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Management of alcohol withdrawal symptoms

The current epidemic of alcoholism shows no sign of abating, and with 20-30% of hospital patients currently thought to be excessive drinkers[1 2] doctors are likely to have to deal increasingly with the alcohol withdrawal syndrome.

Dependent drinkers react in different ways to the sudden withdrawal of alcohol, so that even if an individual's drinking habits are known his response may be difficult to predict. Some people seem to be scarcely affected, while at the other end of the scale a few, put at less than 5% of the total, develop the dramatic features of delirium tremens, especially if they are also stressed by infection, injury, or operation. Most display some features of anxiety, agitation, tremor, and sweating, accompanied by fever, nausea, and retching. The general hyperactivity is often associated with varying degrees of insomnia, itching, cramps, hyperacusis and tinnitus, and perceptual disturbances; and both tachycardia and hypertension are common. Confusion, paranoia, visual hallucinations, and convulsions occur in the more severely affected patients. Gross and his colleagues[3] have devised a method of grading withdrawal symptoms by a series of scales, which can be used for comparative studies and for assessing different treatments.

Symptoms begin six to eight hours after the last drink (which explains why the chronic alcoholic steadies the nerves with the early morning drink), reach a peak at about 48 hours, and then subside over the course of a week. Their cause is not known. Increased secretion of sympathomimetic[4] and other amines has been suggested to explain the hyperactivity, and though the adrenal cortex is overactive (as shown by raised plasma concentrations of cortisol) the response to stress seems to be inadequate.[5] The dehydration and oliguria found in the early phase of withdrawal possibly result from the chronic diuresis caused by alcohol or from increased secretion of antidiuretic hormone. Hypokalaemia may be prominent in severer forms of withdrawal.[6]

Management requires sympathetic handling and careful observation. If possible patients should be treated in a general medical ward, even though they tend to be disruptive. The cause of the syndrome should be established, infections such as pneumonia being the most common, and treated vigorously. If necessary an intravenous drip should be set up to combat dehydration and to facilitate the giving of sedatives. For the first day or two additional supplements of 50-100 mmol potassium should be given in divided doses, and injections of a high-potency vitamin preparation are said to be beneficial. Over 100 drugs have been tried in the treatment of withdrawal symptoms,[7] and the choice is often dictated by personal preference. The current favourite in Britain is chlormethiazole[8] in a dose of 500-1500 g every six hours initially; the aim should be to reduce the dose as symptoms disappear and to stop the drug as soon as possible to avoid dependence. Chlordiazepoxide is a useful alternative and may need to be given in large doses at first—for example, 25-50 mg every six hours; cumulative effects can be avoided by reducing the total amount by a quarter each day. Both drugs will suppress anxiety and prevent convulsions. If convulsions do occur chlordiazepoxide is the drug of choice; phenytoin does not act quickly enough. Beta-blocking agents, such as propranolol 40 mg six-hourly, have been used to treat the hyperactivity, but they will not prevent convulsions and do not augment the action of sedatives.[9]

Since the symptoms arise because of the sudden withdrawal of alcohol, treatment is sometimes advocated with small quantities of alcohol. The equivalent of about 8 g absolute alcohol, (half a pint of beer or a glass of spirits) can be given every four to six hours and gradually tailed off; it has the advantage of being safer than drugs and allows the latter to be used in smaller quantities. Alcohol is also a valuable preventive agent in known alcoholics who are undergoing an operation, for example; and it can be tried as a first step in the patient who unexpectedly becomes confused or delirious while in hospital.

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Multiple sclerosis in the Orkney and Shetland Islands and in north-east Scotland

Sutherland reported that multiple sclerosis in 1954 appeared to be more common in the Orkney and Shetland Islands and in Caithness in the north-east of Scotland than in the west of Scotland, and postulated that there might be a disadvantageous genetic factor responsible for this high prevalence.[1] Since then three further studies of the prevalence of multiple sclerosis, based on prevalence days in 1962, 1970, and 1974, have been undertaken in the Orkney and Shetland Islands, the last two by Poskanzer and his colleagues.[2-9] The population of each group of islands had varied slightly between 17 000 and 20 000 people; and the prevalence of ascertained cases of probable multiple sclerosis increased during 1954-74 from 82 to 258 per 100 000 (from 17 to 45 patients) in the Orkney Islands and from 118 to 152 per 100 000 (from 22 to 28 patients) in the Shetland Islands. Other studies have also shown that repeated