Functional relationships between cricopharyngeal sphincter and oesophageal body in response to graded intraluminal distension

N A ANDREOLLO, D G THOMPSON, G P N KENDALL, AND R J EARLAM

From the Departments of Gastroenterology and Surgery, The London Hospital, Whitechapel, London

SUMMARY Responses of the cricopharyngeal sphincter to graded intraluminal distension were studied in order to determine its response threshold and to define the functional relationship between the sphincter and oesophageal body. Nine normal subjects underwent manometric study using a multilumen tube with an attached inflatable balloon sited 10 cm below the sphincter. Sphincteric and oesophageal motor responses to six graded balloon inflations were recorded in each subject. The sphincter responded to distension with increasing rise in pressure, from a median value of 42.5 mmHg at lowest levels of distension to 95 mmHg at maximal tolerated distension. Non-swallow related contractile activity was stimulated in the oesophageal body proximal to the distension and increased in quantity as inflation progressed. Distal propagation of this secondary activity was progressively inhibited with increasing distension. These interrelated changes thus show the normal upper oesophageal clearance responses to intraluminal distension. It is suggested that their more widespread application, in addition to standard manometric techniques, might provide a more rational evaluation of those patients suspected to have impaired oesophageal clearance, but in whom standard manometry is non-diagnostic.

The cricopharyngeal (upper oesophageal) sphincter, situated at the intersection of the airway and the first part of the alimentary tract, is formed mainly by the cricopharyngeal muscle, with additional fibres from the circular muscle of the oesophagus distally and the inferior pharyngeal constrictor proximally.

The coordinated relaxation and contraction of this sphincter constitutes an integral part of normal deglutition, ensuring the passage of a bolus from the pharynx into the oesophagus, and additionally forms a dynamic barrier to prevent oesophagopharyngeal reflux and spillover into the tracheobronchial tree. This latter function is of particular importance because breakdown of this mechanism may be related to oesophagopharyngeal regurgitation and aspiration pneumonitis.

Measurements of the resting sphincter pressure and its relationships to the oesophageal body have previously been attempted, but the data are incomplete and often contradictory. Some groups have observed an increase in pressure during intraluminal distension, others in contrast have reported relaxation.

Preliminary studies of normal human oesophageal responses to experimental distension in our laboratory have shown that increased secondary peristalsis results. This response was abnormal in some patients with non-organic dysphagia who showed delayed isotope transit despite normal standard manometry. In this study a more systematic investigation of the responses of the upper oesophageal sphincter and body to graded distension was made in order to define the normal pattern more fully and to enable more accurate recognition of disordered function in non-organic dysphagia to be made in the future.

Methods

SUBJECTS
Nine healthy volunteers (eight men, one woman,
The multilumen oesophageal manometric tubes used for the study (Fig. 1) were constructed from triple lumen polyvinyl chloride (PVC) capillary tubing (internal diameter (ID) 0.5 mm, external diameter (ED 1.5 mm, side hole diameter 0.5 mm, Dural Plastics, Australia) bonded around a central PVC tube (ID 1.0 mm, ED 1.6 mm Portex, Hythe, Kent, England) and with a latex balloon constructed from a 5 cm length of condom attached 15 cm from its tip. Three ports 0.7 cm apart were sited at the proximal end of the tube to ensure optimal detection of the upper sphincter, while five others sited at 5 cm intervals above and below the balloon, enabled detection of oesophageal responses to distension. All tubes were continuously perfused at a rate of 0.3 ml/min using a standard Arndorfer type pneumohydraulic infusion system. Transmitted pressure changes were detected proximally using attached strain gauge transducers (Gaeltec, S8b, Skye, Scotland) the outputs of which were recorded on an eight channel chart recorder (Watanabe Linear Corder Mark VII, Tokyo, Japan), run at a paper speed of 100 mm/min to provide a permanent graphic record. Because of the magnitude of the sphincter response, the calibration of the three sphincter channels was adjusted until their amplitudes were half those of the oesophageal body channels.

**APPARATUS**
The multilumen oesophageal manometric tubes used for the study (Fig. 1) were constructed from triple lumen polyvinyl chloride (PVC) capillary tubing (internal diameter (ID) 0.5 mm, external diameter (ED 1.5 mm, side hole diameter 0.5 mm, Dural Plastics, Australia) bonded around a central PVC tube (ID 1.0 mm, ED 1.6 mm Portex, Hythe, Kent, England) and with a latex balloon constructed from a 5 cm length of condom attached 15 cm from its tip. Three ports 0.7 cm apart were sited at the proximal end of the tube to ensure optimal detection of the upper sphincter, while five others sited at 5 cm intervals above and below the balloon, enabled detection of oesophageal responses to distension.

All tubes were continuously perfused at a rate of 0.3 ml/min using a standard Arndorfer type pneumohydraulic infusion system. Transmitted pressure changes were detected proximally using attached strain gauge transducers (Gaeltec, S8b, Skye, Scotland) the outputs of which were recorded on an eight channel chart recorder (Watanabe Linear Corder Mark VII, Tokyo, Japan), run at a paper speed of 100 mm/min to provide a permanent graphic record. Because of the magnitude of the sphincter response, the calibration of the three sphincter channels was adjusted until their amplitudes were half those of the oesophageal body channels.

**ANALYSIS OF DATA**
Resting sphincteric pressure was determined by

---

**Study Protocols**
After a standard manometry study had been carried out on each volunteer to exclude oesophageal motor abnormality and to identify the location of the upper sphincter, the motor responses of the sphincter and proximal oesophagus to six predetermined balloon volumes (1, 2, 4, 6, 8, and 10 ml), were recorded for three minutes at each inflation. Each inflation was achieved by injecting a known volume of tap water into the balloon channel of the tube and was separated by a control period of three minutes, during which the balloon remained deflated. The order in which the six inflations were performed was randomised for each individual. Because the pressure recorded within the sphincter is radially asymmetrical, the sphincter ports on the tube were maintained posteriorly orientated by taping the tube to the side of the mouth throughout the study after the correct position had been achieved.

During each balloon inflation, subjects were asked to indicate the occurrence of any retrosternal discomfort and to report any other sensations.

---

**Measurement of Balloon Diameter**
To provide an estimate of oesophageal diameter at each inflation volume, the diameter of the balloon was measured using calipers, both directly on a laboratory bench, and in three subjects using fluoroscopy during oesophageal distension. Any possible magnification effects of fluoroscopy on balloon diameter were corrected for by comparison with a radioopaque segment of known length attached to the tube.

---

**Fig. 1** The recording tube in situ and the position of the pressure recording sites and balloon relative to the upper oesophageal sphincter (UOS).
Functional relationships between cricopharyngeal sphincter and oesophageal body

Fig. 2  Relationship between the balloon diameters measured directly on the laboratory bench with those measured at fluoroscopy. Values represent median and range of three studies.

measuring the difference between the values recorded from the sphincter ports when placed alongside the subjects neck and those recorded from the tube sited in the sphincter. Responses to distension were calculated by measuring the change from this baseline value to that of the new pressure, selecting for measurement whichever of the three ports showed the maximal response.

Secondary peristaltic responses to distension were determined at 5 cm proximal and at 5 and 10 cm distal to the balloon, by counting the number of non-swallow initiated waves occurring at each site during each inflation period and comparing this number with that found during the preceding control period.

In each subject the amplitude of each primary peristaltic wave was measured proximally and distally to the balloon, between and during each distension period, by direct measurement from the chart.

To determine response thresholds, the first distension volume which induced a significantly different response from the control data, was used. Because the data did not always appear to be normally distributed, Wilcoxon’s rank sum test was used to test the likelihood that observed changes could have been due to chance.

Results
A total of 49 intraluminal oesophageal distensions were carried out in nine normal subjects. All

Fig. 3 (a) Normal manometric pattern of the upper oesophageal sphincter and oesophageal body during two swallows in a normal subject (balloon deflated). (b) The manometric responses of the cricopharyngeal sphincter and oesophageal body during (6 ml) distension, in one subject. Note the increase in pressure at the UOS ports. Proximal to the balloon the number of secondary contractile waves can be seen to increase. There is no coincident distal change in secondary activity but the amplitude of the primary peristaltic wave initiated by swallowing 50 seconds after balloon inflation, can be seen to be attenuated distally.
reported retrosternal discomfort at 8 ml inflation and five were unable to tolerate 10 ml.

**Balloon Volume Diameter Studies**
A good relationship was found between changes in balloon diameter when measured in air and at fluoroscopy (Fig. 2), indicating that at the distension volumes used, no significant distortion of the balloon was produced by the oesophagus. Inflation volume was therefore used as an index of oesophageal diameter for the remaining studies.

**Upper Sphincter Responses**
Basal sphincter pressures ranged between 25 to 50 mmHg (median value 30 mmHg). Oesophageal distension caused a rapid rise in sphincter pressure which was sustained for the duration of the distension period (Figs. 3a, 3b). The least distension required to initiate a sphincteric response in all subjects was 2 ml (1.0 cm diameter). The magnitude of rise in the sphincteric pressure above basal values increased progressively as the distension increased, rising from a median value of 42.5 mmHg (range 37.5-55 mmHg) at 2 ml, to 95 mmHg (range 50-110 mmHg) at 10 ml (Fig. 4). During inflation to 6 ml and above, the most distal sphincter port also showed phasic contractions which often resembled the secondary peristaltic activity in the adjacent oesophagus. In some subjects this phasic change was so large that peak pressure values rose off-scale (Fig. 3b).

**Oesophageal Body Responses**

*Secondary peristalsis*
The proximal oesophagus responded to distension by an increase in the number of secondary contractile waves (Fig. 5); the threshold for this increase being 4 ml (0.5 cm diameter). The number of these proximal non-swallow related contractions increased progressively as distension increased (Fig. 5). In contrast, the proportion of secondary waves detected distal to the balloon fell progressively, indicating a distension dependent inhibition of propagation across the balloon.

*Primary peristalsis*
The amplitude of swallow initiated pressure waves was measured, 5 cm proximal and 5 and 10 cm distal to the undistended balloon, and the average for each individual calculated. Proximally the median amplitude for the group was 28.5 mmHg (range 23-42 mmHg) and distally 28.5 mmHg (range 24-40 mmHg). During distension the proximal amplitude was initially unchanged (median value 26.2 mmHg, range 23.6-33 mmHg, p>0.05 at 2 ml), but increased to 61.7 mmHg (42-70 mmHg) at 10 ml (p<0.05), from a threshold of 6 ml (1.8 cm) (Fig. 6). Distal to the balloon the median amplitude fell progressively from a threshold response of 18.3 mmHg (range 10.3-38.7 mmHg, p<0.05) at 2 ml, to 7.5 mmHg (6.2-12 mmHg) at 10 ml.

**Discussion**
These results show that the normal oesophagus responds to intraluminal distension with a stimula-
Aboral displacement of the sphincter ports out of the sphincter cannot explain these results as the response was seen in channels which also showed the characteristic swallowing pattern of the sphincter. Furthermore, they were occasionally observed in all three sphincter ports.

The progressive modification of primary peristaltic activity with increasing distension, resulting in proximal enhancement and distal inhibition of contractile activity is a new observation although at extremes of distension distal peristaltic inhibition is known to occur.\(^\text{10}\)

The close stimulus response relationship between the primary and secondary peristaltic responses suggests that a final common pathway is influenced by the distension, while the low thresholds for recruitment of all responses indicate that the responses probably represent part of the normal deglutitive process and are not just recruited for clearance of obstructing material.

While our data do not permit comment about the relative sensitivities of upper and lower oesophagus to distension, animal studies have reported\(^\text{11}\) that a gradient of responsiveness to distension exists, which declines in magnitude with distance from the upper sphincter, even after bilateral vagal blockade. This would suggest that the passage of intraluminal material is mediated by an intrinsic aboral gradient extending from the upper to the lower sphincter, which facilitates antegrade propulsion and prevents regurgitation or aspiration.

The in vitro data indicate, as might be expected, that when a highly compliant balloon is distended by indistensible liquid, its volume-diameter relationships are constant once the balloon is stretched to achieve a roughly spherical shape. The close similarity between these relationships in vitro and in vivo suggests that oesophageal forces applied to the balloon were small because in vitro balloon pressures recorded over the range of inflation volumes used were small (10–15 mmHg). If this is so then it suggests that oesophageal distension is accompanied by relaxation of the oesophagus not only below, but also at the site of the distension.

The relationship between degree of balloon distension and oesophageal response also deserves comment. All the measured responses showed greater changes as the balloon volume rose and quite marked changes occurred over a relative small change in diameter at the limit of distension. This pattern of response is probably the result of changes in circumference of the oesophagus which obviously increase proportionally more than diameter as distension increases.

The breakdown of the cooperative responses we have shown here is likely to have clinically important
consequences. Although no specific studies have yet been conducted, it is of relevance to note that patients with symptoms of regurgitation and pulmonary disease have been reported to show upper sphincter hypotension, \(^{13}\) that patients with abnormal oesophageal peristalsis suffer increased pulmonary infections \(^{14}\) and, that patients with unexplained dysphagia may show abnormal secondary peristalsis despite normal primary peristaltic activity. \(^{15}\)

It is possible, therefore, that the measurement of cricopharyngeal sphincter and oesophageal body responses to graded distension might prove to be a useful addition to routine manometry for the investigation of patients with unexplained dysphagia, regurgitation or pulmonary disease, and might uncover impairment of the oesophageal clearance process which would otherwise escape detection.

Dr Andreollo is a Research Fellow of the Brazilian National Council of Scientific and Technological Development (CNPq) and Unicamp (SP). Dr Thompson is a Wellcome Trust Senior Lecturer in Medicine. Dr Kendall was the WEG Knott Research Fellow of the British Digestive Foundation. We would like to thank Miss E Walker for her help in the performance of the studies, Dr D L Wingate (Gastrointestinal Science Research Unit) for his helpful advice and Ms Julie Rostron for typing the manuscript. A preliminary report of some of the data in this paper was presented to the British Society of Gastroenterology in April 1986.

References