Preservation of postural control of transient lower oesophageal sphincter relaxations in patients with reflux oesophagitis

A C Ireland, J Dent, R H Holloway

Abstract

Introduction—In normal subjects, transient lower oesophageal sphincter relaxations (TLOSRs) and gas reflux during belching are suppressed in the supine position. Supine reflux, however, is a feature of reflux disease.

Aims—To investigate whether postural suppression of TLOSRs and gas reflux is impaired in patients with reflux disease.

Patients—Ten patients with erosive oesophagitis.

Methods—Oesophageal manometry was performed during gastric distension with 750 ml carbon dioxide. Measurements were made for 10 minutes before and after distension in both sitting and supine positions.

Results—In the sitting position gastric distension substantially increased the rate of gas reflux (median (interquartile range)) as evidenced by increases in oesophageal common cavities from 1 (0–1)/10 min to 7 (5–10)/10 min and TLOSRs from 1 (1–1.5)/10 min to 6 (2.5–8)/10 min. However, this effect was suppressed in the supine position in all but one patient (TLOSRs 0 (0)/10 min to 1 (0–4.5)/10 min, common cavities 0 (0)/10 min to 0.5 (0–2)/10 min).

Conclusions—Postural suppression of TLOSRs and gas reflux is generally preserved in reflux disease.

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Keywords: gastro-oesophageal reflux; lower oesophageal sphincter; posture; oesophageal manometry

In normal subjects and most patients with gastro-oesophageal reflux disease, reflux of acid occurs predominantly during transient lower oesophageal sphincter (LOS) relaxations (TLOSRs). These relaxations are also the major mechanism underlying the reflux of gas during belching. We have proposed therefore that acid reflux may be a variant of the belch reflex.

In normal subjects, TLOSRs and gas reflux are almost completely suppressed in the supine posture. Reflux disease is characterised by an increased frequency of reflux episodes and, particularly in patients with more severe oesophagitis, by increased levels of recumbent reflux. These observations suggest that there may be defective postural suppression of TLOSRs in patients with reflux disease. The aim of this study therefore was to investigate the influence of posture on TLOSRs and gas reflux in patients with erosive or ulcerative reflux oesophagitis.

Methods

PATIENTS

We studied 10 patients, all men, with gastro-oesophageal reflux disease. Their mean age was 59 (42–71) years and all had erosive oesophagitis (Hetzel grade 2–3). All patients gave written informed consent and the study was approved by the research ethics committee of the Royal Adelaide Hospital.

MANOMETRIC TECHNIQUE

Oesophageal manometry was performed using an eight channel assembly incorporating a sleeve sensor. Intragastric pressure was recorded by a side hole located 1 cm beyond the distal margin of the sleeve. Side holes at the proximal sleeve margin and 4, 8, 12, and 16 cm more proximally recorded motility in the oesophageal body, and a side hole in the pharynx monitored swallowing. The sleeve and gastric and oesophageal side holes were perfused with degassed distilled water at a rate of 0.6 ml/min using a low compliance pneumohydraulic capillary infusion system. The pharyngeal side hole was perfused at a rate of 0.3 ml/min to obtain an accurate indication of the occurrence of swallowing but minimise the effect of the assembly on the swallow rate. Each lumen was connected to external pressure transducers (Deseret Medical Inc, Sandy, Utah, USA), the output of which was displayed on a 12 channel polygraph (model 7D; Grass Instruments, Quincy, Massachusetts, USA) at a paper speed of 2.5 mm/s.

Abbreviations used in this paper: LOS, lower oesophageal sphincter; TLOSR, transient LOS relaxation.
Studies were performed after a four hour fast and, in smokers, after at least six hours abstinence from smoking. Drugs that might affect oesophageal motility were discontinued for 24 hours before the study. After topical nasal anaesthesia, the catheter was introduced and positioned with the sleeve straddling the lower oesophageal sphincter (LOS). The patients were then allowed to accommodate to the assembly for 15 minutes. Figure 1 is a schematic representation of the protocol. With the patients sitting, baseline recordings were made for 10 minutes, after which the stomach was distended abruptly with 750 ml CO₂ by the instillation of a two part radiological mixture (Fields Negative C; Fields Group Chemicals, Sydney, NSW, Australia) into the stomach via the large central channel of the assembly.⁵ ⁶ Recordings were continued for 10 minutes. The patients then lay supine and recordings were made for 10 minutes, after which the stomach was again distended with 750 ml CO₂ and recordings continued for a further 10 minutes. Immediately after the second post-gas period, the patients sat up and another 10 minutes of recordings were made. During the study, subjects were instructed not to belch.

DATA ANALYSIS
Basal LOS pressure was measured at end expiration and referenced to intragastric pressure. A visual mean was taken for each minute of the 10 minute recording period and an overall mean calculated. The tracings were analysed for the presence of common cavities and the occurrence of TLOSRs. TLOSRs were scored according to previously defined criteria.¹⁵ Oesophageal common cavities were used as a marker of gas reflux, and defined as an abrupt increase in intraoesophageal pressure to intragastric pressure in at least the two distal oesophageal recording channels.⁵ ⁶

STATISTICAL ANALYSIS
The frequencies of TLOSRs and oesophageal common cavities were analysed using the Friedman and Wilcoxon signed rank tests. Basal LOS pressure was analysed using analysis of variance and the paired Student’s t test. Data for TLOSRs and common cavities are expressed as median (interquartile range); data for basal LOS pressure are expressed as mean (SEM). p<0.05 was accepted as indicating statistical significance.

Results
When in the sitting position, patients exhibited a low rate of common cavities. Gastric distension produced a prompt and sevenfold increase in the rate of oesophageal common cavities (p<0.01; fig 2). When the patients lay supine, the rate of common cavities fell to predistension levels and subsequent gastric distension induced a small but significant increase in the rate of common cavities (p<0.05). The rate of common cavities in the 10 minutes after distension when the patients were supine was lower in nine of the 10 patients than that when they were sitting. The median was significantly lower as a group (0.5 (0–2)/10 min; p<0.01) when supine than when sitting (7 (5–10)/10 min). One patient, however, retained a high rate of gas reflux in the supine position. On resuming the sitting position, there was no significant increase in the rate of common cavities (3 (2–4)/10 min).
Figure 3 summarises the patterns of LOS and oesophageal motility associated with gas reflux. A total of 138 common cavity episodes was scored, 116 while sitting and 22 while supine. About half (51%) of common cavities occurred during TLOSRs which was the most common mechanism overall; 16% occurred during swallow induced TLOSRs and 30% during periods of absent LOS pressure. Posture had a significant effect on the patterns of motor activity associated with common cavities (p < 0.01); absent LOS pressure was more common while sitting, and swallow induced LOS relaxation more common while supine. However, almost all (9/10) of the common cavities that occurred during swallow induced LOS relaxation while supine occurred in one patient who was also the patient with the highest rate of supine common cavities.

Data on the rate of TLOSRs were available from only eight of the 10 patients because basal LOS pressure was too low in two patients to allow identification of these events. Gastric distension increased significantly the rate of TLOSRs in the sitting position from 1 (1–1.5)/10 min to 6 (2.5–8)/10 min (p < 0.03), and caused a smaller but significant increase when supine (0 (0) to 1 (0–4.5)/10 min; p < 0.05; fig 4). Two patients had rates of TLOSR while supine that approached those when sitting. On resumption of the sitting position, there was no change in the rate of TLOSRs (3 (1.5–3.5)/10 min) compared with that during the post-distension supine period.

Discussion
This study was designed to test the hypothesis that postural control of TLOSRs and gas reflux is defective in patients with reflux oesophagitis. Our findings do not support this, at least as a general rule. Although gastric distension did increase the rates of both these events when the patient was in the supine posture, the rates in this position were significantly lower than when the patient was sitting, findings consistent with significant postural control. Moreover, TLOSRs and gas reflux were almost completely suppressed in all but three patients, results similar to those in normal subjects.5,6 These findings suggest that postural suppression of TLOSRs and gas reflux is generally preserved in patients with reflux disease although it may be less potent than in normal subjects and may even be defective in a small subgroup of patients.

Although our data argue against the notion of defective postural control of TLOSRs, they are not necessarily inconsistent with the observation of increased supine reflux in reflux disease. So-called “supine” reflux is an imprecise term in that patients are not necessarily supine. A more appropriate term would be “recumbent” reflux because patients may spend as much of their recumbent period in the lateral recumbent or even the semi-prone position rather than strictly supine as in the present study. TLOSRs and acid reflux occur in the lateral recumbent position in both normal subjects and in patients with reflux disease, and in this position rates of TLOSRs and acid reflux are generally preserved in patients with reflux disease although it may be less potent than in normal subjects and may even be defective in a small subgroup of patients.

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Our previous studies in normal subjects have shown that suppression of TLOSRs in the supine posture is virtually complete in all normal subjects. One of our patients, however, failed to show any suppression of TLOSRs or common cavities in the supine posture, and exhibited rates of these events that were substantially higher than that observed in our previous studies in normal subjects. Two other patients also had supine rates of TLOSRs that were higher than that in our previous controls, although, in both patients, their rate when supine was lower than that when sitting. These observations suggest that a subgroup of patients, for example those with predominant recumbent reflux, may have defective postural control of TLOSRs. Our patients were chosen on the basis of proven oesophagitis and not on the basis of supine reflux on 24 hour oesophageal pH monitoring. In patients with endoscopy-negative reflux disease or mild reflux oesophagitis, who constitute the majority of patients with reflux disease, the acid exposure is predominantly upright, diurnal, and postprandial. Nevertheless, the degree of recumbent acid exposure in such patients is higher than that in controls. It is also possible that supine reflux occurs predominately through mechanisms other than TLOSRs that are not susceptible to postural suppression. Patients with reflux oesophagitis exhibit a heterogeneity of reflux mechanisms such that, although TLOSRs are the most common mechanism of reflux in most patients, another mechanism such as absent basal LOS pressure is the most important mechanism in some patients. In the present study, a higher proportion of common cavities occurred during swallow induced LOS relaxation in the supine posture. This mechanism occurred almost entirely in the one patient with the highest rate of common cavities when supine. Interestingly, however, we observed a reduction in the occurrence of gas reflux during absent basal LOS pressure.

The mechanism of postural suppression of TLOSRs remains unclear. The concept that fluid bathing the gastric cardia triggers inhibitory reflexes has not been supported by subsequent studies in dogs, in which removal of the fluid did not remove the strong postural suppression. Likewise, the effects do not appear to be mediated through the vestibular system. It is possible that the supine posture imposes mechanical effects on the cardia that may influence patterns of LOS relaxation or the sensory mechanisms responsible for triggering TLOSRs. Postural suppression of reflux as the result of absent basal LOS tone may also be a reflection of mechanical effects on the LOS segment when supine.

In summary, we have shown preservation of postural suppression of TLOSRs and gas reflux in reflux disease. The mechanisms underlying this postural suppression, however, remain to be determined.

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