Unreviewed Reports

Two cases of hereditary haemorrhagic telangiectasia of the bronchus

Though gastrointestinal and urinary tract lesions are well documented in hereditary haemorrhagic telangiectasia, lower respiratory tract lesions are not. A 74 year old man, admitted with acute chronic bronchitis and known hereditary haemorrhagic telangiectasia, had tracheal telangiectasia on bronchoscopy[1]; at necropsy some months later widespread bronchial telangiectasia was found. A 35 year old man with profuse haemoptysis had telangiectasia inside his lower lip but no other abnormal findings. Bronchoscopy showed telangiectasis at the carina and in the main bronchi, which was profuse at the origin of the bronchus of the left upper lobe. Bronchial anteriography showed a large feeding artery and a hypervascular segmental bronchial supply in the left upper lobe.—S M C HUGHES, E T PEEL, *et al*, Llandough Hospital, Penarth CF6 1XX. *(Accepted 24 January 1984)*

[1] Ona FV, Ahluwalia M. Endoscopic appearance of gastric angiodysplasia in hereditary haemorrhagic telangiectasia. *Am J Gastroenterol* 1980;**73**:148-9.

Atherosclerotic infarction of the rectum

Atherosclerosis is the commonest factor predisposing towards bowel ischaemia. Nevertheless, the rectum has an excellent collateral circulation and pure selective infarction of it has not been described. A 77 year old man with a past history of myocardial infarction and stroke had bloody diarrhoea, severe abdominal pain, and absent bowel sounds. Laparotomy disclosed almost complete infarction of the rectum and subsequent examination showed thrombus occluding the terminal branches of the inferior mesenteric vessels supplying it. Given that there were no other factors predisposing to large bowel ischaemia, such as hypotension and hypovolaemia, atherosclerosis is the only identifiable cause.—M WEAVER, Department of Surgery, Queen Elizabeth Hospital, Birmingham B15 2TH. *(Accepted 25 January 1984)*

Cimetidine in acute upper gastrointestinal haemorrhage in elderly patients

Because mortality of upper gastrointestinal haemorrhage increases sharply in patients over 60 we performed a multicentre study of a five day course of cimetidine or placebo in such patients. Patients with duodenal ulcer (20 cimetidine, 23 placebo) showed no difference in the outcome with the two regimens; in those with gastric ulcer, however, none of the 10 given cimetidine died but 4 out of 12 given placebo did—a statistically significant difference ($p<0{\cdot}05$). Possibly cimetidine improves the prognosis in two ways: reduction of acid output and reduction of blood flow in the gastric mucosa, and should be considered in elderly patients with gastric ulcer haemorrhage.—N DARLE, B ALMSKOG, *et al*, East Hospital, Gothenburg, Sweden. *(Accepted 23 January 1984)*

Hypotensive effect of eicosapentaenoic acid in normotensive and hypertensive subjects

N-3 fatty acids have a beneficial effect on two risk factors for atherosclerosis—namely, lipoproteins and platelet function[1]—and may have on a third—namely, hypertension. Blood pressure was measured before and after mackerel and herring diets (taken for two weeks and equivalent to 2·2 g and 1·0 g eicosapentaenoic acid respectively) in 15 normotensive men and 10 men with mild hypertension. After the mackerel diet systolic blood pressure had decreased from 128 (12) to 113 (11) mm Hg and diastolic blood pressure from 153 (13) to 140 (13) mm Hg. After the herring diet the changes were minor.—P SINGER, H HEINE, Central Institute for Cardiovascular Research, Academy of Sciences of the German Democratic Republic, 1115 Berlin, East Germany. *(Accepted 23 January 1984)*

[1] Goodnight SH, Harris WS, Connor WE, Illingworth DR. Polyunsaturated fatty acids, hyperlipidemia, and thrombosis. *Arteriosclerosis* 1982;2:87-113.

Profound hypocalcaemia after high doses of intravenous fusidic acid

A 68 year old woman with an aortic valve prosthesis was admitted with confirmed bacterial endocarditis. On admission she was severely hypocalcaemic (serum calcium concentration 1·25 mmol/l (5·01 mg/100 ml), ionised calcium concentration 0·7 mmol/l (2·8 mg/100 ml)). The onset of hypocalcaemia had coincided with the start of treatment with intravenous fusidic acid 4 g daily five days previously. Intravenous fusidic acid is prepared in a phosphate buffer (5·5 mmol (214 mg) phosphate/500 mg sodium fusidate). The 44 mmol (1·67 g) of phosphate daily accompanying this high dose of fusidic acid would be sufficient to lower serum calcium concentrations.[1] The manufacturer's recommended dose of 1·5 g fusidic acid intravenously daily should provide adequate therapeutic concentrations and should not be exceeded as severe hypocalcaemia may result.—J C COWAN, S HUDSON, *et al*, Regional Cardiothoracic Centre, Freeman Hospital, Newcastle upon Tyne NE7 7DN. *(Accepted 23 January 1984)*

[1] Fulmer DH, Dimich AB, Rothschild EO, Myers WPL. Treatment of hypercalcaemia. *Arch Intern Med* 1972;**129**:923-30.

Exacerbation of IgA nephropathy after influenza vaccination

Adverse effects of influenza vaccination are unusual,[1] but we report exacerbation of IgA nephropathy. A 37 year old man who had had IgA nephropathy during viral infections since 1979 had no features of renal dysfunction in between. Eight hours after influenza vaccination he developed fever, the urine turned dark, and two days later serum creatinine was 132 μmol/l with macroscopic haematuria. After a week these abnormalities disappeared and the urine contained only a few erythrocytes and traces of protein.—RISTO SIPILÄ, MARTIN VON BONSDORFF, Fourth Department of Medicine, Helsinki University Central Hospital, Helsinki, Finland. *(Accepted 2 February 1984)*

[1] Retailliau HF, Curtis AC, Stopp G, Caesar G, Eddins DL, Hattwick MAW. Illness after influenza vaccination reported through a nationwide surveillance system, 1976-1977. *Am J Epidem* 1980;**111**:270-8.

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