

26.5.4 Alcohol misuse 6486

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SECTION 26 Psychiatric and drug-related disorders 6486 *Treponema pallidum* infection have been increasing. Given this re- surge, some recommend that patients with new onset psychosis should be screened. The human immunodeficiency virus (HIV) penetrates the central nervous system, although the extent to which HIV disrupts neuronal function and neurotransmission is unknown. As with other organic psychoses, treatment of the underlying cause is paramount and in this case preventative; antiretroviral therapy reduces the rate of new onset psychosis in HIV infected persons. Organic psychosis from HIV infection is more frequent in individuals with prior psychiatric disorders, lower global cognitive performance, and brain atrophy, suggesting it may be modulated through a dementia syndrome. Other infectious agents causing encephalitis can present as or- ganic psychosis. In these cases, early encephalitis may have mostly nonfocal findings and lumbar puncture with cerebrospinal fluid analysis is required. Typically these conditions progress from initial organic psychosis to a more clearly recognizable encephalitis picture so early detection is important but difficult. FURTHER READING Clancy MJ, et al. (2014). The prevalence of psychosis in epilepsy: a sys- tematic review and meta-analysis. *BMC Psychiatry*, 14, 75. Dalmau J, Rosenfeld MR (2014). Autoimmune encephalitis update. *Neuro Oncol*, 16, 771–8. Demily C, Sedel F (2014). Psychiatric manifestations of treatable her- editary metabolic disorders in adults. *Ann Gen Psychiatry*, 13, 27. Fricchione GL, Carbone L, Bennett WI (1995). Psychotic disorder caused by a general medical condition, with delusions: secondary ‘organic’ delusional syndromes. *Psychiatr Clin North Am*, 18, 363–78. Friedman JH (2013). Parkinson disease psychosis: update. *Behav Neurol*, 27, 469–77. Friedrich F, et al. (2014). Psychosis in neurosyphilis—clinical aspects and implications. *Psychopathology*, 47, 3–9. Lee SW, Donlon S, Caplan JP (2011). Steroid responsive enceph- alopathy associated with autoimmune thyroiditis (SREAT) or Hashimoto’s encephalopathy: a case and review. *Psychosomatics*, 52, 99–108. 26.5.4 Alcohol misuse Jonathan Wood ESSENTIALS Excessive intake of alcohol is common and increases the risk of developing many medical conditions, as well as leading to psycho- logical and social harm. Medical conditions commonly associated with harmful use include upper gastrointestinal and liver disease, hypertension, and accidents. The psychological state of alcohol de- pendency makes it more difficult to reduce intake and physical de- pendency may lead to a withdrawal syndrome after admission to hospital.

Withdrawal symptoms range from mild to severe and will commonly require treatment to prevent complications of alcohol withdrawal such as seizures. Delirium tremens is a life-threatening consequence of alcohol withdrawal that requires immediate active management. The role of alcohol in the development of Wernicke–Korsakoff syndrome means patients will also need prophylaxis or treatment for this in the acute medical setting.

Introduction The use of fermentation to make alcohol out of sugars has been a human activity for thousands of years. Drinking alcohol is currently seen as a normal part of Western society. The current World Health Organization classification ICD-10 divides abnormal alcohol use into harmful use (F10.1) and alcohol dependence (F10.2). The most recent American Psychiatric Association classification of psychiatric disorders, DSM-5, combines these into a single diagnosis of Alcohol Use Disorder (AUD) with a spectrum of mild to severe. Alcohol misuse is a significant cause of both psychiatric and medical morbidity and mortality. As many as 20% of admissions to medical wards in the United Kingdom are directly related to alcohol consumption or to alcohol-related illnesses.

Aetiology Alcohol is an addictive drug, but not everyone who uses it develops misuse. The aetiology of AUD includes genetic, psychological, and social factors.

Genetic factors The genetic transmission of misuse is polygenic, with many susceptibility loci, each with small or medium effects and low penetrance. There are also genes involved in the metabolism of alcohol that are negatively associated with misuse. ALDH2 is an enzyme that catabolizes acetaldehyde. People with less effective variants of this enzyme have a transient accumulation of acetaldehyde when alcohol is ingested which leads to aversive symptoms such as facial flushing and tachycardia, hence they are less likely to drink excessively.

Box 26.5.3.3 Some infections associated with psychosis

Syphilis	Human immunodeficiency virus
West Nile Virus	Rabies
Lyme disease	Epstein–Barr virus
Herpes encephalitis	Japanese encephalitis
St. Louis encephalitis	Eastern equine encephalitis
Subacute sclerosing panencephalitis	Brucellosis
Cryptococcus	Mycoplasma pneumoniae
Leptospirosis	Hepatitis B

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6487 Psychological factors Alcohol ingestion is pleasurable for most people and therefore likely to be repeated. Other psychological factors such as depression or anxiety may play a role in the development of alcohol use disorders. A particular association is with social anxiety as those who feel anxious in social situations can ‘self-medicate’ with the often readily available alcohol.

Social factors General social factors including the availability and price of alcohol, as well as the acceptability of drinking are clearly important in determining drinking behaviour. Individual factors such as adversity, family interactions, and peer group influence may also favour the development of alcohol misuse.

Epidemiology In England, the 2007 Adult Psychiatric Morbidity Survey (APMS) estimated the prevalence of hazardous drinking in adults aged over 16 as 24% (33% of men, 16% of women). This included 4% of adults (6% of men, 2% of women) whose drinking could be categorized as harmful. The level of excessive drinking in the United Kingdom is higher than in most other countries of the world. Binge drinking is generally more common in the young and less common in older adults, but can often persist into middle age. In 2012, 3.3 million (or 6%) of all global deaths were attributable to alcohol misuse, making it the fifth leading risk factor for premature death and disability; among people between the ages of 15 and 49, it is the leading factor.

Alcohol harm and dependence Harmful alcohol use is defined as a pattern of excess alcohol use that damages the person’s physical or mental health. Alcohol dependence is defined by meeting three of the following criteria over the past year:

- cravings or compulsion to drink
- tolerance
- withdrawal symptoms
- difficulties controlling use
- neglect of alternative activities

continuation of use despite harmful consequences

Alcohol problems commonly coexist with other

mental illnesses and social problems. A comprehensive assessment of the patient is therefore required, covering all the areas listed in Box 26.5.4.1. Medical consequences of excess alcohol intake Heavy use of alcohol leads to health-related problems in most bodily systems, with the risk of premature death increasing with intakes over 100g per week and substantially increasing at over 200g per week. Liver The liver is the primary site for alcohol metabolism and alcohol is one of the most common causes of chronic liver disease. Excessive alcohol use leads to a spectrum of changes, which range from fatty liver to hepatic inflammation and necrosis (alcoholic hepatitis) to progressive fibrosis (cirrhosis). Only a relatively small number of heavy drinkers progress to liver disease and so genetic and other factors must also play a role. The association between alcoholic liver disease and the polymorphism of genes encoding for alcohol metabolizing enzymes such as ADH, ALDH2, and CYP2E1 is not clear. Although men have higher rates of alcoholic liver disease, women have a greater susceptibility to it. Gastrointestinal With regular, heavy alcohol use a variety of medical complications involving the gastrointestinal tract and related organ systems can develop. Associated oesophageal disorders include oesophagitis, oesophageal varices related to liver disease, and oesophageal mucosal tears. Other common upper gastrointestinal problems are gastritis, duodenitis, and ulcer disease. Alcohol is also a common cause of pancreatitis and the incidence among heavy alcohol consumers is around 3% per year. Cardiovascular There is a well-documented association between heavy alcohol consumption and hypertension. There is an ongoing debate as to whether moderate alcohol consumption has a beneficial effect on blood pressure and cardiovascular risk. Heavy drinking is also a risk factor for arrhythmias, cardiomyopathy, sudden cardiac death, and stroke. Musculoskeletal High alcohol intake is associated with osteoporosis. This is likely to be due to reduced bone formation during ongoing remodelling and perhaps mediated by parathyroid hormone and calcium metabolism. Consuming more than two units of alcohol per day is a risk factor for both osteoporotic and other fractures. In addition, alcohol inhibits repair following a fracture, probably through suppression of synthesis of an ossifiable matrix. Alcohol can cause acute rhabdomyolysis (breakdown of muscle tissue) following a period of more heavy use or in relation to withdrawal. This typically advances over a few hours then recedes as the Box 26.5.4.1 Areas covered in comprehensive assessment Alcohol use, including: – consumption: historical and recent patterns of drinking, and if possible a collateral history from family member or friend – dependence symptoms – alcohol-related problems Other drug misuse Physical health problems Psychological and social problems Cognitive functioning Readiness and belief in ability to change drinking behaviour

SECTION 26 Psychiatric and drug-related disorders 6488 muscles heal. Chronic alcoholic myopathy generally affects proximal muscles and may develop over years. Biopsy shows muscle atrophy affecting predominantly type II, especially type IIB, muscle fibres. The myopathy recovers gradually with a reduced alcohol intake. Pulmonary Alcohol use increases the risk of adult respiratory distress syndrome (ARDS), probably via increasing alveolar epithelium permeability. Alcohol also increases the rates and the morbidity and mortality associated with community-acquired pneumonia, particularly of gram negative organisms. Neurological Alcohol can cause several neurological problems, including: Delirium tremens Delirium tremens may be the presenting picture of alcohol dependence. This extreme reaction to withdrawal from alcohol typically occurs 24–48 hours after cessation. The delirium and is characterized by acute confusion and agitation commonly coexisting with persecutory delusions and hallucinations. It requires hospital treatment and has a mortality of 1–4%. Withdrawal seizures Seizures are a common complication of alcohol withdrawal. These commonly occur 12–48 hours after discontinuing alcohol but may occur up to 10 days after

stopping. They take the form of generalized convulsions. Subsequent electroencephalograms (EEGs) after the seizure and withdrawal period are normal. Alcoholic dementia and cerebral degeneration Cross-sectional magnetic resonance imaging (MRI) studies have shown smaller volumes of both grey and white matter in the cerebral cortex in those with a history of excessive alcohol use. The area of greatest loss appears to be the frontal lobes. Chronic heavy alcohol consumption is associated with several neuropsychological deficits in abstract thinking, memory, learning, attention, and psychomotor skills. These deficits can progress to a dementia. The dementia associated with heavy alcohol use manifests as deficits in executive functioning to short- and long-term memory problems, as well as behavioural abnormalities such as disinhibition, apathy, or irritability.

Wernicke-Korsakoff syndrome Wernicke-Korsakoff syndrome is caused by a deficiency of thiamine in the central nervous system. It has an abrupt onset with an abnormal mental state, ophthalmoplegia (weakness or paralysis of extraocular muscles), and truncal ataxia (ataxia affecting the proximal musculature especially that involved in gait stability). Untreated it may progress to a chronic syndrome with severe anterograde amnesia, retrograde amnesia, and other cognitive deficits.

Cerebellar degeneration Damage to the cerebellum can contribute to deficits of gait and balance in chronic heavy alcohol users. Commonly there is atrophy in the anterior superior vermis.

Peripheral neuropathy Alcoholic neuropathy is caused by a combination of the direct toxic effects of alcohol and its metabolites on neurons and associated nutritional deficiencies. Onset is usually slow and begins with tingling or burning sensations in the peripheries. Later, sensory loss or distal weakness may also appear which progress proximally.

Endocrinological Alcohol has widespread effects on the body's hormonal systems. Alcohol decreases testosterone secretion, which may lead to gynaecomastia, impotence, and testicular atrophy. In women, even moderate consumption can lead to significant reproductive problems by increasing oestrogen, inhibiting follicle-stimulating hormone, and disrupting folliculogenesis. In addition, alcohol increases the release of prolactin in both sexes, which may contribute to some of the aforementioned abnormalities. Both acute and chronic alcohol use activates the hypothalamic-pituitary-gonadal axis. In some drinkers a condition called alcohol-induced pseudo-Cushing's syndrome may develop. This is often clinically indistinguishable from Cushing's syndrome but present more mildly. These symptoms will often disappear within a few months of abstinence. Both acute and chronic alcohol exposure to alcohol diminish serum growth and insulin-like growth factor-1 levels in animals and humans of both sexes. Alcohol also has effects on the thyroid system with thyroid stimulating hormone response blunted to thyrotropin releasing hormone. Alcohol may lead to higher levels of parathyroid hormone by decreasing gut absorption of calcium. Chronic heavy alcohol use may increase insulin resistance in diabetics reflected in higher haemoglobin A1C than matched patients.

Cancer Heavy alcohol use increases the risk of oesophageal cancer around tenfold. It is also associated with an increased risk of oral, pharyngeal, laryngeal, breast, bowel, and liver tumours.

Injuries Alcohol is a cause of accidental injuries in both chronic heavy drinkers and episodic drinkers. Alcohol-impaired driving is estimated to account for a third of all driving fatalities. There is a strong link with accidents involving fires; two-thirds of those who required admission with burns were intoxicated at the time. Alcohol is also implicated in drowning and falls, and is the biggest single cause of accidents in the home.

Alcohol-related complications of pregnancy Although most women cease drinking excess alcohol in pregnancy, alcohol-related birth problems are not uncommon. Birth defects include reductions in weight, height, and head circumference; decreased cognitive abilities and an increased risk of behavioural problems such as attention deficits and impulsiveness. The so-called fetal alcohol syndrome is characterized by growth deficiency, facial dysmorphism, and central nervous system (CNS) disorders. The current

global estimated incidence is about one in 1000 live births in the general obstetric population and

26.5.4 Alcohol misuse 6489 25 per 1000 births among alcohol-dependent women. It is the most common preventable cause of intellectual disability in the Western world. Clinical investigation Investigations may indicate excess alcohol use by showing the effect the alcohol intake has had on the body. A blood count may show macrocytosis, potentially with thrombocytopenia, due to effects on the bone marrow. Liver enzymes, prothrombin time, and bilirubin may all be raised, with a particular elevation in γ -glutamyltransferase. Treatment Alcohol withdrawal The most common situation where a general physician may come across alcohol-related problems is after admission to hospital. The characteristic withdrawal symptoms related to alcohol can be both unpleasant and medically dangerous; they are listed in Box 26.5.4.2. Treatment of withdrawal is usually with benzodiazepines in a fixed progressively reducing dose regimen (see Table 26.5.4.1).

Benzodiazepines ameliorate the imbalance between excitatory and inhibitory neurotransmitter systems, treating unpleasant symptoms, and reducing the risk of complications such as seizure. The doses used may need to be greater where there is severe dependence and lower in patients whose benzodiazepine metabolism may be suboptimal, such as older people and those with liver disease. As an alternative to a fixed reducing regimen, a symptom-triggered regimen may be used; the drug is given according to the symptoms. The revised Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) can be used to monitor withdrawal. Carbamazepine is also used extensively in Europe to manage alcohol withdrawal and is an effective alternative to benzodiazepines. A treatment regimen for outpatients might start at 800 mg in divided doses on the first day, tapering to 200 mg on the fifth day. Delirium tremens The treatment of delirium tremens is with a high-dose benzodiazepine; lorazepam or diazepam are commonly used.

Treatment with antipsychotic agents such as haloperidol or olanzapine may also be required. Refractory delirium tremens treatment may necessitate treatment with general anaesthetic agents, such as propofol, with appropriate airway management. Wernicke's encephalopathy Wernicke's encephalopathy is best prevented by giving prophylactic parenteral thiamine to all alcohol-dependent patients admitted to hospital. If Wernicke's encephalopathy develops, high doses of parenteral thiamine should be given. Psychological treatment of alcohol use disorders Less than 10% of people with moderate to severe alcohol problems receive treatment in a given year and only a third ever receive treatment. The main treatment for alcohol problems is a talking therapy which focuses on supporting the individual to change their behaviour and remain abstinent. The most commonly used intervention for hazardous drinking that is given by physicians is brief simple education and advice. For those with a special interest in alcohol problems an extended brief intervention based on motivational interviewing can be beneficial. The principles of motivational interviewing are listed in Table 26.5.4.2. For those with alcohol dependence, Alcoholics Anonymous (AA) provides the most commonly available group-based therapy. AA is a fellowship for mutual support. The AA programme consists of studying and following the 'twelve steps' which aim to help the alcoholic to achieve a sober way of life. AA also advocates sponsorship: support from another AA member who offers one-to-one guidance to those working through the programme. Pharmacological treatment of alcohol dependence Drugs are only used in combination with psychological treatment, and few patients with alcohol use disorders receive pharmacotherapy, which is intended to reduce the return to drinking in those who are abstinent. Box 26.5.4.2 Symptoms of alcohol withdrawal Autonomic hyperactivity (e.g. sweating, tachycardia, or anxiety) Hand tremors Headache Insomnia Nausea or vomiting Short-lived hallucinations or illusions Psychomotor agitation Grand mal seizures Table 26.5.4.1 Sample alcohol withdrawal regimen for severe dependence Day Dose of chlordiazepoxide 1 30 mg qds 2 25 mg qds 3 20 mg

qds 4 15 mg qds 5 10 mg qds 6 10 mg tds 7 10 mg bd 8 5 mgs bd 9 5 mgs nocte Table 26.5.4.2
Principles of motivational interviewing Expressing empathy Expressing empathy and acceptance
helps build rapport Developing discrepancy This helps the person to see that the addictive
behaviours do not fit with their longer-term aims and aspirations Rolling with resistance Avoiding
getting into arguments, but offering new perspectives helps prevent a breakdown in
communication Supporting self-belief Supporting the person's confidence that they can make
changes makes them seem possible

Revision #1

Created 2026-01-22 16:44:01 UTC by Omar Ayman

Updated 2026-01-22 16:44:01 UTC by Omar Ayman