Leading article

Duodenogastroduodenal reflux

Many gastroenterologists respond with interest to a colleague who begins a discussion about duodenogastroduodenal reflux or biliary gastritis. In part, the interest stems from a knowledge of controversies in the clinical and scientific literature, but it seems to be reinforced when, from time to time, endoscopy allows direct observation of bile stained duodenal fluid flowing back through the pylorus into a patient’s stomach. There is an immediate sense of observing something potentially nasty for the gastric mucosa. After all, is it not a credo of gastroenterology that bile in the stomach is both unnatural and unhealthy? Cold logic will then remind the endoscopist that the circumstances of endoscopy probably render his observation irrelevant to the usual behaviour of his patient’s gastric outlet, but, in defiance of logic, the feeling that duodenogastroduodenal reflux may have clinical significance will persist.

In this issue of Gut, Muller-Lissner et al describe a complex and sophisticated procedure they have used to measure duodenogastroduodenal reflux in normal subjects and patients with gastric ulcer. Based on the use of $^{99m}$Tc-HIDA as a tracer of bile, they have attempted to overcome problems of previously described scintigraphic methods by intubating the stomach to obtain samples of gastric contents and by using a gamma camera to measure the amount of $^{99m}$Tc within the descending duodenum. They have then been able to calculate duodenogastroduodenal reflux rate, defined as the quantity of technetium which enters the stomach each minute, expressed as a fraction of the quantity within the duodenum. As with any complicated method, the paper needs careful study if the reader is to understand the details of the procedure, but investigators with experience of scintigraphic duodenogastroduodenal reflux studies will recognise that this new procedure has circumvented some major problems, not least the difficulty of overlap between stomach and liver, or stomach and duodenum.

The authors’ findings lead them to some conclusions which merit attention. Firstly, they assert that duodenogastroduodenal reflux occurred in all their healthy subjects – that is, that it is a normal event. Other recent studies of duodenogastroduodenal reflux based on intraduodenal infusion of marker substances have come to the same conclusion, but their validity is substantially dependent on evidence that transpyloric intubation does not itself induce an abnormality. It now seems clear that duodenogastroduodenal reflux is indeed a normal phenomenon and thus the identification of abnormal reflux must depend on its quantitation.

Muller-Lissner et al have compared duodenogastroduodenal reflux in their normal subjects and patients with gastric ulcer and they challenge the long established belief that reflux is increased in the latter. No doubt their conclusion will be disputed by supporters of the hypothesis which states that an increased amount of intragastric bile is a factor contributing to the development of Type I gastric ulcer; a wish to see measurements of
duodenogastric reflux in fasting ulcer patients may be anticipated. Much of
the evidence in favour of duodenogastric reflux being a feature of gastric
ulcer stems from non-quantitative studies using the Capper technique. The
qualitative nature of these observations does not invalidate the finding
that ulcer patients behave differently from normal subjects. Nevertheless,
the challenge to the hypothesis is clear and the point in question is certainly
sufficiently important to need further investigation.

Of course, it is gastritis rather than chronic gastric ulcer that is the usual
accompaniment of the major duodenogastric reflux which may follow
gastric surgery. Much uncertainty has been repeatedly expressed regarding
its clinical significance, given that some patients with this 'biliary gastritis'
are completely free of symptoms, while, in others, gastritis is the only
abnormality to be found which might explain distressing epigastric
discomfort, nausea, and vomiting. It appears, however, that there is a
quantitative difference in reflux between symptomatic and asymptomatic
postoperative patients. Bile acid concentrations in fasting gastric aspirates
are higher in the symptomatic group and recent evidence indicates that
postprandial reflux is also greater in those with symptoms. In patients
who underwent revisional surgery which created a Roux loop, decreased
reflux was associated with clinical improvement. While these findings are
not particularly surprising, they do constitute an objective demonstration
that the magnitude of reflux can have clinical significance.

How does duodenogastric reflux in the fasting subject compare with
reflux during emptying of a meal? In normal subjects the reflux rate
calculated by Muller-Lissner et al was much the same in the fasting and
postprandial studies. At first sight this observation is in conflict with other
evidence that there is less reflux after meals than during fasting. The
disagreement almost certainly depends upon the different methods used in
the two investigations and the consequent difference in the way reflux was
defined. Clearly, this makes comparison difficult. Even comparison of
different Tc-HIDA studies is not easy, as at least three ways of calculating
duodenogastric reflux have now been used by different investigators.

Some thought should be given to the best definition of reflux, not only in
the interests of clear communication, but also to allow the important
concepts of its physiology and pathophysiology to emerge. The contractile
activity of the distal stomach and duodenum which generates retrograde
flow at the pylorus (duodenogastric reflux) obviously differs from the
activity which expels gastric contents back to the duodenum.

Accumulation of duodenal contents within the stomach (duodenogastric
reflux) depends on their interaction. In the context of gastro-oesophageal
reflux, it is generally recognised that delayed clearing of refluxed fluid may
be as important as the refluxing process itself in the aetiology of mucosal
injury – presumably the same is true in the stomach. If so, the mechanisms
responsible for gastric emptying are of interest, particularly those
correctly concerned with fluids. In the fasting state, clearance of liquid from
the stomach is apparently accomplished during Phase III of the interdigestive
motor complex (the phase of powerful, repetitive antral and duodenal
contractions), which suggests that antral contractile activity is largely
responsible. After meals, however, gastric emptying of liquids is thought to
be substantially independent of antral function. It is therefore interesting
that although patients with gastric ulcer empty liquid meals normally,
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emptying of solids which is dependent on antral function, is delayed.\textsuperscript{12} The inference that clearance of fluid from the fasting stomach may also be impaired in such patients deserves more investigation.

There are now signs of progress in the study of duodenogastric reflux. With the development of methods for its quantitation, we may now accept that some reflux occurs as a normal event and that greatly increased reflux is indeed responsible for gastric mucosal injury and for symptoms, at least in post-gastrectomy patients. It is now necessary to put on trial some of our cherished notions about its importance in relation to gastric ulcer and Type B gastritis in the intact stomach. Once we establish when abnormal reflux occurs and when it matters, rational treatment will become a possibility.

R C HEADING

University Department of Therapeutics and Clinical Pharmacology, Royal Infirmary, Edinburgh.

References