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SHORT REPORTS

24 Hour intragastric acidity during maintenance treatment with ranitidine

The recommended dose of ranitidine for the maintenance treatment of duodenal ulceration is 150 mg at bedtime.¹ Ranitidine is assumed to protect the patient from relapse of ulceration by decreasing nocturnal intragastric acidity, but the effect of this dose over 24 hours is not known.

Patients, methods, and results

We studied eight men with a history of endoscopically proved duodenal ulceration on two occasions when they were in symptomatic remission—before and on the seventh day of treatment with ranitidine 150 mg at night. Their mean age was 46.5 (range 30-64) years and mean weight 75.1 (range 63-91) kg. Six were smokers and each consumed an average of 17 cigarettes during each 24 hour study period. No patient received any antisecretory drug within 14 days of the start of the study, which was approved by the hospital ethical committee.

Mean hourly intragastric H^+ concentrations in eight patients with duodenal ulcer before and during treatment with ranitidine 150 mg at night. (*= $p < 0.001$, †= $p < 0.01$; Wilcoxon's rank sum test.)

Our established technique for measuring intragastric acidity^{2,3} was modified to take account of the single evening maintenance dose of ranitidine: the patients were admitted to the ward at 1900, a 10 FG nasogastric tube was passed at 1915, and a standard dinner was eaten at 1930. The 24 hour study period was from 2200 until 2100 the next day. Samples of gastric juice were aspirated every hour on the hour and their acidity measured using a combined glass electrode repeatedly calibrated with standard buffers of pH 7.00, 4.01, and 1.09 (Radiometer, Copenhagen). After the first study period each patient took ranitidine 150 mg at night by mouth for seven days, the last tablet being taken at 2215 during the second study period.

Mean 24 hour intragastric acidity decreased by 42% from 42.9 mmol/l to 24.8 mmol/l ($p < 0.001$, two way analysis of variance). This change was due to a significant decrease in hourly intragastric acidity from 2400 to 0900 (figure). From 1000 to 2300 there was no significant change in mean hourly acidity. Median intragastric pH over 24 hours increased from pH 1.4 to pH 1.8 during maintenance treatment with ranitidine. No adverse drug effects were reported by any patient during the study.

Comment

Ranitidine 150 mg twice a day decreases both daytime and nocturnal intragastric acidity, with median 24 hour intragastric pH rising from 1.4 to 2.4 during treatment.^{3,4} This study showed that ranitidine 150 mg at night caused a decrease in acidity that was restricted to the night and early morning.

Duodenal ulceration is a chronic illness and many patients require maintenance treatment with an H_2 -antagonist. It is reassuring that intragastric acidity was decreased for only 10 out of 24 hours after each maintenance dose of ranitidine 150 mg at night. For most of the day and during the evening the patients receiving maintenance treatment with ranitidine showed no change in intragastric acidity. The presence of normal concentrations of acid in the stomach for this prolonged period makes bacterial colonisation of the stomach unlikely during maintenance treatment.⁵

It may be relevant to the pathogenesis of duodenal ulceration that the short lived decrease in nocturnal acidity observed in this study is sufficient to prevent relapse of ulceration in most patients.

There is no report of the effect of maintenance treatment with cimetidine 400 mg at night on 24 hour intragastric acidity in patients with duodenal ulcers, but its effect may be even more transient than that of ranitidine.

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Acceptance of viewdata for poisons information

We recently suggested that a viewdata system could present basic poisons information effectively and ease the work of poisons information services by facilitating the storage and retrieval of information and reducing the need for staff to answer telephone inquiries.¹ Since its inauguration at the beginning of April 1983, our viewdata system has been the information source used by staff of the Scottish Poisons Information Bureau to answer conventional telephone inquiries and by centres throughout Britain which obtained terminals for direct

access to the data base. The viewdata computer records the date, time, and source of every viewdata inquiry, and a similar record of conventional telephone inquiries has been kept. We now report on the impact of the viewdata service on the workload of the bureau after the first 14 months.

Use of service

Users—During April 1983 to May 1984, 52 users were registered, 27 in Scotland, 21 in England, one in Wales, and three in Northern Ireland. The Scottish centres using the service included eight accident and emergency departments, two intensive care units, one clinical pharmacology unit, and 11 general practices. The non-Scottish users included the poisons information centres in Leeds, Newcastle, Northern Ireland, and the West Midlands; nine general practices; and four accident and emergency departments.

Total inquiries—The bureau received 13 999 inquiries during the 14 months, 58% of which were answered directly by the computer and 42% by the staff. The mean monthly number of inquiries answered by staff fell from 461 (SD 53) in the first seven months of viewdata to 388 (52) in the second seven months ($t=2.58$; $df=12$; $0.05 > p > 0.02$). The West Midlands, Leeds, and Newcastle poisons information services made 49% of the viewdata inquiries.

Scottish inquiries—During the period studied 8093 inquiries originated from Scotland. The number each month tended to rise, but an increasing proportion were answered by viewdata (figure). Eight accident and emergency departments made a total of 2665 inquiries (mean 333 (SD 202), range 55-645), including 2482 viewdata calls (89% of Scottish viewdata inquiries). After being registered for use of viewdata, the least user of the service made 75% of its poisons inquiries in this way, while the others used it for 92-95%.

Total monthly Scottish inquiries and those made by conventional telephone calls and by viewdata.

Comment

The total number of inquiries to the Scottish Poisons Information Bureau did not exceed 5480 a year up to 1982, and the 13 999 in the 14 months after introduction of its viewdata service was therefore a very considerable increase. Most of the additional inquiries originated from English poisons information services and were answered by the new system. Scottish calls also increased, but by March 1984 the computer directly answered more than half (figure). The attractiveness, novelty, and educational value of viewdata almost certainly accounted for some of the increase but in our view were unlikely to have been the sole or major reason. A substantial proportion must have related to patients presenting at hospitals. Despite the overall increase in inquiries, the number of conventional telephone calls fell significantly in the second seven months after introduction of viewdata.

We intended the new service to be accessed directly from hospitals

and general practices, and the Scottish inquiries are therefore of particular interest, since hospital emergency departments (the source of about two thirds of all poisons inquiries¹) are the main Scottish users of viewdata. Eight emergency departments accounted for 33% of Scottish calls, and 89% of these were answered by viewdata. A cross check on conventional telephone inquiries confirmed that the hospitals with viewdata terminals used them almost exclusively.

The main factor limiting the speed of adoption of viewdata by hospitals is finding the funds (£300-£600) for a terminal. Concern about paying for computer time and the information accessed has been expressed on occasions but is unwarranted; the only cost (apart from the terminal) is that of the telephone call to Edinburgh, which would have been made even if viewdata did not exist.

We appear to have established a service which is of value to doctors working in emergency departments and also to other poisons information centres. Since it also reduces the number of routine telephone calls to be answered, staff have more time to devote to improving the quality of the service.

We acknowledge the continuing support of the Lothian Health Board. The viewdata system was established with funds from the Chief Scientist and Scottish Home and Health Department and received the British Computer Society 1983 award for social benefit.

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Fine bore enteral feeding and pulmonary aspiration

We report two cases of pulmonary aspiration in patients undergoing enteral feeding through fine bore nasogastric tubes. We analyse the possible mechanisms and suggest a form of treatment.

Case reports

Case 1—An 83 year old man was admitted from a referring hospital with a chest drain in situ after a diagnosis of a pneumothorax. He had an undiagnosed traumatic diaphragmatic hernia that had been missed after a road traffic accident seven years previously. Repair of an iatrogenic perforation of transverse colon and repair of the diaphragmatic hernia necessitated a transverse colectomy and end colostomy and later a thoracotomy for pleural toilet. He required ventilation in the postoperative period, initially with a cuffed endotracheal tube and later with a cuffed tracheostomy tube. Fine bore enteral feeding was begun two weeks after admission once gut motility and absorption had been established, correct placement of the tube being checked on each routine chest radiograph. On three occasions over the ensuing two months a milky substance, indistinguishable from the feed, was aspirated from the bronchi despite the feeding tube being correctly positioned. On three other occasions feed was also seen in the mouth and the tube noticed to be coiled up in the pharynx. Two months after admission he suffered a cardiorespiratory arrest. During attempted resuscitation feed was aspirated from his tracheostomy tube and the feeding tube found in his oropharynx, suggesting that pulmonary aspiration may have been contributory. Unfortunately, it proved impossible to resuscitate him.

Case 2—A 23 year old man was admitted after a road traffic accident. He had sustained a fracture-dislocation of T6 and T7 with paraplegia and no sensation below T7. This necessitated him being nursed on a Stryker frame. Other injuries included a continuous pneumothorax and atelectasis. Two weeks after the accident he underwent an emergency thoracotomy with resection of infarcted lung. After two days he began enteral feeding through a fine bore nasogastric tube. Twelve hours later he became cold, clammy, and cyanosed with laboured respiration. Coarse crepitations were heard on the right side with inspiratory and expiratory wheezes and stridor. Earlier that day breath sounds had been normal. Chest x ray appearances were unchanged from a routine morning radiograph taken four hours previously. Measurement of blood gases confirmed hypoxaemia, hypercapnia, and a respiratory acidosis, suggesting intrapulmonary shunting associated with hypoventilation. The feeding tube was noted to be in the mouth. Immediate