PAIN IN GASTRIC ULCER

SIR,—I should like to support the suggestion by Dr James and Mr Kenefick (March 30, p. 562) that gastric-ulcer pain cannot usually be reproduced by stimulating it with weak hydrochloric acid. A similar situation probably occurs in duodenal ulceration and I have suggested [1] that the commonest type of pain, in the epigastrium just below the xiphisternum, arises from the lower œsophagus. This was based on evidence gained from perfusing the lower œsophagus with 0·1*N* HCl under accurate manometric control. It must be emphasised that I have no evidence to enable me to state that pain never arises directly from either a gastric or a duodenal ulcer, but it is rare. The same technique has been used during the last four years to investigate over 50 patients with gastric ulceration; in addition 200–300 ml. of acid was then instilled into the stomach and pressures were recorded for 30 minutes. The results have deliberately not yet been published since they are so controversial, as indeed was the original concept that the commonest duodenal-ulcer pain could arise from the lower œsophagus, and it is important to be certain of the evidence by investigating large numbers of gastric ulcers. In the past, failure to do this has led to many statements which have later proved wrong.

However, a preliminary analysis shows that the epigastric pain of gastric ulceration can also arise from the lower œsophagus. Direct stimulation of the ulcer is usually without effect, but rarely pain in other abdominal sites may be reproduced and occasionally a vague upper abdominal pain is associated with type-III gastric contractions. In direct contradistinction to what our textbooks tell us, the majority of gastric ulcers, when their whole natural history is considered, are either painless or associated with nausea, vomiting, and loss of appetite. Many of these vague symptoms may be caused by reflux of bile into the stomach, but such patients always have a negative epigastric-pain reproduction test.

I should like to suggest that Dr James and Mr Kenefick continue their important studies, but they must have numerous patients, and pay careful attention to the anatomical site of the pain and when it last occurred. In my experience, eventually local stimulation will cause pain in the rare subject who is sensitive. Perhaps it would be wise to use 0·1*N* HCl and also try the effect of bile-salts in combination.

Balloon distension and acid perfusion tests have now demonstrated that œsophageal pain can be referred to the epigastrium as well as retrosternally. The gastric mucosa is luckily insensitive to stimulation by acid in the normal subject, but the lower œsophagus responds to heat, cold, trauma, and acid in a manner reminiscent of the skin which is supplied by the somatic nervous system. There is no doubt that the gastro-œsophageal squamocolumnar junction is the site of a sharp transition in pain sensation. This should not come as a surprise to anyone who has injected piles and knows the sharp difference in sensation that occurs at the anal squamocolumnar junction.

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1. *Br. med. J.* 1972, i, 683.