & Wodak, A. (submitted), 'A randomized controlled trial of methadone maintenance treatment vs. wait list control in an Australian prison'.
- Dolan, K. & Wodak, A. 1999, 'HIV transmission in a prison system in an Australian State', *Medical Journal of Australia*, vol. 171, pp. 14–17.
- Dolan, K., Wodak, A. & Hall, W. 1998, 'A bleach program for inmates in NSW: an HIV prevention strategy', *Australian & New Zealand Journal of Public Health*, vol. 22, no. 7, pp. 838–840.
- Dolan, K., Wodak, A. & Hall, W. 1999, 'HIV risk behaviour and prevention in prison: a bleach programme for inmates in NSW'. *Drug and Alcohol Review*, vol. 18, no. 2, pp. 139–143.
- Gaughwin, M.D., Douglas, R.M. & Wodak, A.D. 1991, 'Behind bars — risk behaviours for HIV transmission in prisons, a review' in *HIV/AIDS and Prisons Conference Proceedings*, Norberry, J., Gerull S.A. and Gaughwin M.D. (eds.), Australian Institute of Criminology, Canberra.
- Haber, P., Parsons, S., Harper, S., White, P., Robinson, W. & Lloyd, 1999, 'A. Transmission of hepatitis C within Australian prisons', *Medical Journal of Australia*, vol. 171, no. 1, p. 31.
- Lowe, D. 1996, *Evaluation of the condom trial in three Correctional Centres in New South Wales: Final Report for the Department of Corrective Services*, Sydney.
- NCHECR (National Centre in HIV Epidemiology and Clinical Research) 2001, *HIV/AIDS, Hepatitis C and Sexually Transmissible Infections in Australia: Annual Surveillance Report 2001*, NCHECR, Sydney.

Post, J.J., Dolan, K.A., Whybin, L.R., Carter, I.W.J., Haber, P.S. & Lloyd A.R. 2001, 'Acute hepatitis C virus infection in an Australian prison inmate: tattooing as a possible transmission route', *Medical Journal of Australia*, vol. 174, pp. 183–184.

Rutter, S., Dolan, K., Wodak, A. & Heilpern, H. 2001, *Prison-based Syringe Exchange. A Review of International Research and Program Development*, Technical Report No. 112, National Drug and Alcohol Research Centre, Sydney.

# Health Professionals As Patients

**H**EALTH professionals are not immune to problems with drugs or alcohol. Apart from the general risk factors they share with the rest of the community, health professionals may be exposed to particular risks relating to:

- the demands, responsibilities and stresses of their professional lives
- conflict between their professional and personal lives
- easy access to prescription drugs
- self-treatment (often following self-diagnosis and self-investigation)
- reluctance of colleagues to confront or deal with early warning signs

The stakes are considerably higher in the case of health professionals with drug or alcohol problems where the well-being, and even the lives of patients under their care may potentially be at-risk. There are real and tragic examples of patients dying as a direct result of their treating doctor's dependence, and we are all aware of health professionals whose own lives are devastated by drug or alcohol problems.

Note: All health professionals should have a GP with whom they can develop a professional relationship. Self-treatment and corridor or tea-room consultations with colleagues are not appropriate or effective health care.

## TREATING A HEALTH PROFESSIONAL WITH A DRUG OR ALCOHOL PROBLEM

If you are treating another health professional you should:

- recognise that it has taken a great deal of courage (and perhaps some not-so-gentle persuasion) for a health professional to present to you for treatment
- treat them as a patient, not a colleague. This may be self-evident, but you should not assume anything about their knowledge of their problem, or expect them to take more responsibility for their management than you would expect from any other patient
- assess them in the same way that you would assess any other patient. A detailed history and appropriate physical examination are crucial and should never be circumvented
- treat them in the same way that you would treat any other patient. In these circumstances, they are your patient first and a health professional second
- provide them with the same information that you give to other patients. Assume nothing
- never allow them to prescribe or procure their own medications, no matter how convenient it may be
- be directive about their follow-up. Do not leave it up to them. Ensure provision of adequate after-care with an ongoing care manager
- consider the impact of their problem upon their work. If you believe that patient safety may be at risk, you should advise the health professional accordingly. If they are not receptive to your advice, you should seek the advice of their registering authority such as the Medical Board or Nursing Board

### Registering Authorities

Registering authorities are charged with responsibility for public protection. In some jurisdictions, there is a statutory responsibility to notify a registering authority of an impaired health professional. Most have established programs for dealing with registrants who have drug and alcohol problems and pose a current or potential risk to the public. These programs are non-disciplinary, and aimed at supporting the health professional in practice while monitoring their progress and ensuring that they are adequately treated. Contact the relevant registering authority in your state to clarify the definition of 'impairment' and find out whether you have a statutory responsibility.

## DEALING WITH A COLLEAGUE WITH A DRUG OR ALCOHOL PROBLEM

As a health professional, you should:

- be alert to the possibility that a colleague may have a drug or alcohol problem. The general indicators are discussed elsewhere in this handbook, and apply equally to health professionals. There are additional indicators that may alert you to a problem. These include:
  - unexplained behaviour changes
  - inappropriate prescribing
  - administering patient medication in a secretive manner
  - drug wastage, particularly in the case of illicit drugs
  - poor compliance with documentation requirements; e.g. drug register
  - patients complaining of inadequate pain relief

- collecting patient medications from the pharmacy
- unwillingness to respond to on-call responsibilities; e.g. refusing to return after-hours
- take action, or make sure that someone else does! It is a regrettable truth that for a variety of reasons colleagues do not act, and the consequences can be tragic for the individual and their patients. The reasons include:
  - not wanting to create waves
  - hoping that someone else will take action
  - unfounded fear of legal action
  - not knowing what to do
  - feeling intimidated by the person concerned

### The Steps to Take

1. If you feel unable to deal with the matter yourself, make your supervisor aware of your concerns. Do not let the matter drop until you are sure that you have been taken seriously
2. If you feel able to talk to the colleague yourself, *do not take on a treating role*, but:
  - arrange to meet with them privately
  - let them know that you are concerned and why
  - ask for their version of events
  - ask them to consult with an appropriate specialist
  - provide them with contact information
3. Consider alerting their head of department or supervisor
4. Follow-up to make sure that they have taken your advice. Be aware that your colleague may tell you what they think you want to hear, having taken no positive steps

Consider the impact of their problem upon their work. If you believe that patient safety may be at risk, you should advise the health professional accordingly and alert their head of department. If they are not receptive to your advice, you should seek the advice of their registering authority. Please refer to the note *Registering Authorities* above. These are actions of professional responsibility and concerned assistance for a colleague who may be in genuine distress.

### BEING A HEALTH PROFESSIONAL WITH A DRUG OR ALCOHOL PROBLEM

Health professionals may experience drug and alcohol problems just like any other member of the community.

You may feel that with your professional knowledge and skill you should be able to control and manage your problem. Experience shows that this is rarely the case.

You may feel that asking for help is an admission of personal or professional inadequacy. Unfortunately, the consequences of failing to seek help may be far more detrimental to your personal and professional life.

If you have developed a functional relationship with your GP then they are the appropriate person to help you with your problems in the first instance and you are encouraged to seek their assistance sooner rather than later.

Do not try to 'go it alone'. You will need professional support and advice. In the absence of appropriate GP support contact the Doctors' Health Advisory Service.



The Doctors' Health Advisory Services provide a 24-hour service to impaired doctors. Table 21-1 Doctors' Health Advisory Services gives contact details for each state.

In Victoria, the Victorian Doctors Health Program has been established as a full time service to assist doctors and medical students with health concerns including alcohol and other drug problems.

Contact: (03) 9495 6011

**Table 21-1**  
**Doctors' Health Advisory Services**

New South Wales	(02) 9437 6552
Queensland	(07) 3833 4352
South Australia	(08) 8273 4111
Tasmania	(03) 6223 2047 (03) 6235 4165 (after hours)
Victoria	(03) 9280 8722
Western Australia	(08) 9321 3098

# Gambling

**I**N ADDITION to problems encountered with psychoactive substances (i.e. alcohol and other drugs) there is a range of behaviours that exhibit similar patterns of problems and harms that may have the same underlying or antecedent factors. Such behaviours include eating, exercise, sexual behaviour and gambling. The latter is an area of increasing concern across Australia. Collectively, such behaviours are referred to as appetitive behaviours, that is the desires or inclinations — appetites—that are basic to life which can get out of hand (Orford, 2001).

Problematic gambling is included here because of its increasing prevalence, likelihood of presentation to health care workers and its receptiveness to similar forms of intervention as covered elsewhere in this Handbook.

## PROBLEM GAMBLING

*Problem gambling* is gambling behaviour that results in harm:

- to the individual
- to family and friends
- that may extend into the community

The harm may include:

- financial loss — even bankruptcy
- loss of employment
- criminal activity
- breakdown of relationships
- health problems including anxiety, depression and suicidal thoughts

Over the past 20 years Australian governments have legislated to allow major increases in the availability of legal gambling facilities, firstly by casinos and more recently by the introduction of electronic gaming machines into local hotels and clubs. It is well known that as the opportunities for legal gambling increase, so do the number of people who take part.

It is generally accepted that 2% of the population experience gambling problems with a further 1–3% at risk of developing a problem. Beyond this are the many thousands of family members, friends and associates adversely affected by another's gambling, with estimates of up to 10 significant others affected by any one problem gambler. In addition there are the demands on community and public resources.

## Identifying Problem Gamblers

Only a small proportion of problem gamblers have presented for help and the prevalence of problem gambling is difficult to determine.

While there is no consensus on diagnostic criteria or screening tools the 'EIGHT' Gambling Screen (see Figure 22–1) is a useful tool for patients to use and discuss with their GP. Four or more *yes* answers suggest that the patient's gambling may be affecting his/her wellbeing.

Research has examined the rates of comorbidity of problem gambling with other psychological disorders and noted:

- high rates of comorbidity with major depressive disorders
- bipolar disorder
- anxiety disorders
- drug and alcohol problems

## PRESENTATION OF PROBLEM GAMBLERS

Patients may not see a connection between their gambling and their current health concerns, or may minimise the connection out of guilt. Studies have shown that gamblers will commonly present to GPs with:

- depression
- anxiety
- headaches
- sleep difficulties
- heavy alcohol use or other drug problems
- indigestion
- back and neck pains

Broaching the subject of gambling can be done in a number of ways. While gathering a history of the complaint it may be appropriate to ask generally:

*'How are you spending your leisure time?'*

If framed as a health issue, patients may feel more comfortable to disclose that their gam-

1. Sometimes I've felt depressed or anxious after a session of gambling.  
 Yes, that's true.  
 No, I haven't.
2. Sometimes I've felt guilt about the way I gamble.  
 Yes, that's so.  
 No, that isn't so.
3. When I think about it, gambling has sometimes caused me problems.  
 Yes, that's so.  
 No, that isn't so.
4. Sometimes I've found it better not to tell others, especially my family, about the amount of time or money I spend gambling.  
 Yes, that's true.  
 No, I haven't.
5. I often find that when I stop gambling I've run out of money.  
 Yes, that's so.  
 No, that isn't so.
6. Often I get the urge to return to gambling to win back losses from a past session.  
 Yes, that's so.  
 No, that isn't so.
7. I have received criticism about my gambling in the past.  
 Yes, that's true.  
 No, I haven't.
8. I have tried to win money to pay debts.  
 Yes, that's true.  
 No, I haven't.

**Figure 22-1**  
**EIGHT Gambling Screen**

Source: *Early Intervention Gambling Health Test* (no date)

Developed by Sean Sullivan, Goodfellow Unit, Auckland Medical School (unpub.)

bling is causing them personal problems such as relationship and financial difficulties. Or that someone else's gambling problem may be the cause of the trouble.

## TREATMENT/REFERRAL OPTIONS

Discuss how:

- gambling is a system over which the gambler has little or no control
- losing is the most probable outcome
- winning in many forms of gambling is totally random
- the only true winners are the gambling industry

Indicate that gambling problems can:

- cause stress
- cause depression and anxiety
- affect physical and emotional health

Ask patients to describe the positive and negative outcomes from their gambling.

Most states offer free face-to-face counselling through agencies such as BreakEven. They offer a variety of counselling services to problem gamblers and/or their families including:

- personal counselling
- financial counselling
- relationship counselling

If a patient recognises that their gambling may be causing some harm make an appointment for them to attend one of these services and/or consider prescribing antidepressants as they can help reduce the urge to gamble. Follow-up their progress at the next planned visit.

## REFERENCES

Orford, J. 2001, *Excessive Appetites. A Psychological View of Addictions*, 2<sup>nd</sup> edn., John Wiley & Sons, Chichester.



# Appendices & Glossary





**NHMRC ALCOHOL GUIDELINES – SHORT- AND LONG-TERM RISK**

**Table A-1**  
Risk of harm in the short term

	<b>Low Risk</b> <i>minimal risk, potential health benefits</i>	<b>Risky</b> <i>regularly drinking to intoxication</i>	<b>High Risk</b> <i>sustaining moderate to high levels of drinking over time</i>
<b>Males (on any one day)</b>	up to 6 standard drinks on any one day, no more than 3 days per week	7–10 standard drinks on any one day	11 or more standard drinks on any one day
<b>Females (on any one day)</b>	up to 4 standard drinks on any one day, no more than 3 days per week	5–6 standard drinks on any one day	7 or more standard drinks on any one day
		<b>HIGH RISK OF HARM FROM INTOXICATION</b>	

Source: NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: Health risks and benefits*, National Health and Medical Research Council, Canberra.

**Table A-2**  
Risk of harm in the long term

	<b>Low Risk</b> <i>minimal risk, potential health benefits</i>	<b>Risky</b> <i>regularly drinking to intoxication</i>	<b>High Risk</b> <i>sustaining moderate to high levels of drinking over time</i>
<b>Males (on an average day)</b>	up to 4 standard drinks per day	5–6 standard drinks per day	7 or more standard drinks on any one day
<b>Males (overall weekly level)</b>	up to 28 standard drinks per week	29–42 standard drinks per week <b>HAZARDOUS</b>	43 or more standard drinks per week <b>HARMFUL</b>
<b>Females (on an average day)</b>	up to 2 standard drinks per day	3–4 standard drinks on any one day	5 or more standard drinks on any one day
<b>Females (overall weekly level)</b>	up to 14 standard drinks per week	15–28 standard drinks per week <b>HAZARDOUS</b>	29 or more standard drinks per week <b>HARMFUL</b>

Source: NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: Health risks and benefits*, National Health and Medical Research Council, Canberra.

# Appendix A

**LABORATORY MARKERS FOR ALCOHOL-RELATED DAMAGE**

Test	Advantages	Disadvantages
<p><b>(GGT) Serum Gamma-Glutamyl Transferase</b> (enzyme in liver, blood and brain)</p>	<ul style="list-style-type: none"> <li>• non-specific indicator of liver disease</li> <li>• sensitivity 20–50% for consumption of 40g alcohol per day or more</li> <li>• raised before AST and ALT</li> <li>• has half-life of 14–26 days</li> </ul>	<ul style="list-style-type: none"> <li>• low sensitivity — GGT may be elevated by medications, non-alcoholic liver disease, diabetes, obesity. A standard measure of liver function</li> </ul>
<p><b>AST/SGOT (Aspartate aminoTransferase)</b> <b>ALT/SGPT Alanine aminoTransferase</b></p>	<ul style="list-style-type: none"> <li>• reflects overall liver health</li> <li>• can be routinely obtained using standard laboratory measures</li> </ul>	<ul style="list-style-type: none"> <li>• like GGT, elevation in one of these measures alone may not necessarily be due to alcohol consumption</li> </ul>
<p><b>(CDT) Carbohydrate Deficient Transferrin</b> (variant of the protein that transports iron)</p>	<ul style="list-style-type: none"> <li>• sensitivity is 60–70%; specificity 95% (few false positives)</li> <li>• elevated levels specifically associated with the metabolism of alcohol and dependent on quantity consumed (detected at &gt; 60g per day)</li> <li>• returns to normal on reduction of consumption (half-life of 15 days)</li> </ul>	<ul style="list-style-type: none"> <li>• need to exclude uncommon liver disease e.g. primary biliary cirrhosis</li> </ul> <p>(note: this test is not routinely available in clinical practice)</p>
<p><b>(MCV) Mean red Cell Volume</b></p>	<ul style="list-style-type: none"> <li>• supportive diagnostic tool when used with LFTs</li> </ul>	<ul style="list-style-type: none"> <li>• less sensitive than GGT – can be elevated by medications e.g. valproate, azathioprine, folate and Vit B 12 deficiency, liver disease, hypothyroidism, smoking</li> </ul>
<p><b>Other laboratory measures</b></p> <p>Thrombocytopenia, elevated bilirubin, low albumin and a prolonged INR all indicate significant organ damage related to high alcohol intake (Dawe et al., 2002).</p>		

## REFERENCES

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*, 2<sup>nd</sup> edn., Commonwealth Department of Health and Ageing, Canberra.

## AUDIT – INTERVIEW VERSION

### The Alcohol Use Disorders Identification Test: Interview Version

<p>1. How often do you have a drink containing alcohol?</p> <p>(0) Never [skip to questions 9–10]            (1) Monthly or less            (2) 2 to 4 times a month            (3) 2 to 3 times a week            (4) 4 or more times a week</p> <input type="text"/>	<p>6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/>
<p>2. How many drinks containing alcohol do you have on a typical day when you are drinking?</p> <p>(0) 1 or 2            (1) 3 or 4            (2) 5 or 6            (3) 7, 8 or 9            (4) 10 or more</p> <input type="text"/>	<p>7. How often during the last year have you had a feeling of guilt or remorse after drinking?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/>
<p>3. How often do you have six or more drinks on one occasion?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/> <p>Skip to Questions 9 and 10 if Total Score for Questions 2 and 3 = 0</p>	<p>8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/>
<p>4. How often during the last year have you found that you were not able to stop drinking once you had started?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/>	<p>9. Have you or someone else been injured as a result of your drinking?</p> <p>(0) No            (2) Yes, but not in the last year            (4) Yes, during the last year</p> <input type="text"/>
<p>5. How often during the last year have you failed to do what was normally expected from you because of drinking?</p> <p>(0) Never            (1) Less than monthly            (2) Monthly            (3) Weekly            (4) Daily or almost daily</p> <input type="text"/>	<p>10. Has a relative or friend or a doctor or another health worker been concerned about your drinking or suggested you cut down?</p> <p>(0) No            (2) Yes, but not in the last year            (4) Yes, during the last year</p> <input type="text"/>
<p style="text-align: right;">Record total of specific items here</p> <p style="text-align: center;">If total is greater than recommended cut-off, consult User's Manual</p> <input type="text"/>	

Source: 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* (2<sup>nd</sup> edn.) WHO, Department of Mental Health and Substance Dependence, Geneva, [www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS](http://www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS).

# Appendix C

## AUDIT – SELF-REPORT VERSION

Scoring instructions: Each response is scored using the numbers at the top of each response column. Write the appropriate number associated with each answer in the column at the right. Then add all numbers in that column to obtain the total score.

The Questionnaire appears overleaf.



## The Alcohol Use Disorders Identification Test: Self-report Version

GP: Because alcohol use can affect your health and can interfere with certain medications and treatments, it is important that we ask some questions about your use of alcohol. Your answers will remain confidential so please be honest.

Questions	0	1	2	3	4	
1. How often do you have a drink containing alcohol?	never	monthly or less	2–4 times a month	2–3 times a week	4 or more times a week	
2. How many drinks containing alcohol do you have on a typical day when you are drinking?	1 or 2	3 or 4	5 or 6	7 to 9	10 or more	
3. How often do you have six or more drinks on one occasion?	never	less than monthly	monthly	weekly	daily or almost daily	
4. How often during the last year have you found that you were not able to stop drinking once you had started?	never	less than monthly	monthly	weekly	daily or almost daily	
5. How often during the last year have you failed to do what was normally expected of you because of drinking?	never	less than monthly	monthly	weekly	daily or almost daily	
6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	never	less than monthly	monthly	weekly	daily or almost daily	
7. How often during the last year have you had a feeling of guilt or remorse after drinking?	never	less than monthly	monthly	weekly	daily or almost daily	
8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?	never	less than monthly	monthly	weekly	daily or almost daily	
9. Have you or someone else been injured because of your drinking?	no		yes, but not in the last year		yes, during the last year	
10. Has a relative, friend, doctor or other health care worker been concerned about your drinking or suggested you cut down?	no		yes, but not in the last year		yes, during the last year	
<b>Total</b>						

Source: 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* (2<sup>nd</sup> edn.) WHO, Department of Mental Health and Substance Dependence, Geneva, [www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS](http://www.stir.ac.uk/departments/humansciences/appsocsci/audit/DRUGS), pp. 30-31.

### TIP SHEET FOR REDUCING ALCOHOL CONSUMPTION

- Drink slowly, sip, don't gulp, take smaller mouthfuls.
- Place glass on the table between each sip.  
Drink slowly, and concentrate on each mouthful.
- Alternate alcoholic drinks with non-alcoholic spacers.
- If thirsty, quench thirst with water, then have an alcoholic drink.
- Ensure there are plenty of non-alcohol or low-alcohol drinks available for yourself and your friends.
- Eat when, or before you drink as it helps to slow down the rate of absorption.
- Avoid drinking in rounds, or keeping up with your mates.  
Alternatively, buy the first round then opt out and buy your own drinks from then on.
- Plan your drinking time — begin drinking later and go home earlier.
- Drink a full glass each time and say no to top-ups.  
This makes it easier to count your drinks.
- Avoid salty snacks, no matter how tempting.
- Limit the number of drinks and money for each drinking occasion.  
Ensure you have enough food to eat, and taxi money to get home.

Source: adapted from WAADA, 1995; NSW Health, 2000

## REFERENCES

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](http://www.health.nsw.gov.au).

WAADA 1995, *The Drinker's Guide to Cutting Down or Cutting Out*, Drug and Alcohol Services Council (DASC), Adelaide.

**ALCOHOL WITHDRAWAL ASSESSMENT SCALE (CIWA-AR)**

<p><b>Nausea and vomiting</b></p> <p>Ask ‘Do you feel sick in the stomach? Have you vomited?’ Observation:</p> <ul style="list-style-type: none"> <li>(0) No nausea and no vomiting</li> <li>(1) Mild nausea with vomiting</li> <li>(2)</li> <li>(3)</li> <li>(4) Intermittent nausea, with dry retching</li> <li>(5)</li> <li>(6)</li> <li>(7) Constant nausea, frequent dry retching and vomiting</li> </ul>	<p><b>Tactile disturbances</b></p> <p>Ask ‘Have you any itching, pins and needles sensations, any burning, any numbness or do you feel bugs crawling on or under your skin?’ Observation:</p> <ul style="list-style-type: none"> <li>(0) None</li> <li>(1) Very mild itching, pins and needles, burning or numbness</li> <li>(2) Mild itching, pins and needles, burning or numbness</li> <li>(3) Moderate itching, pins and needles, burning or numbness</li> <li>(4) Moderately severe hallucinations</li> <li>(5) Severe hallucinations</li> <li>(6) Extremely severe hallucinations</li> <li>(7) Continuous hallucinations</li> </ul>
<p><b>Tremor</b></p> <p>Arms extended, elbows slightly flexed and fingers spread. Observation:</p> <ul style="list-style-type: none"> <li>(0) No tremor</li> <li>(1) Not visible, but can be felt fingertip to fingertip</li> <li>(2)</li> <li>(3)</li> <li>(4) Moderate</li> <li>(5)</li> <li>(6)</li> <li>(7) Severe, even with arms not extended</li> </ul>	<p><b>Auditory disturbances</b></p> <p>Ask ‘Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things you know are not there?’ Observation:</p> <ul style="list-style-type: none"> <li>(0) Not present</li> <li>(1) Very mild sensitivity</li> <li>(2) Mild sensitivity</li> <li>(3) Moderate sensitivity</li> <li>(4) Moderately severe hallucinations</li> <li>(5) Severe hallucinations</li> <li>(6) Extremely severe hallucinations</li> <li>(7) Continuous hallucinations</li> </ul>
<p><b>Paroxysmal sweats</b></p> <p>Observation:</p> <ul style="list-style-type: none"> <li>(0) No sweat visible</li> <li>(1) Barely perceptible sweating, palms moist</li> <li>(2)</li> <li>(3)</li> <li>(4) Beads of sweat obvious on forehead</li> <li>(5)</li> <li>(6)</li> <li>(7) Drenching sweats</li> </ul>	<p><b>Visual disturbances</b></p> <p>Ask ‘Does the light appear to be too bright? Is its colour different? Does it hurt your eyes? Are you seeing things you know are not there?’ Observation:</p> <ul style="list-style-type: none"> <li>(0) Not present</li> <li>(1) Very mild sensitivity</li> <li>(2) Mild sensitivity</li> <li>(3) Moderate sensitivity</li> <li>(4) Moderately severe hallucinations</li> <li>(5) Severe hallucinations</li> <li>(6) Extremely severe hallucinations</li> <li>(7) Continuous hallucinations</li> </ul>

continued over page

## ALCOHOL WITHDRAWAL ASSESSMENT SCALE (CIWA-AR) (CONTINUED)

<p><b>Anxiety</b></p> <p>Ask '<i>Do you feel nervous?</i>' Observation:</p> <ul style="list-style-type: none"> <li>(0) No anxiety, at ease</li> <li>(1) Mildly anxious</li> <li>(2)</li> <li>(3)</li> <li>(4) Moderately anxious or guarded so anxiety is inferred</li> <li>(5)</li> <li>(6)</li> <li>(7) Equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions</li> </ul>	<p><b>Headache, fullness in the head</b></p> <p>Ask '<i>Does your head feel different? Does it feel as though there is a band around your head?</i>' Do not rate for dizziness or light headedness. Otherwise rate severity.</p> <ul style="list-style-type: none"> <li>(0) Not present</li> <li>(1) Very mild</li> <li>(2) Mild</li> <li>(3) Moderate</li> <li>(4) Moderately severe</li> <li>(5) Severe</li> <li>(6) Very severe</li> <li>(7) Extremely severe</li> </ul>
<p><b>Agitation</b></p> <p>Observation:</p> <ul style="list-style-type: none"> <li>(0) Normal activity</li> <li>(1) Somewhat more than normal activity</li> <li>(2)</li> <li>(3)</li> <li>(4) Moderately fidgety and restless</li> <li>(5)</li> <li>(6)</li> <li>(7) Paces back and forth during most of the interview or constantly thrashes about</li> </ul>	<p><b>Orientation and clouding of sensorium</b></p> <p>Ask '<i>What day is this? Where are you? Who am I?</i>' Observation:</p> <ul style="list-style-type: none"> <li>(0) Oriented and can do serial additions Ask person to perform serial addition of 3s up to 30 e.g. 3, 6, 9...</li> <li>(1) Cannot do serial addition or is uncertain about date</li> <li>(2) Disoriented for date by no more than 2 calendar days</li> <li>(3) Disoriented for date by more than 2 calendar days</li> <li>(4) Disoriented for place and/or person</li> </ul>

## ALCOHOL WITHDRAWAL OBSERVATION CHART

### Observations

<b>Surname</b>	<b>First name</b>	<b>Age</b>
----------------	-------------------	------------

	Date								
	Time								
Breath alcohol reading									
Blood glucose reading									
Temperature (per axilla)									
Pulse									
Respiration rate									
Blood pressure									

### Alcohol Withdrawal Assessment Score

Nausea and vomiting									
Tremor									
Paroxysmal sweats									
Anxiety									
Agitation									
Tactile disturbances									
Auditory disturbances									
Visual disturbances									
Headache, fullness in head									
Orientation and clouding of sensorium									
<b>Total score</b>									

### Nursing Management:

- Nurse in a quiet, evenly lit environment
- Provide reassurance and explanation
- Re-orientate the person if confused
- Ensure adequate hydration

## Vitamin Administration

- thiamine 100 mg and multivitamins daily
- give thiamine orally unless parenteral administration indicated (e.g. malnutrition, acute Wernicke's Syndrome)
- persons receiving intravenous dextrose or glucose should first receive parenteral thiamine to prevent acute Wernicke's Syndrome.

## Medical Management of Acute Alcohol Withdrawal

When significant withdrawal is predicted preferred drug treatment is:

- diazepam 20 mg orally 2 hourly by weight (i.e. total 60 mg if weight < 75 kg; total 80 mg if weight 75–90 kg; total 100 mg if weight > 90 kg). Thereafter diazepam 20 mg orally by 2 hourly until Alcohol Withdrawal Scale (AWS) score is 10 or less
- further medical assessment is required for doses beyond 120 mg
- if AWS score rises to 15 or more recommence diazepam loading after further medical assessment

## Withdrawal Convulsion Prophylaxis

- preferred drug treatment is:
  - Day 0 diazepam by weight related loading to a minimum of 75 mg (i.e. if < 75 kg, additional 15 mg 2 hours after last dose)
  - Day 1, 2 diazepam 10 mg orally b.d.
  - Day 3 diazepam 5 mg orally b.d.
- Note: If high AWS scores occur during the Day 0 loading phase, doses should be continued 2 hourly until the score is 10 or less. Ensure that a minimum total of 75 mg diazepam has been given on Day 0 unless the patient is excessively drowsy.

## Combined Alcohol and Benzodiazepine Withdrawal

- where a combined alcohol and benzodiazepine dependence exists, the minimum dose of diazepam given during Day 0 should be equivalent to the stated dose of benzodiazepine intake, to a maximum of 80 mg. This dose should be given at a rate of 20 mg per 2 hours until the total first day dose has been given
- in the initial stages, more diazepam may be required to manage acute alcohol withdrawal symptoms or to prevent withdrawal convulsions. This should be given at a rate of 20 mg per 2 hours until the score has settled
- during subsequent days, inpatient clients will require a continuous, gradual diazepam withdrawal regime in accordance with the recommended guidelines (see *Guidelines II: Drugs: Hospital Management of Intoxication & Withdrawal*)
- such regimes feature QID doses of diazepam with the total daily dose decreasing by 5–10 mg per day over a period of 7–14 days.

## General

- symptomatic treatment (e.g. paracetamol for headache, metoclopramide for nausea or vomiting) may be useful

## THE FIVE 'A's

### 1. Ask

Which of these best describes you?

- Smoker      Thinking of quitting
- Smoker      Not thinking of quitting
- Ex-smoker      Quit in the last 6 months
- Ex-smoker      Quit for more than 6 months
- Never smoked

*Intervention Level: Brief; Moderate (Mod.); Intensive (Intens.)*

### 2. Assess

Not Interested in Quitting		Interested in Quitting		Recently Quit	
Brief	"How do you feel about your smoking right now?"  "Have you considered quitting?"	Brief	"How important is quitting for you right now?"	Brief	"Any slips, even one puff?"
Mod.	Explore difficulties and what would be the hardest thing about quitting.	Mod. Experience from past quit attempts: "What worked?" "What didn't?"  Explore motivation and confidence (use scale 1–10). Assess nicotine dependence.	Mod.	Check how they are managing. "Any benefits or problems?"	
Intens.	Systematically explore likes and dislikes; e.g. decisional balance.	Intens.	Assess high-risk situations.	Intens.	Explore high-risk situations.



3. Advise					
Not Interested in Quitting		Interested in Quitting		Recently Quit	
Brief	State importance of considering quitting and acknowledge their right to choose, handle reactivity.	Brief	Set quit date. Support decision to quit.	Brief	Affirm decision: "That's great, it's the most important thing that you could do for your health."
Mod./ Intens.	Provide personalised feedback on any objective adverse health effects.	Mod./ Intens.	Brainstorm solutions: "What has worked?"  "What didn't work?"  "What tipped you back?"  "How can I help?"	Mod./ Intens.	Summarise benefits accrued to date.

4. Assist					
Not Interested in Quitting		Interested in Quitting		Recently Quit	
Brief	Express interest: "I'm interested in your long term health and I'm here to help if you need it."	Brief	Offer Quitbook and Quitline Card.	Brief	Offer support e.g. Quitbook and Quitline Card.
Mod.	Mention Quitbook and Quitline card.	Mod./ Intens.	Develop a plan and quit date. Review dealing with nicotine withdrawal, triggers, social support, habit, negative emotions and social situations. Discuss pharmacotherapy e.g. NRT, Zyban, covering type, side effects, dosage, monitoring.	Mod.	Express ongoing interest: "I'll make a note to see how you are getting along."
Intens.	Discuss options e.g. Quitline, Pharmacotherapy (NRT, Zyban). Offer Quitbook and Quitline card. Acknowledge difficulties, most smokers take 5–6 attempts to quit.			Intens.	Help with specific situations e.g. weight gain, withdrawal, negative moods, and stress. Assist with high-risk situations.

5. Arrange					
Not Interested in Quitting		Interested in Quitting		Recently Quit	
Brief	Offer help in future.	Brief	Suggest follow-up appointment, ideally in the next seven days.	Brief	Offer follow-up appointment if there is a likelihood (or occurrence) of relapse.
Mod.		Mod.		Mod.	
Intens.	Offer appointment and explain how you can help.	Intens.	Discuss a plan highlighting the value of follow-up appointments. Discuss Quitline 12 week support program.	Intens.	

### CREATE

- C** **Coordinated** — is there a clear plan, identified role and allocated responsibilities?
- R** **Receptive** — what will stimulate interest in doing this e.g. feedback, incentives, benefits greater than costs
- E** **Effective** — are evidence based strategies for implementation being used e.g. reminder systems?
- A** **Ability** — is there adequate knowledge, appropriate skills, sufficient resources and time?
- T** **Targeted** — can all smokers be identified especially those who are interested in quitting?
- E** **Efficient** — what steps have been taken to make the process a part of routine practice?

# Appendix I

PROFORMA FOR DECISION BALANCE WORKSHEET

	Like (benefit)	Dislike (cost)
Smoke		
Quit		

# Appendix J

**HEPATITIS C REFERRAL CHECKLIST**

Date: / /

To Doctor: \_\_\_\_\_

**Referring Medical Officer**

Name: \_\_\_\_\_

Surgery: \_\_\_\_\_

Provider No: \_\_\_\_\_

**Patient Details**

Name: \_\_\_\_\_

Address: \_\_\_\_\_

Date of Birth: \_\_\_\_\_

**The following laboratory results accompany this referral:**

- HCV Antibody (confirmed +ve)
- ALT (3 abnormal results over 6 months)
- LFTs
- Prothrombin Time
- FBC
- Alpha feto protein baseline
- TSH
- HBV serology including
  - HbsAg
  - HbsAb
  - HbcAb
- HAV IgG
- HIV Ab
- Fe studies
- EUC
- Past Liver Biopsy results

Comments:

\_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

Signature:

\_\_\_\_\_

Source: Northern Rivers Area Health Service



# Appendix K

## A TO Z DRUG GLOSSARY

This has been compiled from a number of Australian and international sources. It is focussed on the AOD sector with emphasis on those concepts and terms that may be of specific interest to health professionals.

### QUICK LISTING

#### A

AA  
abstinence  
abstinence syndrome  
addict  
addiction  
ADIN  
ADIS  
administration – routes of  
agonist  
alcohol  
amphetamines  
ANCD  
antagonist  
anticraving agents  
assessment  
AUDIT  
aversion therapy

#### B

barbiturates  
behaviour change  
benzodiazepines  
binge  
brain function  
brief intervention  
brief motivational interviewing  
buprenorphine

#### C

cannabis  
CBT  
CDSR  
chroming  
cocaine  
Cochrane Library  
communication style  
comorbidity or dual diagnosis  
controlled use  
classes of psychoactive drugs  
cravings

#### D

dependence – DSM–IV & ICD–10  
depressants  
detox/detoxification  
'doctor shopping'  
drug abuse  
drug interactions  
drug-related behaviours and harms  
drug states

#### E

'E' or ecstasy  
education (community focus)  
education (patient focus)  
education (schools)  
effectiveness  
efficacy  
Evidence Based Practice (EBP)

#### F

FLAGS  
follow-up  
FRAMES

**G**

GABA  
GHB  
gold standards

**H**

half-life  
hallucinogens  
harm minimisation  
harm reduction

**I**

illicit drugs  
inhalants  
interventions  
intoxication

**K**

ketamines

**L**

LAAM  
lapse/lapse-relapse cycle  
licit drugs

**M**

maintenance  
maintenance pharmacotherapy  
methadone  
models of drug use  
motivational interviewing  
mesolimbic dopamine system/reward  
centre of the brain

**N**

National Drug Strategy (NDS)  
National Drug Strategy Household Survey  
(NDSHS)  
NEPOD  
neuro-adaptation  
neurotransmitters

**O**

opioids/opiates  
overdose

**P**

party drugs  
patient-centred approach  
patient information  
patterns of drug use  
pharmacotherapies  
polydrug use  
pregnancy and drugs  
prevention – primary, secondary & tertiary  
psychoactive drugs

**R**

relapse/relapse prevention  
risk/ 'at risk' groups

**S**

salience  
screening tools  
serotonin  
setting limits  
shared care  
social rehabilitation  
stages of change  
stimulants  
street language/names  
supply reduction

**T**

THC  
Therapeutic Community (TC)  
tolerance  
treatment options/modalities

**U**

unsanctioned drug use

**W**

withdrawal syndrome

**Y**

youth

**Z**

zero tolerance

## GLOSSARY

### A

#### AA

**Alcoholics Anonymous** is a voluntary, anonymous self-help organisation which promotes abstinence as a goal. Abstinence is achieved by commitment to a 12-step program, requiring active involvement and regular attendance at meetings. AA is not affiliated with any particular religion but is spiritually based.

#### **abstinence**

Refraining from using drugs.

#### **abstinence syndrome**

See [withdrawal syndrome](#)

#### **addict**

A term arising from the disease model of addiction. Generally considered judgmental as it stereotypes or labels a person. 'Addict' doesn't provide useful information regarding a person's pattern of AOD use (i.e. high risk, low risk). Alternatives: he/she has problems related to their drug use; she/he uses drugs.

#### **addiction**

Physical and psychological craving for a drug or drugs and related behaviours. The process of addiction is progressive and chronic. Addiction is more commonly referred to as psychological and physical dependence.

### ADIN

#### **Australian Drug Information Network**

This provides a central point of access to quality Internet-based alcohol and drug information provided by prominent Australian and international organisations. It is funded by the Australian Government Department of Health and Ageing under the National Illicit Drug Strategy – 'Tough on Drugs'



[www.adin.com.au](http://www.adin.com.au)

ADIN's large collection of quality assessed websites and databases enables organisations and individuals to search and share relevant information on licit and illicit drug issues. Info on conference events, latest publications and links to AOD services.

## ADIS

### Alcohol and Drug Information Services

Each state provides AOD assessment, referral or advisory/counselling services. Resources include printed information for people experiencing AOD related harms, and their friends, families and carers. Some states offer access to experienced clinicians on specific clinical matters; e.g. withdrawal and maintenance therapies. Generally operate a 24-hour service.

ACT	02 6205 4545	SA	1300 131 340 (Adelaide)
NSW	02 9361 8000 (Sydney) 1800 422 599 (NSW)	TAS	1800 811 994
NT	08 8981 8030 (Darwin) 1800 629 683 (NT)	VIC	13 15 70
QLD	07 3236 2414 (Brisbane) 1800 177 833 (QLD)	WA	08 9442 5000 (Perth) 1800 198 024 (WA) 1800 198 024 (WA)

### administration – routes of

Important information when taking a patient history since the way a drug (or drugs) enters the body will affect how quickly the drug has an effect, how the drug is metabolised and potential harms. Changing from one route to another may be a useful stepping stone to cutting down and quitting. Methods of administration include oral, nasal, smoked, rectal and injected.

### agonist

A drug that 'mimics' naturally occurring chemicals which stimulate receptors in the brain and CNS periphery (endogenous agonists). Agonists bind to and activate receptor sites. Their effect depends upon the drug's affinity for the receptor site and its concentration at the site.

See also [antagonist](#), [pharmacotherapies](#)

### alcohol

Classified as a sedative/hypnotic drug. Ethanol (ethyl alcohol C<sub>2</sub>H<sub>5</sub>OH) is the main *psychoactive* ingredient in alcoholic beverages. Alcohol is second to tobacco in its effect on morbidity, mortality and health care costs from drugs. Routinely asking about patterns of alcohol (&OD) use assists GPs to identify alcohol-related harms or risky patterns of drinking and provides opportunities for intervention. Screening tools for alcohol use, can be self-administered, such as the *AUDIT* have good reliability and validity.

Alcohol is commonly called *grog*, *piss* and *booze*.

### amphetamines

A category of CNS stimulants that include amphetamine (commonly known as 'speed'), methamphetamine (speed, crystal, meth, ice) as well as *d*-amphetamine and prescription drugs; e.g. Ritalin. Effects include wakefulness, perceived increases in awareness and greater energy. Other effects may include dilation of the pupils, tachycardia, elevated blood pressure, sweating, chills, loss of appetite, nausea or vomiting. *Exaggerated behaviours associated with use include aggression, agitation and impaired judgement. Chronic use can result in permanent personality and behavioural changes (WHO, 1994).*

Commonly called *go-ey, speed, go-fast, crystal, amphetts, ox blood* (mixed with iodine), *dexies, uppers, pep pills, quick, fast, ice*.

### ANCD

#### Australian National Council on Drugs

Key advisory body established by the Prime Minister that supports the Ministerial Council on Drug Strategy (MCDS). ANCD has broad representation from volunteer and community organisations and law enforcement, education, health and social welfare interests.



[www.ancd.org.au](http://www.ancd.org.au)

The ANCD website contains information about national projects, research publications including evidence supporting treatment, funding opportunities, drug use information and links to AOD related sites.

### antagonist

A drug that can bind with a receptor site in the brain, producing no pharmacological response but inhibiting the actions of agonists for that receptor

Examples: naltrexone at opioid receptors; buprenorphine, an opioid antagonist and a partial opioid agonist.

See also [agonist](#), [pharmacotherapies](#)

### anticraving agents

Pharmacotherapeutic medications that reduce craving for a drug, thus promoting abstinence.

Examples: Acamprosate [Campral] suppresses alcohol craving by facilitating **GABA** transmission and reducing glutamate so that a balance is restored.

See also [pharmacotherapies](#)

**assessment**

Taking a good history, opportunistic communication, observations and investigations and the use of appropriate screening tools are generic assessment strategies. Assessment of AOD problems includes:

- history of drug use and treatment, medical and psychiatric problems, psychosocial factors,
- physical examination and where needed laboratory tests to confirm drug use/investigate abnormalities and/or screen for illnesses predisposed by the drugs used.
- create opportunities for harm reduction (injecting behaviour, sexual behaviour, immunisation).

**AUDIT**

**Alcohol Use Disorders Identification Test**, a ten item validated questionnaire that takes approximately 2-5 minutes to complete. See Appendices C & D.

See also screening tools

**aversion therapy**

Therapy based on the use of aversive stimuli, such as electrical shock linked with drug related behaviour so that the patient is unwilling to continue certain behaviours.

**B****barbiturates**

A sedative–hypnotic group of drugs that are now rarely seen in Australia. Increasing dosage produces progressive CNS depression, ranging from mild sedation to anaesthesia. *Very narrow margin between therapeutic and lethal dose, especially with presence of tolerance, hence dangerous in overdose.*

**behaviour change**

A response to a specific behaviour (adoption/quitting). Behaviour change is influenced by beliefs and prior knowledge of the behaviour, attitudes and subjective norms surrounding the behaviour, exposure to the behaviour (weighing up the pros and cons) and the perceived ability to adopt the new behaviour.

See also stages of change motivational interviewing

**benzodiazepines**

Introduced as safer alternatives to barbiturates. They are used as anti-anxiety medications, anti-epileptics and muscle relaxants. They are classified according to speed of onset and duration of effects. *Have significant potential for dependence in a relatively short time (2–4 weeks).* Commonly called *rohies, serras, vals, moggies, sleepers, footballs, tamazies, tranx.*

Examples: *Valium, Librium, Halcion, Xanax, Ativan, Serax, and Klonopin*

Enhances inhibitory neurotransmitter GABA at post-synaptic receptors by 'bending' the receptor so that GABA molecules attach to and activate their receptors more effectively and more often. They do not act directly to open chloride ion channels, and as such are not as lethal in overdose but alcohol and benzos is potentially fatal, as ethanol opens this channel and adds synergistically to generalised depressant effects.



[www.dasc.sa.gov.au/site/page.cfm?site\\_page\\_id=88](http://www.dasc.sa.gov.au/site/page.cfm?site_page_id=88)

Benzodiazepines GP Handbook (21 pages) covering who needs help to stop? Management of withdrawal, referral, prevention of dependence – pdf 726kb. Patient resources 'Benzodiazepines 1: Reasons to Stop' and 'Benzodiazepines 2: Stopping Use' OR try the links, *Publications & Resources, Professional* from:



[www.dasc.sa.gov.au/site/page.cfm](http://www.dasc.sa.gov.au/site/page.cfm)

See also [doctor shopping](#)

### **binge**

An episode of intense (concentrated) or excessive alcohol or drug use. A prolonged binge is called a bender. Heavy drug use over a limited time period that can result in intoxication and sometimes overdose.

### **brain function**

Studies on psychoactive drugs indicate common patterns of altered brain function across all addictive substances; e.g. drug induced activation of particular brain pathways and regions; persistent changes in brain function even after prolonged abstinence; and the brain's memory of the drug experience, triggered by exposure to environmental cues that induce drug-craving and drug-seeking behaviour.

See also [mesolimbic dopamine system/reward centre of the brain](#) [neuro-adaptation](#)

### **brief intervention (BI)**

'any intervention that involves a minimum of professional time in an attempt to change drug use ... requiring a total of between five minutes and two hours' (Heather, 1990). BI provides GPs with a unique opportunity to reduce and prevent problematic use of AOD as part of their continuing and holistic care, especially for patients with low levels of problem severity and low levels of dependence. It is an effective, realistic, efficient and flexible treatment option in a primary care setting.

Examples: brief assessment, self-help materials, information on safe levels of consumption (alcohol), [harm reduction](#), [relapse prevention](#), assessment of readiness to change including [brief motivational interviewing](#), brief counselling including problem solving and goal setting, follow-up.



**brief motivational interviewing**

A technique developed for time limited (less than 5 minutes) and opportunistic interventions. Two central concepts are determining:

- the *importance* to the patient about changing their drug use behaviour and
- *confidence* about their ability to do so.

The technique consists of using scaling questions, exploring importance, summarising, building confidence, exchanging information, reducing resistance and summarising and inviting action.

See also [motivational interviewing](#) [patient centred approach](#) [stages of change](#)

**buprenorphine**

A synthetic partial opioid agonist and antagonist; blocks the effects of other opioids and has demonstrated effectiveness in opioid maintenance and withdrawal. Duration of action is dose dependent, 24–48 hours at higher doses making it useful as a maintenance treatment for opioid dependence. Withdrawal from short acting opioids (e.g. heroin) can be managed using short duration (5–7days) treatment. Withdrawal from buprenorphine is milder and the overdose risk is lower than with other opioid agonists such as methadone.

State and territory regulations restrict prescribing to accredited medical practitioners with specific training.

See also [pharmacotherapies](#)

**C****cannabis**

A generic term for the various [psychoactive](#) preparations of the marijuana plant, *Cannabis sativa*. Cannabis is both a depressant (at low doses), and a hallucinogen (at high doses). The principal psychoactive constituent is delta-9-tetra-hydrocannabinol (THC). Acute effects may include relaxation, euphoria, disinhibition, heightened visual and auditory perceptions, increased appetite, altered time perception and difficulties with concentration. Other effects may include anxiety and panic, paranoia, visual or auditory hallucinations, impaired coordination, short-term memory loss, tachycardia and supraventricular arrhythmia.

Cannabis is the most prevalent illicit drug used today (AIHW, 2001).

Commonly called *pot*, *mull*, *gunja*, *kiff*, *hooch*, *THC*, *heads*, *dope*, *grass*, *yarndi*

### **CBT**

**Cognitive Behaviour Therapy.** A broad array of therapeutic interventions that aim to provide patients with coping and living skills to function in their environment. Specific techniques include social skills training, anger management, and behavioural self-management. After-care or ongoing follow-up sessions are likely components of such a program.

### **CDSR**

See [Cochrane Library](#)

### **chroming**

The practice of inhaling vapours from spray paint. Also sometimes refers to sniffing volatile substances. Other modes of administration are huffing (saturated material is held against the mouth or nose) and bagging (vapours inhaled from a plastic or paper bag held over the nose or mouth). Both methods increase vapour concentration and euphoric effects.

See also [inhalants](#)

### **cocaine**

A central nervous system stimulant, derived from the South American coca plant. It produces euphoria, increased confidence and feelings of control, energy, reduces the need for sleep and suppresses appetite. Cocaine blocks the reuptake of dopamine, noradrenaline and serotonin at presynaptic locations, thereby increasing these neurotransmitters at postsynaptic receptor sites. Tolerance to the rewarding effects of cocaine develops extremely quickly because of rapid [neuro-adaptation](#). Crack (purest and most potent form of cocaine) is obtained by heating cocaine salt combined with baking soda (freebasing). Cocaine is snorted, usually smoked (crack) or injected intravenously. The most common clinical problems associated with cocaine use are anxiety, temporary psychosis and cardiovascular problems.

Commonly called *coke*, *snow*, *C.flake*, *stardust*, *white lady*, *crack*

### **Cochrane Library**

A regularly updated electronic library of databases and a register. It includes **CDSR**, **Cochrane Database of Systematic Reviews**, a collection of regularly updated health care reviews by an international network of experts, (named the Cochrane Collaboration). Its aim is to produce, maintain and disseminate systematic reviews of the evidence about the prevention and treatment/control of health problems.



[www.cochrane.org.au](http://www.cochrane.org.au)

The **Australasian Cochrane Centre** website.  
Email: [cochrane@med.monash.edu.au](mailto:cochrane@med.monash.edu.au).



[www.cochrane.org/cochrane/revabstr/g360index.htm](http://www.cochrane.org/cochrane/revabstr/g360index.htm)

The Drugs and Alcohol Review group listing.

### **communication style**

A communication style which is open and empathetic and non-judgmental facilitates effective responses to drug problems. In attempting to bring about behaviour change, active listening and guidance rather than prescriptions are known to be effective.

See also [motivational interviewing](#)

### **comorbidity or dual diagnosis**

Refers to a person who has a substance use problem(s) and a mental health problem(s) (e.g. depression or anxiety) at the same time. Interaction between the two can have serious consequences for a person's health and wellbeing; therefore appropriate diagnosis is essential in the management of comorbidity. Comorbid problems generally require long-term management approaches and an *integrated approach with other services*.



[www.health.gov.au/pubhlth/nds/new/comorbidity.htm](http://www.health.gov.au/pubhlth/nds/new/comorbidity.htm)

Describes the 1<sup>st</sup> phase of the National Comorbidity Project and provides discussion papers in PDF format. Includes a General Practice perspective, what comorbidity is, how common, prevention and treatment.



[som.flinders.edu.au/FUSA/PARC/toolkitcomorbidresource](http://som.flinders.edu.au/FUSA/PARC/toolkitcomorbidresource)

Treatment principles guide for GPs.



[som.flinders.edu.au/FUSA/PARC/Publications](http://som.flinders.edu.au/FUSA/PARC/Publications)

Also in PDF format on the PARC website.



[www.ceida.net.au/training\\_forum/website/midasc.htm](http://www.ceida.net.au/training_forum/website/midasc.htm)

Provides a set of info slides for trainers & GPs including comorbidity; i.e. which comes first, approaches to dealing with dual disorder, what frontline staff need to learn, treatment goals.

See also [shared care](#)

### **controlled use**

Treatment goal of moderation (cut down) as opposed to abstinence.

### **classes of psychoactive drugs**

Psychoactive drugs affect moods, thoughts and behaviours. Despite the range of drugs commonly used, broad groupings (depressant, stimulant or hallucinogenic) can assist health professionals to identify or anticipate problems related to drug type. Table G-1 lists the major effects.

**Table G-1**  
Major effects of psychoactive drugs

Depressants	Stimulants	Hallucinogens
decrease consciousness, awareness & coordination; cause relaxation & euphoria, respiratory depression, lowered HR, BP, slower reactions  e.g. alcohol, barbiturates, benzodiazepines, opioids, solvents	increase alertness, activity, confidence, heart rate, anorexia  e.g. amphetamines, nicotine, cocaine, caffeine, volatile nitrites	distort perceptions and subjective awareness  e.g. LSD, psilocybin

NOTE: Some of the limitations of classifying drugs in this way include: many drugs produce different effects with different dosages, or when combined with other drugs; individuals vary in their response to a drug(s).

**cravings**

A strong desire or urge to use drugs, most apparent during drug withdrawal and may persist long after cessation of drug use. Symptoms are both psychological and physiological. Cravings may be triggered by a number of cues; e.g. seeing a dealer, music/object association such as walking past a pub.

See also anticraving agents

**D**

**dependence**

A person a strong desire to take a drug or drugs and cannot control their use despite harmful effects. Using the DSM-IV or ICD-10 diagnostic criteria assists diagnosis of dependence. Physical dependence is referred to as neuro-adaptation. The criteria appear overleaf.

**Dependence is characterised by three (or more) of the following occurring at any time in the same 12-month period.**

1. Tolerance – the need for markedly increased amounts of the substance to achieve intoxication or desired effect or a markedly diminished effect with continued use of the same amount of the substance
2. Withdrawal – the characteristic withdrawal syndrome for the substance or where the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
3. The substance is often taken in larger amounts or over a longer period than was intended
4. There is a persistent desire or unsuccessful attempts to cut down or control substance use
5. A great deal of time is spent in activities necessary to obtain the substance or to recover from its effects
6. Social, occupational or recreational activities are given up or reduced
7. Substance use is continued despite awareness of recurrent problems associated with use.

NOTE: The World Health Organization International Classification of Diseases, 10th Edition (ICD–10) suggests that the person must possess a strong desire to take the substance and is consuming it (Proudfoot & Teesson, 2000).

#### **dependence (continued)**

**DSM–IV** Diagnostic and Statistical Manual of Mental Disorders (4<sup>th</sup> edn) of the American Psychiatric Association. Provides a widely used definition for dependence. See Table G–2.

See Table G–3 for the **ICD–10** International Classification of Diseases, 10<sup>th</sup> edition definition of dependence.

#### **depressants**

Describes a group of psychoactive drugs which effect the CNS by suppressing functions resulting in decreased consciousness, awareness and coordination. High levels of intoxication may result in stupor or coma. Included in this category is alcohol, benzodiazepines, barbiturates, small doses of cannabis, opiates (i.e. codeine, methadone).

**detox/detoxification**

Is synonymous with and more commonly termed withdrawal. Usually it refers to supervised withdrawal for a person who is dependent. It may or may not involve medication.

**‘doctor shopping’**

Common term for describing the practice of visiting many doctors over a period of time in order to obtain larger quantities of prescription drugs.

The HIC (Health Insurance Commission) defines a ‘doctor shopper’ as someone who in one year sees 15 or more different GPs, has 30 or more Medicare consults, and obtains more PBS prescriptions than appears to be clinically necessary.

**drug abuse**

Although substance abuse is a DSM–IV diagnosis the term ‘drug abuse’ is subjective and often has little relative meaning. It is probably more useful to look at patterns of use and problems of use. Alternatives: this person experiences AOD related harm; has risky patterns of use, is a dependent user etc.

See also models of drug use

**Table G–3**  
**ICD - 10 definition of dependence**

Dependence is characterised by three (or more) of the following occurring at any time in the same 12-month period.

1. Tolerance – evidence, such that increased doses of the substance are required to achieve the same effects originally produced by lower doses.
2. Withdrawal – physiological state when substance use has reduced/ ceased, evidenced by the characteristic withdrawal syndrome for the substance or use of the same substance with the intention of relieving/ avoiding withdrawal symptoms.
3. Compulsion to use – strong desire or compulsion to take the substance
4. Difficulties controlling use – difficulties in controlling substance behaviour, such as onset, termination and levels of use.
5. Substance use prioritised – neglect of other interests due to substance use (increased amount of time obtaining, using and recovering).
6. Continued use despite harmful consequences – persisting with substance use despite evidence of harmful consequences. Efforts should be taken to determine that the user was actually, or could be expected to be, aware of the extent of harm.

### drug interactions

Two or more psychoactive drugs interact with each other in one of three ways:

- *antagonism*, one drug cancels out the effects of the other, see [antagonist](#);
- *potentiation*, combined effects that exaggerate and increase the effects of each drug; e.g. alcohol and benzodiazepines enhance sedation;
- *interference*, one drug disrupts the action of the other; e.g. disulfiram prevents the metabolism of alcohol beyond acetaldehyde.

### drug-related behaviours and harms

This has been described in various models (e.g. Thorley's model – intoxication, regular use, and dependence). It can also be viewed temporally as a cycle of drug-related behaviour that starts with the acquisition of drugs and moves through administration of drugs, drug affected behaviour, recovering from drug use and withdrawal.

See also [patterns of drug use](#)

### drug states

Conditions induced by drug use that may impact on physical, cognitive and social/emotional functioning. Drug states – [intoxication](#), [overdose](#), [dependence/tolerance](#) and [withdrawal](#) require consideration when working with people who have drug-related issues. Some patients will exhibit multiple states over a period of time.

## E

### 'E' or ecstasy

The street name generally used for 3,4-methylenedioxymethamphetamine (MDMA). However other drugs may be sold as 'ecstasy', and 'ecstasy' tablets often contain a range of drugs such as amphetamine, amphetamine derivatives, caffeine, aspirin, paracetamol, ketamine in addition to, or in place of MDMA. It is usually sold as a tablet or capsule, typically with a symbol impressed on the surface.

Commonly called *white doves*, *love hearts*, *ecstasy*, *Adam*, *XTC*

### education (community focus)

The National Illicit Drug Strategy stresses the importance of increased education, counselling and referral services through community based programs as well as augmenting existing community wide education and information campaigns on illicit drugs. One of the community initiatives can be accessed at the following web address. The Community Partnerships Kit web site is a resource for groups wishing to undertake community action to prevent harms related to illicit drug use. It uses an action research model: Look, Think, Act & Reflect.



[www.communitypartnerships.health.gov.au/](http://www.communitypartnerships.health.gov.au/)

**education (patient focus)**

GPs are well placed to provide information to patients so that they are better informed about their condition and can make an informed choice about what to do. Educating patients implies being an active listener and responding to gaps in a patient's knowledge and skill base. This exchange of information can be reinforced and extended by providing published patient information.

**education (schools)**

There is a range of State and National drug strategy initiatives from Reception to Year 12 (R–12). These include teacher support materials, learning activities for students and take-home activities to involve parents and other adults in the learning process as well as early intervention approaches.

**effectiveness**

The degree to which an intervention produces desired outcomes under everyday, normal conditions in contrast to efficacy, i.e. interventions under 'ideal' conditions. To search for evidence regarding effectiveness, try the following databases: CDSR (Cochrane Database of Systematic Reviews), DARE (Database of Abstracts of Reviews of Effectiveness), EMBASE and MEDLINE.

**efficacy**

The degree to which an intervention produces desired outcomes under optimal or ideal conditions such as those carried out in a research setting with high levels of resourcing. Caution should be exercised in interpreting the relevancy of the results to a GP's setting, to the local population or service.

**Evidence Based Practice (EBP)**

**Evidence Based Practice** integrates and reviews the best available research evidence, with professional expertise and practical wisdom, to apply it to decision-making practices. It promotes the explicit, conscientious and judicious use of the best, most up-to-date research evidence to guide health care decisions.



[www.cochrane.org/cochrane/revabstr/g360index.htm](http://www.cochrane.org/cochrane/revabstr/g360index.htm)

For the most up-to-date listing of Drugs and Alcohol Reviews and protocols go to the Cochrane website.

See also gold standards



## F

### FLAGS

A treatment model in relation to AOD patients. The model consists of five steps:

Feedback (give results of screening)

Listen to patient's concerns

Advise patients about continued use of drugs

Goals of treatment should be defined

Strategies for treatment are discussed, implemented and monitored.

See also [FRAMES](#)

### follow-up

An essential process in monitoring the outcomes of treatment, providing ongoing psychological/social support and adapting treatment plans according to patient needs.

### FRAMES

A treatment model (Miller & Sanchez, 1993) with six therapeutic steps that are common to successful brief interventions:

Feedback

Responsibility

Advice

Menu

Empathy

Self-efficiency

## G

### GABA

**Gamma-aminobutyric acid** is the most important inhibitory neurotransmitter in the CNS. By gating negative chloride (Cl<sup>-</sup>) ions into the interior of nerve cells, GABA inhibits the presynaptic release of neurotransmitters due to a positive voltage polarization pulse. GABA receptors can be found at 60–80% of CNS neurons. Benzodiazepines enhance the effect of GABA at post-synaptic receptors, hence their depressant action.

### GHB

**Gamma hydroxybutyric acid (GHB)** is a naturally occurring short-chain fatty acid metabolite of gamma amino butyric acid ([GABA](#)). It is found in all body tissues, the highest concentration being in the brain where (unlike GABA) it crosses the blood–brain barrier to affect the

activity and levels of neurotransmitters. It is involved in the regulation of GABA, dopamine, 5-hydroxytryptamine, acetylcholine and affects the rate of serotonin metabolism. Research indicates that GHB produces deep reversible depression of cerebral metabolism, increases dopamine concentrations and induces hypothermia.

GHB is used as a 'party drug' and a 'drug-rape' drug. It comes in a liquid form, is easy to manufacture (recipes are available on websites), has no colour or smell, and can be easily slipped into drinks and food. It has been described as 'like alcohol without the hangover'. There is a narrow margin of safety with overdose being common. It is typically taken with other drugs (ecstasy, alcohol, amphetamines, cannabis).

Common names include *Fantasy*, *Grievous Bodily Harm*, *GHB*, *Liquid ecstasy*, *Liquid E*, *Liquid X*.



[ndarc.med.unsw.edu.au/  
ndarc.nsf/website/  
DrugInfo.resprojfactsheet](http://ndarc.med.unsw.edu.au/ndarc.nsf/website/DrugInfo.resprojfactsheet)

GHB use, patterns and associated harms (2001) based on interviews with 76 GBH users OR try 'Drug Info, Research Project Sheets' link:



[ndarc.med.unsw.edu.au/  
ndarc.nsf/website/home](http://ndarc.med.unsw.edu.au/ndarc.nsf/website/home)

### gold standards

The gold standard demonstrates a high level of confidence that the result will re-occur. A 'gold standard' is said to have the highest quality and validity at the present time and become accepted as such by the medical community.

Examples: AUDIT as a screening tool to assess alcohol risk  
Methadone as a pharmacotherapy for opiate dependent patients

## H

### half-life

The time it takes for the concentration of a drug in the blood to be reduced by 50%. Drugs with a short half-life and short duration of action are commonly used (e.g. heroin) because of their 'peak effects' than drugs with a longer duration of action and longer half-lives.

### hallucinogens

Substances that interact with the CNS affecting state of consciousness, and producing disturbances in thought and perception (e.g. illusions, and occasionally hallucinations).

Examples: LSD, Psilocybin (Magic Mushrooms)

**harm minimisation**

Underpinned the National Drug Strategy and aims to promote better health, social and economic outcomes for the community and individual. Harm minimisation includes preventing anticipated harm and reducing actual harm from licit and illicit drugs. It is a comprehensive approach to drug-related harm, involving *demand reduction*, *supply reduction* and *harm reduction* strategies. It takes into account three interacting factors: the individual, the environment/ setting and the drug(s).

**harm reduction**

Aims to reduce the impact of drug-related harm within society, at an individual and community level. It includes reducing the physical and social harms associated with drug use, encompassing the prevention of disease, death, incarceration and isolation. It acknowledges that drug use exists and will continue to, and therefore it focuses on promoting harm reduction methods.

Examples: clean needles and syringe programs, methadone as a treatment option for opioid dependency, experimental, supervised injecting facilities, brief advice, info pamphlets

**I****illicit drugs**

A drug whose production, sale or possession is prohibited. An alternative term is 'illegal drug'. Classification of legal status varies over time according to societal attitudes and legislation.

**inhalants**

A group of psychoactive substances which are CNS depressants, rapidly changing from a liquid or semisolid state to vapour when exposed to air. The most commonly used inhalants include petrol, lacquers and varnishes containing benzene and adhesives, spray paint, glue and paint thinners containing toluene. Alternatively called solvents or volatile substances. Their appeal is linked to being inexpensive, readily obtainable, easy concealment and rapid intoxication with accompanying rapid resolution of intoxication.

**interventions**

A set of sequenced and planned actions designed to reduce risky behaviours in society. Intervention often targets a specific group (*risk group*) in order to reduce the adoption of potentially harmful behaviours (such as drug use). In the GP setting interventions are synonymous with treatment plan activities, which are negotiated with the patient.

**intoxication**

The acute effects of a drug when taken on a single occasion that produce behavioural and/or physical changes. When intoxicated the amount of a drug(s) exceeds the individual's tolerance. The capacity to think and act within a normal range of ability diminishes.

See also [drug-related behaviours and harms](#)

**K****ketamines**

Fast-acting dissociative anaesthetics commonly used as animal tranquillisers. They interfere with pain pathways, leaving the respiratory and circulatory functions intact. The effects are rapid and usually last from 45–90 minutes. Low doses produce stimulant effects; medium to high doses a powerful paralysing psychedelic – and possible out-of-body or near death experiences at high doses. Ketamine is synthetically produced by the pharmaceutical industry in liquid form; however it is easy to convert into powder form by heating and grinding. It comes in liquid, pill, powder and tablet form. Injecting ketamines may cause immediate loss of consciousness.

Commonly called *special k*, *kitkat*, *vitamin k*, *'k'* and *ket*.



[www.thegooddrugsguide.com/ketamine/effects.htm](http://www.thegooddrugsguide.com/ketamine/effects.htm)

A consumer-oriented information site on ketamine and other drugs.

**L****LAAM**

Levo-alpha-acetyl-methadol, an opioid agonist used for the management of opiate dependence. LAAM has duration of action of 48–72 hours, considerably longer than other opioids such as methadone. Currently (2003) not available in Australia.

**lapse/lapse–relapse cycle**

The first use of drugs after a period of abstinence. A relapse refers to a return to uncontrolled use or use at levels similar to those prior to abstinence. It is important to recognise that lapse/relapse is not 'failure' but a step towards behaviour change. Research indicates that the absence of coping skills and certain belief systems (e.g. 'I'm an addict and can't stop') are major predictors of relapse risk.

See also [relapse](#)

### **licit drugs**

A drug whose production, sale or possession is not prohibited. 'Legal' drug is an alternative term.

Examples: Alcohol, tobacco, benzodiazepines, some inhalants

## **M**

### **maintenance**

A stage of behaviour change, in which a dependent user actively tries to remain abstinent.

### **maintenance pharmacotherapy/drug substitution**

A drug (e.g. methadone or buprenorphine) is prescribed on a long-term basis to provide pharmacological stability (i.e. relief from withdrawal/intoxication), allowing the patient to make lifestyle changes.

See also [relapse/relapse prevention](#)

### **methadone**

A synthetic opioid agonist predominantly used in maintenance therapy with patients who are dependent on opioids. Methadone liquid is usually administered once daily as the effects last 24 hours with regular dosing. It may also be used short-term (5–30 days) to relieve heroin withdrawal discomfort. GPs can become methadone prescribers by completing a nationally accredited methadone prescribers' course.

### **models of drug use**

Frameworks to assist in understanding different perspectives about drug use at an individual and community level.

Examples: *Thorley's model* identifies drug-related problems/harms, derived from three interactive and overlapping areas of drug use – dependence (being 'STUCK'), regular use (DRIP, DRIP) and intoxication.

*Zinberg's model* (based on Social Learning Theory) considers three interactive factors – the drug itself, the person, and the social setting or environment in which drug usage occurs. Zinberg's model suggests that drug use is functional with both positive and negative consequences, it is learned (therefore can be 'unlearned') and the social setting is important. Controlled use is possible.

*The Medical model* emphasises drug use (addiction) as a disease due to genetic/biological causes and the physiological and psychological effects that are induced by drug taking. This model stresses the importance of pharmacotherapy in treating drug use problems.

## models of drug use (continued)



[www.sociology101.net/system/page2ofcrack](http://www.sociology101.net/system/page2ofcrack)



[www.bali3000.com/drugnet/models/-20k-](http://www.bali3000.com/drugnet/models/-20k-)

## motivational interviewing

This is a counselling technique developed by Miller and Rollnick. It assists GPs to work with ambivalence in their patients and explore their patient's reasons to change drug use – the patient is encouraged to argue for change (not the GP) and to provide their own solutions with support from the GP.

Examples of motivational interviewing activities – exploring positive and negative consequences of drug use, exploring patient's concerns, using reflective listening and summarising to communicate understanding, eliciting self-motivational statements (e.g. *'what are the things you like and dislike about your cannabis use?'*) helping the patient to decide whether to change (e.g. *'where does this leave you now?'*).

See also [brief motivational interviewing stages of change](#)

## mesolimbic dopamine system/reward centre of the brain

A pathway in the brain comprising the ventral tegmental area (VTA), nucleus accumbens and the prefrontal cortex. It is activated by natural rewards such as sexual activity as well as psychoactive substances. The intense feeling from activation of the reward or pleasure experienced previously leads to a desire for or repetition of the behaviour. In chronic substance users this can lead to chronic and intense cravings which may be activated by anticipatory dopamine release in response to cues; e.g. drug use implements.

## National Drug Strategy (NDS)

Refers to a framework of policies and programs aimed at reducing drug-related harm in the community. The NDS aims to prevent and reduce the uptake of harmful drug use and minimise the harmful effects of licit and illicit drug use in Australian society. The NDS adopts a comprehensive approach, which encompasses the use of both licit and illicit drugs.



[www.health.gov.au/pubhlth/strateg/drugs/nds/index.htm](http://www.health.gov.au/pubhlth/strateg/drugs/nds/index.htm)

Describes the National Drug Strategy 1998–99 to 2002–03. PDF file, 393 Kb.



[www.health.gov.au/pubhlth/strateg/drugs/illicit/index.htm](http://www.health.gov.au/pubhlth/strateg/drugs/illicit/index.htm)

Describes the National Illicit Drug Strategy 1998–99 to 2002–03

### **National Drug Strategy Household Survey (NDSHS)**

This is the most current and comprehensive survey concerning licit and illicit drug use undertaken in Australia. The 2001 survey gathered information from almost 27,000 persons aged 14 years and over. Previous surveys were conducted in 1998, 1995, 1993, 1991, 1988 and 1985.

2001 National Drug Strategy Household Survey: First Results is published by the Institute of Health and Welfare and can be found on their website



[www.aihw.gov.au/](http://www.aihw.gov.au/)

Summary of drug use: proportion of the population aged 14 years and over, and mean age of initiation, Australia, 1998, 2001 OR try 'Fact Sheets' link from:



[ndarc.med.unsw.edu.au/  
ndarc.nsf/website/  
DrugInfo.factsheets](http://ndarc.med.unsw.edu.au/ndarc.nsf/website/DrugInfo.factsheets)



[ndarc.med.unsw.edu.au/  
ndarc.nsf/website/home](http://ndarc.med.unsw.edu.au/ndarc.nsf/website/home)

### **NEPOD (1998–2000)**

National Evaluation of Pharmacotherapies for Opiate Dependence.

A three-year clinical trial (1998–2000) which monitored the effectiveness of treatment options for opiate (heroin) users. Thirteen studies were conducted which investigated detox/withdrawal treatments and maintenance treatments. It found that all maintenance treatments provided benefits and that methadone was the most cost-effective; buprenorphine, because of its unique properties should also be considered.

### **neuro-adaptation**

The process whereby the brain adapts to the presence of a drug. The brain becomes relatively insensitive to normal levels of neurotransmitters by a number of postulated mechanisms. This is one explanation for craving. The drug user may experience under-stimulation (without drugs), a reduction in euphoria/pleasure and need to increase the dose to maintain the drug's euphoric effects.

### **neurotransmitters**

Chemical messengers that are released by neurones to communicate across synapses. They bind to particular receptor sites, exciting or inhibiting an action. Psychoactive drug molecules can behave like neurotransmitters, binding to a particular receptor site and occupying the neurotransmitter's position. The interaction between the drug molecule and the receptor site involves two types of action:

**Agonist** The drug's shape may be similar to the neurotransmitter molecule. The drug molecule will therefore occupy the receptor site and imitate the neurotransmitter's action.

**Antagonist** The drug's shape may resemble the shape of the neurotransmitter molecule, and occupy the receptor site, but not imitate its actions. The drug prevents the neurotransmitter from exerting its action.

**O**

**opioids/opiates**

A class of substances with morphine-like effects that can be reversed by the specific antagonist naloxone. Morphine is derived from the opium poppy; others are semi-synthetic; e.g. heroin or synthetic; e.g. pethidine; they are mostly used as analgesics for pain. Opioids stimulate opioid receptors, producing drowsiness, reduced pain perception and euphoria (highly reinforcing). Regular use leads to tolerance accompanied by craving for the drug and physical dependence with accompanying strong withdrawal syndrome on cessation of use. Potential adverse effects include respiratory depression and arrest, nausea, confusion, constipation, sedation, unconsciousness, coma; tolerance and dependence.

Examples: opium, heroin, morphine, codeine, fentanyl, methadone, buprenorphine, pethidine, diconal, palfium

**overdose**

Results from the ingestion of a drug(s) that exceeds a person's tolerance. The result may include acute psychosis (e.g. amphetamines) or potentially life threatening effects; e.g. respiratory depression with opiates.

**Table G-4**  
**Overdose categories of concern**

<p><b>Drugs that depress vital functions</b></p> <p>heroin, benzodiazepines + alcohol</p>	<p><b>Drugs that have toxic effects</b></p> <p>alcohol or analgesics e.g. paracetamol</p>	<p><b>Drugs that cause stress on internal organs</b></p> <p>stimulants which cause organs and systems to work harder</p>
---	---	--



## P

### party drugs

A term that loosely groups drugs used in the pub, club, party or rave scenes. Includes stimulants.

Examples: amphetamines, MDMA (Ecstasy), GHB, ketamine

Individuals who use party drugs often take other drugs; e.g. alcohol, antidepressants, benzodiazepines, cannabis.

### patient centred approach

This is recommended in order to encourage a good therapeutic relationship and trust with the patient. It includes regarding the person's behaviour as their choice, expressing empathy, encouraging the person to decide how much of a problem they have, avoiding arguments/confrontation, encouraging discrepancy and reevaluation of substance use. These strategies have been empirically demonstrated to enhance the quality of support to drug users and enhance the likelihood of behaviour change.

See also [motivational interviewing stages of change](#)

### patient information

Refers to effective strategies in providing patients with relevant information. Information can be provided in passive (pamphlets, posters, booklets) and active (consultation with physician) forms. Passive patient information is usually free or of minimal cost and needs to be provided in different languages to cater for the broad community.

See also [ADIS](#)

### patterns of drug use

This model suggests drug use exists as a continuum and that most drug use is experimental or occasional. Some people gradually move up the 'scale' and progress towards dependent patterns of use.



### pharmacotherapies

Involves the use of medications in responding to problem drug dependence. Medication can be used to alleviate withdrawal discomfort, as maintenance substitution; e.g. methadone or to reduce craving; e.g. naltrexone. Pharmacotherapies are best used as part of a comprehensive treatment plan that includes psychosocial therapies and support.

### polydrug use

The simultaneous or sequential non-medical use of more than one drug.

**pregnancy and drugs**

Drug and alcohol use throughout pregnancy is associated with a range of adverse effects.

- Examples:
- Increased incidence of premature labour, risk of spontaneous abortion (miscarriage) and stillbirth
  - Reduced birth size and weight, often leads to breathing difficulties and vulnerability to infection
  - Neonatal Abstinence Syndrome (NAS)
  - Fetal Alcohol Syndrome (FAS)

Assessment includes questions about history of alcohol and other drug use from the date of the woman’s last menstrual period and whether or not substance use is continuing. Management strategies include providing patient information about the effects of drug and alcohol use during pregnancy and exploring a range of choices for action.

**prevention**

Interventions that are designed to stop or delay the uptake of drugs or reduce further problems among those using drugs. Interventions can be categorised as primary, secondary or tertiary.

Table G-5

TYPE AND TARGET	FOCUS
<p><b>Primary</b></p> <p>Individuals who are likely to – or who have already begun experimenting with drugs</p>	<p>Educate about drug-related harm, and aims to prevent uptake of drug use or reduce frequency of use</p>
<p><b>Secondary</b></p> <p>Individuals who are already using drugs</p>	<p>Increase awareness about the risks involved, in order to reduce the amount of drug-related harm</p>
<p><b>Tertiary</b></p> <p>Individuals who are dependent or heavy substance users.</p>	<p>Treat and rehabilitate</p>

**psychoactive drugs**

Refers to any chemical substance which when taken into the body, alters mood, cognition and behaviour. The term ‘drug’ usually includes tobacco, alcohol, pharmaceutical drugs and illicit drugs. It also refers to other substances that have psychoactive effects; e.g. solvents.

## R

### **relapse/relapse prevention**

Relapse is a commonly described feature of those patients who are drug-dependent. It takes time for anyone to change their behaviour. Encourage patients to view relapse as just another step in a journey, not as a failure.

Relapse prevention describes a set of strategies that aim to equip patients to cope with high-risk situations that may lead to previous patterns of drug use.

See also [motivational interviewing](#)

### **risk/'at-risk' of AOD related harms**

Surveys, reviews and epidemiological data indicate that certain groups in society are potentially at greater risk of harm through intended/actual drug use, relative to the general population. These 'at-risk' groups include outpatient groups, youth, gay, lesbian, bisexual, transgender and intersex groups, Aboriginal and Torres Strait Islanders and health professionals.

## S

### **saliency**

A preoccupation with substance use, or seeking the substance. It dominates the user's thoughts or actions.

### **screening tools**

Questionnaires that assist in screening for drug use and related harms. They provide useful information and facilitate further discussion between the patient and GP. Screening tools are not designed to replace a good history but are complementary to, and time saving.

Examples    AUDIT, FAST (based on the AUDIT) & CAGE for alcohol use  
                  SDS for psychological dependence  
                  ASSIST for alcohol and other drug use

### **serotonin (5 hydroxytryptamine)**

A phenolic amine neurotransmitter that has a prominent role in sleep regulation and mood. Serotonin is affected by a number of psychoactive substances. Its synaptic concentration is increased by stimulants, especially MDMA. Its release is inhibited by opioid receptors. Many antidepressants act via their effects on serotonin; e.g. inhibition of reuptake.

### setting limits

Setting limits can have benefit for both GPs and patients. Consequences are clear and there is less room for feeling compromised or used. The basic principles are being clear, concrete and 'up front' in what you say, mean what you say and say what you mean, and follow through with what you say.

### shared care

An integrated approach to provide effective, planned delivery of care for patients with chronic or complex conditions. The AOD shared care model focuses on joint provision of clinical services by GPs and specialist AOD agencies to those patients with AOD problems and ongoing education and training for GPs.

### social rehabilitation

An intervention to integrate clients into society through education, work or housing. Traditionally social rehab was provided after completion of a treatment program but it is increasingly viewed as an intervention that can be used at any stage throughout the treatment process.



[www.emcdda.org/  
responses/themes/  
social\\_reintegration.shtm](http://www.emcdda.org/responses/themes/social_reintegration.shtm)

The European Monitoring Centre for Drugs & Drug Addiction website describes social rehabilitation, an in-depth study on social reintegration facilities and has an evaluation instruments bank that can be downloaded; European Union focus.

### stages of change

Prochaska and DiClemente described a model with five distinct stages to behaviour change:

*Pre-contemplation* – the individual may not have recognised they have a problem

*Contemplation* – the individual is beginning to question the behaviour (weigh up pros and cons).

*Action* – the individual is at the stage where they want to do something about the behaviour. They become actively involved in attempting to change their behaviour.

*Maintenance* – the individual attempts to maintain their progress by replacing the behaviour with an alternative one.

*Relapse*

See also [motivational interviewing patient centred approach](#)

**stimulants**

Any drugs that activate, enhance or increase neural activity of the CNS. Includes the amphetamines, caffeine, cocaine, nicotine and synthetic appetite-suppressants. Some other drugs such as antidepressants and certain opioids have stimulant effects in high doses or after chronic use.

**street language/names**

There are good listings of street language and their meanings in a number of publications. It is recommended that GPs use medical terminology since this is standard, less subject to change and is less likely to result in misunderstanding.

**supply reduction**

Activities that aim to disrupt the production and supply of illicit drugs. It may also be used to impose limits on access to and the availability of licit drugs such as legislation regulating the sale of alcohol and tobacco to people under the age of 18.

**T****THC**

Delta-9-tetrahydrocannabinol, the primary psychoactive constituent in cannabis.

**Therapeutic Community (TC)**

'A structured residential environment in which people live whilst undergoing drug treatment' (National Drug Strategic Framework, 1998). Therapeutic communities are long-term residential programs (often at least 3 months) that provide a holistic approach to therapy via counselling, group therapy, and other self-help strategies.

**tolerance**

A state in which continued use of a drug results in a decreased response to the drug dose. Increased doses are required to achieve the same level of effect previously produced by a lower dosage.

**treatment options/modalities**

The choice of treatment option(s) depends upon the nature and severity of the drug problem/habit, the social and environmental context in which the patient lives and the resources that exist within and outside the GP setting. There is a range of treatment options.

**U****unsanctioned drug use**

Drug use that is proscribed by law, school authorities or school policies and/or guidelines. It includes illicit, social and prescription drugs.

### W

#### **withdrawal syndrome**

Describes a range of physical and psychological symptoms that occur when a person stops or substantially reduces substance use if they have been using for a long period or/and at high doses. Generally, signs and symptoms are opposite of the acute effects of the drug. The course of the withdrawal depends upon the level of tolerance developed, other illnesses and the psychosocial environment.

### Y

#### **Youth**

Youth are a high-risk group for drug-related harms. GPs are viewed as credible information sources and are in a good position to deliver information on illicit/licit drug use to youth, parents and/or carers. Although young people have access to illicit drug information (especially on the Internet) they need information on the health consequences associated with illicit drug use, especially the long-term health effects.



[www.ysas.org.au/](http://www.ysas.org.au/)

Youth Substance Abuse Service, Victoria

See also [patterns of drug use](#)

### Z

#### **zero tolerance**

A policy that promotes the idea that 'no drugs', 'no drug use' is the aim of education and intervention activities. This contrasts with the policy of [harm minimisation](#).

## REFERENCES

AIHW (Australian Institute of Health & Welfare) 2001, *2001 National Drug Strategy Household Survey: Detailed Findings*, Drug Statistics Series No.11, AIHW cat. no. PHE 41, AIHW, Canberra.

Heather, N. 1990, cited in Ali, R., Miller, M. & Cormack, S. 1992, *Future Directions for Alcohol and Other Drug Treatment in Australia*, AGPS, Canberra.

Miller, W.R. & Sanchez M.C. 1993, 'Motivating young adults for treatment and lifestyle change' cited in Howard, G. (ed.), *Issues in Alcohol Misuse by Young Adults* University of Notre Dame Press, Notre Dame, pp. 55–79.

Proudfoot, H. & Teesson, M. 2000, *Investing in Drug and Alcohol Treatment*, National Drug and Alcohol Research Centre, Sydney, [www.ndarc.med.unsw.edu.au](http://www.ndarc.med.unsw.edu.au).

WHO (World Health Organization) 1994, *Lexicon of Alcohol and Drug Terms*, WHO, Geneva.