

Alcohol and anaesthesia

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Key points

Consumption of alcohol is widespread in British society and a common co-factor in emergency hospital admissions.

Morbidity associated with chronic alcohol abuse appears to be increasing and affecting younger patients.

Anaesthetists must consider the acute and chronic effects of alcohol at all stages of the patient pathway.

Alcohol withdrawal is a potentially life-threatening complication that must be diagnosed and actively managed.

Anaesthetists are as susceptible to alcohol-related disease as others in the same socio-economic group.

Two-thirds of adults in England drink alcohol on a weekly basis, and 30% drink more than the recommended daily level.¹ Among children, 46% of 15 yr olds and 3% of 11 yr olds admit to drinking periodically.²

Alcohol misuse is estimated to cost the NHS £3 billion per year. Alcohol-related disease was the primary or secondary diagnosis for over 180 000 NHS hospital admissions in 2004/2005.³ This includes a doubling in the number of admissions for alcoholic liver disease over the past 10 yr. Casualties in road traffic accidents involving a driver over the legal limit for alcohol numbered 17 000 in 2004, representing 6% of the total and including 590 fatalities.⁴ Twelve per cent of A&E attendances are for alcohol-related problems, and 22% of attendees have recently consumed alcohol. Risk of injury is greatest with episodic, heavy drinking, a pattern that is increasing in Britain.

The high prevalence of alcohol-related disease means that the anaesthetist will frequently encounter such patients and must consider both the acute and chronic effects of alcohol consumption.

Acute intoxication

The differential diagnosis of decreased conscious level in an intoxicated patient may be challenging. Alcohol intoxication alone can cause coma, but other causes must also be considered, particularly head injury. Risk of vomiting, exacerbated by the decreased rate of gastric emptying, and the need to secure the airway for imaging or ongoing management means that intubation is often indicated.

Even in the absence of a depressed conscious level, alcohol consumption can produce psychomotor impairment equivalent to commonly used sedation regimens.⁵ This makes it difficult to obtain informed consent. If a procedure cannot be delayed until the effects of intoxication have gone, the patient may have to be treated as if lacking the capacity to make an informed choice.

Confusion, aggression, and psychomotor impairment mean the intoxicated patient remains at risk of causing injury to himself or others.

Chronic alcohol misuse

Patients suffering from chronic alcohol misuse can present with acute deterioration, with or without concurrent illness, and necessitating intensive care. Alternatively, they may present acutely or electively for any number of operations, and the wide-ranging effects of the chronic condition must be taken into account. Recovery may be complicated by alcohol withdrawal. Table 1 summarizes the medical disorders associated with alcoholism.

Anaesthetic considerations

Pre-operative

A history of alcohol use should be sought in all adults and adolescents presenting for surgery. The CAGE questionnaire (Table 2) is concise and specific; a score of >2 is strongly indicative of alcohol use at a level likely to have adverse medical or social consequences. If chronic alcohol misuse is suspected, then examination should concentrate on the cardiovascular (hypertension, arrhythmias, and signs of cardiac failure) and nervous systems (disturbance of vision, co-ordination or cognitive function, or evidence of autonomic or peripheral neuropathies). Specific signs of liver disease should also be sought.

Investigations may reveal a pancytopenia as a result of ethanol-induced bone marrow toxicity. Folate deficiency is common. Electrolyte, glucose, and haematological abnormalities may be present without overt liver disease. A prolonged prothrombin time is an early manifestation of impaired hepatic synthetic function. Chest X-ray and ECG are mandatory. Echocardiogram is indicated if there is a history or clinical signs of impaired cardiac function.

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Table 1 Medical disorders associated with alcoholism

CNS	Wernicke–Korsakoff syndrome	Metabolic	Hyperlipidaemia
	Peripheral neuropathy		Obesity. Hypoglycaemia
	Autonomic dysfunction		Hypokalaemia Hypomagnesaemia Hyperuricaemia
CVS	Cardiomyopathy	Haematological	Macrocytosis
	Heart failure		Thrombocytopenia
	Hypertension		Leucopenia
	Arrhythmias (e.g. AF, SVT, VT)		
GI	Alcoholic liver disease	Musculoskeletal	Myopathy
	Pancreatitis		Osteoporosis
	Gastritis		Osteomalacia
	Oesophageal and bowel carcinoma		

Table 2 The Cage questionnaire

C	Have you ever felt you should cut down on your drinking?
A	Have you been annoyed by other people criticizing your drinking?
G	Have you ever felt guilty about drinking?
E	Have you ever taken a drink in the morning to steady your nerves or ease a hangover (eye-opener)?

Before regional anaesthesia is undertaken, pre-existing neurological or musculoskeletal abnormalities, such as weakness because of neuropathy or myopathy, or Dupuytren's contractures, should be documented in detail. Regional techniques may be technically more difficult with increased risk of inadvertent injury. The sequelae of such injuries need to be differentiated from the pre-existing deficits.

Before surgery, administration of parenteral B vitamins (Pabrinex) may be indicated to prevent Wernicke–Korsakoff syndrome. Vitamin K, clotting factors, fresh frozen plasma, or platelets may also be necessary to correct coagulopathy.

Very nervous patients benefit from anxiolytic pre-medication; however, it should be remembered that anxiety may be an early manifestation of the alcohol withdrawal syndrome (AWS).

Intra-operative

Rapid sequence induction is indicated for reasons highlighted above. Chronic alcohol use increases dose requirements for general anaesthetic agents. This is thought to be because in part of enzyme induction; for example, the microsomal ethanol-oxidizing system (particularly Cytochrome P-450 2E1), or through the development of cross tolerance.⁶ Conversely, if the blood ethanol concentration is elevated, then competitive inhibition of metabolic enzymes can increase the sensitivity to other drugs. Volatile agents compete with ethanol for binding on neuronal gamma-aminobutyric acid (GABA) and glycine receptors. The effective doses of propofol, thiopental, and opioids such as alfentanil are increased. These increased anaesthetic requirements can exacerbate the risk of cardiovascular instability in patients who may be suffering from cardiomyopathy, heart failure, or dehydration.

The distribution and metabolism of anaesthetic drugs is altered by hypoalbuminaemia and hepatic impairment. Neuromuscular blocking agents that undergo hepatic metabolism may have a prolonged duration of action.

Post-operative

Chronic heavy alcohol use is associated with a 2–5-fold increase in post-operative complications, with higher rates of admission to high-dependency or intensive care units and increased length of hospital stay. Depletion of coagulation factors and thrombocytopenia increase the incidence of post-operative bleeding. The adrenocortical stress response to major surgery is attenuated. Immune deficiency as a result of leucopenia and altered cytokine production increase the risk of post-operative infection (especially, surgical wounds, respiratory system, or urinary tract). Experimentally, chronic ethanol ingestion has been shown to deplete pulmonary glutathione, thus reducing surfactant production and altering epithelial cell permeability. This may explain the increased susceptibility to acute lung injury shown by alcohol abusers, especially after thoracic surgery.⁷ Underlying cardiac disease limits the ability to meet the increased metabolic requirement after major surgery, and arrhythmias and acute coronary syndrome are more common after operation. Electrolyte disturbances or periods of relative hypotension exacerbate the risk.

Alcohol use is an independent risk factor for the development of acute confusion or delirium after operation. This can be reduced by meticulous pain control, oxygenation, and correction of metabolic disturbances. Neuroleptic drugs are preferred to benzodiazepines when alcohol withdrawal is not the primary cause of delirium. Haloperidol can be given i.v.; it is less sedating and causes less hypotension than other antipsychotic drugs. Extrapyramidal side-effects are rare when it is used i.v. for delirium. The dose given should take into account the level of disturbance and the age and physical status of the patient. An initial dose of 0.5–10 mg may be repeated after 20–30 min. Haloperidol can also be given as an infusion. A daily limit of 100 mg is generally safe, reduced to 60 mg if benzodiazepines are used concurrently. Other neuroleptics that have been used in the treatment of delirium include chlorpromazine and the atypical antipsychotic drugs risperidone and olanzapine.

Alcohol withdrawal syndrome

Delirium may be a sign of the AWS. The syndrome results from neurological receptor changes after long-term alcohol use. Ethanol binds to post-synaptic GABA_A receptors, enhancing their inhibitory effect. The resulting chronic excitatory suppression, coupled with a direct inhibition of excitatory glutamate *N*-methyl-D-aspartate (NMDA) receptors, leads to an increased brain synthesis of excitatory neurotransmitters such as norepinephrine, 5-hydroxytryptamine, and dopamine. When the inhibitory effects of ethanol are withdrawn, the brain becomes exposed to increased levels of excitatory neurotransmitters.

Alcohol withdrawal syndrome is characterized by tremor, gastric upset, sweating, hypertension, hyper-reflexia, anxiety, and agitation progressing to delirium, hallucinations, and seizures. The syndrome typically develops after 6–24 h without alcohol, but can

Table 3 Pharmacological therapies in alcohol withdrawal. *Wide dose variations reflect variation in symptom severity. Prophylactic doses require regular administration (e.g. 6 hourly)

	Duration of action	Route of administration	Dose*
Benzodiazepines			
Chlordiazepoxide	Long	p.o.	5–25 mg (prophylaxis) 50–100 mg (treatment)
Lorazepam	Short	p.o./i.v.	0.5–2 mg (prophylaxis) 1–8 mg (treatment)
Diazepam	Long	p.o./i.v.	2.5–10 mg (prophylaxis) 10–40 mg (treatment)
Other agents			
Clomethiazole		p.o.	9–12 capsules in 24 h (prophylaxis)
Haloperidol		p.o./i.v./i.m.	0.5–20 mg (treatment)
Clonidine		i.v.	0.1–1 mg bolus/0.1–4 µg kg ⁻¹ h ⁻¹ infusion (treatment)

be delayed for up to 5 days. Although it may occur before operation, it is more common in the post-operative period. It can be fatal if not treated appropriately. Prophylactic treatment may help prevent AWS.⁶ The most commonly used agents are benzodiazepines, although clomethiazole is an alternative. Table 3 lists the drugs used for both prophylaxis and treatment of AWS.

Long-acting agents may be better at preventing seizures and rebound symptoms but are associated with more sedation than short-acting agents. An observational structured withdrawal severity scale such as the Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised (CIWA-Ar) is useful for monitoring the effectiveness of prophylaxis or fixed-schedule treatment regimens, or guiding administration in symptom-triggered treatment regimens. The severity of withdrawal symptoms can vary greatly, as can the dosage of medication needed for control of these symptoms.⁸

Non-benzodiazepine agents are not recommended for monotherapy but may be used in conjunction with benzodiazepines in AWS. Symptomatic control has been achieved with β -adrenergic blockers and centrally acting α -adrenergic agonists (clonidine and dexmedetomidine), but they do not reduce the incidence of delirium or seizures. Haloperidol may be useful for severe agitation or hallucinations but, conversely, may increase the risk of seizures. Future management may include the use of non-benzodiazepine anti-convulsant agents such as carbamazepine, sodium valproate, and topiramate. Additionally, animal studies have identified centrally acting natriuretic peptides as another potentially useful mechanism for modifying AWS.

Alcohol misuse and doctors

There is a widely held perception that doctors suffer from a higher than average incidence of alcohol misuse. This is based on epidemiological evidence of an increased incidence of cirrhosis among doctors. More recent studies have tended to indicate that doctors

are no more likely to abuse alcohol than those from matched socio-economic populations. This still means, however, that there are a large number of practicing doctors whose alcohol intake adversely affects their health and, potentially, their professional performance. Hazardous drinking behaviour has been shown to be common among medical students, and to continue into the junior doctor years and beyond.

Anaesthetists and alcohol misuse

Data from Australia and New Zealand showed 1.3% of doctors entering anaesthetic training became chemically dependent (alcohol or other drugs) over the 10 yr study period.⁹ In the UK and Ireland, the number of anaesthetists with alcohol misuse problems warranting intervention was reported as 77 over a 10 yr period.¹⁰ It is probably that this underestimates the true prevalence of problem drinking, as it represents only the most serious situations.

Anaesthetists do not have the highest incidence of alcohol misuse among doctors, but they are over-represented as a group. Particular risk factors for drinking problem include stress, working patterns (often alone and out of hours), a feeling of lack of control over workload, and fatigue. Individuals who misuse other substances can often also drink alcohol to excess. Anaesthetists as a group are prone to drug dependence as a result of easy access to these substances, particularly opioids.

Rehabilitation

The success of rehabilitation programmes is enhanced if there is early recognition of the problem and instigation of expert treatment. Unfortunately, alcoholics are often very adept at concealing their drinking problem and may be in denial of needing help. The stigma attached to alcohol misuse can also delay presentation. All doctors should be aware of the signs of alcohol misuse among colleagues and also their own risk factors. Trusts should likewise have a policy for support and treatment of their staff.

Successful treatment of alcohol abuse can allow a full and productive return to clinical duties. The support and encouragement of colleagues is essential in preventing relapse.

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Please see multiple choice questions 8–11