CP eradication from the gastric antrum or the duodenal bulb.

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Bile acid malabsorption in progressive systemic sclerosis

SIR,—We have read with interest the paper by Stellaard and coworkers[1] on the increase of unconjugated serum bile acids in patients suffering from progressive systemic sclerosis (PSS). We have recently studied the serum pattern of primary bile acids in PSS-patients and the results have been partly reported on the journal of the Italian Association of Rheumathology[2] and at international symposia.[3,4]

Our data confirm that a bile acid malabsorption is, in these patients, much more frequent than it is usually believed. We also think that malabsorption can not be caused only by bowel bacterial overgrowth. Very often the clinical picture of malabsorption is not reversed by wide spectrum antibiotics and in most patients steatorrhoea persists despite treatment.

In a group of 11 patients with 'classical' PSS (ARA criteria) we evaluated serum conjugated primary bile acids concentrations (by RIA) after an overnight fast and during three hour after a standard semiliquid meal (450 cal.). Postprandial curves were compared with 10 curves from sex and age matched healthy controls. Fasting serum concentrations of chenodeoxycholic acid conjugated (CDCA) were similar in the two groups [1·76 (1·0)μmol/l in controls, 1·42±0·75 μmol/l in PSS], while the concentrations of cholic acid conjugated (CCA) were lower in the PSS-group [0·67 (0·23) *v* 0·48 (0·17) μmol/l. $p<0·05$]. A similar pattern was exhibited by the postprandial increases postprandial maximal peaks of CDCA were almost normal in patients with PSS [6·59 (2·18) in controls, 5·19 (2·99) in PSS], while CCA peaks were strongly reduced in PSS-patients [3·36 (0·65) *v* 1·35 (0·44), $p<0·001$].

Similarly, the area under the curve calculated for serum post-prandial concentrations of CDCA was not significantly different in the two groups, while for CCA concentrations, the area was significantly smaller in PSS-patients ($p<0·01$).

In five patients we also studied the abdominal retention of ^{75}SeHCAT (^{75}Se-homocholic acid taurine) at the fourth and the seventh day after the ingestion of a 370 kBq (10 μCi) capsule (Amersham). PSS-patients exhibited strongly reduced ^{75}SeHCAT abdominal retention (at seventh day: mean 5·53%, range 1–14) when compared with five sex and aged matched healthy controls (mean 23·30%, range 11·4–33). The results of ^{75}SeHCAT–test were in good agreement with serum postprandial patterns of CCA.

Because it is well known that negligible bacterial deconjugation of SeHCAT occurs *in vivo,* bowel bacterial overgrowth does not seem to be important in determining bile acid malabsorption in PSS.

Our data are consistent with a bile acid malabsorption of first type, very similar to that found to be related to Crohn's disease or to ileal resection[5] and the severity of malabsorption may depend on the extent of ileal involvement in PPS.

In conclusion, we suggest that fasting and postprandial serum concentrations of CCA and ^{75}SeHCAT abdominal retention test represent good markers of ileal disease in these patients. We therefore agree with Stellaard and coworkers, that further studies should be done to investigate the specific role of bacterial overgrowth in PSS, particularly with cholyl-glycine-1-^{14}C breath test.

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Small intestinal adenocarcinoma, duodenal carcinoid tumour, and von Recklinghausen's neurofibromatosis

SIR,—We would like to comment on the report by Jones and Marshall (*Gut* 1987; **28:** 1173–6) of small intestinal adenocarcinoma occurring in neurofibromatosis. On the basis of their own and four additional published cases, they suggest the existence of a specific association between these two conditions. Surprisingly, as two of the five tumours were duodenal, they did not mention the now established link between duodenal carcinoid and neurofibromatosis.

In recent years we[1 2] and others[3 4] have drawn attention to an association between von Recklinghausen's disease and a distinctive carcinoid tumour of the duodenum – a review of 27 such cases has recently been published.[5] This duodenal carcinoid tumour is distinguished by containing somatostatin, and on microscopy commonly has psammoma bodies and a glandular growth pattern. The latter feature of the tumour makes it easily confused with duodenal adenocarcinoma by the histopathologist – indeed of nine duodenal carcinoids studied by ourselves, four had not previously been investigated with immunocytochemistry and were diagnosed originally as adenocarcinomas.[5] In two of the cases of small bowel adenocarcinoma in neurofibromatosis cited by Jones and Marshall the intestinal tumour was in the duodenum, yet the authors do not appear to have considered the possibility that they might be glandular carcinoids. In fact, we have studied personally the duodenal tumour from one of the cases[6] (Jones and Marshall's fourth case) and have indeed found it to be a somatostatin containing glandular carcinoid; this case has also been studied by Dayal *et al*[4] who agree with this interpretation.

Jones and Marshall base their suggestion of an association between neurofibromatosis and small bowel adenocarcinoma on five cases – now known to be four. While there may be such a link, it remains a tenous one, particularly since rare conditions occurring together are much more likely to be reported than when they occur singly. We feel that it is important for clinicians to be aware of the more certain association of neurofibromatosis with duodenal glandular carcinoids, so that this diagnosis is the first to be considered in a patient with von Recklinghausen's disease and a duodenal (especially a periampullary) tumour. The histopathologist should be aware of the association also, the mimicry of adenocarcinoma may otherwise lead to misdiagnosis. The distinction is an important one – duodenal carcinoids are much less aggressive than duodenal adenocarcinomas. Consequently their surgical extirpation is far more likely to lead to cure.

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Preservation of faecal continence during rises in ultra-abdominal pressure

SIR,—I was pleased to see the paper by Bannister, Gibbons, and Read[1] confirming that continence is sphincteric and not as previously thought dependent on an anorectal flap valve. Parks popularised the flap valve theory of continence which has gained wide acceptance.[2] In a recent study[3] we addressed the