Correspondence

Effect of morphine on the human oesophagus

...—How bitter the memory, but how sweet the vindication.

Dowlatshahi et al (Gut 1985: 26: 802–6) have described it and, more importantly, got it published. Five years ago my colleagues and I tried, but failed. In their study of the effect of morphine on the human oesophagus they found that morphine decreased lower oesophageal sphincter relaxation in response to swallowing and that, contrary to accepted medical dogma, it caused a small, albeit non-significant, increase in lower oesophageal sphincter pressure and in the amplitude of distal oesophageal peristaltic contraction.

Some years ago, in a very similar study (which was never accepted for publication) we carried out oesophageal manometry in six healthy volunteers on three separate occasions. After steady state basal recordings were obtained intravenously injections of saline, naloxone 0.8 mg or morphine 7.5 mg were given in random order to each subject on different days. Five minutes after the injection of the test drug, contraction of the body and sphincter of the oesophagus was stimulated by a subcutaneous injection of 12.5 mg of methacholine. Fifteen minutes later, after the effect of methacholine had worn off, maximal sphincteric contraction was stimulated by an intramuscular injection of 500 µg of pentagastrin.

Basal LOS pressure and oesophageal peristaltic pressure were not significantly different in the three study periods and neither saline nor naloxone affected the basal motility of the oesophageal body and sphincter or their response to methacholine or pentagastrin. Morphine significantly increased LOS pressure, (18±3 to 23±3 mmHg, mean±1 SD, p<0.05) and as in Dowlatshahi’s study there was a significant decrease in LOS relaxation (50±15%, mean±1 SD) in response to swallowing. In addition, morphine caused a non-significant increase in peristaltic pressure 10 cm above the sphincter (67±40 to 75±50 mmHg, mean±SD). When peristalsis was stimulated by methacholine, however, morphine premedication markedly augmented the peristaltic pressure immediately above the LOS (128±88 mmHg for morphine, 76±36 mmHg for saline and 63±16 mmHg for naloxone; mean±SD, p<0.025). Peristaltic pressures in the striated muscle portion of the oesophagus were not affected by morphine premedication and morphine did not affect the sphincter’s response to either methacholine or pentagastrin.

As the authors of the recent study aptly point out, the divergent results of their study when compared with others is not entirely unexpected, for if the oesophagus does indeed contain five different types of opioid receptors, why should they all behave in

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the same fashion in response to different opioid medications? Unfortunately, I and my colleagues were unable to convince reviewers of other journals of that important fact. Perhaps our observations, belatedly conveyed here in the form of a letter, will lend support to the careful study of Dowlatshahi et al. I wonder how many other research drawers contain ‘dead’ yet viable data?

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Books

Precancerous lesions of the gastrointestinal tract. Edited by B C Mason and J R Jass. (Pp. 174; illustrated; £22.50.) London: Bailliere Tindal, 1985. Pathologists are often amused when, having presented a paper, they are congratulated on the beautiful pictures with the scientific content forgotten. I am in danger of doing the same with this book. The colour photomicrographs are of the highest standard and the transparencies, which can be bought separately, will be even better. If pathology is not to be regarded as simply pattern recognition, the text of an atlas is important. In a short space the essential points are covered but perhaps in the interest of brevity, misleading and inaccurate statements have crept in. The difficult subjects of severe dysplasia, in situ and intramucosal carcinoma are not well tackled. In the gastric section we are told that a distinction between severe dysplasia and intramucosal carcinoma has important treatment implications yet it appears that severe dysplasia amounting to in situ carcinoma is almost always associated with invasive carcinoma. Again in the colorectal area severe dysplasia without invasion of the muscularis mucosa is regarded as in situ carcinoma when any form of invasion eliminates an in situ lesion. It is stated that no lymphatics are present in the colorectal mucosa when they are known to occur around the crypts. These cannot be regarded as major faults and may help in an important function – the promotion of discussion.

When another edition is contemplated a section on cytology would indicate the value of correlating cytological and histological appearances. This book will be useful to pathologists and clinicians and is good value.

D J POLLOCK

Corrections

Correction
In line 6 of the BSG abstract on toddler diarrhoea by Guerro, Brown and McNeish (Gut, October 1985, T.22) the words ‘mouth to caecum transit’ should read ‘mouth to anus transit’.

Correction
In the paper entitled ‘Effect of warfarin on cell kinetics . . . (Gut 1985; 26: 807–15) Figures a and b on p. 812 have been reversed in error.

In the leading article by J B Elder (Gut December 1985) p. 1280, second paragraph, line two should read ‘(2000 mg/kg/day) lasting from 875–903 days with plasma blood concentrations . . .’

News

Third European Symposium on Gastrointestinal Motility
This meeting will be held from 16–18 June 1986. Closing date for abstracts is 8 March 1986. Further details from Prof G Vantrappen, University Hospital, St Rafael-Gathuisberg, Herestraat 49, 3000 Leuven, Belgium.

Emergency in Gastroenterology
A symposium dealing with recent developments in this field will be held from 30 April to 2 May 1986 at Klinikum rechts der Isar, Munich, FRG. Details from PD Dr med G E Vogel, Ismaningerstrasse 22, D-8000 Munich 80, FRG.

FASEB Summer Research Conference
To be held from 20–25 July 1986 in Vail, Colorado, USA, on physiology and pathology of the splanchnic circulations. Details from the FASEB, Splanchnic Circulation Conference, 9650 Rockville Pike, Bethesda, Maryland 20814, USA.