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Chapter 14

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566 Chapter 14 Substance misuse The psychiatry of substance misuse The subspecialty of substance misuse is concerned with the assessment and treatment of patients with problems arising from the misuse of harmful or addictive substances. These include: (1) alcohol; (2) illegal or ‘street’ drugs; (3) prescription and over-the-counter medicines; and (4) volatile chem icals. The

resultant problems include both mental and physical illnesses and family, housing, employment, and legal difficulties. Both psychological and pharmacological interventions are used in treatment, which may include de toxification and substitute prescribing. The majority of medical interventions in patients with substance use prob lems are undertaken by GPs. In areas where there are no substance misuse specialists, more complex cases are seen by general psychiatrists, with man agement of acute medical problems, including OD and withdrawals, treated in the general hospital. All psychiatrists will have ample opportunity to see and develop skills in treating patients with substance misuse. Around the UK, there is variable service provision for drug and alcohol misuse. Some services will restrict themselves to the primary substance misuse, while others will address all mental health needs. Specialists tend to work alongside voluntary and non-medical treatment agencies, many of which provide a good and vital service. Strong links between psychiatry/ substance use services and non-medical agencies should be fostered. Drug treatment services within the healthcare system make up only one part of the wider range of centrally and locally funded and volunteer services for problem drug users. Within the health service, the majority of service provision is within primary care, which will have a variable degree of experience of (and enthusiasm for) such work. The availability of specialist services will vary by area and setting (e.g. rural/urban) and may range from the special interest of an individual psychiatrist or GP to a specialist service with support staff and dedicated facilities. Local pharmacists can also be a useful resource in supervising consumption of substitute drugs. Non-healthcare provision will also vary by setting, although it may in clude: advice shops offering leaflets and education about drugs and harm reduction strategies; self-help groups, with some adhering to an Alcoholics Anonymous (AA)-style ‘12-step approach’, usually involving peer support from ex-users; and residential rehabilitation facilities, offering detoxification and abstinence programmes. The practitioner working in the field of drug misuse should develop an awareness of these services and their referral criteria and encourage a collaborative and coordinated approach to patient management. The skills required for those working in the field of substance misuse are: • Knowledge of the psychiatric symptoms and syndromes associated with substance misuse This includes the effects of substance misuse on the brain in causing psychiatric symptoms and the effects of substance misuse on pre-existing mental and physical illness. • Knowledge and understanding of the influence of psychological and social factors on substance misuse and relapse. • Experience of interviewing and counselling methods Skills in interviewing and motivating patients who may have very ambivalent feelings about changing their behaviour.

The psychiatry of substance misuse • Experience of available pharmacological and psychological treatment methods An area undergoing constant development where there is a need to keep abreast of changes in evidence. • Awareness of the culture and pattern of drug use within a community Patterns of drug use change over time, and the types and strengths of drugs available in a community will also change dynamically. Information from the police and voluntary sector can be helpful here. • Willingness to be involved with other agencies Valuable work in the field of substance misuse is done by agencies outside the healthcare system. Practitioners should attempt to understand the work of these agencies and refer to them where appropriate. • Understanding of the natural history of substance misuse/addiction Substance misuse disorders can be chronic, and at times lifelong, with a relapsing/remitting course (like many psychiatric conditions). Taking a long-term approach is therefore essential. • Ability to consider health in its wider context Substance misuse gives rise to health risks beyond the effect of the drug (e.g. drink-driving deaths, HIV infection). In addition, it is a community problem, leading to lost productivity, crime, road

accidents, violence, and family break-up. • Consideration of change beyond change in an individual patient Patterns of substance misuse in a society are susceptible to political manipulation (e.g. licensing hours, decriminalization, legalization, availability of treatment services). One role of substance misuse specialists is to understand these factors and to present the case for political change. A non-judgemental approach Sometimes there is a perception that drug or alcohol users are ‘difficult’ patients to treat. Bear in mind the General Medical Council (GMC) guide lines direct that it is ‘unethical for a doctor to withhold treatment from any patient on the basis of a moral judgement that the patient’s activities or lifestyle may have contributed to the conditions for which treatment was being sought’. Note: for the purposes of this chapter, we refer to alcohol misuse and drug misuse separately and refer to them collectively as substance misuse. Alcohol is, of course, a drug and should be thought of as such, but we be lieve this terminology to be clearer and more understandable to patients.

568 Chapter 14 Substance misuse Substance use and misuse ‘Humankind cannot bear very much reality.’ TS Eliot ‘The urge to escape, the longing to transcend themselves, if only for a few minutes, is and always has been one of the principal appetites of the soul.’ Aldous Huxley People in all cultures, at all times throughout history, have sought out mood or perception-altering substances. Twenty-five per cent of adults smoke; 90% drink alcohol; 33% have lifetime experience of one illegal drug (mostly cannabis). Society’s attitude to substance use and to those with substance use problems has varied, from prohibition and condemnation to tolerance and treatment. Within the British society, at the moment, caffeine use is legal and accepted; alcohol and tobacco use are accepted with legal limita tions; and other substances have severe legal limitations—some available only on prescription, others not at all. Despite this, the harmful effects of alcohol dwarf those of other drugs. Many of the abused substances subsequently described have been used in their naturally occurring form throughout history (e.g. the chewing of coca leaves by Peruvian Indians). There has been a tendency for the development of more potent drug preparations, which contain a higher concentration of the active ingredient (e.g. freebase cocaine), and the development of routes of administration which produce more rapid and intense effects (e.g. IV use). This has generally been associated with an increase in the attendant problems. Patients presenting with drug misuse problems represent only a small percentage of those who take drugs. Little is known about the non- presenting drug users. Their numbers may be hinted at by community sur veys, but they are otherwise poorly studied. It is clear, however, that the normal route from use of a substance to its abstinence is the individual deciding to discontinue use and then doing so, without medical consult ation or help. Reasons given for substance use are varied and may change over the course of a patient’s life. They include: a search for a ‘high’; a search for a repeat of initial pleasurable effects; cultural norm in some subcultures; self- medication for anxiety, social phobia, insomnia, and symptoms of psychotic illness; and to prevent the development of withdrawal symptoms. There is evidence for i vulnerability to substance use in those with a family history of substance misuse, and the role of environmental stressors in perpetu ating use cannot be underplayed.

Substance use and misuse The pattern of risks associated with substance use varies with the sub stance taken, the dose and route of administration, and the setting. They in clude: acute toxicity; behavioural toxicity (e.g. jumping from a height due to believing one can fly); toxic effects of drug contaminants; secondary medical problems; secondary psychiatric problems; risk of development of depend ency; and negative social, occupational, marital, and forensic consequences.

570 Chapter 14 Substance misuse Substance misuse disorders (See Boxes 14.1 and 14.2.) Acute intoxication The pattern of reversible physical and mental abnor malities caused by the direct effects of a substance. These are specific and characteristic for each substance (e.g. disinhibition and ataxia for alcohol, euphoria and visual sensory distortions for LSD). Most substances have both pleasurable and unpleasant acute effects; for some, the balance of positive and negative effects is situation-, dose-, and route-dependent. At-risk use A pattern of substance use where the person is at i risk of harming their physical or mental health. This is not a discrete point, but shades into both normal consumption and harmful use. At-risk use depends not only on absolute amounts taken, but also on the situations Box 14.1 ICD-11 'Disorders due to substance use' ICD-11 groups these together with 'Disorders due to addictive be haviours' (for further discussion, see E Impulse-control disorders 2, p. 424) as 'mental and behavioural disorders that develop as a result of the use of predominantly psychoactive substances, including medi cations, or specific repetitive rewarding and reinforcing behaviours'. Subcategories include: single episodes of harmful substance use, sub stance use disorders (harmful substance use and substance depend ence), and substance-induced disorders such as substance intoxication, substance withdrawal and substance-induced mental disorders, sexual dysfunctions, and sleep–wake disorders. The somewhat arbitrary sub stance list in ICD-10 has been brought up-to-date and now includes: al cohol; cannabis; synthetic cannabinoids; opioids; sedatives, hypnotics or anxiolytics; cocaine; stimulants, including amphetamines, methampheta mine, or methcathinone; synthetic cathinones; caffeine; hallucinogens; volatile inhalants; MDMA or related drugs, including MDA; dissocia tive drugs, including ketamine and PCP; other specified psychoactive substances, including medications; multiple specified psychoactive sub stances, including medications; unknown or unspecified psychoactive substances; and non-psychoactive substances. Box 14.2 DSM-5 'Substance use disorders' With the release of DSM-5 in May 2013, there were changes in the way substance misuse disorders were classified. Although core features of dependence, as per ICD-10 (based on Edward and Gross criteria), were largely retained as descriptive features, disorders were re--classified. The specific substance or substances are labelled as the clinically rele vant 'Substance use disorder' (e.g. alcohol use disorder, stimulant use disorder) with subclassification of 'mild', 'moderate', and 'severe'. Mild disorders require the presence of 2–3 symptoms from the core 11. Moderate disorders require the presence of 4–5 symptoms, and severe disorders require six or more symptoms.

Substance misuse disorders and associated behaviours (e.g. any alcohol use is risky if associated with driving). Harmful use The continuation of substance use despite evidence of damage to the user's physical or mental health or to their social, occupa tional, and familial well-being. This damage may be denied or minimized by the individual concerned. Dependence The layman's 'addiction'. Encompasses a range of features initially described in connection with alcohol abuse (E The dependence syndrome, p. 574), now recognized as a syndrome associated with a range of substances. Dependence includes both physical dependence (the phys ical adaptations to chronic, regular use) and psychological dependence (the behavioural adaptations). In some drugs (e.g. hallucinogens), no physical dependence features are seen. Withdrawal Where there is physical dependence on a drug, abstinence will generally lead to features of withdrawal. These are characteristic for each drug. Some drugs are not associated with any withdrawals, some with mild symptoms only, and some with significant withdrawal syndromes. Clinically significant withdrawals are recognized in dependence on alcohol, opiates, nicotine, BDZs, amphetamines, and cocaine. Symptoms of with drawal are often the 'opposite' of the acute effects of the drug (e.g. agita tion

and insomnia on BDZ withdrawal). Complicated withdrawal Withdrawals can be simple, or complicated by the development of seizures, delirium, or psychotic features. Substance-induced psychotic disorder Illness characterized by hallucin ations and/or delusions occurring as a direct result of substance-induced neurotoxicity. Psychotic features may occur during intoxication and with drawal states, or develop on a background of harmful or dependent use. There may be diagnostic confusion between these patients and those with primary psychotic illness and comorbid substance misuse. Substance- induced illnesses will be associated in time with episodes of substance misuse, will occur more readily with specific substances (e.g. cocaine), and may have atypical clinical features (e.g. late first presentation with psychosis, prominence of non-auditory hallucinations). Cognitive impairment syndromes Reversible cognitive deficits occur during intoxication. Persisting impairment (in some cases, amounting to de mentia) caused by chronic substance use is recognized for alcohol, volatile chemicals, BDZs, and, debatably, cannabis. Cognitive impairment is associ ated with heavy chronic harmful use/dependence and shows gradual de terioration with continued use and either a halt in the rate of decline or a gradual improvement with abstinence. Residual disorders Several conditions exist (e.g. alcoholic hallucinosis, E Alcohol misuse disorders 2, p. 602; persisting drug-induced psychosis, E Psychotic illnesses and substance misuse, p. 640; LSD flashbacks, E Hallucinogens, p. 624) where there are continuing symptoms despite continuing abstinence from the drug. Exacerbation of pre-existing disorder All other psychiatric illnesses, espe cially anxiety and panic disorders, mood disorders, and psychotic illnesses, may be associated with comorbid substance use. Although this may result

572 Chapter 14 Substance misuse in exacerbation of the patient’s symptoms and a decline in treatment effect iveness, it can be understood as a desire to self-medicate (e.g. alcohol taken as a hypnotic in depressive illness) or to escape unpleasant symptoms (e.g. opiates taken to ‘blot out’ derogatory auditory hallucinations). Sometimes there is debate about whether there is, for example, a primary mood dis order with secondary alcohol use, or vice versa. Careful examination of the time course of the illness may reveal the answer. In any case, it is advisable to address substance misuse problems first, as this may produce secondary mood improvements and continuing substance misuse will limit antidepres sant treatment effectiveness.

Substance misuse disorders 573

574 Chapter 14 Substance misuse The dependence syndrome This is a clinical syndrome describing the features of substance dependence. It was described initially by Edwards and Gross1 as a provisional descrip tion of alcohol dependence but may be applied to the description of drug dependence. • Primacy of drug-seeking behaviour Also called ‘salience’ of drug use. The drug and the need to obtain it become the most important things in the person’s life, taking priority over all other activities and interests. Thus, drug use becomes more important than retaining job or relationships or remaining financially solvent and in good physical health, and may diminish the moral sense, leading to criminal activity and fraud. This diminishes the ‘holds’ on a person’s continued use. If he rates drug use above health, then stern warnings about impending illness are likely to mean little. • Narrowing of the drug-taking repertoire The user moves from a range of drugs to a single drug taken in preference to all others. The setting of drug use, the route of use, and the individuals with whom the drug is taken may also become stereotyped. • i tolerance to the effects of the drug The user finds that more of the drug must be taken to achieve the same effects. They may also attempt to combat increasing tolerance by choosing a more rapidly acting route of

administration (e.g. IV, rather than smoked) or by choosing a more rapidly acting form (e.g. freebase cocaine, rather than cocaine hydrochloride). In advanced dependence, there may be a sudden loss of previous tolerance; the mechanism for this is unknown. Clinically, tolerance is exhibited by individuals who are able to display no or few signs of intoxication, while at a blood level in which intoxication would be evident in a non-dependent individual. • Loss of control of consumption A subjective sense of inability to restrict further consumption once the drug is taken. • Signs of withdrawal on attempted abstinence A withdrawal syndrome, characteristic for each drug, may develop. This may be only regularly experienced in the mornings because at all other times, the blood level is kept above the required level. • Drug taking to avoid development of withdrawal symptoms The user learns to anticipate and avoid withdrawals (e.g. having the drug available on waking). • Continued drug use despite negative consequences The user persists in drug use, even when threatened with significant losses as a direct consequence of continued use (e.g. marital break-up, prison term, loss of job). • Rapid reinstatement of previous pattern of drug use after abstinence Characteristically, when the user relapses to drug use after a period of abstinence, they are at risk of a return to the dependent pattern in a much shorter period than the time initially taken to reach dependent use. 1 Edwards G, Gross MM (1976) Alcohol dependence: provisional description of a clinical syndrome. BMJ 1:1058–61.

Stages of change and harm reduction Stages of change and harm reduction Stages of change A model for understanding motivation and action towards change in harmful patterns of drug use was proposed by Prochaska and DiClemente.2 Motivation is regarded as a prerequisite for, and a precursor to, action to wards abstinence or more controlled drug use. This model can be used when trying to tailor treatments to the individual. • Pre-contemplation The user does not recognize that problem use exists, although this may be increasingly obvious to those around them. • Contemplation The user may accept that there is a problem and begins to look at both the positive and negative aspects of continued drug use. • Decision The point at which the user decides on whether to continue drug use or attempt change. • Action The point of motivation where the user attempts change. A variety of routes exist by which change may be attempted, which may or may not include medical services. • Maintenance A stage of maintaining gains made and attempting to improve those areas of life harmed by drug use. • Relapse A return to previous behaviour, but with the possibility of gaining useful strategies to extend the maintenance period on the user’s next attempt. Harm reduction Harm reduction is a method of managing drug users, in which it is accepted that steps can be taken to reduce the mortality and morbidity for the user without necessarily insisting on abstinence from drugs. This approach gained currency during the 1980s in an attempt to halt the projected AIDS epidemic. The majority of patients will present before abstinence is a real istic or achievable goal for them. Optimum care for this group of patients will involve engaging them with the service, exploring and encouraging mo tivation to change, and suggesting harm reduction strategies. Examples of such strategies include: • Advice directed at use of safer drugs or routes of administration. • Advice regarding safer injecting practice (E Box 14.6, p. 619). • Advice regarding safe sex. • Prescription of maintenance opiates (substitution prescribing) or BDZs. • Assessment and treatment of comorbid physical or mental illness. • Engagement with other sources of help (e.g. social work, housing). Drug misuse is a community problem. Some aspects of harm reduction include consideration of reduction of morbidity to the community more generally. Prescription of methadone may reduce criminality in a dependent individual, with consequent community benefit. Equally, there is a responsi bility with the prescriber to consider the potential for community harm via leakage and accidental OD when

monitoring the prescription of any drug. 2 Prochaska JO, DiClemente CC (1986) Towards a comprehensive model of change. In: Miller WR, Heather N (eds). Treating Addictive Behaviours: Processes of Change, pp. 3–27. New York, NY: Plenum Press.

576 Chapter 14 Substance misuse Alcohol misuse In the UK, roughly 93% of men and 87% of women drink alcohol. Minimal alcohol consumption can, of course, be pleasurable, socially enjoyable, and associated with health benefits (reduction in deaths from coronary artery disease). There is a tendency to view most people as normal drinkers and a subset as vulnerable to the development of alcohol problems. In fact, on a population level, increasing the overall alcohol consumption (e.g. by reducing the real price of alcohol) tends to increase the total number of problem drinkers. Alcohol consumption in the community is roughly normally distributed, with a long 'tail' to the right. The distinction between normal and heavy drinking is arbitrary. On both a population and an individual level, i con sumption is associated with i risk of harm of all kinds. However, the fact that normal drinkers heavily outnumber heavy drinkers means that, despite their lower rates of problems, greater numbers of alcohol-related prob lems occur in normal, rather than heavy, drinkers. This gives rise to the so-called 'prevention paradox'—that to significantly reduce overall alcohol- related morbidity, we must look to reduce problems in normal, rather than heavy, drinkers. This applies more to problems such as drink-driving and drink-related trauma, rather than to medical complications of heavy use such as liver cirrhosis. The term 'alcoholic' is often used by patients themselves and is the pre ferred term of AA. It has unfortunately acquired a pejorative meaning to the general public, and images of the 'down and out' or 'skid row' alcoholic, drinking strong drinks from brown paper bags have damaged this word's use in clinical contexts. It is not used in DSM-5 or ICD-10 where the pref erence is to make the diagnosis of alcohol dependence or harmful use (al cohol use disorder in DSM-5; dependence and harmful pattern of use in ICD-11). A history of alcohol use Alcohol has been used in all societies throughout recorded history, with documentary evidence of brewing and wine-making as early as 3000 BC. The intoxicating effects of alcohol were most probably discovered inde pendently in many cultures around the time of the evolution of agriculture, possibly on noting fermentation in fruit. Ancient peoples produced alcoholic beverages from a wide variety of materials, including fruits, berries, honey, corn, barley, wheat, sugar cane, and potatoes. The use of alcohol by indi viduals has been variously regarded, from complete tolerance through to outright prohibition. Alcohol has always had a place in the lifestyles and formal rituals of many peoples around the world. It was used as an intoxicant in religious rituals, as a celebration, as a gift, as a greeting, and to mark births and deaths. For al most as long as alcohol use is recorded, there are recorded attempts at con trol on its use by the authorities. In 92 AD, the Roman emperor Domitian attempted to restrict wine production and its distribution and sale. Similar restrictions were attempted at various times by other leaders, sometimes accompanied by moral disapproval of drinking or drunkenness in particular. In medieval Britain, ale was a staple part of the diet and was consumed

Alcohol misuse in huge quantities, while drunkenness, particularly among the clergy, was frowned upon by the Christian churches. Consumption of wine, however, continued to play a role in Christian worship. After initially preaching mod eration, Mohammed later forbade the use of alcohol to followers of his religion, possibly as a way of differentiating his converts from the Christians around them. The process of natural fermentation of alcohol by yeasts can produce beverages of up to 13% proof; above this concentration, the yeast dies. Stronger concentrations of alcohol are produced by the process of distilla tion, which was discovered in the Middle East in 1000 AD. Public

consump tion of distilled liquor became prevalent in the eighteenth century, and the accompanying social problems, together with the conservative attitudes of the emerging Protestant clergy, led to a developing moral disapproval of alcohol consumption. In the mid-eighteenth century, as part of a continuing military and trade dispute with France, the British government imposed heavy taxes on French wine imports and encouraged the distillation of cheap domestic spirits—in particular, gin. This change in the drinking practice in the general popula tion from low- to high-strength alcohol produced significant alcohol-related problems in the general public, immortalized in the lithographs of the ‘gin palaces’ by George Cruikshank. In an effort to control the problem, the government passed laws to restrict the time and place at which alcohol could be sold and began to levy increasing taxes on distilled spirits. This had the positive effect of reducing consumption, but the negative effect of introducing a government interest in continuing consumption. The late eighteenth-century writings of Benjamin Rush describe habitual drunken ness as a ‘disease of the mind’. Eighteenth-century America saw the development of an increasingly widespread temperance movement (those signing a pledge ‘TA’ for total abstinence becoming known as teetotallers). The temperance movement lobbied for a complete ban on alcohol consumption and succeeded in 1921, following the passing of the eighteenth amendment to the US Constitution which provided for prohibition. The period of 11 years, until the repeal of prohibition in the twenty-first amendment, did indeed see a reduction in so cial problems and mortality; however, its unpopularity, widespread flouting of the law, and the flourishing of illegal activity in gangsterism led to its repeal. Today, in most Western countries, alcohol use is widely tolerated and socially accepted. Interestingly, moral disapproval of drinking during preg nancy and drinking while driving a motor vehicle has resulted in substantial decreases in these activities. Despite improvement in these limited areas, most Western countries have seen an increase in absolute consumption and alcohol-related medical harm, compounded by an increasing passion for drug misuse.

578 Chapter 14 Substance misuse Alcohol as a drug 1 Preparations The active ingredient in alcoholic drinks is ethyl alcohol, which makes up a variable percentage of the volume (see Box 14.3 for pricing). The flavour of drinks comes from ‘congeners’—the additional organic sub stances derived from the brewing materials. Pattern of use Of all drugs, alcohol has the widest range of patterns of use, ranging from yearly light consumption to continuous consumption throughout the waking hours. Drug actions The effects of alcohol on the CNS were traditionally de scribed as being due to non-specific effects on neuronal cell wall fluidity and permeability. It is now believed that, in addition to these general ef fects, there are neurotransmitter-specific effects, including: enhancement of GABA-A transmission (anxiolytic effects), release of DA in the mesolimbic system (euphoriant and ‘reward’ effects), and inhibition of NMDA-mediated glutaminergic transmission (amnesic effects). Ethyl alcohol is oxidized by al cohol dehydrogenase (ADH) to acetaldehyde, which, in turn, is oxidized by acetaldehyde dehydrogenase (ALDH) to CO_2 and water. Ninety-eight per cent of alcohol metabolism takes place in the liver. Approximately 1 unit (or 8g) of alcohol can be metabolized per hour. Illicitly brewed alcohol may contain methanol, which is broken down to formaldehyde that has marked toxic effects on the retina. Acute effects Alcohol is absorbed rapidly from the mouth, stomach, and small intestine, and from a single consumption, maximum blood levels are obtained in 760min. Absorption is slowed by the presence of food in the stomach and is sped up by taking effervescent drinks. Alcohol is hydrophilic and widely distributed throughout the body organs, including the brain, pla centa, lungs, and kidneys. Blood alcohol concentration (BAC) is consistent throughout the body, with the exception of fat, and can be estimated from breath samples. In normal drinkers, BAC correlates with the subjective and

the observable CNS effects of alcohol. Heavy drinkers may have a high BAC with limited outward signs of intoxication, due to the development of tolerance. Because of their different body fat distribution, women will have a higher BAC than men following the same oral intake. Initial symptoms of alcohol intoxication are subjective elevation of mood, i socialization, and disinhibition. Continuing consumption, intended to prolong these effects, can lead to lability of mood, impaired judgement, aggressiveness, slurred speech, unsteady gait, and ataxia.

Alcohol as a drug 1 Box 14.3 Minimum unit pricing (MUP) for alcohol Alcohol-related harm in psychological, medical, and social terms con tributes to high levels of morbidity and mortality globally. International bodies, such as WHO and the Organization for Economic Cooperation and Development, have long advocated for MUP for alcohol as an ef fective tool to reduce morbidity and mortality and the associated cost to public services. NICE guidelines also recommend introducing a minimum price per unit of alcohol as a very effective way of harm reduction among populations with higher rates of hazardous drinking. Evidence within published litera ture and economic analysis backs support for this guidance. The guidance is aimed at people who drink harmful amounts in the form of cheaper alcohol drinks and is based on the premise that minimal alcohol pricing curbs wider accessibility, and therefore consumption of larger quantities of cheap products. While NICE1 recognizes the potential unfair impact on people who are from disadvantaged groups in terms of accessing al cohol, it also notes the vulnerability of these groups to the impact of alcohol-related problems. When the guidance was developed, NICE con cluded that the longer-term benefit of MUP would outweigh the potential disadvantages and contribute towards reducing overall health inequalities within the population. Alcohol prices in Scotland have been deemed to be at historically low levels in recent years. This was backed up by a recent report from Alcohol Health Alliance UK's Cheap alcohol: the price we pay.2 It reported that al cohol can be purchased for as cheap as 18p per unit (a 3L bottle of White Ace™ cider). In June 2012, the Scottish Government passed the Alcohol (Minimum Pricing) (Scotland) Act 2012 for the introduction of a pre ferred minimum price of 50p per unit.3 Due to various legal challenges by a consortium of global alcohol producers, fronted by the Scotch Whisky Association (SWA) via the Court of Session and the Court of Justice of the European Union, the Act could not be implemented initially. In October 2016, the Court of Session ruled that the Scottish Government's MUP policy was legal, and with a ruling by the UK Supreme Court in November 2017 of the plans as 'a proportionate means of achieving a legitimate aim', the way is clear for the Scottish Government to bring the legislation into action in 2018. The stage is set for England, Wales, and Northern Ireland to follow suit. 1 National Institute for Health and Care Excellence (2010) Alcohol-use disorders: prevention. Public health guideline [PH24]. M https://www.nice.org.uk/guidance/ph24/resources/alcoholuse- disorders-prevention-pdf-1996237007557 [accessed 12 July 2018]. 2 Alcohol Health Alliance UK. Cheap alcohol: the price we pay—AHA report October 2016. M http://ahauk.org/cheap-alcohol-price-pay-aha-report-october-2016/ [accessed 12 July 2018]. 3 The Scottish Government (2012) Minimum unit pricing. M http://www.scotland.gov.uk/ Topics/Health/Services/Alcohol/minimum-pricing [accessed 12 July 2018].

 Alcohol as a drug 2 Societal factors The prevalence of alcohol-related harm increases with mean population consumption. This mean consumption is i by i availability of alcohol, i societal tolerance of drinking, d restrictions on the sale of alcohol, and a d 'real price' of alcohol. Price is the most influential factor in demand, with the real price of a pint of beer or bottle of whisky having dropped considerably since the war (see Box 14.3). Where societies

forbid all alcohol consumption (e.g. prohibition America, Islamic countries), there is a decrease in alcohol-related problems, but an increase in the level of personality abnormality in those who continue to drink. Risk factors Heavy drinking is more common in men, in lower socio- economic groups, in those with lower educational levels, and in the young. Some professions are also associated with heavy drinking and drink-related harm. These include: drinks industry workers (easy availability and effect of heavy drinkers seeking out jobs here); travelling salesmen (boredom, periods away from home, acceptance of drinking on the job); and doctors (stress, freedom from direct supervision, reluctance to seek help with in cipient problems). Genetics First-degree relatives of alcoholics have double the risk of al cohol problems themselves. Significantly higher rates in identical, compared with fraternal, twins (although not 100% concordance). Children of alco holics have i risk of development of alcohol problems themselves, even when adopted into families without alcohol problems. A metabolically rela tively inactive form of ALDH is common in South East Asian people, leading to accumulation of acetaldehyde and an unpleasant 'flushing' reaction in affected individuals who take alcohol. This may account for the significantly lower rate of alcohol problems found in affected individuals. No causative genes for alcoholism have been identified, and it is expected that it will show polygenic inheritance. Problem drinkers contain a significant subgroup of in dividuals with dissocial personality traits, which predisposes to alcoholism, and is itself heritable. Medical complications Acute toxicity occurs at levels over 300mg% (E Alcohol misuse disorders 1, p. 600), with clouding of consciousness and coma, risk of aspiration, hypoglycaemia, and acute renal failure. Associated with a wide range of chronic medical problems (E Medical complications of alcohol misuse, p. 608). Psychiatric complications Harmful use and dependent use (E Alcohol misuse disorders 1, p. 600), distinguished by the presence of withdrawals on abstinence; withdrawals may be complicated by seizures and devel opment of an acute confusional state—DT (E Alcohol withdrawal syn dromes, p. 590); acute alcohol-induced amnesia; alcoholic hallucinosis (E Alcohol misuse disorders 2, p. 602); alcohol-induced delusional dis order (E Alcohol misuse disorders 2, p. 602); Wernicke–Korsakoff syn drome (E Wernicke–Korsakoff syndrome, p. 606); pathological jealousy (E p. 603); alcohol-related cognitive impairment and alcoholic dementia (E Alcohol-related cognitive impairment/alcohol-related brain damage, p. 602). Alcohol misuse is also associated with the development of, or ex acerbation of, anxiety/depressive symptoms and with deliberate self-harm and suicidal behaviour.

Alcohol as a drug 2 Interventions Advice and 'brief interventions' regarding safer drinking patterns in those with 'at-risk' or harmful use (E Alcohol misuse disorders 1, p. 600); strategies towards encouraging and maintaining abstinence in those with dependency and those with established medical or psychiatric damage; medically managed detoxification (E Management of alcohol withdrawal 1, p. 592); psychological and pharmacological support of abstin ence or changed drinking pattern (E Maintenance interventions in alcohol misuse 1: psychological methods, p. 596; E Maintenance interventions in alcohol misuse 2: pharmacological methods, p. 598).

582 Chapter 14 Substance misuse Screening for alcohol problems Diseases related to alcohol abuse are common, significant, and amenable to improvement by early detection and intervention. Screening is therefore indicated. There are low rates of detection in primary care and hospital settings, which may be improved by i vigilance, i awareness of alcohol problems, awareness of routes of referral, asking routine alcohol-screening questions (e.g. CAGE; see Box 14.4), and paying special attention to at-risk groups. Many patients give reasonably accurate drinking histories if asked, although some may underestimate consumption. A combination of clinical history, screening

measure, and a biomarker is the optimal approach to detection. Disorders suggesting underlying alcohol abuse Hepatitis; cryptogenic (medically unexplained) cirrhosis; seizures—especially late onset; gastritis; anaemia; unexplained raised MCV or deranged LFTs; cardiomyopathy; accidents, particularly repeated and poorly explained; TB; head injury; hypertension persisting despite apparently adequate treatment; treatment resistance in other psychiatric conditions; impotence in men. Breath testing BAC measures recent alcohol consumption, in mg of al cohol per 100mL of blood (mg%). Correlates with breath alcohol measured by a breathalyser (see Table 14.1). Useful in assessing recent drinking (e.g. in supervised detoxication regimes) and as an objective measure of intoxi cation [e.g. in Accident and Emergency (A&E)]. Discrepancy between high BAC and a lack of apparent intoxication suggests tolerance. Measurement is dependent on adequate technique and reasonable cooperation. Blood tests Elevated red cell MCV, GGT, and carbohydrate-deficient transferrin (CDT) are markers for excess alcohol consumption. They are best used to monitor consumption in patients at follow-up. Not sensitive/ specific enough for routine screening purposes. • MCV Sensitivity 20–50%, specificity 55–100%. Remains raised for 3– 6mths due to 120-day lifespan of red blood cells (RBCs). False positive in B12 and folate deficiencies. Box 14.4 CAGE questionnaire A brief screening questionnaire for identification of at-risk drinking: C: Have you ever felt you should Cut back on your drinking? A: Has anyone ever Annoyed you by criticizing your drinking? G: Have you ever felt Guilty about your drinking? E: Have you ever had a drink early in the morning as an Eye-opener? More than two positive responses suggests possible at-risk drinking and should prompt further assessment. Note: the ‘Cage +2’ adds two additional questions: • What is the most alcohol you have drunk in a single day? • What is the most alcohol you have drunk in a single week?

Screening for alcohol problems • GGT Sensitivity 20–90%, specificity 55–100%. Raised for 2–3wks. Other LFTs are less specific for alcoholic-related liver damage. False positive in liver diseases of other cause, obesity, diabetes, smoking, and medication (e.g. anticonvulsants), and may remain raised in chronic alcoholic liver disease despite abstinence. • CDT Sensitivity 70%, specificity 95%. i in response to heavy drinking (7– 10 days), 2–3wks to return to normal, can be used to monitor relapse. More expensive than GGT and not available in all areas. Urinary tests Urinary ethyl glucuronide (an alcohol metabolite) has been proposed as a measure of alcohol intake, being sensitive to ingestion of one or two drinks, remaining elevated for several days. It has still to be used routinely, although it has been used in forensic settings. Hair testing Testing of hair for ethyl glucuronide or fatty acid ethyl es ters has been proposed as a method for detecting alcohol use over prior months, although this requires further research and validation. AUDIT/FAST Alcohol Assessment Scales NICE guideline (CG115)3 recommends various tools, including the Alcohol Use Disorders Identification Test (AUDIT) or the abbreviated AUDIT-C and the Fast Alcohol Screening Test (FAST). Others recommended as more appropriate for Emergency Departments include the Paddington Alcohol Test (PAT) or the Single Alcohol Screening Questionnaire (SASQ). For those patients referred to specialist alcohol services, validated tools recom mended for administration, additional to clinical assessment, are the AUDIT and the Severity of Alcohol Dependence Questionnaire (SADQ) (to assess the severity of dependence). Table 14.1 Breath and blood alcohol levels Breath alcohol reading (mcg%) BAC (mg%) 0.35 0.52 0.70 0.87 1.05 1.40 1.75 Note: measurement should form part of the routine assessment of a patient presenting with alcohol problems and of patients in follow-up (e.g. supervised detox), rather than being prompted by a suspicion of inaccuracy of oral report. 3 National Institute for Health and Care Excellence (2011) Alcohol-use disorders: diagnosis, assessment and management of harmful drinking and alcohol

dependence. Clinical guideline (CG115). M https:// www.nice.org.uk/guidance/cg115 [accessed 12 July 2018].

584 Chapter 14 Substance misuse Assessment of the patient with alcohol problems Patients with a primary alcohol problem, or where it is thought that alcohol consumption is a contributory factor in their presentation, should have a more detailed assessment of their alcohol use, in addition to standard psy chiatric history and MSE. Lifetime pattern of alcohol consumption Age at first alcoholic drink. Age when began to drink regularly. Age when first drinking most weekends. Age when first drinking most days. When did they first begin to drink more than their peers? When (if ever) did they first feel they had an alcohol problem? Pattern of drinking throughout life until present—describe periods of ab stinence and more heavy drinking and the reasons for these (including en vironmental/psychosocial stressors). Current alcohol consumption Describe a current day's drinking. When is the first drink taken? What types of drink are taken and in what setting? What is the total number and volume of drinks taken in a day? Some pa tients find it hard to describe a typical day or easy to over-rationalize recent heavy consumption. Ask them to describe the previous day's drinking, then the day before that, etc., until a pattern emerges. Describe a typical and a 'heavy' day's drinking (see Table 14.2). Table 14.2 Amounts of alcohol in common drinks The amount of alcohol in drinks is measured in units. One unit contains 78g of alcohol. In alcoholic drinks where the percentage of alcohol by volume is given: number of units = volume in litres × % of alcohol. The numbers of units in common drinks are given below. In calculating the numbers of units in an alcohol history, remember that home measures of drinks are usually more generous than those in pubs. Drink Alcohol % by volume Measure Alcohol units Beer and stout 4.0 Pint 2.0 Continental lager 5.0 440mL can 2.2 Strong lager 9.0 440mL can 4.0 Normal cider 4.5 Pint 1L 2.5 4.5 Strong cider 8.4 1L 8.4 Wine 9–14 125mL glass 750mL bottle 1.5 6.8–10.5 Gin/vodka/rum 37.5 25mL measure 700mL bottle 26.3

Assessment of the patient with alcohol problems Signs of dependence Do they experience withdrawals in the morning or when unable to obtain alcohol? Have they ever drunk more alcohol as a way of relieving withdrawals? Are they having to drink more to get the same intoxicating effect? Do they no longer get 'drunk' at all? Do they find it difficult to stop drinking once started? Have they tried and failed to give up, and if so, why? Do they have episodes of 'lost' memory/'blackouts'? Physical/mental health Have they been told of any physical health prob lems due to drinking? Have they previously been told to stop drinking by a doctor? Any previous or current psychiatric diagnoses? Problems related to alcohol Have they missed days at work, or had warnings about poor performance, or lost a job as a result of alcohol? Are there relationship difficulties or a relationship breakdown due to drinking? Are there financial problems? Have they been in trouble with the police, or do they have outstanding charges against them? Previous treatment attempts Describe the nature and type of previous treatments. Describe the subsequent return to drinking. Describe any periods of abstinence since the development of the drinking problem. How were they maintained and what ended them? Family history Drinking problems in parents and extended family. Quality of relationships in past and present. Childhood environment. Attitude to referral Why have they attended the appointment today? Do they feel they have an alcohol problem, and if so, will they accept help for it? What sort of help do they want, and are there types of treatment they will not accept? At what stage of change are they (pre-contemplative, con templative, decision, action)? Patient goals What (if anything) do they want to change about their drinking? What pattern of drinking do they aspire to? Physical examination Note

general condition; evidence of with drawals, including tremor in hands or protruded tongue; degree of facial capilliarization; stigmata of liver disease (palpable liver edge, jaundice, spider naevi, ascites, palmar erythema); evidence of peripheral neuropathy; ataxia of gait; breath alcohol reading. Blood testing FBC, LFTs, other blood tests, as indicated on history/ examination. Cognitive testing Although not generally indicated until 4wks of abstin ence, it is helpful to get a feel for the patient's level of cognition, especially if there is a suggestion they may be experiencing delirium or have significant alcohol-related brain damage.

586 Chapter 14 Substance misuse Giving drinking advice There are a variety of situations where the doctor will be called on to give 'safe drinking' advice: individuals whose histories reveal evolving risky drinking patterns; patients with comorbid psychiatric illness; and individuals with alcohol problems who are attempting controlled drinking, rather than abstinence. There is a wide variety of types of alcoholic drink, each of a different 'strength' (i.e. percentage alcohol by volume; see Table 14.2). It is the amount of alcohol taken, rather than the type of drink, which contributes to physical/mental health effects—avoiding spirits or other drinks perceived as 'strong' will not protect from health risks if the absolute amount of al cohol is above safe limits. Low-risk drinking guidelines In August 2016,4 new revised low-risk drinking guidelines were published UK-wide. This included new guidance for regular and single-episode con sumption and drinking during pregnancy. They proposed that to minimize health risks from alcohol, a new limit of 14 units per week for both men and women who drink regularly was recommended. This constitutes a re duction for men, the previous limit being 21 units per week. For those drinking up to 14 units, spreading the drinking over 3 or more days is advised. Drink-free days per week are also recommended. For pregnant women, no alcohol during pregnancy is advised as the safest approach. For single-occasion drinking, advice is to limit the total amount in one sit ting. Drinking at a slower pace with food and alternating with water is recommended. Certain groups that may be more vulnerable to the effects of alcohol, e.g. those at risk of falls, those with medical or mental health conditions, or those on prescribed medication with the potential to interact with alcohol, are advised to be more cautious with their levels of drinking on any single occasion. Risks encountered from heavier drinking are highlighted, including a range of medical problems (cancers of the mouth and throat) and risks of death from long-term illnesses. It also advocates drinking in a safe en vironment (getting home safely, risk of accidents and injuries) and avoiding risk-taking behaviours such as engaging in unprotected sex. Brief interventions for hazardous and harmful drinking Low-intensity, short interventions, based predominantly at primary care level, to reduce hazardous drinking. Techniques include presenting patients with screening results, identifying risks, giving medical advice, assessing the patient's goals/commitment, and working collaboratively to support the patient. 4 The Scottish Government (2016) UK Chief Medical Officers' alcohol guidelines review. M http:// www.gov.scot/Topics/Health/Services/Alcohol/safer-drinking [accessed 12 July 2018].

Giving drinking advice Techniques of controlled drinking Patients who are seeking advice about avoiding potential alcohol problems and those individuals who are seeking to change from 'at-risk' or harmful drinking patterns to controlled drinking patterns may find a selection of the following strategies helpful: • Set a weekly and daily alcohol limit and keep to it. • Do not drink alone. • Do not drink with individuals who drink heavily themselves. • Pace drinking, matching the consumption of a light or slow drinker. • Do not buy rounds. • Alternate soft and alcoholic drinks. Drink with a meal. • Rehearse what to say if offered a drink that you do not want. • Plan alternative, enjoyable non-drinking activities to replace drinking periods (e.g. cinema, sports).

588 Chapter 14 Substance misuse Planning treatment in alcohol misuse Patients presenting with alcohol problems often display marked ambiva lence about whether there is even a problem, let alone about the need for change. This reflects both the perceived positive, as well as negative, roles alcohol plays in their lives and the memory of previous failure or difficulties in attempting change. The aim in counselling such patients is to guide them in making their own decision towards change or, if change is not likely or possible now, to guide them towards harm reduction and considering the possibility of future change. Motivational interviewing This is a technique aimed at enabling a patient to move through the stages of change (E Stages of change and harm re duction, p. 575) to the point where action can be contemplated. It is based on the principle that: ‘people believe what they hear themselves say’. The interviewer aims to aid the patient in explaining why they should change their behaviour and how this will be achieved. • The therapist does not take a directive or prescriptive role but expresses interest and concern for the patient’s problems and explores the consequences of their behaviour. • Uses open-ended questions, reflective listening, and summarizing with identification of discrepancy between individual statements. • Aids the assessment of the pros and cons of current behaviour, avoiding confrontation or direct challenge. • Emphasizes the patient’s own perceptions of the degree of risk, rather than telling them about the risks which they may not believe. • Encourages personal responsibility and patient’s choice of treatment options. Planning interventions The initial assessment interview forms the beginning of intervention. Its aims are to gather and impart information, promote the possibility of positive action, and plan treatment. The ongoing thera peutic relationship aims to maintain purpose, monitor progress, and aid self- monitoring and self-awareness. The process of planning treatment should proceed along the following lines: • Make the diagnosis (alcohol dependence, harmful or at-risk use). • Assess the stage of change (E Stages of change and harm reduction, p. 575). • Decide with the patient the goal of intervention: • Continue current drinking pattern In some patients, there will be no need for change at all. In others, there will be a clear history of alcohol problems, but the patient presents as ‘pre-contemplative’ regarding change. In these cases, give harm reduction advice and ‘leave the door open’ to further assessment and help, rather than alienating the patient. • Change to a safer drinking pattern Many individuals will be able to modify risky or harmful drinking patterns, given appropriate advice and help (perhaps monitored by a ‘drinking diary’, which is later reviewed). • Attempt abstinence from alcohol In some individuals, the only safe course is to aim to abstain from alcohol completely. • For abstinence in a dependent drinker, consider the need for, and the setting of, detoxification (E Management of alcohol withdrawal 1, p. 592).

Planning treatment in alcohol misuse Plan support methods and follow-up (E Maintenance interventions in al cohol misuse 1: psychological methods, p. 596). • At follow-up contact, review progress, emphasize changes made, and review mental health. • Anticipate and deal with relapse if it occurs. Abstinence vs controlled drinking The decision to try for controlled drinking, rather than abstinence, is one for individual patient choice. The doctor should offer suitable advice. • Factors suggesting the possibility of success of controlled drinking: previous prolonged periods of controlled drinking, alcohol misuse primarily in the context of other mental disorder which has responded to treatment, otherwise stable lifestyle, absence of drinking problem in family and friends. • Factors against controlled drinking: previously alcohol-dependent, previous failure at controlled drinking, comorbid mental illness, comorbid drug use, established organ damage, risk of job loss/marriage loss. Relapse Alcohol misuse is a chronic illness, and many patients will ‘fall off the wagon’ several times before achieving long-standing change. The pos sibility of relapse should

be anticipated with the patient, and appropriate strategies should be in place to deal with it (e.g. early review). Causes of relapse: ambivalent motivation, insufficient support, novel events, over-confidence, mental illness, environmental stressors. Counselling families The family of a patient with alcohol problems may con tact you directly to ask for advice regarding their relative. • The patient's relatives sometimes request that their relative be detained in hospital 'to stop them drinking'. The Mental Health Acts in the UK specifically do not allow detention of patients solely for the reason of drug or alcohol dependency. • Aim to encourage and reward moves by the drinker to achieve change in their drinking pattern, while avoiding rewarding, and hence reinforcing, drinking, but avoiding confrontation or ultimatums. • Sometimes continued family involvement, despite their best intentions, serves only to support the drinker in their chosen lifestyle. In this case, the family may have to be aided to step back (AA calls this 'disengaging with love'). Prognostic factors There is 73.6-fold excess mortality, compared with age- matched controls. Of 100 45-yr-old patients at 20-yr follow-up: 40% dead, 30% abstinent, 30% problem drinking. Positive factors: motivated to change; supportive family or relationship; in employment; treatable comorbid illness (e.g. anxiety disorder, social phobia); accepting of appropriate treatment goal; AA involvement. Negative factors: ambivalent about change; unstable accommodation or homeless; drinking embedded into lifestyle (e.g. limited pursuits outside alcohol, all friends are drinkers); repeated treatment fail ures; cognitive impairment.

590 Chapter 14 Substance misuse Alcohol withdrawal syndromes In a patient with alcohol dependence, stopping alcohol completely or sub stantially reducing the usual amount causes the development of character istic withdrawal syndromes. These syndromes should be anticipated, and prophylaxis considered in any patient: • With a history of dependence. • Who has previously experienced withdrawal syndromes. • Who has consumed >10 units of alcohol on a daily basis for the previous 10 days. • Currently experiencing withdrawals. Uncomplicated alcohol withdrawal syndrome • Occurs 4–12hrs after the last alcoholic drink. • Features: coarse tremor, sweating, insomnia, tachycardia (pulse >100), nausea and vomiting, psychomotor agitation, and generalized anxiety. • Occasionally, transitory visual, tactile, or auditory hallucinations or illusions. • There may be increasing craving for alcohol both in itself and as a relief from withdrawal symptoms. • Symptoms increase in severity in rough proportion to the habitual alcohol consumption, peaking at 48hrs and lasting 2–5 days, with symptoms being more prolonged in heavier drinkers. Alcohol withdrawal syndrome with seizures • In 5–15% of cases, withdrawals are complicated by grand mal seizures occurring 6–48hrs after the last drink. • If seizures occur only during withdrawal, they do not signify the development of idiopathic epilepsy. • Predisposing factors: previous history of withdrawal seizures, idiopathic epilepsy, history of head injury, hypokalaemia. Delirium tremens Acute confusional state (E Acute confusional state (delirium), p. 854) secondary to alcohol withdrawal. A medical emergency requiring inpatient medical care. • Occurs in 75% of episodes of withdrawal. Onset 1–7 days after the last drink, with a peak incidence at 48hrs. • Risk is i by severe dependence, comorbid infection, and pre-existing liver damage. • In addition to the features of uncomplicated withdrawal, there is: • Clouding of consciousness. • Disorientation. • Amnesia for recent events. • Marked psychomotor agitation. • Visual, auditory, and tactile hallucinations (characteristically of diminutive people or animals—'Lilliputian' hallucinations). • Marked fluctuations in severity hour by hour, usually worse at night. • In severe cases: heavy sweating, fear, paranoid delusions, agitation, suggestibility, raised temperature, sudden cardiovascular collapse.

Alcohol withdrawal syndromes • Reported mortality of 5–10%. It is most risky when it develops unexpectedly and its initial manifestations are misinterpreted (e.g. in a patient not known to be alcohol-dependent developing symptoms post-operatively). • Differential diagnosis: hepatic encephalopathy, head injury, pneumonia, acute psychotic illness, acute confusional state with other primary cause.

592 Chapter 14 Substance misuse Management of alcohol withdrawal 1 Detoxification (detox) is the medical management of withdrawal symp toms in a patient with substance dependence. Alcohol detox involves: psy chological support; medication to relieve withdrawal symptoms (usually via a reducing BDZ regime); observation for the development of features of complicated withdrawal; nutritional supplementation; and integration with follow-up. Detox may be carried out as inpatient or, with support, in the community. The need to medically manage the complications of al cohol withdrawal can also arise in an unplanned fashion (e.g. in an alcohol- dependent patient in police custody or following emergency surgery). Most of the problems of alcohol use are related to the inability to maintain abstin ence, rather than to the initial problems of withdrawal. Detoxification procedure • Decide on the setting. • Assess the need for a BDZ-reducing regime. • Consider the need for other medications. • Provide verbal and written advice. • Inform the GP of the plans. • Give the patient a contact in case of emergency. • Decide on explicit follow-up after detox. Setting Outpatient detoxification • Treatment of choice for most uncomplicated alcohol-dependent patients, with comparable completion rates to inpatient detox and comparable percentage of those remaining abstinent at 6mths. • Where there are doubts about compliance or concerns about drinking ‘on top of’ the prescribed drug, the patient should be seen daily in the morning and breathalysed before dispensing that day’s and the following morning’s supply of the drug. Indications for inpatient detoxification • Past history of complicated withdrawals (seizures or delirium). • Current symptoms of confusion or delirium. • Comorbid mental/physical illness, polydrug misuse, or suicide risk. • Symptoms of Wernicke–Korsakoff syndrome (E Wernicke–Korsakoff syndrome, p. 606). • Severe nausea/vomiting; severe malnutrition. • Lack of stable home environment. Reducing regime BDZs are prescribed in alcohol withdrawal to ameliorate unpleasant with drawal symptoms (e.g. tremor, anxiety) and to reduce the risk of with drawal seizures. They are prescribed in a rapidly reducing regime, in order to avoid the development of secondary, iatrogenic dependence, while covering the period of maximum risk (see Table 14.3). • Many units prefer chlordiazepoxide to diazepam for outpatient use, as it has lower abuse potential.

Management of alcohol withdrawal 1 • Diazepam is often preferred for inpatient use, as it is faster-acting, allowing dose titration against effect, and can be given parenterally. Preferred in those with a history of alcohol withdrawal seizures. • BDZs remain the first-line pharmacological treatment for acute alcohol withdrawals in hospital settings, but NICE also includes carbamazepine or, in select cases, chlormethiazole as treatment options. Indications for prescribing a reducing regime • Clinical symptoms of withdrawal. • History of alcohol dependence syndrome. • Consumption is >10 units/day over the previous 10 days. Not required if • <10 units daily. • No history of withdrawals/drinking to avoid anticipated withdrawals. • BAC = 0 and no withdrawal symptoms. Symptom monitoring Review patients regularly to assess withdrawals. Continuing symptoms should be managed by increasing the next day’s planned dosages, rather than increasing the length of the course or relying on ‘as required (PRN)’ dosage. Table 14.3 Benzodiazepine withdrawal regime Suggested outpatient reducing regime using chlordiazepoxide On waking

Midday Early evening At bedtime Day 1 – 30mg 30mg 30mg Day 2 20mg 20mg 20mg 20mg Day 3 20mg 10mg 10mg 10mg Day 4 10mg 10mg – 20mg Day 5 10mg – – 10mg

594 Chapter 14 Substance misuse Management of alcohol withdrawal 2 Fixed-dose regimens and symptom-triggered regimens NICE guidelines (CG100)5 recommend the use of symptom-triggered regimens as an adjunct to clinical assessment and monitoring of acute al cohol withdrawal for patients in hospital or other 24-hr assessment set tings. These are regimens tailored to patient-specific symptoms that trigger medication administration for symptom alleviation, based on severity. Medication is withheld if no symptoms are observed. NICE cites the Clinical Institute Withdrawal Assessment—Alcohol, revised (CIWA–Ar) scale as an example—a 10-item assessment tool used to assess, monitor, and treat alcohol withdrawal. Clinically, this scale is widely used in hospitals. Fixed- dose, rather than symptom-triggered, regimens are preferred in community detox settings. Other medications • Anticonvulsants BDZs in sufficient dosage are the most effective anticonvulsants in alcohol withdrawal. Other oral drugs (e.g. phenytoin, carbamazepine) do not reach therapeutic level until after the time of maximal risk. • Antipsychotics Where hallucinations or delusions develop, they can usually be managed by temporarily increasing the BDZ dose. Addition of an antipsychotic [e.g. haloperidol 5–10mg orally (PO) up to tds] should be considered if this fails. Antipsychotics reduce seizure threshold; with sufficient BDZ cover, this should not be a concern. • Supplementary vitamins Where there are symptoms suggestive of Wernicke–Korsakoff syndrome or evidence of malnourishment, give parenteral B vitamins (E Wernicke–Korsakoff syndrome, p. 606). In other patients, give a 4-wk course of 100mg thiamine tds, in addition to multivitamins (mineral deficiencies, e.g. magnesium, are commonly seen in this group, and can predispose to withdrawal seizures). • Other psychotropics While many patients withdrawing from alcohol complain of anxiety and/or depressive symptoms, many will be directly secondary to alcohol use/withdrawal. Do not treat with psychotropics until the patient has been assessed when abstinent from alcohol. Generally speaking, do not start new psychotropics at this time. Post-alcohol detoxification Before prescription of pharmacotherapy for post-detox patients within specialist alcohol services, appropriate medical pre-assessment should be undertaken. NICE guidelines (CG115)6 particularly recommend blood tests—U&Es, LFTs, and GGT. For those patients with moderate to severe alcohol dependence, NICE recommends acamprosate and naltrexone, in 5 National Institute for Health and Care Excellence (2010) Alcohol-use disorders: diagnosis and man agement of physical complications. Clinical guideline [CG100]. M https://www.nice.org.uk/guid ance/cg100 [accessed 12 July 2018]. 6 National Institute for Health and Care Excellence (2011) Alcohol-use disorders: diagnosis, assessment and management of harmful drinking and alcohol dependence. Clinical guideline [CG115]. M https:// www.nice.org.uk/guidance/cg115 [accessed 12 July 2018].

Management of alcohol withdrawal 2 combination with additional alcohol-focused psychological therapy, as first- line post-detox treatment. They only recommend disulfiram for those in whom naltrexone and/or acamprosate are unsuitable or if patients spe cifically request disulfiram and have a sound understanding of the risks (E Aversive drugs, p. 598). Inpatient or residential alcohol detoxification programmes For patients requiring inpatient or residential alcohol detox, NICE recom mends that those consuming between 15 and 30 units of alcohol daily should be considered for inpatient/residential detox if they also meet various other criteria. These criteria include: significant medical comorbidities (including alcohol-related, e.g. withdrawal seizures, DT); psychiatric comorbidities (including learning disabilities and cognitive impairment), severe

malnutri tion; a score of >30 on the SADQ; and vulnerable adults.

596 Chapter 14 Substance misuse Maintenance interventions in alcohol misuse 1: psychological methods In planning treatment in alcohol problems, attention should be focused not only on achieving, but also on maintaining, change. Many patients find the initial change (e.g. moving to abstinence or controlled drinking) surpris ingly easy but find it difficult to maintain change in the longer term. Alcohol misuse is a chronic illness characterized by relapse, and in dependent drinkers, there is the tendency for dependent drinking patterns to recur rapidly on abstinence. For this reason, maintenance interventions should support change, and in every patient, relapse should be anticipated and strategies to deal with it should be in place. Individual counselling In addition to monitoring agreed change, individual counselling can address the following: • Social skills training (e.g. 'saying no'). • Problem-solving skills. • Relaxation training. • Anger management. • Cognitive restructuring. • Relapse prevention. In selected patients, there may be a role for more formal psychotherapies. Group support A variety of group methods, both within the health ser vice and in the voluntary sector. Variable local provision. Most widespread and best known is AA (see Box 14.5). Pharmacological support (E Maintenance interventions in alcohol misuse 2: pharmacological methods, p. 598). Residential abstinence In selected patients, time in a residential facility may offer a period of abstinence which is unachievable 'outside', allowing interventions in physical and mental health and a chance to plan social change to permit continued abstinence on discharge. A variety of facilities exist, usually outside healthcare provision; some offer detox, while others will only accept patients following detox. Most residential rehabilitation centres will utilize group therapies and follow the '12-step' approach, ad vocated by AA (see Box 14.5). Residential rehabilitation is used in patients where home environment is unsupportive of abstinence and there has been failure of previous treatment options. Advice to all patients regarding relapse Returning to drinking is the most common outcome in patients (and some consider relapse as pathogno monic of addiction). The stages of change model (E Stages of change and harm reduction, p. 575) considers relapse to be at the beginning of a further process of change, but with i knowledge as to future strategies to combat relapse. A relapse can be motivated by over-confidence or forget ting gains. A 'slip' does not mean a full-blown relapse is inevitable, and all patients should have strategies to deal with relapse discussed and agreed 'ahead of time'.

Maintenance interventions in alcohol misuse 1: psychological methods ALCOHOL MISUSE 1: PSYCHOLOGICAL METHODS Box 14.5 Alcoholics Anonymous (AA) Alcoholics Anonymous (AA) is the best known and most widespread of the voluntary self-help organizations for problem drinkers. It was founded in 1935 in the USA by Bill Wilson and Dr Bob Smith, themselves both problem drinkers. Currently, there are 73,000 groups in the UK and 788,000 groups worldwide. Associated organizations are Al-Anon (for relatives of problem drinkers); Al-Ateen (for teenage children of problem drinkers); and Narcotics Anonymous (NA) (for addicts of illicit drugs). AA views alcoholism as a lifelong, incurable disease, the symptoms of which can be arrested by lifelong abstinence. Many other groups will use a variant of the AA model—the '12-step' programme. AA is a useful, ef fective intervention for many problem drinkers, and all patients should be encouraged to consider attendance. An AA meeting will generally follow a standard routine—there will be 10–20 people in each group, and only first names are used; a rotating chairman will introduce himself with 'My name is X, and I am an alcoholic', then will read the AA preamble; a number of speakers are called from the floor who give an account of their stories and recovery, if possible, leading to general discussion; the meeting ends with a prayer and is fol lowed by informal discussions and contact

between new members and sponsors who may offer emotional and practical support and perhaps a phone number. Open meetings are held where friends, family, and inter ested professionals can attend. Closed meetings are for members only. (See E Useful resources, p. 1075 for contacts in the UK and Ireland.) The ‘12 steps’

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a power higher than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God as we understood him.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of the persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory, and when we were wrong promptly to admit it.
11. Sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as a result of these steps, we tried to carry this message to alcoholics and to practise these principles in our affairs. Reprinted with kind permission of AAWS.

598 Chapter 14 Substance misuse Maintenance interventions in alcohol misuse
2: pharmacological methods (See Linford-Hughes et al., 2012.)7 Aversive drugs Disulfiram (Antabuse®, Esperdal®) Action Irreversible inhibition of ALDH which converts alcohol to CO2 and water. If alcohol is taken, there is a build-up of acetaldehyde in the blood stream, causing unpleasant symptoms of flushing, headache, nausea and vomiting, and tachycardia. There is also recent evidence to suggest it may block dopamine B hydroxylase, increasing DA and decreasing NA. Indication Can act as a helpful adjunct to therapy and allow the patient’s relatives/employers to regain confidence in their ability to remain abstinent (evidence for i efficacy with supervised administration). Dose Prescribe once abstinence achieved. Loading dose 800mg; then re duce over 5 days to 100–200mg daily or 200–400mg on alternate days. Side effects Halitosis and headache. Rare reports of psychotic reactions and hepatotoxicity. Notes • Patients should be counselled as to the nature and purpose of the drug and the likely side effects if they drink. • It is no longer recommended to give an alcohol ‘challenge’ to a patient newly started on disulfiram. • Compliance is i if the taking of the drug is monitored by another person (e.g. spouse). Anti-craving drugs Acamprosate (Campral EC®) Action Believed to act through enhancing GABA transmission in the brain. Has been found to reduce alcohol consumption in animal models of alcohol addiction, with possible neuroprotective effects. Patients taking it report diminished alcohol craving. In an RCT, a cohort treated with acamprosate showed an i percentage of those remaining abstinent and a doubling of time to first relapse. The majority of trials have been conducted with ad junctive psychosocial treatments, and there, these should accompany treatment. Indications

Patients who wish to remain abstinent from alcohol. Dose Once abstinence achieved/at end of detox: 666mg tds. Side effects GI upset, itch, rash, altered libido. (Generally well tolerated.) 7 Lingford-Hughes AR, Welch H, Peters L, et al. (2012) BAP updated guidelines: evidence-based guidelines for the pharmacological management of substance abuse, harmful use, addiction and comorbidity: recommendations from BAP. J Psychopharmacol 26:899–952. M https://www.bap.org.uk/pdfs/BAP_Guidelines-Addiction.pdf [accessed 12 July 2018].

599 ALCOHOL MISUSE 2: PHARMACOLOGICAL METHODS Notes • Discontinue if the patient returns to regular drinking or relapses more than once, while on the drug. • Has no role in assisting with controlled drinking. • Has no aversive action if alcohol is taken (though can be used in conjunction with disulfiram). • Has no addictive potential itself. Nalmefene (Selincro®) Action Acts at the opioid receptor as a μ and δ receptor antagonist, and a partial agonist at the κ receptor, reducing reward when patients consume alcohol, therefore reducing its reinforcing effect. Indications Recommended by NICE8 for use in conjunction with psycho social interventions for people with alcohol dependence who are heavy drinkers (drinking >5 units daily for women and 7.5 units daily for men persistent 2wks following initial assessment), with no requirement for detox for physical withdrawals. Complete abstinence from alcohol is not required, as the aim is to reduce overall alcohol intake. Dose 18mg/day. Side effects Nausea, dizziness, insomnia, and headaches. Naltrexone (Adepend®) Action Antagonizes the effects of endogenous endorphins released by al cohol consumption. It is believed that this diminishes both the desirable ‘high’ experienced on taking alcohol and the loss of control reported by most dependent drinkers. Indications In motivated subgroups of alcohol-dependent patients, it ap pears to be effective in reducing total alcohol consumed and the number of drinking days. Dose Once abstinence achieved, give 25mg od initially, maintenance 50mg od. Side effects GI upset, feeling anxious/‘on edge’, headache, fatigue, sleep disturbance, flu-like symptoms. Notes • Does not have an aversive or dependence-producing effect. • Not currently licensed in the UK for treatment of alcohol dependence. Baclofen Action Baclofen is a GABA-B agonist, mainly used to treat neurological con ditions that cause muscle spasticity. Indications There is some evidence in the literature that it may be effective for relapse prevention in alcohol dependence, particularly in those with cir rhotic liver disease, due to its limited liver metabolism and short half-life of 1–2hrs. Although not licensed, it is recognized as a potential intervention in the BAP guidelines.7 8 National Institute for Health and Care Excellence (2014). Nalmefene for reducing alcohol consump tion in people with alcohol dependence. Technology appraisal guidance [TA325]. M https://www.nice. org.uk/guidance/ta325 [accessed 12 July 2018].

600 Chapter 14 Substance misuse Alcohol misuse disorders 1 Acute intoxication The symptoms of alcohol intoxication will vary, de pending on the BAC, individual alcohol tolerance, and, to some extent, the setting in which the alcohol is taken. In general, as BAC rises from mild in toxication (BAC <100mg%) to moderate intoxication (BAC 100–200mg%) to severe intoxication (BAC >200mg%), a characteristic syndrome of acute intoxication is observed. Initial symptoms are elevated mood, disinhibition, and impaired judgement, followed by slurred speech, unsteady gait, nys tagmus, ataxia, aggressiveness, lability of mood and impaired concentration, and eventually sopor and coma. At-risk drinking There are reported benefits to health (lowered risk of coronary artery disease and strokes) associated with low levels of alcohol consumption, as compared with those who are abstinent (the ‘J-shaped curve’), although this remains a contentious area. Above this low level, health risks increase with increasing alcohol consumption. It is therefore arbitrary at

which point drinking is considered ‘at risk’. Patient and situ ational factors are important (e.g. any alcohol consumption while driving or in pregnancy carries i risks; for patients with established alcohol-related organ damage, any consumption is risky). Harmful drinking (DSM-5—alcohol use disorder) Non-dependent drinking which continues, despite established harm to the patient’s physical or mental health secondary to the alcohol consumption. ICD-10 diagnosis considers only physical and mental health harm, not harm related to social sanction. Alcohol dependence Harmful use of alcohol + established dependence syndrome (E The dependence syndrome, p. 574). Usually, daily stereo typed drinking pattern, with i tolerance, withdrawal features on abstin ence, and ‘relief drinking’ (i.e. further drinking to alleviate the effects of withdrawals). Pathological intoxication (‘mania à potu’) This is a medically and legally disputed syndrome which was not included in DSM-5 (or ICD-11), due to lack of empirical evidence, but is found in ICD-10. It is described as an idio syncratic reaction to a small amount of alcohol, characterized by severe agi tation, belligerence, and violent behaviour, followed by collapse, profoundly deep sleep, and amnesia for the events which followed the alcohol con sumption. It is a dubious diagnosis which is mainly sought after by defence lawyers, as most legal systems do not regard normal self-induced intoxica tion as a valid defence. There is, of course, a strong association between alcohol and violent crime. Careful re-examination of the history will usually demonstrate that significant quantities of alcohol have been consumed. Alcohol-induced amnesia (‘blackouts’ or ‘palimpsest’) This term re fers to transient amnesic episodes related to periods of intoxication. Characteristically, the patient will report a ‘gap’ in their memory lasting sev eral hours, with global or partial amnesia for their actions during that time. The patient’s behaviour, as reported by witnesses, is usually characteristic of their normal behaviour when intoxicated. This amnesia seems to be a failure of recall, rather than initial registration, and represents a reversible

Alcohol misuse disorders 1 form of brain damage. Its occurrence is not predictive of longer-term cog nitive impairment. It occurs in the later stages of a drinking career, if at all, and tends to recur once established. Two forms are described: • ‘En bloc’—dense amnesia with well-demarcated start and finish points. • Partial—episodes with indistinct start and end points, with islands of preserved memory and variable degrees of recall. There is some degree of state-dependent recall in blackouts, and a return to intoxication may aid recall. Because of the potential confusion of the term ‘blackout’ with periods of loss of consciousness, the term ‘alcoholic palimpsest’ is to be preferred.

602 Chapter 14 Substance misuse Alcohol misuse disorders 2 Alcoholic hallucinosis This is a substance-induced psychotic illness (de fined in ICD-10), which is a rare complication of prolonged heavy alcohol abuse. The sufferer experiences hallucinations—usually auditory—in clear consciousness and while sober. The auditory hallucinations may begin as elemental hallucinations (e.g. bangs or murmurings) before, with continued alcohol use, being experienced as formed voices, most usually derogatory in nature. There may be secondary delusional elaboration. The nature of hal lucinations tends to worsen during periods of alcohol detox, and, at times, when intoxicated with alcohol. • Differential diagnosis Transitory hallucinatory or illusionary experiences while intoxicated, DT, psychotic illnesses. • Course In 795% of patients, there is rapid resolution of these symptoms on ceasing alcohol consumption, but the symptoms rapidly recur on restarting drinking. In 75%, there are prolonged symptoms (<6mths after abstinence) and an emergence of more typical schizophrenic symptomatology. • Management Persisting symptoms may be treated with antipsychotic medication (bearing in mind the medical comorbidities seen in this population).

Alcohol-induced psychotic disorder with delusions Long recognized, but only recently included in diagnostic guidelines—DSM-5 now includes substance-/medication-induced psychotic disorders in its chapter for schizophrenia spectrum and other psychotic disorders. Development of persecutory or grandiose delusions after a long history of heavy drinking. No other features of DT. Resolves on abstinence. Delirium tremens (E Alcohol withdrawal syndromes, p. 590.) Alcohol-related cognitive impairment/alcohol-related brain damage The classification of alcohol-related cognitive impairment is unclear. ICD-10 views it as a number of discrete entities, as opposed to a continuum: am nesic disorder (F10.6), dementia (F10.73), and other persisting cognitive disorder (F10.74). DSM-5 has moved all cognitive impairment diagnoses to a separate section 'Neurocognitive disorders'. In this section, ARBD is now 'Substance-/medication-induced major or mild neurocognitive dis order'. Neurocognitive impairments are defined as being persistent out with periods of acute withdrawal or delirium and consistent with deficits that would be caused by alcohol, based on the chronological history of abuse and onset of symptoms. ICD-11 also groups the 'Neurocognitive disorders' together and allows amnestic disorder and dementia to be 'due to psychoactive substances including medications'. The majority (50–60%) of heavy drinkers display some degree of cog nitive impairment on cognitive testing while sober. There is impairment in short-term memory, long-term memory recall, new skill acquisition, execu tive function, relative preservation of language ability, and mildly impaired

Alcohol misuse disorders 2 visuospatial function. IQ [measured by the Wechsler Adult Intelligence Scale (WAIS)] is generally preserved [in comparison with premorbid IQ, meas ured using the National Adult Reading Test (NART)]. CT/MRI examination of the brain of heavy drinkers reveals cortical and subcortical atrophy. White matter loss is prominent, which correlates with neuropathology findings. The degree of structural abnormality poorly correlated with the degree of functional impairment. In all patients with ARBD (including those with 'alcohol dementia' and Korsakoff syndrome), a significant amount of medical comorbidity is seen, including small vessel disease, repetitive head injuries, and comorbid alcohol liver disease (ALD). The neurotoxic effects of alcohol on the brain are exacerbated significantly by thiamine deficiency, and there is evidence to suggest earlier onset in women. Abstinence from alcohol use has been shown to correlate with functional and MRI improve ment at 1yr. The term alcohol dementia is used at times, describing a generalized dementia syndrome, in which there is intellectual decline and more pro nounced neuropsychological deficits. The changes correlate with total life time drinking and the length of drinking history. Wernicke–Korsakoff syndrome (E Wernicke–Korsakoff syndrome, p. 606.) Pathological jealousy (Othello syndrome) This is a monosymptomatic delusional disorder (E Delusional disorder 1: clinical features, p. 230) seen most commonly secondary to current or previous alcohol abuse. The form is a primary delusion in which the con tent is that the patient's spouse or partner has been, or is being, unfaithful. Delusional evidence may be provided to back up this belief, and the patient may go to great lengths to obtain 'evidence' (e.g. following her, planting tape recorders, examining discarded clothing). There is a significant association with violence and even homicide towards the supposedly unfaithful partner. Management • Abstinence from alcohol with the addition of antipsychotic medication. • It may be necessary for the couple to separate, and advice to this effect may have to be given to the at-risk partner.

604 Chapter 14 Substance misuse Psychiatric comorbidity Anxiety and depressive disorders Symptoms such as generalized anx iety, panic attacks, and low mood are very frequently reported in alcohol abusers. Many patients with alcohol problems also merit diagnoses of de pressive illness

(750%) or anxiety disorder (775%). The phenomenology of these disorders is similar to that found when the disorders occur in isola tion. The difficulty is deciding the sequence of events, as, in some cases, the alcohol problem is secondary to the patient ‘self-medicating’ with alcohol in order to relieve primary anxiety or depressive symptoms. Nonetheless, chronic alcohol use will act as a direct depressant; its secondary effects will produce depressogenic life events (e.g. loss of job) and alcohol-related ef fects such as waking at 4 a.m. due to withdrawal, or weight loss related to nausea may masquerade as, or mask, biological depressive features. Patients may emphasize the primacy of the mood or anxiety features and seek their resolution before tackling the alcohol problem. Generally, a pri mary mood or anxiety disorder diagnosis should not be made in the pres ence of continuing alcohol misuse, and psychological or pharmacological treatment for mood disorder is unlikely to be effective. The correct course is to initiate detox if indicated and to reassess mood/anxiety symptoms after 4wks of abstinence, treating residual symptoms at this point. Only a minority will require formal treatment. An undiagnosed depressive illness preceding the alcohol problem is more common in women. Alcohol prob lems can also arise as a result of self-medication of agoraphobia and social phobia. Suicide Classically quoted as a lifetime risk of 10–15% in dependent drinkers. Now estimated at 74% lifetime risk of suicide in those with alcohol problems. Psychiatric comorbidity is important, as are social isolation, phys ical ill health, and repeated failed attempts at abstinence. Schizophrenia High rates of alcohol and substance use found in schizo phrenic patients (750%). i risk of violence, EPSEs, TD, non-compliance, relapses, and rehospitalizations. Alcohol is an easily available temporary treatment for some of the distressing symptoms of psychotic illness. Drug misuse Comorbid alcohol and drug misuse can be used to enhance effects (e.g. euphoriant effect of alcohol and cocaine combined) or to min imize unpleasant side effects (e.g. alcohol to relax after taking stimulants), or as a substitute when the primary drug is unavailable. Comorbid drug misuse is associated with poorer outcome. Some comorbidities can have an iatro genic component where there is mixed abuse or substitution of BDZs for alcohol. This can result from inappropriate prescribing of anxiolytics, mis diagnosis of alcohol problems as anxiety disorders, and repeated unsuper vised withdrawals with hoarding of tablets. Aim to limit new prescriptions, review the diagnosis in patients with treatment-resistant anxiety disorders, and avoid short-acting BDZs (e.g. lorazepam).

 Wernicke–Korsakoff syndrome Wernicke encephalopathy and Korsakoff psychosis represent the acute and chronic phases of a single disease process—Wernicke–Korsakoff syndrome—which is caused by neuronal degeneration secondary to thia mine deficiency, most commonly seen in heavy drinkers. Wernicke encephalopathy Clinical features Acute onset of tetrad of: (1) acute confusional state; (2) ocular signs (ophthalmoplegia, nystagmus); and (3) ataxic gait. Associated features of: peripheral neuropathy, resting tachycardia, and evi dence of nutritional deficiency. Ophthalmoplegia is most commonly due to sixth nerve palsy (paralysis of lateral gaze). Triad only seen in 10% of cases; confusion in 780% of cases. Aetiology Occurs secondary to thiamine (vitamin B1) deficiency. Heavy drinkers are especially vulnerable due to poor intake (alcohol is calorie- rich, but vitamin-poor), reduced absorption, and impaired hepatic storage. Other rare causes of thiamine deficiency are starvation, post-gastric resec tion, anorexia nervosa, and hyperemesis gravidarum. Pathology Haemorrhages and secondary gliosis in periventricular and periaqueductal grey matter involving the mamillary bodies, hypothalamus, mediodorsal thalamic nucleus, colliculi, and tegmentum of the midbrain.

Treatment • Give high-potency parenteral B1 replacement—IV Pabrinex®, two ampoules by infusion over 30min bd for 3–7 days. Specialist use. (Note: associated with allergic reactions; facilities for treatment of anaphylaxis must be available, although recent evidence suggests negligible risk with recent preparations.) Avoid carbohydrate load until thiamine replacement is complete (i.e. do not rehydrate with glucose solutions prior to thiamine). • Treat immediately when the diagnosis is made or strongly suspected. In addition, consider treating all those at high risk (alcohol-dependent patients with poor nutrition) prophylactically with parenteral vitamins. • All patients with symptoms of Wernicke encephalopathy and those at high risk should have parenteral vitamins as just described above. All other patients undergoing detox or being assessed for alcohol problems should receive oral replacement—thiamine 100mg tds for 1mth. • Assess and treat for alcohol withdrawal syndrome (E Alcohol withdrawal syndromes, p. 590). Prognosis • Untreated, the acute phase lasts 72 weeks, with 84% of cases developing features of Korsakoff psychosis. Mortality of 715% in untreated cases. • With treatment, ophthalmoplegia and confusion resolve within days, but ataxia, neuropathy, and nystagmus may be prolonged or permanent. Korsakoff syndrome Clinical features Absence or significant impairment in the ability to lay down new memories, together with a variable length of retrograde amnesia. Working memory (e.g. ability to remember a sequence of numbers) is

Wernicke–Korsakoff syndrome unimpaired, as is procedural and ‘emotional’ memory. Thus, the affected individual may be able to go with a psychologist to an interview room, perform adequately on working memory testing, show evidence of a new skill (e.g. mirror writing) they practised the day before, and yet later have no memory of ever having been in that room or having seen that psych ologist before (although, on returning to the room, they may be more re laxed on subsequent occasions, due to state-related emotional memories). Confabulation for the episodes of amnesia may be prominent. Other neuropsychological deficits associated with ARBD may be seen. Aetiology Most commonly due to thiamine deficiency, secondary to heavy alcohol use. Rarer causes are head injury, post-anaesthesia, basal/temporal lobe encephalitis, CO poisoning, and thiamine deficiency secondary to other causes. It should be remembered that Korsakoff syndrome is not invariably preceded by Wernicke encephalopathy and can present in a ‘chronic’ form. Pathology Pathological features are those of Wernicke encephalopathy. The presumed mechanism is disconnection of a mamillothalamic pathway crucial for memory formation. Treatment • Continue oral thiamine replacement and multivitamin supplementation for up to 2yrs. • Treat psychiatric comorbidity (e.g. depression). • OT assessment, cognitive rehabilitation within an appropriate setting, acknowledging that some patients improve and may progress to independent living. Therefore, these patients will require continuous assessment of their functioning, bearing in mind that improvement occurs in 750% of those presenting with Korsakoff syndrome. Prognosis • Twenty per cent of cases show complete recovery, and 25% significant recovery over time, with the remainder largely unchanged. • The degree of functional impairment is directly related to the degree of memory impairment which may be incompatible with independent living.

608 Chapter 14 Substance misuse Medical complications of alcohol misuse Hepatic • ALD is the most common cause of liver damage in the developed world. Presents as fatty change, alcoholic hepatis, and finally as cirrhosis. • Fatty change seen in >90% of heavy drinkers, can emerge after a single heavy bout, may be asymptomatic, or may present as lethargy, malaise, painful and swollen liver, and obstructive jaundice. Reverses with abstinence. • Alcoholic hepatitis—40% of heavy drinkers. • Cirrhosis—up to 30% of heavy drinkers after 10–30yrs. Predisposed to by genetic

variation (reduced alcohol oxidation and i acetaldehyde accumulation), ♀ sex (less first-pass metabolism and lower body water content for alcohol dispersal), and comorbid hepatitis B or C infection. Gastrointestinal • Gastritis/gastric erosions, with consequent haematemesis. • Metaplasia of the lower third of the oesophagus (Barrett's oesophagus). • Mallory–Weiss oesophageal tears secondary to vomiting. • Peptic ulceration. • Chronic diarrhoea. • Chronic pancreatitis (alcohol is the most common cause), with chronic fluctuating abdominal pain and steatorrhoea. Cancers • Hepatocellular, oesophagus, stomach, mouth, tongue, and pharynx. Cardiovascular • Hypertension. • Dilated cardiomyopathy. • Cardiac arrhythmias (especially AF). • CVA. • Non- or very light drinkers have a higher risk than light drinkers, even after controlling for smoking, hypertension, etc. (i.e. 'the J-shaped curve' for mortality); no specific drink type (i.e. not red wine); mechanism may be an increase in protective high-density lipoprotein cholesterol (HDL- C) and reduced platelet adhesion. Respiratory • TB. • Klebsiella and streptococcal pneumonia. • i vulnerability is related to d immunity, poor nutrition, and self-neglect. Neurological • Wernicke–Korsakoff syndrome (E Wernicke–Korsakoff syndrome, p. 606). • Peripheral neuropathy. • Central pontine myelinolysis (pseudobulbar palsy + quadriplegia). • Marchiafava–Bignami disease (corpus callosum degeneration).

Medical complications of alcohol misuse • Cerebellar degeneration. • Optic atrophy. • Alcoholic myopathy. Genitourinary • Erectile problems. • Hypogonadism in men. Other • Fetal alcohol syndrome (FAS) (E Non-genetic causes of learning disability, p. 818). • Gout. • Osteoporosis. • Impaired absorption and diminished intake of specific vitamins and food overall. • Contribution to accidents, particularly RTA. • Exacerbating factor in violent crime and assaults. • Diminished compliance with treatment for other medical and psychiatric disorders.

610 Chapter 14 Substance misuse Tobacco 1—background Tobacco has been used recreationally worldwide for centuries in various forms. It can be smoked in the form of cigarettes, via a pipe or hookah, or as shisha; it can also be chewed or 'snuffed'. Tobacco use became more widespread in the 1800s, when the implementation of automatic cigarette rolling via machine allowed mass production and a shift in market availability. Smoking cigarettes is the most common method of use worldwide. The WHO formed the WHO Framework Convention on Tobacco Control (WHO FCTC) in 2005. They have issued a series of reports on the 'global tobacco epidemic'—the most recent being in 20159—giving an update on world tobacco use and measures to tackle it. Current levels and previous levels of use have been hard to quantify due to variable levels of monitoring globally. WHO prevalence estimates in 2013 quoted 21%—950 million men and 177 million women—of adults globally are to bacco smokers. Compared to the 2007 estimate of 23%, this constitutes a reduction. Over the decades, tobacco use has started to reduce due to a number of factors, which form the basis for global policy for tackling tobacco use. Many of these are outlined as below: • Raising taxes on tobacco products. • Implementing smoke-free environments. • Cessation programmes. • Warning labels on cigarette packets. • Education and awareness of the risks of tobacco use. • Reducing tobacco product advertising. Smoking-related disease The morbidity and mortality associated with smoking-related diseases are summarized in Table 14.4. Additional concerns centre around: • Perinatal and postnatal disease Maternal smoking in pregnancy increases the risk of miscarriage, premature delivery, and a small-for-dates baby. Postnatally, there is an i risk of sudden infant death syndrome (SIDS), asthma, and other respiratory-related disease in the infant. • Second-hand smoke Inhalation by non-smokers in the vicinity of smokers also causes an i risk of the aforementioned conditions. • Environmental risks Accidents related to

smoking increases morbidity and mortality and burns caused directly or indirectly. Pharmacology The main neurochemical in tobacco that drives its ongoing use and the addiction to it is nicotine. When smoked, this is rapidly absorbed by the alveoli due to their large surface area. If chewed or snuffed, nicotine is ab sorbed across the mucous membranes. Most nicotine metabolism occurs in the liver, but it also occurs in the brain and lungs. Nicotine is extensively metabolized to a number of metabolites in liver. Quantitatively, the most 9 World Health Organization (2015) WHO Report on the Global Tobacco Epidemic, 2015. M http://apps.who.int/iris/bitstream/10665/178574/1/9789240694606_eng.pdf [accessed 12 July 2018].

Tobacco 1—background important metabolite of nicotine in most mammalian species is cotinine. In humans, 70–80% of nicotine is converted to cotinine. This involves two steps—the first mediated by the cytochrome P450 system (mainly CYP2A6 and CYP2B6) to produce nicotine iminium ion; the second step is cata lysed by aldehyde oxidase (AOX). Other metabolic pathways include oxi dation to nicotine N′-oxide (NNO) and glucuronidation to an N-quaternary glucuronide. High levels of nicotine hit the brain within 10–20s, following inhalation. Nicotine is subject to renal clearance, and there is also much heterogeneity in terms of slow and fast metabolizers of nicotine, and other factors, such as age, medical comorbidities, genetics, and medications, can affect metabolism. Nicotine acts on nicotinic ACh receptors in the CNS and peripheral ner vous system, causing flux of cations and depolarization of the plasma mem brane and cell excitability, in turn regulating neurotransmitter release. This then mediates the effects of nicotine such as arousal, anxiety, alertness, and relaxation. Table 14.4 Smoking-related diseases Pathophysiology Disease/conditions Respiratory disease Impaired ciliary function and mucus clearance, structural damage to alveoli, direct carcinogen exposure via smoke inhalation, free radical exposure, other immune responses Asthma, bronchitis, COPD, lung cancer, recurrent respiratory infection Cardiovascular disease Endothelial inflammation and formation of atheroma, lipid profile alteration (iHDL-C, iLDL cholesterol, itriglycerides, iserum cholesterol) Coronary artery disease, stroke, peripheral vascular disease Gastro-oesophageal disease ihistaminic receptor activation, igastro-oesophageal reflux, other immune responses Carcinogen exposure, free radical exposure Gastric and duodenal ulceration, alimentary canal cancers

612 Chapter 14 Substance misuse Tobacco 2—dependence and interventions Nicotine dependence Nicotine is highly addictive and caused i tolerance with repeated use. Users show compulsion to use and suffer withdrawals on cessation of use. Nicotine itself has a relatively short half-life of 1–2hrs. Withdrawal symp toms occur on cessation of consumption, usually within 24hrs, and include dysphoria, disturbed sleep, irritability, agitation, and i appetite. Users may also suffer from cravings. Relief of withdrawals occurs relatively quickly on recommending smoking. ICD-10 criteria for nicotine dependence falls under F17 coding: • Tolerance to nicotine. • Withdrawal symptoms. • Impaired control. • Ongoing use in spite of risks. • Social adverse effects. While smoking causes less social impairment, compared to other drugs of abuse, e.g. heroin, BDZs, alcohol, it still increases the risk of accidents, such as fires, and causes financial strain due to cost and strain on relationships. Ongoing use in spite of physical disease is common in those with a more long-standing and/or severe dependence to nicotine. Management of smoking Smoking cessation has become a big public health drive in recent years.10,11 There are opportunities for brief interventions in primary care and via phar macies, practice nurses, allied health professionals, and dentists, as well as in secondary care in hospitals. Hospitals often have dedicated smoking cessa tion services. Pharmacological interventions, including nicotine replacement and behavioural

interventions, also play an important role. Some smokers abruptly stop and incur withdrawals without any nicotine replacement. There are higher rates of relapse in any individuals who stop smoking as such. Others use nicotine replacement aids or other medication interven tions in order to help wean off nicotine and help to maintain longer-term abstinence. Nicotine replacement therapies Nicotine replacement therapies (NRTs) are available in the form of nicotine gum, nicotine transdermal patches, nasal spray, lozenges, sublingual tablets, and inhalers. Nicotine vaporizers are relatively new on the market in recent years; the act of using them is commonly known as ‘vaping’. Electronic ‘e- cigarettes’ and electronic nicotine delivery systems (ENDS) are alternatives to personal vaporizers (PVs).These battery-powered vaporizers simulate 10 Saunders JB, Conigrave KM, Latt NC, et al. (2016) Addiction Medicine. Oxford: Oxford University Press. 11 National Institute for Health and Care Excellence (2008) Stop smoking services. Public health guideline [PH10]. M https://www.nice.org.uk/guidance/ph10 [accessed 12 July 2018].

Tobacco 2—dependence and interventions tobacco smoking using a heating element (atomizer) to produce an aerosol of a liquid solution (e-liquid) that usually contains propylene glycol, vege table glycerin, nicotine, and flavourings. Little is yet known about the longer- term effects of ‘vaping’ and ‘e-cigarettes’ (see also E Cannabis, p. 626). Nicotine patches • Release nicotine slowly via transdermal patch at a steady rate. • Dose: 21mg patch l 10ng/mL, plasma level of nicotine. • Can be done via pharmacy supervision in the UK or bought over the counter. Other strengths are available (7/14mg per 24hr; 10/15/25mg per 16hr). Nicotine gum • NRT that can be bought over the counter. • Doses: 2mg gum l 7ng/mL, plasma level of nicotine absorbed via oral mucosa; 4mg gum l 15ng/mL, plasma level of nicotine absorbed via oral mucosa. Other pharmacological interventions Bupropion hydrochloride (Zyban®) Bupropion is a DARI and NARI (also an antidepressant) that is administered orally in tablet form. There is evidence that it aids smoking cessation, in combination with motivational support. Doses: initially 150mg for 6 days, then i to 150mg bd for 7–9wks. It is recommended treatment is com menced 1–2wks before a set stop date. Varenicline tartrate (Champix®) Varenicline tartrate is a selective nicotinic receptor partial agonist recom mended by NICE to aid with smoking cessation. Doses: initially 500mcg od for 3 days, then i to 500mcg bd for 4 days, then 1mg bd. Treatment is recommended for 11wks. It is recommended that treatment is commenced 1–2wks before a set stop date. Behavioural interventions and support Behavioural interventions via individual or group behavioural counselling is recommended by NICE to aid with stopping smoking. Counselling would include psychoeducation, support, and advice on small habitual changes that may be associated with smoking behaviours, e.g. smoking outside only, ra ther than in the house; avoiding cues for smoking; and reducing associated behaviours such as drinking alcohol or caffeine.

614 Chapter 14 Substance misuse Illegal drugs In the UK, community surveys indicate that one-third of adults have tried illegal drugs in their lifetime, with 10% having used them in the previous year. The rates for those aged under 25 are higher, with 50% lifetime use and 33% in the previous year. At all ages, ♂ have higher rates of drug use than ♀ (♂:♀ = 3–4:1). Cannabis is the most commonly used illegal drug, while community rates for the other drugs of abuse are low. Users show a variable pattern of consumption with episodic and situational use for drugs with low dependence potential and a tendency to continuous dependent use for more ‘addictive’ drugs. Among some users, particularly those in the dance scene, polydrug use is the norm with individuals consuming >10 dif ferent drugs. Use of illegal drugs is more common in the young, in the lower socio-economic classes, and in those with psychiatric illness. At any one time, <33% of dependent

users will be in contact with treatment services; the mean length of dependent use before seeking help is 9yrs. There are as many patterns of drug use as drug users, and individual pa tient assessment is mandatory; nonetheless, a number of patterns of use of illegal drugs can be recognized: • Experimental use Use of drug in order to explore effects. Common among the young and heavily driven by drug availability and drug use among peers. Very common for 'softer' drugs (e.g. cannabis, volatile chemicals); rarer for more 'hard' drugs (e.g. heroin). • Situational use Drug use limited to certain situations (e.g. parties, raves). Mainly drugs with stimulant/hallucinogenic properties. • Recreational use Regular, but non-dependent use. May be limited in time by the period of life (e.g. ending at the end of university life) or may progress to dependent use. • Polydrug use Non-dependent use of a variety of drugs. One drug may be taken to potentiate the effects of another or to manage unpleasant after-effects of drug use. Risks can be additive or multiplicative. • Dependent use Use of a drug for which a dependence syndrome (E The dependence syndrome, p. 574) has developed. Continued use may be motivated more by the desire to avoid withdrawals than by positive drug effects, which may have diminished due to the development of tolerance. Tendency is for use of the dependent drug to predominate, with other drugs being taken only if the primary drug is unavailable. • Dual diagnosis Drug users who also suffer from a major mental illness. An important group for therapeutic intervention. Categories of drugs of abuse • Opiates: e.g. heroin, dihydrocodeine, methadone, codeine, buprenorphine, pethidine. • Depressants: e.g. BDZs, barbiturates, alcohol, gamma-hydroxybutyrate (GHB). • Stimulants: e.g. amphetamines, cocaine, MDMA. • Hallucinogens: e.g. LSD, PCP, mushrooms, ketamine. • Others: e.g. cannabis, volatile substances, anabolic steroids.

Illegal drugs 615

616 Chapter 14 Substance misuse Slang terms related to drugs (See Tables 14.5 and 14.6.)
Table 14.5 Drug slang terms relating to use Slang term Meaning Backtrack Allow blood to flow back into IV syringe and then re-inject Chasing Consume heroin by heating on foil and inhaling the fumes Cold turkey Withdrawal symptoms (referring to piloerection) Cooking up Melting down heroin prior to injection Fix The required regular dose of drug in a dependent user Gouching Apparent somnolence following heroin use Jag up To inject drugs IV Juggling Selling drugs to finance one's own dependency Junkie An individual dependent on a drug Mainline To inject drugs IV Nodding, on the nod Apparent somnolence following heroin use Rattling Suffering from withdrawals Score Obtain drugs Script Legitimate prescription for drugs Shooting gallery Place where individuals meet to use drugs IV Skin popping To inject drugs subdermally Sorted Having obtained sufficient drug for one's own needs Spliff Cannabis cigarette Tab Dose of LSD impregnated onto paper Works Syringe and needles

Slang terms related to drugs Table 14.6 Drug 'street names' Conventional name Street slang Amphetamine Billy/Whizz, Speed, Sulph Amphetamine-like 'bath salts' Bliss, Bloom, Blue Silk, Cloud 9, Drone, Energy-1, Lunar Wave, M-CAT, Meow Meow, Mephadrone, Pure Ivory, Scarface, Stardust, Vanilla Sky, White Lightning, Wicked X Anabolic steroids Roids Cannabis Bud, Chronic, Dope, Ganja, Grass, Green, Hash, Hashish, Hemp, Herb, Kush, Marijuana, Mary Jane, Pot, Purple Haze, Reefer, Sinsemilla, Skunk (potent), Trees, Weed Cocaine Bernice, Blow, C, Charlie, Coke, Crack (freebase), Dust, Flake, Line, Nose Candy, Rock, Sneeze, Sniff, Snow, Toot, White, Yayo Depressant drugs Downers Diazepam Vallies GHB GBH, grievous bodily harm Heroin Big H, Black Tar, Boy, Brown Sugar, China White, Dope, Dragon, Gear, H, Horse, Junk, Mexican Brown, Mud, Scag, Skag, Skunk,

Smack, Thunder Ketamine Cat Valium, Green K, Honey Oil, Jet, Ket, Kit Kat, Purple, Special K, Special LA Coke, Super Acid, Super C, Vitamin K LSD Acid, Battery Acid, Blotter, California Sunshine, Cid, Doses, Dots, L, Looney Toons, Lucy, Lucy in the Sky with Diamonds, Superman, Tabs, Window Pane, Yellow Sunshine Methamphetamine Chalk, Crank, Crissy, Cristy, Crystal, Crystal Meth, Glass, Go, Ice, Meth, Shards, Tina, Tweak, Whizz MDMA Adam, Beans, Candy, Clarity, Dancing Shoes, E, Ecstasy, Eccies, Happy Pill, Hug, Hug Drug, Love Drug, Lover's Speed, Molly, Moon Rocks, Rolls, Scooby Snacks, X, XTC PCP Angel dust, Embalming fluid, Hog, Love boat, Ozone, Rocket fuel, Superweed, Wack, Wet (a marijuana joint dipped in PCP) Psilocybin mushrooms Blue Meanies, Boomers, Buttons, Caps, Cubes, Liberties, Liberty Caps, Magic Mushrooms, Magics, Mushies, Shrooms Rohypnol® Roofies (flunitrazepam) Stimulant drugs Uppers Synthetic cannabinoids Black Mamba, Bliss, Bombay Blue, Genie, Joker, K2, K2 Drug, K3 Drug, Kroni, Kush, Skunk, Genie, Solar Flare, Spice, Yucatan Fire, Zohai Temazepam Jellies Volatile nitrates Poppers Volatile solvents Air Blast, Bold, Discorama, Glad, Hippie Crack, Huff, Laughing Gas, Moon Gas, Nitrous, Oz, Poor Man's Pot, Poppers, Rush, Snappers, Whippets, Whiteout

618 Chapter 14 Substance misuse Opiates/opioids Opiates are a group of chemicals derived from the opium poppy (Papaver somniferum); synthetic compounds with similar properties are called opi oids. They have potent analgesic properties and, as such, have wide legit imate uses in medicine. They are widely abused for their euphoriant and anxiolytic properties. Heroin is the most frequently abused opiate. Heroin Illicit heroin is sold as a brown or white powder in 'bags' or 'wraps', costing £50–100/g, with a typical dependent user taking 0.25– 2.0g/day. It is most commonly consumed by smoking ('chasing') but is also taken orally, occasionally snorted, and parenterally by IV, IM, or sub cutaneous (SC) routes. Street supplies are of variable purity (25–50% by volume); occasionally, a particularly pure batch is associated with a series of deaths and ODs from users used to a less concentrated form. In common with other opiates, heroin binds to specific receptors, for which there are endogenous ligands (endorphins). There are overall cor tical inhibitory effects, with diminished pain sensation. After consumption, effects are virtually immediate, with euphoria amounting to ecstasy, intense relaxation, and untethering from worries and cares. Although recreational use is not unknown, the tendency is for progres sion to dependent use and this is the most usual pattern by the time of pres entation to treatment services. An established dependent user may move from smoking to occasional or regular IV use to potentiate effects. Users develop tolerance with regular use, and there is cross-tolerance to other opiates. Dependent patients may describe limited euphoriant effects, with the drug being mainly taken to avoid unpleasant withdrawals. Acute medical problems associated with heroin use by any route include nausea and vomiting, constipation, respiratory depression, and loss of con sciousness with aspiration (the cause of many fatalities). Injected use adds risks of local abscesses, cellulitis, osteomyelitis, bacterial endocarditis, septi caemia, and transmission of viral infections (hepatitis B and C, HIV). Opiate dependency develops after weeks of regular use and is associated with an unpleasant (but not generally medically dangerous) withdrawal syndrome (E Substitute prescribing 1: principles, p. 634). Interventions Give harm reduction advice to users who continue to use opiates—do not use opiates while alone; do not use in combination with other drugs; avoid the IV route—and if injecting, give safe injecting advice (see Box 14.6). Consider managed detox (E Substitute prescribing 2: opi ates, p. 636) or transfer to maintenance prescribing (E Substitute pre scribing 2: opiates, p. 636) in established dependence. Other opiates/opioids These include dihydrocodeine, morphine, metha done, pethidine, buprenorphine, and codeine. They may be taken in their pre-prepared tablet or liquid form or prepared for injection. Their acute and chronic

risks are similar to heroin.

Opiates/opioids Box 14.6 Safer injecting advice If using heroin, it is safest to avoid IV use which has the greatest risk of OD and other complications. If using heroin IV: • Use new sterile needles and syringes on each occasion (give details of local needle exchange services, if available). • Use sterile water (water from a running cold kitchen tap is the closest). • Never share needles, syringes, spoons, or filters with another user. • Rotate injection sites. • Avoid injecting into the neck, groin, or breast. • Avoid injection into infected areas. • Ensure that the drug is completely dissolved before injecting. • Always inject with, not against, the blood flow. • Do not take heroin while alone. It is safest to use new sterile needles and syringe on each occasion. Failing this, rather than use dirty equipment, flush both needles and syringes sev eral times with thin bleach, then several times with clean water.

620 Chapter 14 Substance misuse Depressants Drugs of this group produce their effects by generalized or specific cor tical depression. They include BDZs, alcohol, barbiturates, and other drugs that act through GABA receptors. They can be taken for their pleasurable anxiolytic and relaxant properties alone, or as a way of counteracting un pleasant side effects of other drugs of abuse (e.g. to ‘come down’ after stimulant use). BDZs A class of chemicals initially synthesized in the 1950s. Largely re placed barbiturates in clinical practice, as they did not cause fatal respira tory depression. They have therapeutic uses as anxiolytics, hypnotics, anticonvulsants, and muscle relaxants. Problems of dependency arising from long-term use became recognized in the 1980s, leading to a fall in their legitimate prescription, but did nothing to diminish their popularity as drugs of abuse. All BDZs have similar effects and are distinguished by their length of action: short-acting (e.g. temazepam, oxazepam), medium-acting (e.g. lorazepam, alprazolam), and long-acting (e.g. diazepam, nitrazepam, chlordiazepoxide). BDZs are taken orally or, less commonly, by injection. There is hepatic metabolism to active compounds, some with long half-lives. They enhance GABA transmission and produce marked anxiolytic and euphoriant effects. Tolerance develops rapidly (with cross-tolerance to all drugs in the BDZ group), so requiring increasing doses to achieve similar effects. Acutely, they cause forgetfulness, drowsiness, and impaired concentra tion and coordination, with consequent risk of accidents. Use by injection is associated with the same infective risks as IV heroin (see Box 14.6). An add itional problem seen in IV BDZ users is limb ischaemia secondary to IV use of melted tablet contents. Chronic use is associated with impaired concen tration and memory and depressed mood, all of which are more severe in the elderly. BDZ dependency develops after 3–6wks of regular use. There is a withdrawal syndrome (E Substitute prescribing 3: benzodiazepines, p. 638), which can be complicated by seizures and delirium. Interventions Harm reduction advice to user as for opiates (E Opiates/ opioids, p. 618), specifying safe injecting advice (see Box 14.6) if using via the IV route. Consider managed detox or transfer to maintenance pre scribing (E Substitute prescribing 3: benzodiazepines, p. 638) in established dependence. Flunitrazepam (Rohypnol®) A short-acting, potent BDZ seen particularly in dance settings with intoxicant and (probably apocryphal) aphrodisiac ef fects. As it can produce impaired judgement and anterograde amnesia and is tasteless in solution, it has been implicated in cases of ‘date rape’. GHB A synthetic compound originally developed as an anaesthetic which is a probable intrinsic neurotransmitter. Particularly seen in dance settings, usually in combination with other drugs or alcohol. Produces a sense of dissociation, euphoria, and intoxication. Taken as liquid, in 5–10mg dosage, with effects coming on in 15–30min and lasting several hours. Side effects of nausea and vomiting, seizures, and respiratory depression. Usually taken episodically, but a cohort

of patients is increasingly seen with consump tion of the drug multiple times daily, with consequent physical depend ence. Withdrawal from established dependence can present as a medical

Depressants emergency and is associated with delirium, severe behavioural disturb ance, psychotic features, autonomic instability, and occasionally acute renal failure. Such patients will usually require joint psychiatric and medical man agement. Drug treatment of these withdrawals is via reducing BDZ regime, as per alcohol withdrawal (higher doses usually required), with the addition of regular baclofen given as a reducing regime, starting at 20mg five times daily, reducing over the subsequent week. Barbiturates Group of compounds used as hypnotics/anxiolytics in clin ical practice prior to the introduction of BDZs. Now rarely prescribed and rarely seen as drugs of abuse. They act by facilitating GABA neurotrans mission. There is rapidly increasing tolerance to their anxiolytic effects in regular use, but not to the associated respiratory depression. Gabapentin and pregabalin Gabapentin and pregabalin, collectively known as ‘gabapentinoids’, are drugs that are prescribed to treat a variety of conditions such as neuro pathic pain, epilepsy, and anxiety disorders. They work by binding to calcium channels and reducing excitatory neurotransmitter release, indirectly al lowing a more ‘GABA-ergic’ effect because of i availability of endogenous GABA. There is growing abuse of these medications reported in the litera ture and anecdotally across a range of countries, and they are subject to pharmacovigilance by the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA).

622 Chapter 14 Substance misuse Stimulants These drugs potentiate neurotransmission and increase cortical excitability, producing effects of i alertness and endurance, diminished need for sleep, and a subjective sense of well-being. They include cocaine (and crack co caine), amphetamines, 3,4,-methylenedioxymethamphetamine (MDMA or ecstasy), and caffeine. Cocaine The mild stimulant/euphoriant effects of the chewed leaves of the coca shrub have been known to the people of South America for thousands of years, but in its refined form, cocaine is a potent and highly addictive drug. Cocaine hydrochloride is refined to a white powder, which may be inhaled (‘snorted’) or dissolved and injected. The main route of in take is by inhalation, as it undergoes rapid ‘first-pass’ liver metabolism. The user forms the powder into ‘lines’ and inhales via a rolled paper tube (clas sically, a high denomination banknote). Each line contains 725mg of cocaine. Freebase (‘crack’) cocaine (produced by alkalinization, which produces the hydrochloride-free ion form) has a lower vaporization temperature than cocaine hydrochloride and can be smoked. In terms of rapidity of action and peak blood levels, this compares with IV use. Cocaine acts as a local anaesthetic at mucous membranes. It has widespread effects in potentiating dopaminergic, serotonergic, and noradrenalinergic neurotransmission by blocking neurotransmitter re uptake. Its actions begin a few minutes after consumption. There is i en ergy, i confidence, euphoria, and diminished need for sleep, but with rapid fall-off in effects due to rapid metabolism, leading to repeated use. There are very intense effects from freebase cocaine use with rapid and intense ‘high’ with subsequent dysphoria. Cocaine is usually taken in an opportun istic way, sometimes in association with other stimulant drugs. Acute harmful effects include arrhythmias, intense anxiety, hypertension l CVA, acute impulsivity, and impaired judgement. Chronic harmful effects include necrosis of the nasal septum, fetal damage (‘crack babies’), panic and anxiety disorders, persecutory delusions, and psychosis. It is not asso ciated with classical dependence, but a minority of users will consume in a regular ‘compulsive’ pattern. Interventions Harm reduction advice, including safe injecting advice, if ap propriate (see Box 14.6). No role for substitute prescribing in managing withdrawal or for maintenance prescribing. Amphetamines A group of

compounds synthesized in the late nineteenth century, with current legitimate uses in child psychiatry (E Attention- deficit/hyperactivity disorder 2: medication, p. 670) and in narcolepsy (E Hypersomnia 2: narcolepsy, p. 450). Sold as 5mg tablets or as a white powder (£10 per gram). The powder may be swallowed, inhaled, or dis solved and injected. Use is usually situational or recreational, although very regular use with dependence is recognized. There is chemical similarity to NA and DA, producing similar pharmacological effects to cocaine, but its slower metabolism gives a longer duration of action. Acute harmful effects include tachycardia, arrhythmias, hyperpyrexia. irritability, post-use depression, and a quasi-psychotic state with visual, auditory, and tactile hallucinations. Dependency is not seen, but marked psychological addiction occurs, particularly in situations associated with

Stimulants drug use. Anxiety and depressive symptoms are frequently seen in users; their proper assessment requires a period of abstinence. Interventions Harm reduction advice (including safe injecting advice, if ap propriate). No role for substitute prescribing in managing withdrawals. Very limited role for maintenance prescribing of dexamfetamine sulfate in the management of chronic, primary, heavy IV users (specialist instigation only). MDMA (ecstasy) This compound was synthesized in 1914. Initially, it was occasionally used as an adjunct to psychotherapy. Initially legal, it became widely used in the mid-1980s in association with house, rave, and techno music. It is taken orally as 50–200mg tablets. A typical pattern of use is two or more tablets taken at weekends. MDMA causes serotonin release and blocks reuptake. It has structural similarities to mescaline and amphetamine; therefore, it has both hallucino genic and stimulant properties, with these effects appearing 730mins after ingestion. The initial ‘rush’ period of intoxication lasts 73hrs and is charac terized by a feeling of i camaraderie and ‘closeness’ to others, a pleasurable agitation relieved by dancing, and d fatigue. Acute harmful effects include i sweating, nausea and vomiting, and di minished potency despite i libido. Deaths have occurred, associated with dehydration and hyperthermia [a toxic reaction similar to serotonin syn drome (SS) appears to exist; E Serotonin syndrome, p. 1022]. Chronic harmful effects include possible neurotoxicity, hepatotoxicity, and possible chronic cognitive impairment. There is tolerance to its effects, but depend ence does not occur. ‘Hangover’ effects develop 24–48hrs after ingestion, including fatigue, anorexia, and depressed mood (which may be severe). Interventions Harm reduction advice regarding maintaining hydration and avoiding overheating during use. No role for substitute prescribing in managing withdrawal or for maintenance prescribing. For all stimulant drugs, there may be a problem of assessing other aspects of mental state, particularly affective and psychotic features, while chaotic use continues. In selected patients, inpatient assessment will be indicated to allow this.

624 Chapter 14 Substance misuse Hallucinogens Hallucinogens (or psychedelics) are a heterogenous group of natural and synthetic substances which produce altered sensory and perceptual ex periences. They include: lysergic acid diethylamide (LSD), phenylcyclidine (PCP), magic mushrooms, ketamine, mescaline, 2,5-dimethoxy-4- methylamphetamine (DOM), and dimethyltriptamine (DMT). LSD A compound synthesized by Hofman while working at Sandoz Pharmaceuticals in 1944. He reported the hallucinatory experiences that followed his initial accidental ingestion. The drug also occurs naturally in seeds of the Morning Glory plant. It became strongly associated with 1960s culture when its use was at its peak. There was early experimentation with its role in psychotherapy, but there is no current legitimate use. It is very sol uble and intensely potent (effective dose 7250mcg). It is sold impregnated onto paper, in tablets,

or as a powder. LSD is an indole alkylamine with structural similarity to serotonin. There are direct and indirect effects on serotonergic and dopaminergic transmitter systems. It is now not thought to provide a good model for endogenous psychosis. Its actions are very markedly situation- and expectation- dependent. Effects develop 15–30mins after ingestion and last up to 6hrs. There is initial euphoria, a sense of detachment, a sense of novelty in the familiar and a sense of wonder at the normal, visual distortions and misper ceptions, synaesthesia, and distorted body image. Somatic effects include dizziness and tremors. Acute harmful effects are behavioural toxicity (i.e. harm related to acting on beliefs such as having the ability to fly) and 'bad trips' (i.e. dis sociation, fear of incipient madness, frightening perceptions). There is no risk of OD, and physiological dependence and withdrawals do not occur. Chronic harmful effects include flashbacks (E Dictionary of psychiatric symptoms, p. 110), even many years after consumption, post-hallucinogenic perceptual disorder, persistent psychosis, and persistent anxiety/depressive symptoms. Interventions Harm reduction advice directed towards maintaining a safe environment during use and avoiding behavioural toxicity—do not use alone, and use accompanied by a non-user if possible. For all hallucino gens, acute psychotic features should, in general, be managed by admission, maintenance of a safe environment, and symptomatic treatment of agita tion (e.g. with BDZ), with expectation of resolution. Continuing psychotic features should be managed as for acute psychosis (E Initial treatment of acute psychosis, p. 200). PCP A hallucinogen rarely seen in the UK, except as a contaminant of other drugs. May be smoked, snorted, taken orally, or, more rarely, paren terally. There is direct binding to opioid and aspartate excitatory receptors, as well as has serotonergic and cholinergic effects, producing acute effects of confusion, visual sensory distortions, aggression, and sudden violence (which may be severe). Intoxication may give way to longer psychotic states. Magic mushrooms About a dozen varieties of hallucinogenic mush rooms grow in the UK, the best known being the Liberty cap (Psilocybe semilanceata). They may be eaten raw or cooked, dried, or prepared as a drink. Possession and consumption of mushrooms are not an offence,

Hallucinogens unless they have been processed or prepared for illicit use. Small doses cause euphoria, while larger doses (>25 mushrooms) cause perceptual ab normalities similar to LSD. They are not associated with dependence or withdrawal features, and tolerance develops quickly, making continuous use unlikely. Harmful effects include nausea and vomiting, dizziness, diar rhoea and abdominal cramps, behavioural toxicity, and risk of accidental consumption of toxic fungi. Ketamine A compound structurally similar to PCP, used as a veterinary anaesthetic and in battlefield surgery. It is a unique anaesthetic, as it does not produce RAS depression; instead it prevents cortical awareness of painful stimuli. It is taken illicitly as a sniffed powder, with a mean dose of 7100mg. Small amounts lead to a sense of dissociation, larger amounts to LSD-like synaesthesia and hallucinations, associated with nausea, ataxia, and slurred speech. Rare late effects are flashbacks, psychosis, and amnesic syndromes.

626 Chapter 14 Substance misuse Cannabis Cannabis is the most commonly used illegal drug, with only a small minority of its users ever using another illegal drug. Used for centuries as a pleas urable mind-altering substance and as a medication for a wide variety of ailments. Clinical trials are under way to clarify its role in the treatment of chronic pain. Its illegal use is of interest to psychiatrists because of its asso ciation with other drugs of abuse (as a 'gateway drug') and because of its exacerbating effect on chronic psychotic illnesses. Cannabis is produced from the dried leaves, flowers, stems, and seeds of the weed Cannabis sativa. It may be distributed as

herbal material (‘grass’ or marijuana), as a resin (‘hash’), or as cannabis oil. Cannabis may be smoked in cigarettes, alone, or mixed with tobacco; the resin form may be eaten directly or incorporated into foodstuffs (e.g. cakes), with a pos sibility of vaping in the future (see Box 14.7). These various forms contain at least 60 psychoactive cannaboids, the most important of which is 9-δ- tetrahydrocannabinol (THC). The dried herb contains 75% THC by weight, resin 710%, and cannabis oil 715%. Usage pattern is very variable, from infrequent situational use to daily heavy use—the latter at highest risk of harmful effects and most likely to take other drugs. There is a specific cannabinoid receptor and a naturally occurring agonist at this receptor—‘anandamide’. The role of this en dogenous system has yet to be defined. In addition, cannabis shows both weak opiate-like and weak barbiturate-like effects. The drug is metabolized to active and inactive metabolites, and their absorption into fat means that urine tests remain positive for up to 4wks after regular use has ceased. The effects of intoxication are apparent within minutes if the drug is smoked, peaking in 730min and lasting 2–5hrs. The effects of orally con sumed cannabis are slower to begin and more prolonged. The immediate effects include mild euphoria (‘the giggles’), a sense of enhanced well-being, a subjective sense of enhanced sensation, relaxation, altered time sense, and i appetite (‘the munchies’). Physically, there is mild tachycardia and variable dysarthria and ataxia. Acute harmful effects include mild paranoia, panic attacks, and accidents associated with delayed reaction time. Cannabis is normally smoked with Box 14.7 Vaping cannabis? With the increasing popularity of ‘vaping’, the development of more sophisticated delivery systems, capable of regulating the evaporation temperature, allows the possibility of these devices being used to ‘vape’ cannabis, NPS, and other recreational drugs. Vaping cannabis could lead to a reduction in tobacco use and depend ence among cannabis users, with the potential to reduce the harm as sociated with cannabis that relates to smoking. Should this become a future trend, there may be a generation of cannabis users who are not nicotine-dependent.1 1 Blundell MS, Dargan PI, Wood DM (2018) The dark cloud of recreational drugs and vaping. QJM 111:145–8.

Cannabis tobacco; therefore, all of the health risks associated with tobacco will also apply. The tendency of cannabis smokers to inhale deeply and to retain the smoke in the lungs for as long as possible will exacerbate this risk. There are no reports of fatal OD. Chronic harmful effects include dysthymia, anxiety/ depressive illnesses, the disputed amotivational syndrome (possibly repre senting a combination of chronic intoxication in a heavy user and a long half-life). The drug is not usually associated with physical dependency, but there is a mild, but characteristic, withdrawal syndrome in the previously heavy regular user who stops suddenly, consisting of insomnia, anxiety, and irritability. Cannabis use can precipitate an episode or a relapse of schizo phrenia. In addition, in regular users, it is associated with dose-related para noid ideation and other psychotic features. Interventions As an illegal drug, there are no set guidelines on safe use. Clinical experience suggests that irregular use can be free from major problems. Abstinence is indicated in those with major mental illness, and continuing cannabis use may expose those recovering from more serious drug problems to dealers and the drugs subculture.

628 Chapter 14 Substance misuse Volatile substances and anabolic steroids Volatile substances Simple hydrocarbons, such as acetone, toluene, xylene, and butane, have intoxicant properties. These chemicals are found in a variety of common products, including glue, lighter fuel, paint stripper, fire extinguishers, aero sols, paints, petrol, correcting fluid, and nail varnish remover. They are rap idly absorbed when inhaled or by sniffing propellant gases or aerosols. They cause non-specific i permeability of nerve cell membranes and produce eu phoriant effects, disinhibition,

slurred speech and blurred vision, and visual misperceptions. Acute harmful effects include local irritation, headache, cardiac arrhyth mias, acute suffocation by bag or laryngeal oedema, unconsciousness, aspir ation, and sudden death. Chronic harmful effects include liver and kidney damage, memory/concentration impairment, and probable long-term cog nitive impairment. There is a withdrawal syndrome similar to alcohol in very heavy regular users. Interventions Education of users and ‘at-risk’ groups. Most use will be ex perimental, with few going on to regular use. Legal controls on substance availability. Anabolic steroids These prescription-only medicines (e.g. nandrolone and stanozolol) have limited legitimate uses in the treatment of aplastic anaemia and osteopor osis. They can be abused by athletes and bodybuilders seeking competitive advantage or, more rarely, for their euphoriant effects alone. They produce i muscle mass and strength, with i training time and reduced recovery time, as well as euphoriant effects and a sense of i energy levels. (Other drugs misused by athletes include levothyroxine, growth hormone, diur etics, erythropoietin, and amphetamine.) Use of anabolic steroids is associated with physical health problems, including hypertension, hypogonadism, gynaecomastia, amenorrhoea, liver damage, impotence, and ♂ pattern baldness; and with mental health prob lems, including acute emotional instability (sometimes known as ‘roid rage’), i aggressiveness, persecutory/grandiose delusions, depressive illness, and chronic fatigue. If injected, they can also be associated with infection risks (E Depressants, p. 620). There is no withdrawal syndrome. Interventions Education of risks through coaches, teachers, etc. Effective monitoring of individual sports with out-of-season testing.

Novel psychoactive substances (‘legal highs’) Novel psychoactive substances (‘legal highs’) NPS, also known for a time as ‘legal highs’,12 became more popular for rec reational use in the 2000s. They are a heterogenous group of psychoactive substances that, for a few years, were not controlled under the Misuse of Drugs Act and were therefore legal to possess. They were mostly sold over the Internet or in specialist shops, usually labelled as ‘not for human con sumption’ in order to evade the law. A difficulty encountered with these drugs when they first came on the market was that synthesis in factories (usually in China and India) occured at a rate quicker than regulation. In 2010, mephedrone was banned in the UK following a report by the Advisory Council for the Misuse of Drugs (ACMD) submitted to the Home Secretary. In 2016, the Psychoactive Substances Bill was passed in the UK, banning trading of NPS (but not possession) of all existing and newer ana logues that came under the umbrella of NPS. The EMCDDA currently monitors over 560 substances falling into this category. Different types of NPS have been identified, the most commonly en countered being synthetic cathinones (stimulant types) and synthetic canna binoids. Others include hallucinogens NPS, psychedelic NPS and BDZ NPS. Synthetic cannabinoids, such as ‘Spice’, tend to be full cannabinoid re ceptor agonists, causing potent effects such as paranoia, agitation, and psychosis. Synthetic cathinones, such as mephadrone (4′-methyl-cathinone, ‘meow-meow’, ‘M-CAT’), are stimulant-type analogues with equivalent psychiatric sequelae. Other cathinones, often sold as ‘Bath salts’ (e.g. ‘Ivory wave’), also have stimulant effects, causing i release of 5-HT, DA, and/or NA into the synaptic cleft. NPSs are mainly used recreationally and can cause significant mor bidity, including possible fatal consequences. Users present with a range of conditions—medical, psychiatric, and/or both—from mild psychosis to protracted and severe psychotic symptoms, along with acute behavioural disturbance. The more serotenergic agents can cause SS (E Serotonin syndrome, p. 1022), presenting with tachycardia, myoclonus, hyperthermia, agitation, sweating, dilated pupils, and more gravely metabolic acidosis, seizures, and rhabdomyolysis requiring intensive supportive care. Stimulation of the ad renergic system by amphetamine-like agents can lead to tachycardia, vaso spasm, arrhythmia, hypertension, coma,

and seizures, which again may require intensive medical supportive treatment. Unfortunately, routine drug testing does not detect NPS use, although some metabolites can be detected, depending on specific laboratory analyses. 12 Tracy DK, Wood DM, Baumeister D (2017) Novel psychoactive substances: types, mechanisms of action, and effects. BMJ 356:i6848.

630 Chapter 14 Substance misuse Assessment of the drug user Taking a patient's drug use history is part of a standard psychiatric history and is especially important when there is comorbidity. The more detailed assessment described here is appropriate for patients in whom drug use is the primary focus of clinical concern and who are being assessed in spe cialist services. History should cover the following topics. Background information Name, address, next of kin, GP, name of other professionals involved (e.g. social worker, probation officer). Reasons for consultation now Why has the drug user presented now (e.g. pressure from family, pending conviction, 'had enough', increasing dif ficulty injecting)? What does the user seek from the programme? In ♀, is there a possibility of pregnancy? Current drug use Enquire about each drug taken over the previous 4wks. Describe the frequency of use (e.g. daily, most days, at weekends) and the number of times taken daily. Record the amount taken and route. Ask the user about episodes of withdrawal. Include alcohol, tobacco, and cannabis. If there is IV use, inquire about needle or other equipment sharing. Lifetime drug use Record the age at first use of drugs and the chan ging pattern of drug use until the most recent consultation. Enquire about periods of abstinence or stability and the reasons for this (e.g. prison, rela tionship, treatment programme). Complications of drug use ODs—deliberate or accidental. History of cellulitis, abscesses, or phlebitis. Hepatitis B and C and HIV status, if known. Previous treatment episodes Timing, locus, and type of previous drug treatment. How did the treatment end? Was the treatment helpful? Medical and psychiatric history All episodes of medical or psychiatric inpatient care. Contact with hospital specialists. Current health problems. Relationship with the GP. Family history Are there other family members with drug or alcohol problems? Family history of medical or psychiatric problems. Social history Current accommodation. How stable is this accommoda tion? Sexual orientation and number of sexual partners. Enquire about safe sex precautions. Describe the user's relationships—sexual, personal, and family. Note how many of these individuals currently use drugs. Forensic history Previous or pending convictions. Periods of imprison ment. Enquire about continuing criminal activity to support drug use (re mind the patient about confidentiality). Patient's aims in seeking treatment What is the patient's attitude to drug use? What treatment options do they favour? MSE Assess for depressed mood and suicidal thoughts or plans. Inquire directly about generalized anxiety and panic attacks (a BDZ user may be self-medicating a neurotic condition). Inquire directly about paranoid ideas and hallucinatory experiences and the directness or otherwise of their re lationship with drug use. Physical examination General condition. Weight. Condition of teeth. Signs of IV use (especially arms for signs of phlebitis, abscess, or old scar ring). Examine for an enlarged liver. Signs of withdrawals on assessment.

Assessment of the drug user Urine screening This is essential. Several specimens should be taken over several weeks. Repeated absence of evidence of a drug on screening makes its dependent use unlikely (see Table 14.7). Occasionally, testing errors do occur, so do not take action (e.g. stopping maintenance prescription) on the basis of the results of a single sample. Blood testing FBC, LFTs; discuss the need for HIV/hepatitis screening. Table 14.7 Urine drug testing Substance Duration of detectability Amphetamines 48hrs Benzodiazepines Ultra-short-acting (e.g. midazolam) 12hrs Short-acting (e.g. triazolam) 24hrs Intermediate-acting (e.g. temazepam) 40–80hrs Long-

acting (e.g. diazepam) 7 days Cocaine metabolites 2–3 days Methadone (maintenance-dosing) 7–9 days (approximate) Codeine/morphine 48hrs (Heroin is detected in the urine as the metabolite morphine) Cannabis Single use 3 days Moderate use (four times per week) 4 days Heavy use (daily) 10 days Chronic heavy user 21–27 days PCP 8 days (approximate)

632 Chapter 14 Substance misuse Planning treatment in drug misuse The longer-term goal of treatment will be eventual abstinence from drugs, but this may not be an achievable short- or medium-term goal in an in dividual case. Immediate treatment aims are therefore: to reduce drug-related mortality and morbidity; to reduce community infection rates; to reduce criminal activity, including the need for drug users to sell to others to finance their own habit; to optimize the patient's physical and mental health; and to stabilize, where appropriate, on an alternative substitute drug. Make diagnosis Confirm drug use (history, signs of withdrawals, urine testing). Assess the presence and extent of dependence. Assess the se verity of current problems and risk of future complications. Explore social, relationship, and medical problems. Assess the stage of change (E Stages of change and harm reduction, p. 575) and motivation. What are the short- and medium-term aims of treatment? Consider the need for emergency treatment Where there is evidence of psychotic illness or severe depressive illness, the patient may require inpatient assessment. Engage in service Treatment of drug misuse cannot be carried out through 'one-off' interventions. Patients should be engaged in the service by empathic and non-judgemental interviewing, the availability of the ser vice close to the point of need, and the ability of the service to respond to change in a previously ambivalent patient. Substitute prescribing will be a strong motivator for engagement in some patients but should always also have a role in helping the patient achieve some worthwhile change. Decide treatment goals and methods After assessment and diagnosis, the doctor should discuss with the patient their thoughts about treatment options, given the patient's drug history and local treatment availability. The doctor may have strong feelings about the appropriateness of a certain treatment, but this will not be successful unless the patient agrees. Plans may include: • Return to dependent use as previously Where individuals present in withdrawals, without other medical, surgical, or psychiatric reasons for admission, where there is no history of complicated withdrawal, and where there has been no previous involvement in treatment services, it is inappropriate to prescribe. The individual should not receive replacement medication. They should be offered the opportunity to attend for further assessment. • Counselling and support For non-dependent drug use, particularly episodic use, this may be the appropriate course. Give drug information and harm reduction advice, possibly coupled with referral to a community resource. • Detox (E Substitute prescribing 2: opiates, p. 636; E Substitute prescribing 3: benzodiazepines, p. 638) Where there is drug dependence and the patient wishes abstinence, then a plan for detox is considered. This may be community-based, with psychological support, symptomatic medication or reducing substitute medication, or as an inpatient. Consideration should be given to support after detox. How is abstinence to be maintained?

Planning treatment in drug misuse • Supported detox without prescription Some individuals can withdraw from drugs of dependence without use of a prescription. This may occur particularly where other changes in a person's life (e.g. change of area, break from dependent partner) facilitate abstinence. Unsupported detox without any medical help is frequently reported by users. • Supported detox with symptomatic medication Here, in addition to the support mentioned here, the individual is prescribed other non-replacement drugs to ameliorate withdrawal symptoms (e.g. lofexidine in opiate withdrawal). • Conversion to substitute drug with the aim of detox Here the

aim is to convert the individual’s drug use from street-bought to prescribed; then, from a period of stability, attempt supervised reduction in dose, aiming towards abstinence. • Conversion to substitute drug with the aim of maintenance Here the aim again is to convert from street to prescribed drugs, with stabilization via maintenance prescribing in the medium term. In a dependent user who does not feel that they can move to abstinence in the short term, maintenance prescribing to suitably selected patients is useful and associated with overall health benefits. Address other needs The drug treatment service should consider part of its role as being a gateway to other services which the drug user may re quire but be reluctant or unable to approach independently. Patients with social, financial, or physical health needs should have these explored and the need for referral considered. Do not make such referrals without the knowledge and agreement of the patient. Review psychiatric symptoms which have been attributed to drug use to assess their resolution. Consider ‘in-house’ or specialist psychiatric treatment of residual anxiety/depressive symptoms.

634 Chapter 14 Substance misuse Substitute prescribing 1: principles Withdrawal syndromes Any drug consumed regularly and heavily can be associated with withdrawal phenomena on stopping, even if not a classical withdrawal syndrome. The severity of withdrawal symptoms experienced by individual patients does not correlate well with their reported previous consumption, and so it is best to rely on objective evidence of withdrawal severity. Clinically signifi cant withdrawal phenomena occur in dependence on alcohol, opiates, and BDZs and are occasionally seen in cannabis, cocaine, and amphetamine use. In general, drugs with short half-lives will give rise to more rapid, but more transient, withdrawals. Detoxification refers to the process of managed withdrawal from drugs of dependence which can be aided by psychological support, symptomatic prescribing, or prescribing reducing doses of the same or similar drug. Substitute prescribing In many circumstances, the management of a drug user will include pre scription of substitute medication. This may be to enable detox from a de pendent drug or maintenance prescribing—a move from unstable street use to prescribed dependent use, to facilitate change now with abstinence later. The prescription of a drug should not occur in isolation but should be part of a comprehensive management plan, previously agreed with the patient and relevant members of the MDT. Prescribing for drug users should be guided by local procedures and practice, by the Home Office document Drug Misuse and Dependence: Guidelines on Clinical Management, and by the BNF (see Box 14.8). Substitute prescribing may have the following indications • To acutely reduce or prevent withdrawal symptoms: where detox is planned, a first step can be the conversion of all opiate or BDZ use to a single prescribed drug, which can then be reduced in a planned manner. Short-term prescription of a substitute drug may also be indicated to alleviate symptoms of withdrawal complicating the assessment of a dependent patient presenting with a medical or surgical emergency. • To stabilize drug intake and reduce secondary harm associated with street drug use: in patients who are not considering detox in the short term, substitute prescribing can be a means of harm reduction (e.g. by reducing the risk of accidental OD or by changing from IV to prescribed Box 14.8 Requirements for a controlled drug prescription • The prescription may be printed but must be signed by hand. • States the patient’s name, age, and current address. • Gives the name, concentration, and type of preparation required (e.g. methadone, 1mg in 1mL, sugar-free suspension). • States the required dose and frequency. • States the total quantity of drug to be dispensed in both words and figures. • Clearly signed and dated.

Substitute prescribing 1: principles oral use). In addition, having a stable, legitimate supply can reduce the need to resort to criminal activity to fund drug use, reducing the secondary, wider social harms of drug use. • To begin a process of change in drug-taking behaviour: a major aim in substitute prescribing is to fully supply the dependent drug and to move the patient away from extra recreational drug use and chaotic polydrug misuse. After stabilization, the user should be encouraged to discontinue contact with dealers and friends who continue to use drugs in a chaotic fashion. • To provide an incentive to continued patient contact and involvement with treatment services. Substitute prescribing should only be considered where • There is objective evidence of current dependence. This should include a history of daily consumption, a description of withdrawal symptoms, history of drug-seeking to relieve or prevent withdrawals, and consistent presence of the drug on urine screening. • The patient displays realistic motivation to change their drug use in a way which would be aided by prescription (e.g. to cease IV heroin use on instigation of oral methadone prescription). • The doctor believes the patient will cooperate with the prescription and that circumstances exist to allow adequate monitoring. Assessing the need for substitute medication Before prescribing substitute medication for detox or maintenance, the treating doctor should positively confirm dependence via: • Positive history of daily use with features of dependence syndrome. • Presence of the drug in two urine specimens at least 1wk apart. • Objective evidence of withdrawal features at assessment.

636 Chapter 14 Substance misuse Substitute prescribing 2: opiates Opiate detoxification Opiate withdrawal In an opiate-dependent individual, withdrawal symp toms appear 6–24hrs after the last dose and typically last 5–7 days, peaking on the second or third day. Withdrawal following discontinuation of the longer-acting methadone is more prolonged, with symptoms peaking on the seventh day or so and lasting up to 14 days. Symptoms of opiate withdrawal: sweating; dilated pupils; tachycardia; hypertension; piloerection (‘goose flesh’); watering eyes and nose; yawning; abdominal cramping; nausea and vomiting; diarrhoea; tremor; joint pains; muscle cramps. Symptomatic medication Several oral non-opiate medications are effective in ameliorating symptoms of opiate withdrawal. Unlike opiates, they are not liable to abuse or diversion to the black market. • Lofexidine α-adrenergic agonist. Start 200mcg bd, i in 200–400mcg steps up to max 2.4mg daily in 2–4 divided doses. Baseline BP, and monitor BP while raising the dose (risk of symptomatic hypotension); 10-day course; withdraw over 2–4 days. • Loperamide Treatment of diarrhoea. 4mg initially, with 2mg taken after each loose stool, for up to 5 days. Max daily dose: 16mg. • Metoclopramide For nausea/vomiting. 10mg dose, max 30mg daily. • Ibuprofen For headache/muscle pain. 400mg dose, max 1600mg daily. Substitute prescribing Several opiates are used in detox regimes. Where it is planned to continue prescribing on a maintenance basis, currently metha done is the drug of choice. • Methadone Long-acting synthetic opiate. Its half-life is 24hrs, and it is suitable for daily dosing (which can be supervised) (see Table 14.8). At daily dose of >80mg, it produces near saturation of opiate receptors, minimizing the ‘reward’ of further consumption. Prescribed as a coloured liquid, unsuitable for IV use, at concentration of 1mg/1mL. A sugar-free form is available. Licensed for use in opiate withdrawal and maintenance. • Buprenorphine A partial opiate agonist. Licensed for treatment of drug dependence. Available in od sublingual preparation. 8mg 8 30mg methadone. May produce less euphoria at higher doses than methadone. Abuse potential, as tablet can be prepared for injection. Table 14.8 Converting opiate dose to methadone dose Drug Daily dose Methadone equivalent Street heroin 0.5mg–1g 50–80mg Morphine 10mg 10mg Dipipanone (cyclizine) 10mg/30mg 4mg Dihydrocodeine 30mg 3mg Pethidine 50mg 5mg Codeine phosphate 30mg 2mg

Substitute prescribing 2: opiates • Dihydrocodeine Short-acting opiate. Not licensed for use in drug dependence. Occasional use in reduction regimes in patients already on a stable dose of street dihydrocodeine or in the final stages of dose reduction in patients on doses of methadone of <15mg daily. The need for bd–qds doses means that all dosages cannot be supervised. Opiate maintenance Aim is to prevent under-dosing (risk of use of street opiates, withdrawal symptoms) and overdosing (sedation, more drug available than required— with diversion to the black market). There is research evidence that a methadone script reduces street usage, criminality, and drug-related mor tality. For outpatient initiation of methadone maintenance, arrange to re view the patient in the morning, with them having consumed no opiates for 24hrs. Assess withdrawals, and dispense methadone as follows: • None or mild l no prescription. Review following day. • Moderate (aches, dilated pupils, yawning) l 10–20mg methadone. • Severe (vomiting, piloerection, hypertension) l 20–30mg methadone. Review after 4hrs, and repeat the dose if severe withdrawals continue, up to 30mg. Review daily over the first week, with dose increments of 5–10mg daily, if indicated. Methadone reaches a steady state 5 days after the last dose change. Arrange regular review after the first week, making subse quent increases by 10mg on each review, up to 7120mg. Stabilization may take up to 6wks to achieve. For maintenance monitoring, see E Monitoring of maintenance prescribing, p. 639. Dose reduction After stabilization and complete abstinence from street opiates, a decision should be made as to whether the aim is dose reduction or maintenance prescribing. Rapid reduction regimes reduce the dose over 14–21 days (perhaps using the drugs outlined in E Substitute prescribing 2: opiates, Symptomatic medication, p. 636, as adjuncts). Usually reduction is more gradual. Slow reduction is over 4–6 months, reducing by 75–10mg each fortnight. Make the largest absolute cuts at the beginning, and smaller, more gradual cuts as the total dose falls (i.e. keep the percentage drop in dose similar). In general, do not carry out reduction against the wishes of the patient—it is better to carry on a maintenance script than return to street use. Occasionally, ‘tread water’, then restart reduction. Opiate relapse prevention In previously dependent opiate users who have successfully completed detox, the opiate antagonist naltrexone may be used as an aid to relapse prevention. Taken regularly, it will prevent the rewarding, euphoriant effect of opiate consumption. Naltrexone Prescribed to aid abstinence in formerly dependent patients who are drug-free for >7 days. Start at 25mg, i to 50mg daily. Total weekly dose of 350mg may be divided and given 3 days/wk (e.g. to aid compliance or to enable supervision)—give 100mg on Monday and Wednesday and 150mg on Friday. Naltrexone is also used in specialist inpatient facilities to facilitate rapid detox over 5–7 days.

638 Chapter 14 Substance misuse Substitute prescribing 3: benzodiazepines Benzodiazepine detoxification BDZ withdrawal Chronic BDZ use leads to development of dependence, with a characteristic withdrawal syndrome. The symptoms appear within 24hrs of discontinuing a short-acting BDZ but may be delayed for up to 3wks for the longer-acting preparations. Symptoms of BDZ withdrawal: anx iety; insomnia; tremor; agitation; headache; nausea; sweating; depersonal ization; seizures; delirium. Substitute prescribing As for opiates, BDZ substitute prescribing should only be undertaken where there is clinical evidence of dependence, a clear treatment plan, and suitable patient monitoring in place. Substitute pre scribing in BDZ dependency uses long-acting diazepam. In prescribing for patients with BDZ dependency, convert all BDZ doses to diazepam, using Table 14.9. The aim is to find the lowest dose which will prevent with drawal symptoms (which may be well below the amount the patient has been taking). Divide the daily dose to avoid over-sedation. Benzodiazepine maintenance Unlike methadone

maintenance in opiate dependency, there is no evidence that long-term BDZ prescription reduces overall morbidity. There is evi dence that long-term prescription of >30mg of diazepam daily is associated with harm. New prescriptions should be for 30mg or less, with patients already on higher doses reduced to this amount. Dose reduction Cut the dose by 71/8th of the total dose each fortnight. For low dose, 2.5mg fortnightly; for high dose, 5mg fortnightly. Review and halt, or temporarily increase if substantial symptoms re-emerge. If the pa tient is also opiate-dependent and on methadone, keep methadone stable while reducing the BDZ. Table 14.9 Conversion to equivalent diazepam dose Drug Dose Diazepam 5mg Nitrazepam 5mg Temazepam 10mg Chlordiazepoxide 15mg Oxazepam 15mg Loprazolam 500mcg Lorazepam 500mcg Lormetazepam 500–1000mcg

Monitoring of maintenance prescribing Monitoring of maintenance prescribing Detox and stabilization on maintenance medication are often followed by rapid relapse despite successful completion. It is important to build moni toring of compliance into treatment strategies from the beginning. Review Regular review of all patients on maintenance prescription is indi cated at least monthly. At each review: • Is the dose sufficient? Is there evidence of withdrawals? Obtain feedback from the pharmacist/community nurse. • Is the dose insufficient? Consider small weekly increases in dose. Stop if evidence of intoxication. • Confirm use of illegal drugs via history, urine testing, and observation of evidence of IV use. • Plan movement towards goals. • Consider intervention in mental health/other issues. • Consider the need for a period of i supervision. Supervision of substitute prescribing The aim of supervised consumption is to ensure that the drug is being used as prescribed. • Supervised consumption usually for an initial minimum period of 3mths, taking into account work and childcare issues. • Consider ongoing supervised consumption (e.g. in pharmacy). • Once-daily dosing, with daily pick-up of drugs. • No more than 1wk's prescription at a time. • Advice regarding children and methadone. • Close liaison with the pharmacist and GP. • Thorough and clear records should be kept. • No replacement of 'lost' prescriptions. Discontinuing a failing treatment Where there is persistent non-compliance with treatment and where attempts to improve compliance or modify treat ment goals have failed, then maintenance should be discontinued. • Discontinue via a reduction regime. • Offer involvement with other services. • Inform the GP and pharmacist.

640 Chapter 14 Substance misuse Psychotic illnesses and substance misuse The association of substance misuse and psychotic features is common and problematic in clinical practice. The key to management is an accurate diag nosis. Psychotic symptoms represent an underlying psychiatric abnormality in this group of patients, as in any other. There is not a general finding of 'low-grade' psychotic features in substance users, and apparent psychotic features should not be attributed to effects of substance use without fur ther inquiry. Psychotic features during drug intoxication Substances with hallucino genic or stimulant activity can produce psychotic features during acute in toxication. This is not consistent and varies by drug dose and setting. These are characterized by a rapidly changing pattern of symptom type and se verity and include visual and other hallucinations, sensory distortions/illu sions, and persecutory and referential thinking. They are characteristically rapidly fluctuating, hour by hour, and show resolution as the drug level falls. Psychotic features during withdrawal In patients with physiological de pendency on alcohol, BDZs, or cocaine, withdrawals may be complicated by delirium in which variable psychotic features may be prominent. These will occur in the context of the general features of delirium (E Acute confusional state (delirium), p. 854). There may be fluctuating visual or tactile hallucinations and

poorly formed persecutory delusional ideas. Residual psychotic illness (drug-induced psychosis) In some individuals, psychotic features continue after the period of acute intoxication and with drawals has passed. These may be symptomatically more typical of primary psychotic illness and, once established, should be treated as for acute epi sodes of schizophrenia (E Initial treatment of acute psychosis, p. 200). Genuine comorbidity Many individuals with primary psychotic illnesses will misuse substances. In addition to the intrinsic risks of substance misuse, this carries risks in this group of diminished treatment compliance, risk of disinhibition leading to violence, and exacerbation of the primary illness. In view of the sometimes obvious (to others) causal link between drug use and relapse, it is worth asking why patients persist in substance use. Reasons include: • Endemic drug use within the patient's environment (e.g. home or social setting) or within other individuals with mental health problems. • As a means of self-medicating distressing positive and negative symptoms (which may be improved by addressing these symptoms directly).

Psychotic illnesses and substance misuse

642 Chapter 14 Substance misuse Legal issues related to drug and alcohol misuse Fitness to drive It is the patient's responsibility to inform the DVLA of any 'disability likely to affect safe driving'. The Driver and Vehicle Licensing Agency (DVLA) regards drug misuse as a disability in this context. Group 1 licences cover motorcars and motorcycles; group 2 licences cover HGVs and buses. Decisions regarding licensing are made on a case-by-case basis; however, the DVLA's current guidelines are as follows: • Alcohol misuse: loss of licence until 6-mths (group 1) or 1-year (group 2) period of abstinence or controlled drinking has been achieved, with normalization of blood parameters. • Alcohol dependence: loss of licence until 1-year (group 1) or 3- year (group 2) period of abstinence, with normalization of blood parameters. Consultant referral and support may be required. • Dependency/persistent use of cannabis, amphetamines, MDMA, LSD, and hallucinogens: loss of licence until 6-mth (group 1) or 1-yr (group 2) period of abstinence. Medical assessment and urine screening may be required. • Dependency/persistent use of heroin, morphine, cocaine, and methadone: loss of licence until 1-yr (group 1) or 3-yr (group 2) period of abstinence. Independent medical assessment and urine screening prior to relicensing. A favourable consultant report may be required for group 1 and will be required for group 2. Subject to annual review and favourable assessment, drivers complying fully with a consultant- supervised methadone maintenance programme may be licensed. Travel abroad Patients receiving a methadone prescription can travel abroad with a supply. If travelling for <3mths and carrying <3mths' supply, a per sonal import or export licence is not required. However, it is advised that a letter is obtained from the prescribing doctor or drug worker, which should confirm the patient's name, travel itinerary, names of prescribed controlled drugs, dosages, and the total amounts of each to be carried. This advice applies only to the right to take the drug out of the UK and return with any surplus. Travellers are advised to contact the embassy or consulate of the destination country prior to travel to ensure that import of methadone is allowed under local laws—countries' regulations vary widely. If travelling for >3 months and for more detailed information, see M http://www. homeoffice.gov.uk/ [accessed 12 July 2018]. Registration of drug addicts Compulsory registration of all drug addicts to the Home Office register of addicts ceased in 1997. Since then, data have been collected on a regional basis via the anonymized regional drug misuse data bases. Details regarding supply of forms in each area can be found in the BNF. Drug testing and treatment orders (DTTOs) A form of community sen tence introduced in the UK in 2000. The court makes an order requiring offenders with drug problems to undergo treatment and follow-up with a drug

treatment service. This may be part of another community order or a sentence in its own right. The sentencing court monitors compliance via mandatory urine testing. Sentence plans may change in response to indi vidual progress or problems. May last from 6mths to 3yrs.

Legal issues related to drug and alcohol misuse Drink driving limits (See Table 14.10.) On 5 December 2014, the Scottish Government implemented new legis lation for a lower drink driving limit in Scotland. The previous level of 80mg per 100mL of blood was reduced to 50mg. The new legislation was based on the logic model of reducing the BAC level of drivers, with the aims of reducing alcohol-related road traffic injuries and deaths. In 2010, NICE13 re viewed the evidence for the effectiveness of a lowered BAC level in drivers and concluded that there was strong evidence supporting a reduction in road traffic injuries and deaths in certain contexts. Table 14.10 Drink driving limits in different jurisdictions Level of alcohol England, Wales, and Northern Ireland Scotland and Republic of Ireland Micrograms per 100mL of breath 22 Milligrams per 100mL of blood 50 Milligrams per 100mL of urine 67 13 National Institute for Health and Care Excellence (2010) Review of effectiveness of laws limiting blood alcohol concentration levels to reduce alcohol-related road injuries and deaths. M http://www. nice.org.uk/media/default/About/what-we-do/NICE-guidance/NICE-guidelines/Public-health- guidelines/Additional-publications/Blood-alcohol-content-effectiveness-review.pdf [accessed: 12 Jul 2018].

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