symptoms might have some different explanation and should be treated with caution. Another paper showed that it was extremely difficult to measure why some surgeons advised surgery for a duodenal ulcer and that it was probably a random phenomenon.[8] These results were not negative or destructive, although they could be interpreted as such by opponents. They were positive because they showed that the "symptoms ruled OK."

In the mid-1950s and '60s two specific investigations became popular: tests of gastric acid secretion and oesophageal motility. Their clinical use was analysed critically by, respectively, Baron[9] and myself[10]—two people who had spent many years on the subject but could stand back and discuss honestly and intellectually their true value. I believe it is now time that endoscopists critically analysed the accumulating data about endoscopic appearances and the histology of the upper gastrointestinal tract in a similar fashion for the benefit of their colleagues and patients as well as those who pay the bills.

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Endoscopic and histological findings in subjects with dyspepsia

SIR—The paper by Dr Roar Johnsen and colleagues presented further evidence that there was poor correlation between the macroscopic appearances of the upper gastrointestinal tract, microscopic histological findings, and the symptoms of dyspepsia.[1] They misquoted a paper of mine from 1985[2] as claiming that corpus (sic) and antral gastritis could cause symptoms. This is a pity because exactly the opposite was my hypothesis and the conclusions from that paper were entirely in agreement with theirs: "no relationship was found between the length of history, severity of pain and histological abnormalities."[2]

In the past 20 years an increasing number of tests have come on to the market in gastroenterology and we are under an obligation to analyse them for their usefulness. Armed with the latest from the Mayo Clinic in the late 1960s, I and others tackled hiatus hernia as a disease. It became clear that there were three basic abnormalities to account for the symptoms of gastro-oesophageal reflux: an anatomical hiatus hernia, oesophagitis, and reflux. The challenge was to find out a correlation between symptoms and each variable. Try as hard as we could, we could not find much correlation between the investigations and the symptoms. The same approach was applied to the symptoms of duodenal ulcer disease[3] and gall bladder dyspepsia.[4] The symptoms of an ulcer had anyway previously been shown to be non-specific,[3,5] and the radiological appearances of a duodenal ulcer did not correlate with symptoms either.[6]

Research to show that the epigastric pain of duodenal ulceration could be reproduced by acid in the lower oesophagus but not by acid in the duodenum or stomach[7] did not seem to worry anybody, yet it fitted into the overall hypothesis that the results of investigations in relation to