INCIDENCE OF ALDEHYDE SENSITIVITY IN ENDOSCOPY UNITS
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At the present time, aldehyde compounds are the only effective agents for disinfection of endoscopes. However, these substances are toxic and associated with sensitivity reactions.

In order to assess the incidence of sensitivity to commonly used disinfectants, questionnaires were mailed to 400 members of the BSG Endoscopy Associates Group. Replies were received from 173 members in 144 units. 115 units (79%) reported at least one member of staff experiencing some form of sensitivity (27% all staff affected, 20% more than half). Headache (75%), rhinitis (72%), conjunctivitis (65%) and dermatitis (45%) were the most commonly reported symptoms. Cough (11%), excessive tiredness (7%) and nasal bleeding (5%) were also reported.

There was no correlation between the type of ventilation/fume extraction system used and the incidence of sensitivity reactions, nor any relation to the type of disinfectant used (glutaraldehyde solutions 45% sensitivity, formaldehyde based solutions 49%). Personal protective measures were employed in most units, all using protective gloves, but not necessarily impermeable to aldehyde solutions. 75% of units routinely wore aprons, 77% utilised eye protection and 39% regularly wore masks. In the course of the last 5 years 83% of units had changed their cleaning/disinfection procedures as a result of either changes in cleaning machines or as a response to published guidelines on disinfection techniques.

This survey shows that greater attention has been paid to adequate cleaning and disinfection of endoscopy equipment but that there is an unacceptable morbidity to nursing staff from currently used disinfecting agents. There is an urgent need to review the methods of endoscope disinfection in order to reduce the occupational hazard to an highly trained group of individuals.

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RANDOMISED STUDY OF SURVEILLANCE INTERVALS AFTER REMOVAL OF COLORECTAL ADENOMAS AT COLONOSCOPY.

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In a 10-year prospective study, 785 patients were categorised as high or low risk for cancer based on age and number of colorectal adenomas, risk factors determined in a previous pilot study. High-risk patients (437) were randomised to annual (H1) or 3-yearly (H3) intervals and low-risk patients (348) to 5-yearly (L1) or 5-yearly intervals (L5). A total of 2943 person years of follow-up were accrued (mean 4 years).

At follow-up, 48% of high-risk and 36% of low-risk patients had at least one adenoma. Among the high-risk patients, a similar proportion (6%) of H1 and H3 patients developed a large adenoma (≥ 10 mm). However, four H3 and one H1 patient developed cancer, the latter at 10 years. Among the low-risk patients, only 8 developed a large adenoma (3%), but there were 2 cancers (both L3).

For the vast majority of patients, even those perceived to be at high risk, 3 yearly follow-up appears to be adequate. The problem remains to identify at presentation the characteristics predicting the small percentage (5% of this series) who develop large adenomas or cancers at follow-up and therefore require more frequent surveillance. The previously determined risk factors did not adequately delineate all patients at high risk.

THE CUFFED OESOPHAGEAL PROSTHESIS—A USEFUL DEVICE IN DESPERATE SITUATIONS IN OESOPHAGEAL MALIGNANCY

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We have identified 3 groups of patients in whom cuffed Wilson-Cook oesophageal endoprostheses (William Cooke Europe Limited) have proved invaluable. All 13 patients discussed were intubated at endoscopy using an Atkinson introducer (which grips these tubes well) and with fluoroscopy. Group 1 comprised 7 patients with Oesophago-respiratory fistulae with malignancy—the indication for which the tubes are designed. Three primaries were oesophageal, 3 bronchial and one ovarian. Five fistulae were demonstrated radiographically and 2 at endoscopy only. Six closed after intubation and one tube displaced and could not be repositioned. Median survival was 4 weeks (range 1-10) but all those whose fistulae were closed managed liquids and 3 semi-solids.

Group 2 comprised 3 patients with instrumental perforation or tears at endoscopy. Two developed surgical emphysema after dilatation and the third had a large tear following removal of a previously displaced Atkinson tube. All had satisfactory contrast swallows the day after intubation and were started on semi-solid diets, median survival was 10 weeks and one patient is still alive after 11 weeks. The third group included 3 patients with life threatening arterial bleeding from Cardia cancers. No further bleeding occurred in any of the 3 after intubation and 2 survived several more months.

HEATER PROBE VERSUS INJECTION THERAPY FOR BLEEDING PEPTIC ULCER

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One hundred and twenty consecutive patients presenting with major peptic ulcer haemorrhage were randomised in a prospective clinical trial comparing endoscopic injection therapy and thermoagulation using the heater probe. All were either actively bleeding at the time of endoscopy or had a non-bleeding protruberant vessel. In addition all patients had at least one other risk factor from age over 60 years, "shock" and/or HB concentration less than 100g/l. Injection therapy comprised a combination of 4-10mls i:100,000 adrenaline and 0.5-2 ml 5% ethanolamine was used in 56 patients, 57 subjects received a median of 225 joules using the 8F keymed heater probe. All patients received conventional supportive therapy including H2 receptor antagonist drugs.

The two groups were well matched in terms of age, shock, HB concentration, endoscopic findings, concomitant medical disease and NSAID usage. Eight patients in the injected group and 9 in the heater probe group rebled and 7 patients from each group underwent emergency surgery. There were no differences in outcome as determined by median transfusion requirements (5.2 and 5.1 units), median hospital stay (7 days in both groups) and hospital mortality (2 and 3 patients). No significant complications occurred in any patient.

Endoscopic injection and heater probe therapy are equally effective therapy for major peptic ulcer haemorrhage.
The effects of supplemental oxygen on cardiac rhythm during gastroscopy: a randomised controlled double blind trial

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Cardiac arrhythmias have been observed during gastroscopy, and it has been suggested that the hypoxia induced during the procedure is a causal factor.

Methods: 103 patients over 60 years at routine gastroscopy were randomised to receive either supplemental oxygen or air at 2 l/min during the procedure. Pulse, blood pressure, oxygen saturation and ECG were monitored before, during and for one hour after the gastroscopy.

Results: Abnormal ECG's were seen in three cases in oxygen in 94%/88% before, 77%/68% during, and 92%/90% after gastroscopy. Most arrhythmias recorded were supraventricular (SVF) and ventricular ectopies (VE), but there were other arrhythmias seen. Both VE and ST segment changes occurred equally within the two groups. SVE were seen in significantly more patients during gastroscopy (p<0.05) in those receiving oxygen (36%). However, their frequency when present was similar both in the oxygen and air groups, and between the three monitored periods. Although supplemental oxygen during gastroscopy improved oxygen saturation significantly by 3.6% (p<0.001; 95% confidence intervals for the difference between the means: 2.9-4.7), there was no correlation between oxygen saturation or its rise and any ECG changes. Similarly there was no relationship between the incidence of arrhythmias or ST segment change and patient characteristics, such as age or cardiopulmonary history.

Conclusion: ECG abnormalities are common in patients over 60, but this study found no evidence that they are induced by gastroscopy. Supplemental oxygen increases oxygen saturation but does not reduce the incidence of cardiac arrhythmias or ST segment changes.

Liver injury, repair and replacement

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VAGAL DYSFUNCTION AND IMPAIRED SODIUM AND WATER EXCRETION IN CIRRHOSIS

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In order to determine the effect of vagal dysfunction on fluid balance we examined 11 cirrhotics (7 Alcoholic, 2 Primary biliary cirrhosis, 2 Others; 7 Childs A, 4 Childs B) using cardiovascular reflex (CVR) tests and evaluated the response to a standard 20 ml/kg water load. Cirrhotics with vagal dysfunction (n = 6) had a significantly impaired diuresis and urinary sodium excretion compared to those with normal autonomic tests (n = 5): mean ± excretion of water load after 5 hrs 67.8 ± 109.2 p < 0.008; mean 5 hr sodium excretion 32.3 ± 69.4 mmol, p < 0.05. The % water excretion correlated with the number of abnormal CVR tests (r = -0.669 p < 0.02) and urinary sodium excretion correlated with the heart rate response to intravenous atriosine (r = 0.75 p < 0.02) and to deep breathing (r = 0.74 p < 0.013). Vagal and hepatic function and the change in atrial natriuretic peptide was similar between the two groups; however, vagal dysfunction was associated with a significantly elevated plasma noradrenaline (mean at 5 hrs 3.49 vs 1.83 pmol/l, p < 0.003), vasopressin (mean at 2 hrs 0.63 vs 0.23 pmol/l, p < 0.05) and renin (mean at 5 hrs 2.27 vs 0.74 ng/ml/h, p < 0.05).

These results indicate that cirrhotics with vagal dysfunction have impaired sodium and water excretion associated with disturbances in the major vasoactive neurohumoral systems.

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INCREASED INTERLEUKIN-1 AND INTERLEUKIN-6 PRODUCTION IN PATIENTS WITH CHRONIC ALCOHOLIC LIVER DISEASE

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Interleukin-1 and interleukin-6 are monokines released into the circulation and have been found to possess a variety of biologic functions such as inducing an elevation in temperature. Interleukin-6 is also a major inducer of B cell differentiation and stimulates the production of immunglobulins.

We investigated spontaneous and lipopolysaccharide-induced interleukin-1β(IL-1β) and interleukin-6(IL-6) production by peripheral blood mononcytes in 10 patients with chronic alcoholic liver disease and 10 normal controls. Spontaneous production of IL-1β and IL-6 were not different in chronic alcoholic liver disease (43.6 ± 253.2 and 2545.9 ± 817.8 pg/ml respectively) compared to healthy controls (132.9 ± 75.7 and 1201.1 ± 287.7 pg/ml respectively; p>0.05). However, lipopolysaccharide-stimulated monocytes of patients with chronic alcoholic liver disease generate significantly more IL-1β and IL-6 (2908.5 ± 7532.2 and 5854.1 ± 1609.5 pg/ml respectively) than healthy controls (646.6 ± 2437.5 and 2609.9 ± 326.5 pg/ml respectively; p<0.05).

Increased production of cytokines may play a role in the pathogenesis, and might lead to perpetuation and decompensation of the underlying chronic alcoholic liver disease. It may also explain clinical features such as hypergammaglobulinaemia, susceptibility to sepsis and fever even in the absence of infection.