Antacids for duodenal ulcer?

The past 20 years has seen the introduction of a series of drugs which are potent inducers of ulcer healing. Do antacids now have any place in treatment? In Britain they have mainly been used in modest doses as symptomatic remedies, whereas in the United States, with the American practice has been to use high doses to try to promote ulcer healing. Are these differences in prescribing patterns justified? Two recent publications (largely overlapping in content) attest to the continuing interest in antacids and contain useful reviews of current data.

High doses of magnesium-aluminium antacids were clearly shown to promote healing of duodenal ulcers in a study in the United States reported five years ago.3 The findings in that study supplied a possible reason for the apparently higher natural healing rate of duodenal ulcer in the United States than in Britain, though it seemed likely that only an enthusiasm bordering on the obsessional would make people take the regimen needed—30 ml seven times daily of a magnesium-aluminium antacid providing 1008 mmol of neutralising capacity. If there were advantages over H₂-antagonist treatment they did not lie in convenience, or in cost, which was at least as high, or in freedom from short-term adverse effects—many takers of antacids suffering from troublesome diarrhoea—or in reduced relapse rates later.4

Since then antacids in doses ranging down to 280 and 200 mmol daily5,6 have been shown to promote healing. Direct comparison based on the overall neutralising power of antacids between results obtained in different places and with different antacids may be dangerous, but the apparent value of relatively low-dose antacid treatment (which has little effect on intragastric pH) is surprising. So far there have been no comparable studies conducted in Britain.

Attempts to show that antacid treatment gives relief from symptoms have generally failed. In the United States a carboxymethylcellulose placebo proved as useful as a potent antacid in relieving symptoms,7 a finding in agreement with much other data. In one Scandinavian study8 low doses (280 mmol daily) of antacids were found to promote ulcer healing but to have only relatively weak symptomatic effects. Although radically different findings can be obtained between sets of data, even within those obtained in two places in a single study,9 it seems unlikely, taken overall, that failure to show that antacids will relieve symptoms simply stems from methodological difficulties. Quite why antacids should not give simple symptomatic relief if they will promote ulcer healing is hard to understand.

At first sight the evidence is such as to persuade the physician to abandon the use of antacids in treating symptoms but to prescribe them enthusiastically in trying to heal ulcers. If this were done, however, what substitute could be found to manage symptoms? Antacids in ordinary small doses are cheap and safe; and whether they relieve symptoms or whether they act simply as placebos may not be of critical importance provided that they satisfy patients, which in most instances they seem to do. In contrast, histamine H₂-antagonists provide excellent symptomatic relief and, whatever clinicians may wish, are often used as "on-demand" treatment for short periods by patients who have quantities left over in drawers and bathroom cupboards. So far the safety record of H₂-antagonists has proved remarkably good, and such self-treatment seems unlikely to do any harm. Nevertheless, the rational policy is to reserve H₂-antagonists for use in a coherent planned manner where symptoms prove resistant to simple, time-honoured remedies.
If antacids are used then some basic facts need to be remembered. Not only sodium bicarbonate but also magnesium trisilicate mixture and the proprietary compound Gaviscon contain large amounts of sodium—which could precipitate heart failure in predisposed patients. Calcium salts, at least in large doses, can cause hypercalcaemia, and, as calcium ions stimulate release of gastrin, can cause rebound hypersecretion. Magnesium and aluminium salts often bind other drugs, reducing the rate and extent of absorption of cimetidine, digoxin, indomethacin, and levodopa among others,² and in large doses used intensively they can bind phosphate, precipitating osteomalacia.

Given in modest doses magnesium-aluminium antacids remain safe, cheap adjuncts to the treatment of duodenal ulcer, even if in the long term they have little effect on the behaviour of the disease.

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4 Centre for Ulcer Research and Education. Is duodenal ulcer recurrence more common after cimetidine treatment? Gastroenterology 1980;78: A1152.

Hazards of lumbar puncture

As more non-invasive techniques have become available to supplement the clinical management of patients with neurological disorders older techniques should be reappraised. Though lumbar puncture was introduced by Quincke in 1891,¹ the cellular and chemical abnormalities and the changes in hydrodynamics were fully documented only in 1937 by Merritt and Fremont-Smith in their classical monograph.²

In many diseases the “routine” lumbar puncture of old is now superfluous. It is seldom indicated in strokes, since computed tomography will show infarction, haematomas, angiomas, and tumours which may mimic stroke. In suspected compression of the cord an elective myelogram is preferred and has rendered Queckenstedt's test obsolete, except in those hospitals without access to adequate neuroradiology. It is contraindicated in suspected cerebral tumour, abscess, and subdural haematoma.

The routine and generally safe practice of lumbar puncture in cases of suspected spontaneous subarachnoid haemorrhage should also be reassessed. It provides an accurate index of bleeding into the subarachnoid space in all but the exceptional patient in whom the puncture is performed within the first four hours, at which time the blood may have failed to reach the lumbar sac. Computed tomography will show intracranial bleeding and localise the lesion in many cases. The frequency of positive findings varies between 50%, and 100% in different series.³⁶ In a recent survey of 74 cases of proved subarachnoid haemorrhage seven patients showed a dramatic deterioration in conscious level and neurological state after lumbar puncture, and four subsequently died, three showing evidence of cerebral dislocation and tentorial or tonsillar coning. They had all had a lumbar puncture within 12 hours of the event, none had had a computed tomographic scan. In this series computed tomography was performed in 64 cases. Where an aneurysm or angioma was present unenhanced computed tomography showed blood in 59 patients examined within three days. Thus there are rare instances of negative scans in patients with definite intracranial haemorrhage. Nevertheless, those who deteriorated dramatically after lumbar puncture were shown to have haematomas causing coning. In most instances of suspected intracranial haemorrhage computed tomography should be the primary investigation. In those patients in whom a haematoma is not shown and in whom there is no hydrocephalus and no bleeding into the ventricles or basal cisterns, it is safe to do a lumbar puncture. Lumbar puncture is especially important to exclude pyogenic meningitis, which may simulate acute subarachnoid bleeding. Lumbar puncture is contraindicated in a patient with hemiplegia or one who is stuporose or comatose, for these signs suggest the presence of a haematoma with a risk of coning.

Lumbar puncture remains a necessary tool for three main indications. Firstly, to obtain information about the cellular, chemical, microbiological, and pressure changes in many neurological diseases. It is, for example, helpful in the Guillain-Barré syndrome and multiple sclerosis, and is essential in the diagnosis of bacterial, fungal, or viral meningitis. Secondly, it is a necessary route for the injection of radio-opaque media, radioactive agents, and air for the diagnosis of certain cranial and spinal lesions. Thirdly, it remains a route for treatment—for example, antibiotics and antitumour drugs.

The perennial problem of lumbar puncture headache⁷⁸ has recently been reviewed.⁹ The headache is generally attributed to leakage of the cerebrospinal fluid through the hole in the dura, which leads to intracranial hypotension with traction on pain-sensitive nerve endings in the dura and intracranial vessels. If a small needle is used and puncture is performed with the needle bevel in the vertical plane of the dural fibres lumbar puncture headache may be reduced. Epidual blood patching may be followed by complications and is not recommended for routine use for prevention of headache.⁹ The routine of laying the patient flat for 24 hours after puncture is still widely practised, yet anecdotal experience of venereologists performing lumbar puncture in ambulant outpatients suggested that this was not necessary.

Two recent studies¹⁰¹¹ have suggested that the incidence of headache may be reduced by tilting the patient head down, thereby reducing the cerebrospinal fluid pressure in the lumbar sac and decreasing the amount of cerebrospinal leakage. Nevertheless, Hilton-Jones and his colleagues randomly allocated 76 inpatients in whom lumbar puncture was performed for diagnosis into four groups: those treated tilted and supine, tilted and prone, horizontal and supine, or horizontal and prone for four hours after the procedure, after which they were allowed to get up. No significant difference in the incidence of headache was found among the four groups, though the lowest incidence (23%) was in those treated horizontal and supine. These findings parallel the results of another investigation,¹² which showed no beneficial effect of 24 hours’ bed rest after lumbar puncture. Although these