

Alcohol is an available, legal, and frequently used drug in our society. However, its misuse and toxic effects are estimated to cost the British National Health Service £160 million each year in treatment costs. It is estimated that 28 000 people die each year in the UK as a result of their alcohol consumption.¹ Alcohol's associated morbidity impacts greatly on the work of the neurologist.

DETECTION OF ALCOHOL MISUSE

Approximately 25% of male medical admissions may be regarded as problem drinkers, with the group at highest risk being young male patients admitted to medical or orthopaedic wards.²

One of the key messages of this article must be to always ask patients about their alcohol use. This needs to be routinely documented in notes, perhaps most usefully with a full drug use history. People may minimise their alcohol use, so tact is necessary. Table 1 contains a list of questions that may be helpful; clearly it is important to get your own routine and to be guided by what the patient is able to tell you at that time. Relatives may also provide enlightening and not always corroborative histories.

ALCOHOL AT A NEUROTRANSMITTER LEVEL

Alcohol's central nervous system (CNS) effects are mediated through actions on a variety of neurotransmitters. There is a complex interplay between excitatory and inhibitory systems (table 2). The numerous transmitters involved in alcohol's action explain its diverse effects and the large number of drug interactions with both prescribed and illicit drugs.

ACUTE INTOXICATION

Many practitioners reading this article will be aware on both a personal and occupational level of the effects of acute intoxication. Blood alcohol concentrations reflect rate of intake, degree of tolerance, and the simultaneous effects of other drugs.

Extreme intoxication (> 300 mg/100 ml) leads to increasing drowsiness and then coma, with depressed tendon reflexes, hypotension, hypothermia, and slowed respiration. Death may occur with blood alcohol concentrations > 400 mg/100 ml.

Severely intoxicated individuals may require admission to hospital and management in specialist units with close monitoring and respiratory support. In those with a blood alcohol concentration of < 400 mg/100 ml an alternative cause for coma must be considered, such as head injury, other drug usage, hypoglycaemia or meningitis, as outlined by the Medical Council on Alcohol (see key references).

ALCOHOL WITHDRAWAL AND NEUROTOXICITY

When patients are drinking daily, or using alcohol at a very high level, they may become physically dependent on alcohol (table 3). It is sometimes difficult to predict which patients will require detoxification (medication assisted withdrawal), but levels of over 15 units/day for men and 10 units/day for women are often quoted.

At a cellular level daily alcohol intake induces the brain adaptations detailed in table 2, so leaving the brain with a functional increase in NMDA receptor levels, part of an excitatory brain system. When alcohol is stopped these excess receptors combine to cause a large calcium flux into cells, hyperexcitability, and cell death. There is also removal of alcohol mediated inhibitory actions via GABA and the magnesium controlled inhibitory part of the NMDA receptor. The increase in excitatory glutamate combined with a sudden drop in the brain's inhibitory systems combine to give noradrenergic "overdrive", leading to an increase in sympathetic activity.

Patients who stop drinking experience a spectrum of different symptoms ranging from mild sleep disturbance to frank delirium tremens. The severity of these relate to a number of factors, but most importantly the abruptness of withdrawal, level of alcohol intake, and the contribution of residual effects of previous drinking.

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Table 1 The alcohol history**Step 1: estimate consumption**

Do you ever drink alcohol? What do you usually drink?
How many times each week do you drink—could you say how much you would have on those occasions?
Are there times when you would drink more heavily than this?
(The MCA reference in the route map will help you quantify units and qualify them in terms of risk)

Step 2: establish if they are dependent on alcohol

To clarify, did you say that you are drinking every day?
What time of day is your first drink?
If you do not drink for a day, or miss your first drink of the day, how do you feel?
How would you rate alcohol as one of your priorities? Is it sometimes hard to think of anything else?
Have you ever needed medication to stop drinking?

Step 3: elucidate any problems

Has alcohol ever caused you any problems in the past? Could you tell me more about that?
Has anyone close to you expressed worries about your drinking? Did this cause difficulty between you?

Are you concerned about your alcohol use?

Step 4: further qualify experiences and concerns

You mention that you are worried about drinking harming your health—could you tell me more about that? Is it possible that the alcohol you had drunk that evening may have contributed to you becoming involved in the incident that led to your head injury?
Has alcohol ever affected your work or ability to sort things out at home?
Has alcohol ever got you into trouble with the police?
Is your alcohol use leaving you short of money?
Is this difficult to talk about for you?

The clinical manifestations of alcohol withdrawal are detailed in table 4. In hospital inpatients it is important to consider alcohol withdrawal in individuals who become confused in the days following admission. Early signs may be those of autonomic overactivity and tremor which typically peak 6–24 hours after stopping drinking. Early illusory or transient hallucinatory states may be a sign of more severe withdrawal, and an indication that more medication is required.

The peak incidence for seizures is around 36 hours (usually occurring between 12–48 hours) and for delirium around 72 hours.³ The distinguishing features of delirium tremens, the most severe form of alcohol withdrawal, are detailed (table 5).

MANAGEMENT OF ALCOHOL WITHDRAWAL

In patients with minor degrees of alcohol withdrawal there is often no requirement for medication to help with control of

Table 2 Alcohol and neurotransmitters

- ▶ **Dopamine:** alcohol increases dopamine use in the nucleus accumbens, mediating its pleasurable effects via the common reward pathway of the mesolimbic system
- ▶ **Noradrenaline:** alcohol release of noradrenaline (norepinephrine) contributes to the enlivening and activating “party” effects of alcohol
- ▶ **Endogenous opioids:** Alcohol’s analgesic, pleasure, and stress reducing functions are opioid related
- ▶ **GABA:** Alcohol can potentiate GABA (γ aminobutyric acid) activity through certain subunits of the GABA A receptor. This accounts for alcohol’s anxiolytic and ataxic actions, and partially for amnesia and sedation.
- ▶ **Glutamate:** Alcohol acts to block the excitatory NMDA (*N*-methyl-D-aspartate) receptor, opposing glutamate causing amnesia and other cerebral depressant effects
- ▶ **Serotonin:** Alcohol’s stimulation of 5HT3 (5-hydroxytryptamine 3) provides the nausea associated with alcohol use. Serotonin may also be linked to the pleasurable effects of alcohol and differing brain serotonin levels may distinguish between anxious and aggressive alcohol users

Table 3 Alcohol definitions

- ▶ **Unit of alcohol:** In the UK this means a drink with 8 g of ethanol—for example, half a pint of beer or a small (125 ml) glass of wine
- ▶ **Hazardous drinking:** synonymous with “at risk” drinking refers to drinking over 4 units per day for men and 2 units per day for women. These figures are also sometimes expressed as the weekly totals of 21 units per week for men and 14 units per week for women; the former version is more commonly used to dissuade people from the idea that the units can be drunk in one sitting
- ▶ **Harmful drinking** is described in the *International classification of diseases* as a pattern of drinking that causes damage to physical and mental health
- ▶ **Alcohol dependence:** this describes a cluster of symptoms where alcohol comes to dominate an individual’s life with features such as:
 - a strong desire or compulsion to drink
 - difficulty in controlling alcohol use
 - physiological withdrawal when drinking reduces
 - tolerance, where increasing doses of alcohol are required
 - neglect of other aspects of life
 - persisting with alcohol use despite evidence of harm
 Patients will drink on a daily basis, feel unwell if they stop drinking, and may report early morning drinking to relieve withdrawal

symptoms. The medical literature is becoming increasingly consistent that benzodiazepines are the treatment of choice for alcohol withdrawal; however, there is less clarity about which particular compound is superior.⁴

In clinical practice a long acting benzodiazepine is generally recommended, given in a gradually tapering dose (table 6). Courses are time limited, to maximise benefit to the patient and to minimise misuse of the drugs. Higher doses may be required in more unwell patients, and some acutely unwell patients may require parenteral treatment, usually with intravenous diazepam. It is important to also remember that acute withdrawal may precipitate Wernicke’s encephalopathy.

ALCOHOL AND SEIZURES

The relation between alcohol and seizures is complex. It has been estimated that the prevalence of epilepsy in alcohol dependent patients is three times that of the general population, although the prevalence of alcoholism is only slightly higher in patients with epilepsy than in the general population.⁵

As documented above, abrupt cessation after prolonged heavy drinking may trigger alcohol withdrawal seizures, thought to be caused by abrupt decline of the brain alcohol levels. Seizures may occur before the blood alcohol content

Table 4 Clinical features associated with alcohol withdrawal: symptoms usually peak between 10–30 hours, subsiding by 40–50 hours after the last drink

- ▶ Hyperactivity
- ▶ Anxiety
- ▶ Tremor
- ▶ Mild pyrexia
- ▶ Tachycardia
- ▶ Hypertension
- ▶ Sweating
- ▶ Nausea and retching
- ▶ Seizures (see text)
- ▶ Auditory and visual hallucinations, frightening, characteristically of vermin, and last usually 5 or 6 days

Table 5 Distinguishing features of delirium tremens, the most severe end of the alcohol withdrawal spectrum

- ▶ Pronounced withdrawal symptoms: coarse tremor, agitation, tachycardia, and extreme autonomic over activity, complicated by disorientation, hallucinations or delusions
- ▶ Occurs in less than 5% of individuals withdrawing from alcohol
- ▶ May develop from milder episodes of withdrawal, usually 48–72 hours after stopping drinking
- ▶ Hyperpyrexia, ketoacidosis, and circulatory collapse may develop

returns to zero due to partial withdrawal either during sleep or during financial limitations on the level of alcohol provision. Withdrawal seizures can occur after short bouts of drinking (1–6 days). The diagnosis of an alcohol withdrawal seizure is usually made because of the presence of other symptoms of alcohol withdrawal, and a history of recent alcohol misuse. There may be a genetic susceptibility to alcohol withdrawal seizures.

Acutely, partial seizures and epileptic EEG abnormalities are not infrequent in alcohol misusers. These are typical of post-traumatic epilepsy, correlating with the common occurrence of brain injury in alcohol misusers.

It has been suggested that alcohol causes between 9–25% of cases of status epilepticus, and this may often be the first presentation of alcohol related seizures. The outcome of patients with alcohol related status epilepticus appears more favourable, but recovery may be compounded by an unduly prolonged post-ictal state.

Common causes of occult traumatic brain injuries such as parenchymal contusions, subdural haematoma, and sub-arachnoid haemorrhages may coincide with or be caused by alcohol misuse. Brain imaging should therefore be performed in those presenting with their first alcohol related seizure. There should also be evaluation of whether a metabolic cause for seizures such as hypoglycaemia is present or whether they may result from the use of illicit drugs, either stimulant usage or sedative drug withdrawal. Alcohol is increasingly used as part of polypharmacy drug misuse, again requiring careful history taking.

Alcohol dependent patients also have seizures that occur remotely from alcohol withdrawal. The high prevalence of these seizures points to the role of alcohol toxicity in seizure genesis. It has been shown that heavy alcohol use leads to structural brain changes; that alcohol use alone can lead to epilepsy cannot be shown because of the high rate of prior undiagnosed brain injury.

Concern has been expressed that repeated cycles of alcohol exposure and withdrawal may lead to a process termed kindling, which could then precipitate seizures. This hypothesis has some experimental support, but a recent large study did not show a correlation with the number of withdrawal episodes. Previous head injury, however, was predictive of epilepsy.⁵ Clinical experience is that in repeated withdrawal episodes, symptoms tend to progress in severity, culminating in seizures or serious psychiatric sequelae. This may also reflect the fact that larger amounts of alcohol are being systematically consumed.

THIAMINE AND MEMORY

Improvements in imaging technology and our understanding of brain neurochemistry have started to unravel the association between alcohol, thiamine, and memory. It has been

Table 6 Management of alcohol withdrawal

- ▶ Optimise environment; quiet, well ventilated, adequately lit area, with reality orientation and reassurance available
- Moderate symptoms:** tachycardia, nausea, tremor, sweats, anxiety, headache, irritable, flu-like symptoms
- ▶ Oral diazepam 10–40 mg up to four times daily and hourly if necessary (max 160 mg/24 hours) titrated to response. This should be decreased in a stepwise fashion over 7 days
- Severe symptoms:** confusion, bizarre, uncooperative or aggressive behaviour
- ▶ Consider oral medication; if rapid control of the situation is required use intravenous diazepam by slow injection, up to 40 mg in divided doses during the first 30 minutes while monitored
 - ▶ If further adjunctive treatment is required consider haloperidol 5–10 mg intramuscularly. Haloperidol can be given orally 5 mg twice daily up to 20 mg/day if further adjunctive treatment is required to maintain control
 - ▶ Large doses of benzodiazepines may lead to respiratory depression, therefore monitor pulse oximetry and respiratory rate. In complex cases consider occult head injury and other causes of toxic confusion
 - ▶ Consider Wernicke-Korsakoff syndrome

proposed that most organic brain syndromes in alcoholic patients are variants of the Wernicke-Korsakoff syndrome, and that there is no need to consider a separate category of “alcoholic dementia”. Alcohol may have a direct neurotoxic effect on cortical neurons, but much of the damage may be secondary to diencephalic pathology caused by thiamine deficiency.

Scanning studies have shown that Korsakoff patients (including those with wider cognitive impairment as well as memory impairment) have widespread cerebral and subcortical atrophy which is greater than that found in the alcoholic patients without amnesia. This is particularly pronounced subcortically. Magnetic resonance imaging studies have also now suggested that shrinkage of the mamillary bodies is not as pathognomonic of Wernicke-Korsakoff as textbooks suggest, with patients without amnesia having mamillary body damage as frequently as those with amnesia.⁶

It is widely accepted that Wernicke-Korsakoff syndrome is caused by thiamine deficiency. However, research, diagnosis, and treatment in this area are hampered by a lack of accessible means to measure blood and brain thiamine, and misperceptions in diagnosis.

PREVENTION AND MANAGEMENT OF WERNICKE-KORSAKOFF SYNDROME

It is essential to consider dietary and vitamin status in all patients with an alcohol problem, whether they continue to drink or are presenting in the high risk period of alcohol withdrawal. Wernicke’s encephalopathy is characterised as a classical “triad” of ataxia, confusion, and ophthalmoplegia. It is important to move beyond this perception. The majority of diagnoses are made postmortem, and many patients will present not with the classical triad, but co-morbid head injury, abnormal gait, memory disturbance, nystagmus, hypothermia, hypotension, acute confusional state, coma, and of course alcohol withdrawal.

Korsakoff’s psychosis is described as an amnesic syndrome with impaired recent memory and relatively intact intellectual function. Patients rarely have a discrete deficit in forming new memories, often presenting with more global deficits, along a spectrum of severity. Korsakoff’s is not

always preceded by a clear episode of Wernicke's encephalopathy; more often there is an insidious course or a number of undiagnosed subacute episodes.

It is essential to assess nutritional status in patients misusing alcohol. Frequently patients will neglect their diet, deriving all of their calorific needs from alcohol, thus not ensuring an adequate oral intake of thiamine. Repeated vomiting, diarrhoea, and also the actions of alcohol on the gut further serve to impair the availability of thiamine to the body. Thiamine (along with other B vitamins) acts as a coenzyme in glucose metabolism, lipid metabolism, amino acid production, and neurotransmitter synthesis. The body only stores approximately 30 mg of thiamine and deficiency may present within 2–3 weeks of intake ceasing, as daily turnover is 1 mg. The brain is particularly sensitive to a breakdown in the complex B vitamin dependent metabolism of glucose.

Where Wernicke's encephalopathy is suspected prompt high dose parenteral treatment is required (table 7). The importance of this cannot be overstated; this is a life saving treatment. In the early 1990s there was a withdrawal of the parenteral B vitamin preparation Parentrovite, following a warning from the Committee on Safety of Medicines (CSM) regarding serious adverse reactions. There then followed a nine month gap before the reintroduction of parenteral preparations, during which time there was a large shift in clinical practice.

It is now felt that the incidence of anaphylactic reaction has been misperceived; it is reported that in the UK there were four reports to the CSM per million intravenous ampoules sold and one report per five million ampoules sold of the intramuscular preparation.⁷ Nevertheless, the *British National Formulary* (BNF) states that facilities for the treatment of anaphylactic reactions should be available where parenteral preparations are given.

There has been a move to using oral vitamin supplements, even in those acutely unwell with Wernicke's encephalopathy. In alcohol misusers thiamine absorption is very variable, with some patients showing little or no absorption due to a reduction in the sodium dependent transport mechanism caused by alcohol usage and malnutrition. The presence of alcohol in the gut will also decrease absorption. It is unlikely that oral dosing will fulfil the daily requirement for thiamine, never mind replace the large deficits that these patients may have.

Table 7 Management of Wernicke-Korsakoff syndrome

- ▶ **Detection:** Consider in all cases of alcohol withdrawal, alcohol misuse and dependence, head injury, and acute confusional states. The classical triad of symptoms is not required for diagnosis
- ▶ **Assess nutritional status:** Patients should be asked about their diet, recent vomiting/diarrhoea, and weight loss
- ▶ **Treatment of Wernicke-Korsakoff syndrome:** *British National Formulary* recommends 2–3 pairs of high potency intravenous Pabrinex injections every 8 hours. This should be reassessed after 3 days, by when a noticeable improvement should have occurred and treatment should continue as long as clinical improvement continues.⁷
- ▶ **Patients at risk of Wernicke-Korsakoff syndrome:** One pair of ampoules is recommended for 3–5 days in alcohol misusers at risk of Wernicke-Korsakoff syndrome—that is, those with a history of malnutrition, weight loss, diarrhoea, vomiting or physical illness.^{7,8}
- ▶ **Optimal aftercare:** Appropriate and supportive placement; maintenance of abstinence

There is an important subgroup of patients who merit consideration; these are patients who do not manifest clinical signs of Wernicke's, but who are at risk because of poor diet, diarrhoea, vomiting, physical illness, and weight loss. There is conflicting expert opinion in this area, and a paucity of high level evidence, but the balance of probability, and the research findings, suggest that this group of patients should also be given parenteral B vitamins. It is felt that in this group insidious or multiple subacute episodes are occurring.

The debate over the role of oral vitamins continues. A recent evidence based guideline recommended their usage in patients who continued to drink and whose diet may be deficient.⁸ This was on the rationale that these were inexpensive compounds, with little associated harm, which may convey some benefit. Split dosing was recommended, to maximise absorption.

There is a popular misperception that Korsakoff's is a static condition, unamenable to treatment or improvement. Classically the outcome from an episode of Korsakoff syndrome falls roughly into quarters, 25% showing no recovery, 25% slight, 25% significant, and 25% complete recovery of memory.⁹

Often patients with Korsakoff syndrome are first encountered in general medical settings where they have presented with a complication of their alcohol misuse. Once the toxic state has resolved a clearer picture of deficits may be obtained. It may be the case that patients are unable to return home because of their memory deficits. Services are generally poor at placing patients who may be young and "fit", but with pronounced cognitive impairment. This may lead to patients remaining on acute wards, which can be problematic for both staff and patients. Ideally rehabilitation should be in special units, offering to help recovery of cognitive function using special techniques. These include memory aids such as diaries and personal organisers. A technique called "errorless" learning is also used where patient's memories are refined to retain only correct solutions to problems, not the memory of failed attempts.

Table 8 Pharmacological treatments used in alcohol dependence

- ▶ **Acamprosate:** Blocks GABA and reduces NMDA receptor glutamate related excitation. It may have an effect on calcium influx, and it has been proposed as having a potentially neuroprotective role in detoxification. It does not interact with alcohol, and is prescribed post detoxification in specialist centres as an aid to maintaining abstinence
- ▶ **Disulfiram:** Blocks aldehyde dehydrogenase, an enzyme involved in the metabolism of alcohol, leading to a build up of acetaldehyde if alcohol is taken. This leads to an unpleasant reaction where the patient will flush, experience headache, palpitations, nausea, vomiting, and, with large doses, arrhythmias, hypotension, and collapse. It is used in well motivated patients where compliance can be supervised by a partner, colleague or by healthcare staff. Because of the nature of the disulfiram reaction it should not be used in those who would be susceptible to cardiovascular disruption. Psychosis, pregnancy, and breastfeeding are also contraindications. Patients should carry a card warning of the administration of alcohol as it can be present in preparations such as mouthwashes and toiletries.
- ▶ **Naltrexone:** Antagonises endogenous opioids such as β endorphin and enkephalins. By opposing mediators of the pleasurable effects of alcohol it can make alcohol use less rewarding and prevent excessive single session consumption. Naltrexone is not licensed for the treatment of alcohol use in Britain, but is prescribed in some specialist centres and has a growing evidence base. It is mainly used in "binge" drinkers to attenuate the length and severity of their binges

It is essential that these patients are helped to abstain completely from alcohol. They may retain the urge to drink. They have a reduced tolerance to alcohol and are at high risk of injury through severe intoxication or collapse. A further episode of Wernicke-Korsakoff syndrome may be precipitated or recovery further impaired.

These patients explicitly require management by a multi-disciplinary team, with social work and occupational therapy being key players. Liaison psychiatry will be able to offer assessment and advice on management of difficult behaviour, as well as having knowledge of local substance misuse service provision.

NEUROPATHY AND ATAXIA

Alcohol misusers may develop "Saturday night palsy", a focal peripheral nerve palsy as a result of nerve compression when heavily sleeping or stuporose. Recovery is usually complete.

Chronic alcohol misusers may also develop a symmetrical, bilateral mixed sensory and motor peripheral neuropathy, usually of the lower limbs. Individuals may be asymptomatic or present with pain, numbness, burning feet, and hyperaesthesia. There may also be muscle weakness and diminished tendon reflexes. These neuropathies are associated with thiamine deficiency and may show some recovery with abstinence from alcohol and thiamine supplementation.

An acquired cerebellar syndrome with anterosuperior vermal atrophy on imaging (computed tomography or magnetic resonance) is well recognised. Typically gait is broad based and unstable, upper limbs being rarely involved. Similarly dysarthria and disordered eye movements are infrequent and where found should prompt a search for an alternative cause.

Alcohol is also associated with a number of other rarer neurological presentations outwith the scope of this article.

PSYCHIATRIC SEQUELAE OF ALCOHOL MISUSE

Depressive illness is common in alcohol misusers. It is reported that as many as 80% of alcoholics complain of depressive symptoms, including 30% who fulfil criteria for a major depressive disorder.¹⁰

Alcohol intoxication can be accompanied by temporary but severe depressive symptoms. It is recognised that long term alcohol use can induce a depressive disorder indistinguishable from a primary depressive illness. Substance induced disorders will remit 2–4 weeks after drinking has ceased without the need for antidepressants. It is therefore common psychiatric practice to try to reassess mood when patients are alcohol-free, to avoid unnecessary antidepressant prescribing.

Patients often report using alcohol to try to relieve symptoms of low mood. Research has shown some support for this concept in women, and also that depressive symptoms may be predictive of relapse. When a primary depressive illness is present, alcohol may cause further lowering of mood, and its effect of removing inhibitions leaves patients vulnerable to impulsive actions including self harm.

Anxiety disorders and alcohol dependence have a reciprocal causal relation over time. Alcohol is recognised for its anxiolytic and socially enlivening properties and its use as a prop in social situations may lead to dependence. Anxiety disorders are commonly treated using a combination of medication such as serotonin reuptake inhibitors (SSRIs) and talking treatments, with an initial goal of abstinence.

Alcohol use predisposes to psychotic symptoms. These can occur during withdrawal, and classically are visual hallucinations.

Alcohol misusers may develop a hallucinosis and will report hallucinations which may be a continuation of those experienced in acute withdrawal or may start de novo in those who are still drinking. They may start as simple sounds such as glasses clinking, but develop on to form words or sentences. These experiences may lead to the patient forming a delusional network around them. This condition has some similarities to schizophrenia, but differs in its age of onset, family history, and prognosis. Usually, with antipsychotic treatment and abstinence from alcohol, patients will be symptom-free at six months. Alcohol misuse is common in schizophrenic patients, and is often implicated in relapse and exacerbation of symptoms. Again it can be difficult to elucidate the primary condition, but abstinence from alcohol improves symptoms.

TREATMENT AVENUES

Alcohol misuse is a difficult and sometimes frustrating condition to treat, both for the individual involved and also sometimes for clinicians. Services vary between hospitals, with some well served by liaison and addiction psychiatry with specialist alcohol nurses, and others struggling to obtain a psychiatric assessment. Many areas will have cards or leaflets detailing local substance misuse services and how to contact them.

As discussed above, it is important to establish patients' perception of their drinking and to gauge their willingness to change. If they do not regard their drinking as a problem, and are speaking to you under duress, a good outcome can be to establish a rapport and to try to get them to reflect on their drinking. Motivational interviewing approaches, where people are gently helped to discuss their drinking behaviour and encouraged to focus on the associated harms, are now felt to be more helpful than statements like "You are killing yourself with drink". Controlled drinking may be a viable goal for some patients, but generally in those who have reached the levels of harmful or dependent drinking at least a period of sobriety is highly recommended.

Where patients express a readiness to change their behaviour there are a variety of approaches on offer. Post-detoxification there are medications which can be proposed (table 8). These should be used with ongoing supportive treatment. As well as medication there are psychological therapies which have been shown to help prevent relapse: behavioural self control therapy, motivational enhancement therapy, marital/family therapy, and coping skills training. Local alcohol services may run groups for patients that incorporate elements of these approaches.

Lay services have a key role in alcohol treatment. Patients should have access to information about Alcoholics Anonymous and agencies such as Councils on Alcohol. Alcoholics Anonymous maintains the sobriety of thousands of people.

Alcohol misuse has a wider effect on the family. Clinicians should be alert to concerns around domestic violence and child protection. Families may also have a role in helping people move into treatment services and helping them remain there.

SUMMARY

Alcohol is a difficult drug to help people manage. It exerts a myriad of physical and psychiatric effects, many of which will

have implications for the practising neurologist. It is important to recognise when patients are experiencing difficulties with alcohol and sensitively to move them towards help.

In the acute inpatient situation it is important to manage withdrawal with adequate doses of benzodiazepines and consider use of parenteral vitamins. After detoxification patients should be assessed for physical and psychiatric sequelae of their alcohol use and appropriate referral and treatment initiated.

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- ▶ Explains in depth the action of alcohol at a cellular level, discussing also alcohol withdrawal and treatment implications.*
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- ▶ This website of the Medical Council on Alcohol contains the third edition of *The medical students' handbook*. Although this resource has an undergraduate title it is in fact a well written and authoritative text. We recommend downloading and reading it.*