Non-deglutitive motor activity of the oesophagus

Non-deglutitive motor activity of the oesophagus is usually considered a sign of disordered motility. The absence of spontaneous contractions in the normal gutt is related to the nature of the oesophageal smooth muscle cells, which have a stable membrane potential and do not show the characteristic slow wave activity found elsewhere in the gastrointestinal tract. In pathological conditions such as achalasia and, to a lesser degree diffuse oesophageal spasm, non-deglutitive motor activity occurs particularly when there is stasis of food in the oesophagus. When treatment results in disappearance of food stasis, the non-deglutitive simultaneous contractions disappear as well. Recent findings show that, in addition to these well-known tertiary contractions, several other types of non-deglutitive motor activity exist, even in healthy subjects.

In 1986 Clouse and Ferney described a manometric abnormality they termed rhythmic spontaneous peristaltic contractions. This pattern consisted of a sequence of repetitive contractions that originated immediately below the region of the upper oesophageal sphincter. Some rhythmic sequences were initiated by swallows, but others were not related to deglutition. Some rhythmic contraction waves propagated a short distance into the proximal oesophagus. Other rhythmic sequences consisted of repetitive simultaneous contractions in the uppermost portion of the gutt. The rhythms varied from eight to 16 per minute. These contraction sequences were associated with long periods of increased baseline pressure in the distal oesophagus. The technique did not determine whether this was due to failed propulsion of a swallowed bolus or to a common cavity phenomenon due to transient relaxation of the lower oesophageal sphincter.

In 1987 Kahrials et al. reported a patient who was unable to belch. Gastro-oesophageal reflux of gas resulted in a peculiar manometric pattern consisting of repetitive peristaltic pressure waves that were not related to swallowing. This could be precipitated by instillation of 210 ml of air into the stomach. Combined fluoroscopic and manometric findings showed that transient relaxations of the lower oesophageal sphincter resulted in gastro-oesophageal reflux of gas with distension of the oesophageal lumen as was evident by the 10–15 mm Hg oesophageal common cavity pressure. These oesophageal distensions did not cause relaxations of the upper oesophageal sphincter but elicited secondary oesophageal peristaltic contractions, which in turn resulted in clearance of gas from the oesophageal body. This sequence was repeated several times at a rhythm of about nine per minute.

In 1989 Nixon and Koch described a manometric pattern they termed recurrent autonomous oesophageal peristalsis. The pattern was seen in five patients; four of them had reflux oesophagitis, one had symptoms suggestive of gastro-oesophageal reflux. The pattern consisted of repetitive peristaltic contractions in the body of the oesophagus not related to swallowing and occurring at a rhythm of four to eight contractions per minute and lasting for periods of two to eight minutes. In four of the five patients an increase in baseline pressure of 5–10 mm Hg preceded the peristaltic contractions. These repetitive non-deglutitive peristaltic contractions were probably induced by gastro-oesophageal reflux of acid or gas in at least four of the five patients.

In 1989 Shay et al. found a manometric pattern they termed cycling in patients with documented gastro-oesophageal reflux disease. Peristaltic or simultaneous oesophageal contractions occurred in clusters of four or more repetitive waves and lasted for several minutes. The contraction peaks were preceded by a pressure plateau of 5–10 mm Hg. These clustered contractions nearly always developed at a time when the lower oesophageal sphincter pressure was below 5 mm Hg. Scintigraphic findings during simultaneously manometry and pH monitoring indicated that cycling resulted from repeated reflux events and their oesophageal clearance. The clearing contractions were either induced by swallows or elicited by oesophageal distension due to refluxed acid or gas. When repeated reflux episodes resulted in repetitive simultaneous oesophageal contractions that followed each other at short intervals, the pattern resembled that seen in primary oesophageal motility disorders. Cycling was noted in 18 of 52 patients with gastro-oesophageal reflux disease; 17 of them had oesophagitis. Moreover, cycling occurred in nine of 10 patients with Barrett’s oesophagus.

The manometric patterns described in the four papers have some features in common. In all four clustered contractions were reported, which may or may not be related to swallowing, which may be peristaltic in nature, or which develop simultaneously at different levels of the oesophagus. In most patients there is evidence that the pattern is elicited by gastro-oesophageal reflux of acid or gas. The repeated reflux of gas was clearly shown in the patient with dysfunction of the belch reflex. Cycling was also definitely related to gastro-oesophageal reflux. The contractions occurring during episodes of recurrent autonomous oesophageal peristalsis were preceded by a small pressure plateau in four of the five patients. This, together with the fact that all four patients had evidence of reflux oesophagitis, suggests that the contractions were induced by gastro-oesophageal reflux. The rhythmic spontaneous contractions in the upper oesophagus may also have been related to acid gastro-oesophageal reflux, because sometimes the contraction sequences were preceded by increased oesophageal baseline pressure (due to a common cavity phenomenon or inadequate clearance of the swallowed bolus); moreover half of the patients had signs of reflux oesophagitis.

When the repetitive contractions are peristaltic and are preceded by a small pressure plateau suggestive of a common cavity phenomenon, the relation to gastro-oesophageal reflux is clear. In some cases, however, the clustered contractions appear as a series of tertiary contractions of the type often found in primary oesophageal motility disorders. Simultaneous pH measurements or monitoring of lower oesophageal sphincter pressure may then be required to identify the true nature of the phenomenon.

The phenomenon reported in the paper by Janssens et al. in this issue is of a different nature. All subjects included in the study were healthy young persons without any symptoms related to the oesophagus. The contractions were not related to swallowing and usually consisted of several bursts of contractions, each burst composed of two to five simultaneous contractions of rather low amplitude (20 mm Hg). This pattern was limited to the distal, smooth muscle portion of the oesophagus. It usually coincided with the end of phase 2 or with phase 3 of the migrating motor complex in the lower oesophageal sphincter and stomach. Such a pattern of non-
deglutitive, simultaneous repetitive contractions could easily be misinterpreted as a sign of primary or secondary oesophageal motility disorder.

The mechanism of this phenomenon remains unknown. There was no manometric evidence of gastro-oesophageal reflux in these subjects preceding the onset of the contraction bursts. Whether motilin is involved remains to be determined.

It seems, therefore, that a pattern of clustered simultaneous contractions, not induced by deglutition, may be seen not only in disorders of primary oesophageal motility but also in patients with gastro-oesophageal reflux of acid or gas. In both instances the stimulus eliciting this non-deglutitive motor activity is continuous or repeated distention of the oesophagus by swallowed or refluxed material. A third type of non-deglutitive motor activity in the smooth muscle portion of the oesophagus is probably the oesophageal component of phase 2 and phase 3 of the migrating motor complex. Now that ambulatory manometry has become available, it is necessary to identify these patterns if perfectly normal oesophageal migrating motor complex activity is not to be interpreted as a sign of oesophageal motility disorders or as a cause of oesophageal chest pain.

In this context attention is drawn to the fact that 24 hour ambulatory pressure monitoring indicates that non-sequential (simultaneous) pressure peaks are often found in normal subjects. Before redefining the upper limit of normal for the incidence of non-peristaltic deglutitive pressure waves, it seems appropriate to point out some fundamental shortcomings in the technique of prolonged recording that may lead to erroneous conclusions. The most important limitation of presently available systems is the lack of a reliable deglutitive signal. Consequently, available prolonged oesophageal manometric recordings cannot differentiate between deglutitive and non-deglutitive motor activity and they are unable to distinguish multiple pressure peaks induced by a single deglutition from a pressure complex induced by repetitive swallowing, or to separate dry swallows from wet or solid swallows.

Although simultaneous contractions are rare after wet or solid swallows (≤10% of swallows), they occur much more often after dry swallows. Moreover, simultaneous oesophageal pressure peaks may occur as a normal phenomenon when swallows are taken in rapid succession, due to the phenomenon of deglutitive inhibition.10

In conclusion, it is recommended that redefinition of the upper limit of normal incidence of non-peristaltic and non-deglutitive pressure waves should be considered but should be based on recording techniques that reliably signal all deglutitions.

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