disease onset is most likely to have occurred. Longstanding evidence that fetal exposure to medical radiation is a risk factor,[5] together with the report by Gardner and his colleagues[6] linking risk to paternal occupational exposure to radiation immediately before conception, suggest that residence at birth (or before birth) may be more relevant than residence at the time of registration or death. A recent study of the cases arising in children living around Dounreay,[7] however, found that the risk was no higher in the children born in the area than in those moving there after birth.

Has the large amount of research money that has gone into the epidemiology of this small but unquestionably important problem been justified? Certainly it has stimulated the development of new statistical approaches to spatial analyses, which may be applicable to other circumstances. It has also strengthened the validity of what was previously weak evidence on the presence of clusters of cases, even if we have learnt little more about the effects of environmental radiation. Considerably more research is needed, probably on an individual basis, before the causes of these devastating diseases are fully understood.

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Alcohol and cardiac arrhythmias

Patients with unexplained tachyarrythmias should be questioned about their drinking

Heavy drinking increases the risk of cardiac arrhythmias whether or not heart disease is present.[1-8] The evidence has come from clinical observations,[1,2] retrospective case-control studies,[3] controlled studies of consecutive admissions for supraventricular tachyarrhythmias,[4,5] and prospective epidemiological investigations.[6] Finally, electrophysiological studies have shown that alcohol can facilitate the induction of tachyarrhythmias in heavy drinkers with a history of alcohol related heart irregularities.[7,8]

The potential of alcohol misuse to induce arrhythmias is best established in atrial fibrillation. Patients (especially men) admitted for idiopathic atrial fibrillation have been shown to have consumed more alcohol during the week preceding their admission than either control patients[4] or the population outside hospital.[5] In one study 60% of patients with idiopathic atrial fibrillation coming on at the weekend were problem drinkers, according to the CAGE questionnaire,[9] compared with only 12% in the community.[10] The association of alcohol with other types of supraventricular tachyarrhythmias is less clear,[11] but in the Kaiser Permanente study people consuming more than six drinks a day had a higher risk of all supraventricular tachyarrhythmias than people matched for age, sex, race, and smoking but consuming less than one drink a day.[6] Finally, alcohol has occasionally also been shown to promote the onset of ventricular tachyarrhythmias.[1,8]

How might alcohol promote arrhythmias? Subclinical alcoholic disease of the heart muscle may contribute by producing patchy delays in conduction, and potassium and magnesium depletion may also be factors,[1] as may the hyperadrenergic state accompanying alcohol withdrawal. When prolonged alcohol misuse is halted the adrenaline and noradrenaline concentrations in the blood are raised and the density of β adrenoceptors in lymphocytes, which had been reduced, rapidly returns to normal.[12] Studies in animals have shown similar reversal and even an overshoot of the number of β adrenoceptors in the heart after withdrawal of alcohol.[13] And alcohol has an acute electrophysiological action which is also likely to be important: in some heavy drinkers arrhythmias are induced easily after two to three whiskies but not in the sober state.[7,8]

Alcoholics commonly die suddenly and unexpectedly, but the cause of death often remains unexplained even at necropsy.[14] There may be more than one fatal path of events, but primary ventricular arrhythmia culminating in fibrillation ranks high among the possibilities. In one study of victims of sudden death 26% of men under 50 had a history of heavy drinking and myocardial changes compatible with chronic alcoholic injury.[15] Decreased variability of heart rate—a sign of cardiac vagal neuropathy and a factor notorious for increasing the risk of death after myocardial infarction—is a relatively common finding among men dependent on alcohol.[16] By contrast, however, regular modest drinking seems to be associated with a reduced risk of primary cardiac arrest in people without prior cardiovascular disease.[17]

There are no good data on the management of alcohol related cardiac arrhythmias. In our experience most episodes terminate within 24-48 hours either spontaneously or after treatment with β blockers combined with adequate sedation, rehydration, and treatment of any potassium and magnesium depletion. Treatment of the underlying alcohol misuse must not be neglected. In an attempt to help identify covert alcoholism we recommend the administration of the CAGE questions to all patients presenting with otherwise unexplained acute tachyarrhythmias.[9] Studies in people who were not alcoholics have shown that a moderate intake produces varied and partly conflicting changes in impulse conduction and in the number of premature beats. The effects of social drinking on the frequency, severity, and inducibility of clinically important arrhythmias have not been properly studied. Advice to patients who are not alcoholics should therefore be based on clinical judgment and on the understanding that arrhythmogenic effects of alcohol seen in heavy drinkers may not hold for social alcohol consumption.

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Percutaneous endoscopic gastrostomy

The end of the line for nasogastric feeding?

Too often in medicine the sheer enthusiasm generated by technological advance has overtaken proper clinical evaluation. Although the randomised controlled clinical trial is one of the glories of medical science, there is an increasing trend for radical new treatments to be adopted as standard without being subjected to such critical scrutiny.

The technique of percutaneous endoscopic gastrostomy was introduced in 1980 to provide enteral nutrition[1] and became commonplace in North America though less so in the United Kingdom. Although many uncontrolled series have reported its efficacy,[2-4] we have had to wait until now for a controlled trial of the procedure (p 1406).[5]

Park and colleagues compared the success of an endoscopically sited percutaneous gastrostomy with a conventional nasogastric tube in establishing enteral nutrition in patients who had neurological swallowing disorders. Over four weeks, feeding was sustained in all the patients randomised to gastrostomy but in only one of 19 patients in whom nasogastric feeding was attempted. The dismal results for the nasogastric route will not surprise nurses and junior doctors familiar with the problem of tubes that are difficult to site, become displaced, or are pulled out or blocked. In this study these complications occurred in 16 patients in whom nasogastric tubes were used, and in two further instances patients simply refused to continue with this treatment.

Percutaneous endoscopic gastrostomy was developed as an alternative to surgically created gastrostomy, thus avoiding an operation under general anaesthesia in patients who are often frail and elderly. Operative gastrostomy may be hazardous even in skilled hands and has a substantially greater morbidity and mortality than the endoscopic procedure.[6] Advantages of endoscopic gastrostomy include the need for just local anaesthesia, a short procedure time (20 minutes or so), the avoidance of using an operating theatre, and a reduced cost.

The technique of endoscopic gastrostomy should be within the competence of most accomplished gastrointestinal endoscopists, who, over the years, have become accustomed to carrying out ever more complex interventional procedures.[7] Two operators are required. The first passes the endoscope by mouth into the stomach. The site for insertion of the gastrostomy tube is identified by transillumination from within as the stomach is closely applied to the anterior abdominal wall. The second operator passes a cannula through the skin into the stomach and guides a thread into the gastric lumen which is then grasped by the endoscopist using forceps and then drawn back through the mouth. The gastrostomy tube is tied to the thread and is then pulled through the mouth, into the stomach, and back out through the abdominal wall, where it is secured in place. The tube should not be used for 12 hours. If water passes freely an enteric feeding regimen may be started 6-12 hours later.

Some patients are unsuitable for endoscopic gastrostomy. The procedure should be avoided in patients with portal hypertension and ascites, those predisposed to bleed or taking anticoagulants, and those with active gastric ulceration. Previous abdominal surgery may present formidable technical problems in inserting the tube. Apart from the possible complications of upper gastrointestinal endoscopy, the procedure is associated with certain special problems—the most serious of these being peritonitis. This usually presents within the first 24 hours but is uncommon. Antibiotic prophylaxis may lessen the risk, and peritonitis was seen in only 1% of patients in a recent large series.[8] More commonly, superficial infection may surround the tube site, and the tube may become displaced. These complications occurred, respectively, in 6% and 4% of patients.[8]

The main indication for enteral nutrition is difficulty in swallowing in patients with neurological disease. Pulmonary aspiration is a serious hazard for those who are unable to protect their airway, yet it is precisely these patients in whom some form of nutrition must be established. Aspiration is well recognised with nasogastric feeding and may be reduced but certainly not abolished by percutaneous gastrostomy.[9,10] Of the 19 patients with neurological dysphagia studied by Park *et al*, two developed aspiration pneumonia.[5] In another study, however, none of 30 patients with a range of neurological lesions developed pneumonia during 6-12 months of feeding by gastrostomy.[8]

Most importantly, however, the clear superiority of gastrostomy feeding over the nasoenteral route is in allowing patients to receive sustained nutritional support. Successful tube insertion, which occurs in more than 95% of cases in large series, improves body composition and nutritional status with sustained weight gain being the rule during follow up of a year or more. Many patients who were malnourished when they were switched from nasogastric tube to gastrostomy experienced considerable improvements in body mass index within a month of receiving adequate nutrition.[6]

Long term nasogastric feeding may result in nasopharyngeal sepsis and erosion of the oesophageal wall,[11,12] and it is unpleasant for patients. The implication of Park and colleagues' study is for those patients who, instead of a nasogastric tube, could benefit from a gastrostomy. Although most gastroenterologists should be able to learn the technique,

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