The dose of lithium carbonate was increased to 1·2 g and 1·6 g on alternate nights. Two years of unusual calm followed, but after further fits in December 1977 his phenytoin dose was increased to 400 mg daily.

In November 1978 he complained of increasing thirst over the previous two years and polyuria from a much increased fluid intake. He also had a pronounced tremor, which was placing his job in jeopardy. His skin was dry and warm. He was neither depressed nor elated but naturally anxious. Serum concentrations were as follows: lithium 0·8 mmol/l (0·56 mg/100 ml); phenytoin 18 mg/l (toxic range 20-30 mg/l); and thyroid stimulating hormone 4 mU/l. The free thyroxine index on 14 November was 63 and two weeks later 28. Lithium treatment was stopped, and the tremor, polydipsia, and polyuria had disappeared a month later; but by February 1979 the patient was again taking lithium and all his symptoms returned. As an experiment it was decided to change his treatment from phenytoin to carbamazepine 100 mg thrice daily. Five weeks later he felt completely normal for the first time in years. Shortly after changing from phenytoin to carbamazepine his thirst, polyuria, polydipsia, and tremor had disappeared. Spontaneously he added that coming off phenytoin had increased his sexual desires, which were now quite normal. Since there was no clinical evidence of hypothyroidism it was decided to wait to see whether the free thyroxine index improved. It had reverted to a normal concentration of 60 by June 1979.

Comment

In a similar case to this one the lithium treatment and not phenytoin was stopped.[1] My case, however, shows that the phenytoin was causing the lithium-type toxicity. When it was stopped not only did the polyuria, polydipsia, and tremor disappear and the free thyroxine index revert to normal but the patient commented that his libido had returned. Seemingly, in the presence of phenytoin lithium salts at serum concentrations accepted as standard in treating manic-depressive disorder have toxic effects on the renal tubules, thyroid metabolism, and central nervous system centres related to tremor and libido. Reisberg and Gershon,[2] discussing the management of the side effects of lithium, advised caution when there was a history of seizures. But the suggestion that careful monitoring of clinical symptoms and blood concentrations is the best way to minimise toxic symptoms is true only if the emphasis is more on the first than on the second measure.

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Effect of cimetidine on lower oesophageal sphincter pressure in oesophagitis

The lower oesophageal sphincter is one of several factors that prevent abnormal gastro-oesophageal reflux. Eastwood *et al*[1] showed that damage to the oesophageal mucosa from acid infusion interfered with this sphincter's function and decreased its pressure for a long period.

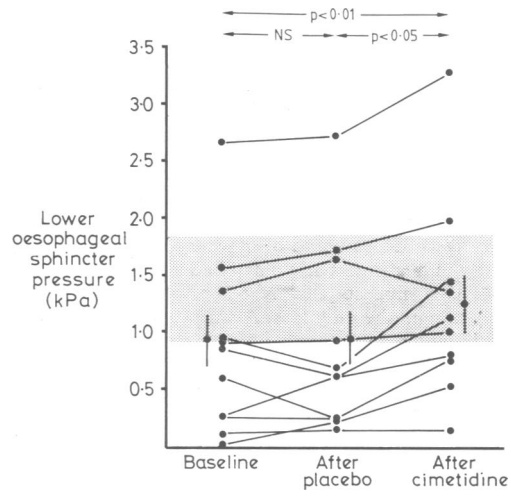
The introduction of cimetidine, a potent inhibitor of gastric acid secretion, has allowed the obverse to be examined—that is, whether suppression of acid reflux in patients with oesophagitis allows the lower oesophageal pressure to increase.

Patients, methods, and results

Informed consent was obtained from seven men and three women (aged 30-61, mean 50·3). All had symptoms of gastro-oesophageal reflux. Fibre-optic endoscopy and biopsy confirmed oesophagitis in all 10 patients. In a randomised, double-blind, crossover trial the patients were given either cimetidine 200 mg or identical placebo tablets three times daily and twice nightly for two six-week periods. On entering the trial and at the end of each six-week period the patients had their reflux symptoms scored on a 0-6 scale (0-3 for both heartburn and regurgitation). At the same time they underwent lower oesophageal manometry and prolonged pH recording as described.[2] The tablets were stopped six hours before these tests.

Statistical analysis was performed with Wilcoxon's signed rank test. The data were also analysed for any carry-over effect; none was detected.

In seven patients the symptoms of reflux improved after cimetidine, whereas in three they were slightly worse. There was a reduction in the mean symptom score from 2·4±SEM 0·5 at the beginning of the trial and 2·1 ±0·5 after placebo to 1·1±0·5 after cimetidine. This reduction was not statistically significant. There was no significant difference between the mean values for lower oesophageal sphincter pressure recorded initially and after placebo (0·93±0·23 kPa and 0·95±0·24 kPa). After cimetidine the mean pressure increased significantly to 1·24±0·25 kPa ($p<0·01$ for difference from pretrial value, $p<0·05$ for difference from placebo; figure). There was no significant change in the mean total length or mean subdiaphragmatic length of the sphincter. Mean distal oesophageal pH showed no significant change throughout the trial. The duration of reflux over the 12-hour test was 86±23 minutes initially, 69±9 minutes after placebo, and 106± 26 minutes after cimetidine. Similarly the number of reflux episodes was 31±7 initially, 26±5 after placebo, and 29±6 after cimetidine.



Lower oesophageal sphincter pressures in 10 patients with oesophagitis recorded on entry to trial and at end of each course of treatment. Means ±SEM are shown beside each column. Shaded area shows normal range for our method of manometry. NS=not significant.
Conversion: SI to traditional units—Pressure: 1 kPa≈7·5 mm Hg.

Comment

Cimetidine had no significant effect on the pressure of the normal lower oesophageal sphincter.[3] In patients with reflux oesophagitis, however, cimetidine provides symptomatic benefit[4] and improves the endoscopic and histological appearances.[5] Neither of these two studies, however, showed any effect on the lower oesophageal sphincter pressure despite a longer course of cimetidine at a higher dose than we used.

Although we have shown an increase in mean lower oesophageal sphincter pressure after cimetidine, this increase was only 0·3 kPa. The pretrial and post-placebo mean pressures were at the lower limit of normal for our method (1·85-0·92 kPa), with six individual values below this limit. After cimetidine the mean pressure was well within the normal range. We were unable to show a concurrent reduction in reflux as measured by prolonged pH monitoring.

Our observation that cimetidine, which decreases gastric acid secretion, led to an increase in lower oesophageal sphincter pressure in patients with oesophagitis, however, supports the experimental observation that acid-induced oesophagitis interferes with sphincter function and thereby creates a vicious circle of oesophagitis.[1]

Cimetidine may provide the potential for breaking this vicious circle, but unless this improved sphincter pressure can be shown to result in decreased reflux of acid, as measured by pH monitoring, the clinical value of our observation remains to be proved.

We are grateful to Smith Kline and French Laboratories Ltd for the cimetidine and placebo tablets.

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Impaired hearing in the elderly

Durham Area Health Authority is developing audiology services and has recently established a centre for issuing hearing aids. Accurate figures of the number of elderly people with hearing problems are not available, for definitions are not always consistent. When more than 4000 persons aged 65 years and over were interviewed one-third had some difficulty in hearing.[1] Only 6·3% had a hearing aid. A summary[2] of statistical data suggests that 3%-3½% of British people have a socially handicapping hearing loss. A variety of available statistics has been collated[3] which give some indication of the numbers affected. We investigated the hearing of patients attending a day hospital, serving a population of nearly 125 000, mainly for hemiplegic patients recovering from strokes. The staff were concerned that some patients had hearing difficulties affecting their behaviour, wellbeing, and response to treatment.

Patients, methods, and results

One of us (GWC) attended the hospital on six consecutive days and produced an audiogram for each of 38 patients (10 men, 28 women). Their ages ranged from 47 to 89 years—18 were between 58 and 69, five between 70 and 79, and 11 were 80 or over. Air and bone conduction tests and, when indicated, a tympanogram were done in each case, using a Kamplex TA155 and an AP61 impedance audiometer. A threshold of 20 dB, except at 6000 and 8000 Hz, was regarded as normal.

Five patients had normal hearing. Five others with a pure conductive loss of 30-35 dB on the lower frequencies had little or no difficulty. Eight out of 27 patients showing the typical curve associated with presbyacusis had little difficulty. Nineteen with a moderate to severe perceptive loss have since been fitted with aids. One patient, deafened in 1941, heard and understood speech at 10 feet (3 metres) when fitted with a commercial aid. Thus over half the patients had very poor hearing. Aids had previously been prescribed for two. One used it successfully, the other did so after proper instruction and fitting. Another patient, having declined an aid two years previously, changed his mind. Shortly after the hearing aids had been fitted the hospital staff noticed an improvement in the patients' attitudes and communication.

Six months later the 20 patients with impaired hearing were reviewed. One had died; one had refused an aid; and out of the 18 fitted with an aid 15 had continued to use it, one used it occasionally, and two did not use it.

Comment

Presbyacusis was expected in these patients but most were unaware of the help that is freely available from hearing aids. Almost all accepted their poor hearing as something they must expect with lengthening years. Attitudes seem to have changed little since Miss Bates described her mother's disability.[4] Our small survey shows that screening the hearing of groups of elderly people would be valuable. But providing a hearing aid is not enough: appropriate back-up facilities are required and about one-quarter of the patients may need additional rehabilitation.[2] Expansion of the services, though clearly desirable, is dependent on resources available.

We thank Dr G Ismay and Mr J S C Munro for their help and Sister Finnigan and her staff for their co-operation.

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Correction

Successful prophylaxis against febrile convulsions with valproic acid or phenobarbitone.

In the study by Sheila J Wallace and J Aldridge Smith (9 February 1980, p 353) the preparation used was Epilim syrup, which contains the sodium salt of valproic acid rather than the acid itself.