

aspects in the pathogenesis of Crohn's disease came from surgeons, who before 1955 treated most patients over many years. Physicians at that time lacking effective drugs had little to offer.

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1 Paulley JW, Pelsler HE. *Psychological managements for psychosomatic disorders*. Heidelberg: Springer, 1989.

### Reply

SIR,—We read with interest the comments by J W Paulley on our paper. We agree that patients with Crohn's disease in addition to medical and surgical care should also have appropriate psychological management, which is within the competence of each physician. In our experience, this is best achieved if the patient has regular contact with a medical team specialising in inflammatory bowel disease treatment. Professional care will relieve much anxiety about the future with this chronic disease, and also enable the patient to cope with the symptoms that most inevitably develop. The disease, however, can no longer be regarded as a psychosomatic disorder. Our studies and others clearly show that Crohn's disease is a chronic gastrointestinal process with a permanently present intestinal inflammation. There should be no antagonism between medical, surgical, and psychological therapy. The best support a patient with Crohn's disease can be given, is for the disease to be recognised as a chronic gastrointestinal disease. Present available medical and surgical treatment, although palliative, may offer a substantial reduction of symptoms. To abstain from such treatment today, using only psychotherapy for recurrent symptoms after surgery, is not adequate. The care of patients with Crohn's disease is a multidisciplinary task. At the University Hospital, Linköping, Sweden, all patients are regularly reviewed by a team including a gastroenterologist, gastrointestinal surgeon, endoscopist, dietitian, stoma therapist, and specially assigned nurse. The team has a joint treatment strategy; the aim of which is to minimise the symptoms during their lifetime, and all decisions on treatment are taken regarding the chronicity of the disease. Medical and surgical treatment are regarded as complementary. The treatment is governed by the patients disease activity, intensity – that is, activity for a longer period – and aggressiveness. Psychological management is a part of the regular care.

We studied 238 patients in 1991 with Crohn's disease. The treatment outlined above resulted in clinical remission of 83% at the end of 1991 (Crohn's disease activity index <150). The patients own experience of their disease was also, in our view, satisfying. Sixty nine per cent considered themselves healthy or almost healthy (>80% healthy, self estimation on a visual analogue scale). We believe that the best help a patient with Crohn's disease can have is the psychological, medical, and surgical support from a knowledgeable team.

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### Osteomalacia after gastrectomy

SIR,—I congratulate Bisballe *et al* (*Gut* 1991; 32: 1303–7) for evaluating their postgastrectomy patients for metabolic bone disease. All too often, we, as physicians caring for patients with gastrointestinal problems that lead to abnormal bone nutrition and metabolism – that is, postgastrectomy, inflammatory bowel disease, small bowel malabsorption, and states of diminished oral intake, ignore the patients' developing bone disease because the process is insidious and the patients are often asymptomatic until the disease is so profound that much of it may be irreversible.

I must take issue, however, with the authors' conclusion advocating supplemental vitamin D for all postgastrectomy patients. The authors have found evidence of bone abnormalities by histomorphometry in up to 62% of 51 patients, but only eight patients were considered to have osteomalacia. It is not clear that vitamin D supplementation is beneficial in non-osteomalacia bone disorders.

The authors suggest that the low mean levels of 25-hydroxyvitamin D are relevant and thus warrant supplementation. Yet, five of eight patients with a diagnosis of osteomalacia had normal levels, highlighting the lack of value serum measurements may have in predicting bone disorder diagnoses. The authors do not state how many of their non-osteomalacia patients had levels that were abnormal (they state only that the mean overall was abnormal). That 1,25-dihydroxyvitamin D levels, the active circulating vitamin D metabolite, were normal in these patients, underscores how complex vitamin D metabolism is and how serum values may not be predictive of an abnormality.

Although the authors agree in their final paragraph that serum biochemical results are of limited value, they use these results as the premise for a therapeutic intervention. They showed that patients who took vitamin D supplementation had higher levels of serum 25-hydroxyvitamin D. It is not sufficient to recommend vitamin D therapy simply because patients have metabolic bone disease. Vitamin D supplements in excess doses can be toxic and may not necessarily be helpful. There is no indication that those patients on supplements had less bony abnormalities on histomorphometry. In fact, half of the patients with osteomalacia were using supplementation.

To determine whether vitamin D is of therapeutic benefit, a study is needed whereby vitamin D therapy is compared with placebo in a randomised blinded group of postgastrectomy patients and not where the use of vitamin D in a group of patients is compared with a non-diseased control group. Oral ingestion of dietary vitamin D needs to be controlled. Investigators of such a study would need to show that bone histomorphometry improved or did not worsen over time while on therapy.

Vogelsang *et al*<sup>1</sup> showed that overall, a group of Crohn's disease patients randomised to vitamin D 1000 IU/day therapy did not have a statistically significant improvement in bone mineral density. Although maintenance doses of vitamin D 400 IU/day with calcium supplementation maintained bone mineral density in a group of corticosteroid using rheumatology patients, higher doses of more potent 1,25-dihydroxyvitamin D were of no benefit and led to raised serum and urine calcium concentrations.<sup>2</sup>

Although low dose vitamin D therapy (400–600 IU/day) in postgastrectomy patients is likely nontoxic – that is, in terms of effects on

calcium, this remains to be proved in a systematic fashion. The benefit of this therapy in postgastrectomy patients certainly needs to be proved and has not been in Bisballe *et al*'s paper.

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- 1 Vogelsang H, Ferenci P, Resch H, *et al*. Randomized controlled trial of longterm vitamin D supplementation in patients with Crohn's disease. *Gastroenterology* 1991; 100: A260.
- 2 Dykman TR, Haralson KM, Gluck OS, *et al*. Effect of oral 1,25-dihydroxyvitamin D and calcium on glucocorticoid-induced osteopenia in patients with rheumatic diseases. *Arthritis Rheum* 1984; 27: 1336–43.

### Reply

SIR,—We thank Dr Bernstein for his constructive comments on our paper on osteomalacia in postgastrectomy patients and his emphasis on the importance of the problem. We also consider bone disease after gastrectomy a problem very often ignored in daily clinical practice.

The diagnosis of osteomalacia in our study was based on very stringent histological criteria, which delineate severe mineralisation defects affecting virtually all bone surfaces. The remaining patients with abnormal bone histology revealed varying degrees of secondary hyperparathyroidism with focal areas of mineralisation defects – that is, earlier stages in the osteomalacic process. Further analysis of serum biochemistry in these patients was hampered by the lack of good parathyroid hormone assays (the study was performed before the advent of assays for intact parathyroid hormone (1–84)). Theoretically, such patients would also benefit from vitamin D supplementation, because the high turnover state associated with secondary hyperparathyroidism leads to osteopenia. We do not think, however, that the study on Crohn's disease by Vogelsang *et al* is of great relevance in this respect. The bone in patients with Crohn's disease is characterised by osteopenia and only very discrete osteomalacia is demonstrable.<sup>1</sup>

We do not advocate the use of more potent analogues (1-alpha-vitamin D<sub>3</sub> or 1,25(OH)<sub>2</sub>D<sub>3</sub>) for postgastrectomy patients. We agree with Dr Bernstein that these compounds demand close monitoring because of the risk for severe intoxication. We have, however, administered daily doses of 18 000 IU calciferol (D<sub>2</sub>) over a five year period to more than 200 osteoporotic women (mean age 65 years) without any cases of vitamin D-intoxication.<sup>2</sup> Thus, we do not think that vitamin D supplements are dangerous as long as they are given in the form of D<sub>2</sub> analogues in a dose range of 400–800 IU. We agree, however, that a controlled clinical study is necessary in order to test for optimal dose, efficacy and side effects.

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- 1 Hessov I, Mosekilde L, Melsen F, Fasth S, Hulten L, Lund B, *et al*. Osteopenia with normal

vitamin D metabolites after small bowel resection for Crohn's disease. *Scand J Gastroenterol* 1984; 19: 579-84.

- 2 Eriksen EF, Mosekilde L, Melsen F. Effect of sodium fluoride, calcium, phosphate and vitamin D, on trabecular bone balance and remodelling in osteoporosis. *Bone* 1985; 6: 381-9.

### Oesophageal strictures in dystrophic epidermolysis bullosa

SIR,—Walton and Bennett in their progress report 'Skin and gullet' (*Gut* 1991; 32: 694-7) declare that diffuse oesophageal involvement in dystrophic epidermolysis bullosa is difficult to manage with a considerable risk of perforation. They recommend colonic interposition either by bypass or replacement of the strictured oesophageal segment. They base their recommendation on a report describing successful surgical oesophageal replacement in two patients.

I am concerned that patients with dystrophic epidermolysis bullosa complicated by oesophageal strictures may be subjected to major surgery when colleagues follow the recommendation of Walton and Bennett and thereby ignore an established non-operative treatment method. Balloon dilatation with balloons of predetermined size, placed in the oesophagus over a pathfinder string, combined with long term tube feeding is an effective and safe procedure.<sup>1</sup> Dilatation even of diffuse strictures in the thoracic oesophagus is no more difficult than in the cervical part. The avoidance of tangential shearing forces induced by bougienage and the use of balloon dilators which produce vertical pressure reduce the risk of mucosal detachment. We have not seen oesophageal perforation with this method and no such complication has to my knowledge been reported with our method. Three of our original four patients treated with balloon dilatation and tube feeding nine or 10 years ago respectively have not experienced recurrence of tight oesophageal strictures. One patient developed severe dysphagia again four and five years after the original treatment<sup>2</sup> and was retreated in 1987,<sup>2,3</sup> with no further recurrence. Oesophageal surgery with its inherent risks has not been necessary in any of our patients.

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- 1 Feurle GE, Weidauer H, Baldauf G, Schulte-Braucks T, Anton-Lamprecht I. Management of oesophageal stenosis in recessive dystrophic epidermolysis bullosa. *Gastroenterology* 1984; 87: 1376-80.  
2 Weidauer H, Feurle GE, Baldauf G, Anton-Lamprecht I. Zur Therapie von Ösophagusstenosen bei rezessiver Epidermolysis bullosa dystrophica. *Laryngorhinootologie* 1985; 64: 522-6.  
3 Seitz HK, Egerer G, Sina R, Baldauf G, Hartschuh W, Anton-Lamprecht I, et al. Dilatationsbehandlung von Ösophagusstenosen bei jungen Patienten mit rezessiver Epidermolysis bullosa dystrophica. *Z Gastroenterol* 1990; 281: 157-9.

### Reply

SIR,—We were most interested in Dr Feurle's success in treating epidermolysis bullosa oesophageal strictures by balloon dilatation and obviously this is a far more desirable

approach than a surgical one — provided the stricture responds as well as his.

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### Papillary cystic neoplasm of the pancreas

SIR,—We were very interested to read of the description by Kingsnorth *et al* of two cases of this unusual primary pancreatic tumour (*Gut* 1992; 33: 421-3). We have also recently seen a case of this rare neoplasm which presented in a 27 year old woman as an abdominal mass approximately eight to 10 weeks post partum. Computed tomography of the abdomen revealed a well defined non-homogeneous mass arising in the body and tail of the pancreas. The tumour was encapsulated, measuring 25×17×13 cm, and partly necrotic, showing solid and papillary areas. The tumour cells showed generalised positive immunohistochemical reactivity with neuron specific enolase, and focal immunoreactivity for insulin and glucagon. The tumour cells showed no evidence of PGP 9-5, somatostatin, or gastrin immunoreactivity. Electron microscopy showed scattered dense core neurosecretory granules and intercellular junctional complexes (desmosomes).

The immunohistochemical pathological findings seen in papillary cystic tumours of the pancreas are not always characteristic; often being quite variable.<sup>1,2</sup> Papillary cystic neoplasms of the exocrine pancreas may sometimes show neuroendocrine features as seen in primary pancreatic islet cell tumours. The immunohistochemical findings are therefore not always characteristic and a final pathological diagnosis rests on knowledge of the site and size of the neoplasm, the young age and female sex of the patient, and the microscopic appearance of the tumour. A failure to appreciate the variable immunohistochemical characteristics of papillary cystic neoplasms of the exocrine pancreas may lead to errors in the interpretation of the pathology of these rare pancreatic tumours by causing potential diagnostic confusion with primary pancreatic islet cell tumours.

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- 1 Miettinen M, Partanen S, Fraki O, Kivilaakso E. Papillary cystic tumor of the pancreas. An analysis of cellular differentiation by electron microscopy and immunohistochemistry. *Am J Surg Pathol* 1987; 11: 855-65.  
2 Bogomeletz WV. Cystic tumours of the pancreas. In: Anthony PP, MacSween RNM, eds. *Recent advances in histopathology*. 15. London: Churchill Livingstone, 1992: 141-55.

## NOTES

### Gastrointestinal Motility

The Sixth European Symposium on Gastrointestinal Motility will be held from 19-21

November 1992 in Barcelona. Further information from Unicongress, Calvet, 55 (4<sup>th</sup>a), 08021 Barcelona, Spain. Tel: 343 414 03 22; Fax: 343 414 02 51.

### Digestive Surgery

The European Workshop on Digestive Surgery will be held on 5-6 November 1992 in Brussels. Further details from Conference Services, Avenue de l'Observatoire 3, Box 17, B-1180 Brussels, Belgium. Tel: 32 2 375 1648; Fax: 32 2 375 3299.

### European Pancreatic Club

The 24th Annual Meeting will be held in Ulm, Germany from 11-14 October 1992. Further details from Marga Wild, Department of General Surgery, University of Ulm, Steinhoevelstr 9, D-7900 Ulm, Germany. Tel: 731 502 7200/1; Fax: 731 502 7209.

### Clinical Nutrition

The Leeds course in Clinical Nutrition will be held from 15-18 September 1992. Further information from Mrs Hilary L Helme, Department of Continuing Professional Education, Continuing Education Building, Springfield Mount, Leeds LS2 9NG. Tel: 0532 333233.

### American Gastroenterological Association 1992 Fall Postgraduate Course

The 1992 Postgraduate Course will be held from 17-19 September, 1992 in San Francisco, California. Further information from SLACK Incorporated, 6900 Grove Road, Thorofare, NJ 08086-9447, USA. Tel: 609 848 1000.

### Sir Francis Avery Jones BSG Research Award 1993

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1993 Award. Applications (15 copies) should include:

- (1) A manuscript (2 A4 pages *only*) describing the work conducted.
- (2) A bibliography of relevant personal publications.
- (3) An outline of the proposed content of the lecture, including title.
- (4) A written statement confirming that all or a substantial part of the work has been personally conducted in the United Kingdom or Eire.

The award consists of a medal and a £100 prize. Entrants must be 40 years or less on 31 December 1993 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Spring Meeting of the Society in 1993. Applications (15 copies) should be made to: The Honorary Secretary, BSG, 3 St Andrew's Place, London NW1 4LB by 1 December 1992.