case 2 the acidosis was compensated for by hyperventilation to correct the arterial hydrogen ion concentration, but presumably cerebral acidosis stimulated this. A poor correlation between hydrogen ion concentration in the blood and cerebrospinal fluid has been described in acetazolamide-induced acidosis. Recovery from the acidosis coincided in both patients with clinical recovery.

There was clinical, haematological, and radiological evidence of pre-existing renal failure in the patient in case 1, and direct biochemical evidence of pre-existing renal failure in the other patient. Acetazolamide treatment was associated with worsening acidosis only and little or no deterioration in other aspects of renal function and no evidence of acute renal failure. The acidosis in the second patient may have precipitated the attack of pyrophosphate arthropathy by reducing the activity of synovial alkaline phosphatase, which may metabolise pyrophosphate.

This has not been reported, though a case of acute gout (the type of crystal was not confirmed) after treatment with acetazolamide has been reported. Acetazolamide normally produces a mild self-limiting acidosis, but a few cases of serious acidosis have been reported in elderly patients, diabetic patients, and patients with renal disease. One patient who had liver damage in addition to acidosis died.

Acetazolamide is excreted by the kidneys, and the frequency of the dose should be adjusted for patients in renal failure. In short-term studies of acetazolamide treatment in renal failure definite retention of hydrogen ions was found in patients with urea concentrations that were comparable to those in our patients. In the only long term study of acetazolamide treatment in renal failure a serious acidosis frequently developed. It is not clear how the acidosis develops in these patients. It has been suggested that in some patients with renal failure excessive bicarbonate may be lost by the tubes. Acetazolamide would be expected to inhibit the partial reabsorption of this bicarbonate.

The manufacturers of acetazolamide warn that it should not be given to patients with "idiopathic renal hyperchloreamic acidosis," but few doctors would relate this obsolete phrase to their elderly patients with mild, acquired renal failure. Many elderly patients have unrecognised mild renal failure, and we recommend that before treatment is started with acetazolamide urea and electrolyte concentrations should be measured—as was indeed suggested when acetazolamide was introduced. If renal function is impaired then further estimations should be carried out and the bicarbonate concentration watched carefully. If this should fall to 20 mmol(mEq)/l or below, oral sodium bicarbonate may be given or, preferably, acetazolamide should be discontinued.

References


What might cause excessive belching? Can air entrapment in the oesophagus cause difficulty in swallowing or cardiac extrasystoles? What treatment is advised?

Excessive belching can result only from abnormal entry of air into the oesophagus or stomach. Fermentation in the stomach is not enough, and intestinal air does not travel backwards. Entry is by aerophagy, in which the upper oesophageal closing mechanism is opened and negative intrathoracic pressure sucks air into the oesophagus. Only rarely does it enter the stomach, and probably only when hiatal herniation can occur. The air is expelled by contraction of the thoracic cage. The stimulus to aerophagy is a sensation of abdominal distension that is a paraesthesia, not a reality, and the patient tries to help up the phantom gas, inadvertently letting it in first. Any obstruction in the oesophagus may produce a sensation of an "air lock" or air entrapment that is not a reality. Occasionally, air swallowed with a bolus will pass down through a stricture ahead of the bolus, which plugs the hole momentarily. Many patients with an obstructed oesophagus from stricture or achalasia belch a small bubble after each swallow. Air is not entrapped above an obstruction because it can be instantly released into the mouth, nor is it entrapped in aerophagy. Extrasystoles may cause a sensation that is misinterpreted as wind needing to come up, leading to aerophagy. There is no good evidence that aerophagy or oesophageal obstruction causes extrasystoles. Treatment is to remove the stimulus to aerophagy. Sometimes holding the chin on to the chest stops the aerophagy sequence because it is then difficult to open the upper oesophageal closing mechanism.—D A W EDWARDS, reader in gastroenterology, London.

Correction

Lithium treatment and preoperative fluid deprivation

An error appeared in this article by Professor M Schoo (7 November, page 1253). The lesson should have read: If patients with lithium-induced polyuria must abstain from drinking before an operation because of the narcotics they should be given intravenous fluids the night before the operation.

ABC of Alcohol: Alcohol in the body

We regret that an error occurred in this article by Drs J B Saunders and A Paton (21 November, p 1380). In the table showing the alcohol content of and peak blood concentration after various drinks the peak blood concentration after 12 single measures of spirits should have read 240 mg/100 ml, not 140.