generally available is etidronate, which is effective in malignant hypercalcaemia, though it is not useful in acute states because of a delay in the onset of its effect.¹⁸ The disadvantage of etidronate is that it may cause impairment of bone mineralisation, which limits its dosage and duration of usage.¹⁹

In the short term all these drawbacks may not be important. The cytotoxic antibiotic mithramycin has a calcium lowering effect, but again this is not immediate.²⁰ Its hepatic, renal, and platelet toxicity largely precludes its use, particularly when the patient may later need other cytotoxic regimens. As a last resort additional measures such as haemodialysis or even a single infusion of phosphate may be considered. In the longer term specific antitumour treatment should be given, and measures for lowering calcium concentrations should be continued until they are satisfactory. Antiresorptive treatment should be continued with either subcutaneous calcitonin, with or without glucocorticoids as necessary, or etidronate.

Future hopes for the management of malignant hypercalcaemia lie in two directions. Firstly, new diphosphonate compounds are under development which have effective calcium lowering properties while not interfering with bone mineralisation.^{21 22} These are likely to prove of great value in long term management. Secondly, the development of specific parathyroid hormone antagonists²³ may well provide a new dimension by counteracting any renal component of the disease.

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The case for compulsory pasteurisation

In August 1983 compulsory pasteurisation of milk was introduced in Scotland, leading to a fall in milk borne infections. Meanwhile, in England and Wales there was no change in legislation and, as Sharp and his colleagues show (p 463), no fall in the number of infections. This amounts to a convincing case for compulsory pasteurisation throughout Britain.

Milk borne salmonellosis was particularly serious in Scotland during the 1970s and early 1980s, when 50 outbreaks affected at least 3518 people and caused 12 deaths. In addition, there were several major outbreaks of campylobacter infection. The costs of one salmonella outbreak were estimated to lie between £236 000 and £3 222 000 and the mid-range average cost for each patient was £2637.¹ But after compulsory pasteurisation, during 1983-4, eight outbreaks of milk borne salmonellosis affected only 46 people—all in dairy farming communities. No outbreaks were reported from the general community, whereas 14 general outbreaks had affected over 1090 people in the previous three years.

In 1983 two household episodes of campylobacter enteritis were attributed to raw milk. One of these happened before compulsory pasteurisation, and the other was in a family who had returned from a holiday in England. In 1984 there were no outbreaks of campylobacter enteritis.

Only temporary exemptions from pasteurisation are granted to a few small dairy farms on outer islands, which produce less than 0.02% of Scottish milk. Concern remains only over dairy farm staff and their families being given free raw milk.

In contrast to Scotland, in England and Wales during 1983-4 some 20 outbreaks of milk borne salmonellosis affected at least 518 people. Twelve of these outbreaks occurred in the general community in people who had bought raw milk direct from dairy farms. In the same years there were six proved outbreaks of campylobacter enteritis and 10 other suspected outbreaks without sufficient microbiological and epidemiological evidence to prove the diagnosis.

Although less than 3% of the 6000 million litres of milk sold annually in England and Wales is untreated, half of this is produced in the north west, where 70% of the 1983-4 outbreaks occurred. Legislation is expected in November 1985 that will reduce the availability of untreated milk from shops—but it will not forbid direct "farm gate" sales or local retail deliveries of farm bottled raw milk. At present legislation covers only cows' milk, but the milk of goats and sheep should be included as soon as possible. To reduce still further the risks of milk borne infection, farm water supplies need to be better controlled and pasteurisation plants must be well maintained, thoroughly cleaned, and efficiently operated. Failures are sometimes due to insufficient pasteurisation or to contamination of pasteurised milk by raw milk or dirty containers. But these are minor matters compared with the need for compulsory pasteurisation throughout Britain of all animal milk intended for human consumption.

Passionate opponents of compulsory pasteurisation are likely to remain unconvinced for ever since they rely on feelings rather than evidence. Often these arguments seem to rest on a desire not to go against what nature intended. It might be argued that nature never intended man to drink

animals' milk anyway, but these arguments count for little in the face of continuing milk borne infections being a costly nuisance and sometimes leading to death.

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Anaesthesia and alcohol

Alcoholism exists at every socioeconomic level, in both sexes, and at most ages. Its prevalence is increasing, and the close association between drinking, related pathological disorders, and acute trauma ensures that large numbers of alcoholics present for emergency surgery. It has long been recognised that alcoholics may be difficult to anaesthetise and that they provide additional intraoperative and perioperative problems. Clearly, early recognition is essential for correct management.

Preoperative assessment of every adult should include direct questioning about smoking and intake of drugs and alcohol. An explanatory rather than moralising approach is more likely to yield the truth. The suspected drinker should be pressed about nutrition, dyspepsia, insomnia, depression, trembling, and cramps. Special note must be made of the facial appearance (plethora, thickening, and so on), the tongue, the voice, the nature of any tremor, blood pressure, and any hepatic tenderness or enlargement. Some French clinicians use the "Le Go" table, into which these specific findings are inserted in an attempt to obtain a more statistical and accurate assessment by a score.¹ Specific haematological investigations have been explored, but none is foolproof. A rise in the mean red cell volume above 93 fl occurs in 85% of heavy drinkers, but there are many clinical red herrings. γ -Glutamyltransferase, transaminase, and other enzyme activities may be abnormal, but results are not specific for alcoholism and may be positive only with cirrhosis or intoxication. A diplomatic approach to a friend or relative may produce much more useful information.

For major surgery alcoholics are at special risk through a diminished adrenocortical response to stress, impaired host defence mechanisms, bone marrow depression, electrolyte and fluid imbalance, abnormal blood glucose concentration, liver disease, cardiomyopathy, drug cross tolerance, and the alcohol withdrawal syndrome. Time allowing, therefore, examination should include measurement of the blood alcohol concentration, a full haematological profile including coagulation studies, urea and electrolyte values, blood glucose concentration, blood gas tensions, an electrocardiogram, and a chest radiograph. Full liver function tests (in emergencies) may be of use only retrospectively.

Clinical examination must include the central nervous system to exclude obvious psychosis (showing cortical damage) and peripheral or autonomic neuropathy. Cardiomyopathy must be suspected and may cause poor exercise tolerance, palpitations, or heart failure. Conduction defects, classical bifid or cloven T waves, digitalis-like ST segments, and arrhythmias may all be seen in the electrocardiogram.² The chest x ray film may confirm evidence of infection or inhalation. A tender, palpable liver usually indicates fatty

infiltration, but the classic features of cirrhosis must be sought.

Even in the absence of liver disease there may be megaloblastic anaemia, neutropenia, and thrombocytopenia due to impaired folate absorption or direct marrow depression.³ Cirrhosis may compound the problems by causing a reduced prothrombin time. Electrolyte concentrations in alcoholics are variable. There may be dehydration, but diuresis occurs only while ethanol concentrations are rising and the alcoholic may be overhydrated if "drying out."⁴ A reduced serum potassium concentration is more constant. Hypoglycaemia in the "hangover" period is also common, and anaesthesia is often required at this time. The natural hyperglycaemic response to anaesthesia should not be relied on to correct this deficit.⁵

The alcohol withdrawal syndrome is characterised by tremulousness, sweating, gastric distress, and weakness and often starts within eight hours of abstinence—and, again, surgery will often be performed at this time. More severe features include hypertension, hyperreflexia, hallucinations, and seizures; these may occur after a delay of up to five days for severe delirium tremens. Physiologically there is increased catecholamine activity associated with tissue hypoxaemia, respiratory alkalosis, and hypokalaemia.⁶

Surgical preparation for the suspected or confirmed alcoholic should include parenteral administration of vitamins, particularly thiamine, pyridoxine, and nicotinic and pantothenic acids. Correction of coagulation abnormalities with vitamin K, fresh frozen plasma, or platelets may be necessary.

A preoperative infusion of 8% weight for volume ethanol in isotonic saline at 0.5 g/kg over 15 minutes has been shown to reduce excitability of the central nervous system and catecholamine mediated effects, and to lower analgesic requirements during the operation, but it has no effect on the underlying diminished adrenocortical response to stress. For this reason both perioperative infusion of alcohol and administration of steroids have been proposed.^{7,8}

Premedication must be given with particular reference to the patient's level of excitation, liver disease, and so on. Alcoholics are generally believed to require greater induction doses and higher maintenance levels for anaesthesia. Definitive studies on minimal alveolar concentration are lacking, however, and the exact parts played by non-specific resistance, cross tolerance, and enzyme induction are ill understood.⁵ Larger doses of thiopentone are often required but may be dangerous in the presence of cardiomyopathy or hypoalbuminaemia—when less barbiturate will be bound to protein. With the increased incidence of chronic gastrointestinal upset and high gastric acidity it may be wise to perform a "crash" induction. Transient hyperkalaemia may result from the administration of suxamethonium in myopathy, and in cirrhosis relaxation may be moderately prolonged. Cirrhosis may also affect the biotransformation of non-depolarising relaxants, with the exception of atracurium.

Maintenance of anaesthesia will depend on the patient's general condition and the nature of the surgery. Volatile agents which appreciably depress the myocardium must be avoided in patients with suspected cardiomyopathy, when fluid balance and cardiac function should be monitored closely. Anaesthesia may precipitate hypotension in patients receiving disulfiram owing to abnormal concentrations of noradrenaline and dopamine.³

The newer, less biotransformed volatile agents are to be preferred in patients suspected of having liver disease.