Letters to the Editor

Recovery from sedation in day units

Sir,—There can be little doubt that day units offer advantages to both hospitals and patients for minor surgical and investigatory procedures. But there is a danger that pursuit of these advantages could erode standards to dangerous levels. The safety and ease of the progress of the patient through day units depends on the medical and administrative practices implemented in such units.

In the case of patients who attend the day unit of this hospital for gastroscopy, the endoscopist administers an intravenous benzodiazepine as sedation. After the procedure, patients rest in a quiet room and are allowed to leave when they feel adequately recovered. All patients are advised that they will not be fit to drive and should arrange to be accompanied home by a responsible adult.

Recently we monitored a group of consecutive patients attending our day unit. Over five weeks 85 patients attended for upper gastrointestinal endoscopy, 40 women and 45 men, aged 17 to 84 years. We noted the times of their arrival, the duration of the sedative, and of their discharge, and the dose of the benzodiazepine they had received. We found that the time from the administration of the intravenous sedative to the time the patient left the unit was unexpectedly short (mean 1 hour 23 minutes ± 30 minutes; range 15 to 530 minutes). For the patients given diazepam there was no correlation between time of dose and time to discharge (r = 0.009, p = 0.05), but there was a significant correlation for those given midazolam (r = 0.41, p = 0.02). For the whole sample (n = 85), however, there was a stronger negative correlation between time to discharge and the time spent in the unit before the procedure (mean 2 hours, range 45 minutes–3 hours 45 minutes (r = 0.3, p = 0.02). It seems overall that the time spent in the unit before the procedure is the best predictor of recovery time.

As psychologists concerned in the assessment of impairment after centrally acting drugs we find these rapid departures startling. We would be most interested in comments from your readers.

CAROLINE WHITEHEAD
LALAGE D SANDERS
Department of Anaesthetics,
University Hospital of Wales,
Heath Park,
Cardiff CF4 4XW

'C-urea breath test for Helicobacter pylori infection

Sir,—We read with interest the recent paper by Dill et al on the 'Evaluation of C-urea breath test in the detection of Helicobacter pylori and in monitoring the effect of tripotassium dicitrato-bismuthate in non-ulcer dyspepsia' (Gut 1990; 31: 1237–41). In this study the authors used 250 mg of C-urea per patient. We have recently conducted a study comparing the C- and C-urea breath tests in Helicobacter pylori positive patients both before and at least one month after treatment. We found 100% agreement between the two tests and obtained just as good discrimination between positive and negative patients with 125 mg (n = 10) and 75 mg (n = 13) of C-urea as with 250 mg (n = 9). We agree with Logan et al.4 that excellent results can be obtained with 100 mg of C-urea per test but think that a further saving without jeopardising accuracy can be achieved by administering only 75 mg of the stable isotope per patient. Others have had similar experience.1

Since the C-urea test was first described by Graham et al in 1987 the analysis of C-urea in breath samples collected during the test has proved to be a major drawback to using the test in routine clinical practice. It is necessary to use isotope ratio mass spectrometry (IRMS) to measure C enrichment in CO2 because C changes of less than 1 part per 1000 need to be determined. Before the actual C measurements take place, CO2 must be purified from other breath gases. This has been achieved by a cryogenic purification unit linked to the IRMS. Breath analysis on such systems is slow (about 20 minutes per sample) and costly (0.5–1 litre of nitrogen per sample) and requires a complex dual inlet IRMS for the final C measurement.

In our own study we have used an automated breath C analyser (ABCA) utilising fast and simple chromatographic purification and a single inlet mass spectrometer. The system consists of a Roboprep-G purification system linked to a TraceMass stable isotope analyser (Europa Scientific, Crewe, UK). Briefly, each breath sample is automatically injected into the purification unit by a continuous flow of helium. Water vapour is removed by a magnesium perchlorate trap. A gas chromatograph (75°C) then separates CO2 from N2 and O2, before the CO2 is swept into the stable isotope analyser to measure C enrichment. Breath samples were measured against a reference gas (5% CO2, balance N2) which had a delta C value of −41.60 per 1000 (ε PDB). The C enrichments of breath samples were expressed in terms of the patient’s own baseline (0 min) delta C value. This technique for analysing C breath samples proved to be easy, fast (5 minutes analytical cycle time), and low consumable cost (GC grade helium)

Patients fasted overnight before the test. A nutrient dense drink (20 g Calogen LCT emulsion, 15 g Maxipro HB powder, 40 g Caloreen glucose polymer, 15 g Crusha syrup, and 300 ml water) was taken followed by the C-urea dose in 50 ml water. Breath samples were collected at 0, 20, 40, and 60 minutes after drinking the C-urea solution by using a saline breath collection bag. At each breath collection 2 × 20 ml aliquots of breath were drawn from the bag to fill two septum capped evacuated tubes. These samples were then sent to Europa Scientific for analysis of C enrichment. The C(CO2) values were performed within 48 hours of the C-urea breath test and the personnel at Europa Scientific had no knowledge of the result of the former when making their own analysis.

All pretreatment H pylori positive patients (positive by the C-urea breath test) showed a C change of >5/1000 in the mean of the 40 and 60 minute breath samples (n = 16) regardless of whether the patient had initially received 250, 125, or 75 mg of C-urea. In contrast, the one month (or greater) post-successful eradication breath tests of previously H pylori patients (n = 16) showed a C change of <3/1000, regardless of the dose of C-urea given.

It has been our previous experience with the C-urea breath test that within as little as 24–48 hours of completing a course of colloidal bismuth (subcitrate) the organism has been temporarily suppressed to undetectable levels rapidly multiplies to levels that again permit its detection using a urea breath test. These rapid results to positivity have been shown by restriction analysis of DNA analysis to be due to recrudescence and not reinfection.2 Logan et al repeated the endoscopy, biopsy, and C-urea breath test 48–72 hours after completing the course of 28 days of colloidal bismuth and found a clearance rate of only 5/28 (18%). When the same group more recently reported their results for 'clearance' of H pylori after one, two, or four weeks of colloidal bismuth (subcitrate) the respective rates of clearance were obtained when the breath tests were performed within 24 hours of stopping treatment. H pylori, however, recurred in all but one patient within one week of stopping treatment. Had 'clearance' been measured by restriction analysis a figure of 1/47 (2.1%) would have been recorded.

In our view the word 'clearance' should be avoided in the context of anti-H pylori treatment. We suggest that instead if research workers wish to study eradication either while on a particular treatment or within 24 hours of discontinuing it they talk about 'suppression' and not 'clearance.' We entirely agree with Dill et al and Logan et al that if one wishes to study H pylori 'eradication' then it is necessary to wait at least one month after completing any given course of treatment before repeating tests. We suggest that assessing the H pylori state more than 24 hours and less than 28 days after treatment has little or no clinical relevance.

G D BELL
K POWELL
J WEIL
G HARRISON
Department of Medicine,
Guy’s Hospital Medical School,
and Medical Physics,
St Thomas’ Hospital,
St Thomas’ Hospital, London SE1 7EH
S BROOKES
E ROBRIN
Europa Scientific,
Europe House,
Eccleston Place,
London SW1

Correspondence to: Dr G D Bell.

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 Reply

Sr,—We would like to thank Dr Bell and his colleagues for their comments. We are pleased to learn that they are now using the 14C-urea breath test (14C-UBT) for the detection of Helicobacter pylori and agree that the chromato-
graphic purification of breath samples for isolation of 14CO2 before mass spectrometry will help reduce the cost of analysis. The European standard protocol, however, using either the pooled or single sample technique for breath collection, provides an even greater reduction in the overall cost of the technique. The quantity of isotope used in the European standard 14C-UBT (100 mg) is less than half that used in Graham’s original description of the 14C-UBT. Smaller quantities of isotope have since been used in several small studies without detrimental effect on the sensitivity or specificity of the test. The ability of the 14C-UBT to detect very low levels of H pylori may, however, be impaired if very small amounts of 14C-urea are used. More specifically, although the trend to use smaller quantities of isotope is wellcome, theoretically the intragastric concentration of the isotope should be slightly greater than the Kmax for the urease of H pylori.

R P H LOGAN
J J MISIEWICZ
Department of Gastroenterology and Nutrition,
Central Middlesex Hospital,
London NW10 7NS

S DILL
Department of Gastroenterology,
Universitatsklinikum,
Kantonsspital Basel,
Basel, Switzerland

Correspondence to: Dr R P H Logan.


NOTES

BOOK REVIEW


There is much in this book which is satisfying to the reader, yet it suffers from a lack of continuity of information between widely separated chapters, which are concerned with complications of the multiple author syndrome. There are two sections, one dealing with concepts of carcinogenesis and the other with the clinical management of premalignant conditions. Under the former there are two well written and instructive chapters on the principles of carcinogenesis and oncogenes. These are followed by contributions on epithelial renewal, DNA flow cytometry, and neoplastic progression in the gastrointestinal tract, and monoclonal antibodies in neoplastic and preneoplastic disorders of the large bowel. In the middle of these we are treated to a lengthy chapter on the subject of dysplasia, which is well written but almost entirely concerned with dysplasia in Barrett’s oesophagus and chronic ulcerative colitis. There is overlap with a subsequent chapter on inflammatory bowel disease in the second section on clinical management. Surprisingly, the chapter on dysplasia includes only a short paragraph on the diagnosis and classification of dysplasia in adenomas. One chapter only is devoted to the whole subject of gastrointestinal polyps and polyposis syndromes. There is inadequate coverage of the epidemiology, genetics, pathology, and evolution of the adenoma-carcinoma sequence. The problems of the malignant potential of juvenile polyposis and the Peutz-Jeghers syndrome are ignored. A major weakness in many of the chapters is the lack of emphasis on the contribution of epidemiology to our understanding of premalignant states. Incipient space is given to meeting, and investigation, particularly endoscopy. The main objective in the study of premalignant conditions and histopathological lesions must be prevention and early detection of cancer with reduced mortality. Yet the book provides no sense of thrust in this direction. It is a collection of essays, most of them individually very good, but without the continuity which makes for easy reading. A last criticism. Please could Dukes’s classification be corrected. It is the Dukes classification not Dukes’s classification. The production of the book is good with clear print and microphotographs of good quality. A pity that it leaves something to be desired.

B C MORSON

Corrections

Effects of olsalazine and sulphasalazine on jejunal and ileal water and electrolyte absorption in normal human subjects by Raimundo et al, March 1991; 32: 270–6. Table II gives data on the effect of olsalazine in the human jejunum; Table V gives data on the effect of sulphasalazine in the human ileum.