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Effect of increasing the fat content but not the energy load of a meal on gastro-oesophageal reflux and lower oesophageal sphincter motor function

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Abstract

Background—Although fatty foods are commonly considered detrimental in patients with reflux disease, no objective data exist that substantiate this belief.

Aims—To investigate the effect of fat on gastro-oesophageal reflux and lower oesophageal sphincter (LOS) motor activity.

Subjects—Thirteen healthy subjects and 14 patients with reflux disease.

Methods—Oesophageal pH, LOS, and oesophageal pressures were recorded for 180 minutes after a high fat (52% fat) and a balanced (24% fat) meal (both 3.18 MJ) on two different occasions. Eight controls and seven patients were studied in the recumbent position and the others in the sitting position.

Results—The percentage of time at pH less than 4 and the rate of reflux episodes were higher (p<0.01) in the patients than in the healthy subjects (mean 14.1% versus 1.7% and 4.4/h versus 0.8/h respectively), as was the percentage of transient LOS relaxations associated with reflux (62% versus 32%, p<0.01). The high fat meal did not increase the rate of reflux episodes nor exposure to oesophageal acid in either group regardless of body posture. The rate of transient LOS relaxations, their association with reflux, and basal LOS pressure were also unaffected.

Conclusions—Increasing fat intake does not affect gastro-oesophageal reflux or oesophagogastric competence for at least three hours after a meal.

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Keywords: oesophagus; oesophagogastric junction; gastro-oesophageal reflux; fat

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Fatty foods are often reported by patients with gastro-oesophageal reflux disease to precipitate heartburn¹ and it is a common belief among physicians that they are contraindicated in these patients. However, there are no data that substantiate these notions.²

The aim of the study was to evaluate the effect of a high fat meal compared with an equal energy balanced meal on gastro-oesophageal reflux and on the main variables of lower oesophageal sphincter (LOS) competence—that is, transient LOS relaxation and basal LOS pressure, ^{3 4} using combined oesophageal pH and pressure monitoring.

Methods

STUDY GROUP

Thirteen healthy subjects (aged 19–38 years; eight men) and 14 patients with gastro-oesophageal reflux disease (aged 23–60 years; 10 men) were enrolled in the study which was approved by the Human Research Review Committee of the Maggiore Hospital, Milan. The healthy subjects had no symptoms or past history of gastrointestinal disease and the patients had either abnormal 24 hour intraoesophageal pH monitoring (more than 5% of time at pH less than 4) (n=8) or erosive oesophagitis (n=6).

MANOMETRIC AND pH RECORDING TECHNIQUE Oesophageal motility was monitored using an assembly of polyvinyl tubes incorporating a sleeve sensor (Dentsleeve, Belair, Australia). The 6 cm long sleeve was positioned so that it straddled the LOS. A side hole 1 cm below the distal margin of the sleeve recorded intragastric pressure. Side holes at the upper margin of the sleeve and 5 and 10 cm more proximally monitored motor activity of the oesophageal body 2, 7, and 12 cm above the LOS. One of two side holes 23 and 28 cm proximal to the upper margin of the sleeve monitored swallows in the pharynx. Each lumen was connected to a pressure transducer (model 4-327-I, Sensormedics, Anaheim, USA) and perfused with distilled water by a low compliance pneumohydraulic infusion pump (Arndorfer, Greendale, Wisconsin, USA; response: >200 mm Hg/s at 0.5 ml/min). The gastric side hole and sleeve were perfused at 0.5 ml/min, whereas the side holes in the oesophageal body and in the pharynx were perfused at 0.13 ml/min in order to minimise the fluid load to the subject. Intraoesophageal pH was monitored using a bipolar intraluminal pH electrode (Ingold 440-M4, Urdorf, Switzerland) positioned 5 cm above the proximal margin of the LOS. Signals from the pressure transducers and pH electrode were processed and recorded on a polygraph (model R711, Sensormedics, Anaheim, USA) at a paper speed of 2.5 mm/s.

EXPERIMENTS

All subjects were studied on two different occasions, in randomised order, at least two days apart. After an overnight fast the subjects swallowed the assembly. When they felt comfortable, the study started with administration of the test meal. On both occasions the subjects ate one sandwich and drank 150 ml of a commercially available diet formula (Ensure, Abbott, Italy); 450 ml of a high fat or balanced

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Table 1 Composition of the two meals

	Balanced meal	High fat meal
Lipids	20 g (24%)	44 g (52%)
Carbohydrates	114 g (60%)	74 g (39%)
Proteins	29 g (16%)	17 g (9%)
MJ	3.16	3.18

Percentage of total energy content of the meal in brackets.

solution, with an osmolarity of 350 mOsm/l in each case, was infused directly into the stomach at a speed of 40 ml/min through the core of the manometric assembly. The balanced solution consisted of 450 ml of Ensure; the high fat solution consisted of 150 ml of Ensure, 150 ml of a lipid emulsion (Lipofundin S 20%, Braun, Italy), and 150 ml of saline solution. Table 1 summarises the composition of the two meals.

After the end of the infusion oesophageal motility and pH were recorded for 180 minutes. Eight healthy subjects and seven patients were studied in the recumbent position, and five healthy subjects and seven patients in the sitting position. Controls and patients were not allowed to sleep.

ANALYSIS OF RECORDS

Oesophageal acid exposure was expressed as percentage of time at pH less than 4. A reflux episode was defined as either a pH drop to less than 4 for at least four seconds or, if basal oesophageal pH was already below 4, a further abrupt drop of more than 1 pH unit. Transient LOS relaxation was defined as already described,5 and reflux was judged to have accompanied a transient LOS relaxation if an abrupt fall in oesophageal pH of more than 1 pH unit occurred during the relaxation. End expiratory basal LOS pressure, referenced to end expiratory intragastric pressure, was calculated as the mean of the average pressure of one minute every five minutes. Analysis of transient LOS relaxation could not be performed in five patients because LOS pressure was too low (less than 3 mm Hg) for most of the recording period to allow assessment.

STATISTICAL ANALYSIS

Data are expressed as mean (SEM). The statistical significance of differences between the high fat and the balanced meal and between healthy subjects and patients was evaluated using paired and unpaired Student's *t* test, respectively.

Table 2 Gastro-oesophageal reflux variables according to body posture

	Balanced meal	High fat meal
Time at pH<4 (%)		
Recumbent controls	2.6 (0.9)	2.5 (1.8)
Sitting controls	0.1 (0.1)	0.1 (0.1)
Recumbent patients	19.5 (6.5)	16.5 (7.5)
Sitting patients	8.6 (2.9)	6.3 (2.4)
Rate of reflux episodes (no/h)		
Recumbent controls	1.0 (0.4)	1.1 (0.7)
Sitting controls	0.4(0.2)	0.6 (0.4)
Recumbent patients	4.8 (1.7)	5.2 (1.9)
Sitting patients	3.9 (1.1)	2.4 (0.7)

Data are expressed as mean (SEM).

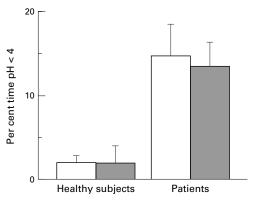


Figure 1 Oesophageal acid exposure after the balanced (open column) and the high fat meals (shaded column). Data are expressed as mean (SEM).

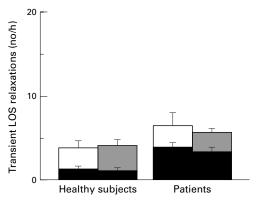


Figure 2 Rate of transient LOS relaxations and proportion associated with reflux, shown in black, after the balanced (open column) and the high fat meals (shaded column). Data are expressed as mean (SEM).

Results

GASTRO-OESOPHAGEAL REFLUX

After the balanced meal the patients had a higher oesophageal acid exposure and rate of reflux episodes (p<0.01) than the healthy subjects (14.1 (3.7)% versus 1.7 (0.6)% and 4.4 (0.9)/h versus 0.8 (0.2)/h; fig 1). The high fat meal did not affect these variables in either group, regardless of body posture (table 2 and fig 1). No difference in response to the high fat meal was found between the five patients showing absent (less than 3 mm Hg) LOS pressure for most of the recording time after the balanced meal and the remainder of the study population.

TRANSIENT LOS RELAXATION

After the balanced meal the rate of transient LOS relaxations showed a trend towards higher values in the patients compared with the healthy subjects (6.4 (1.6)/h versus 3.5 (0.5)/h; p=0.1), and the percentage of these events associated with reflux was higher in the patients (62 (7)% versus 32 (8)%; p<0.01; fig 2). The high fat meal did not affect the rate of transient LOS relaxations or their association with reflux in either group (fig 2).

BASAL LOS PRESSURE

Overall basal LOS pressure did not differ in the patients compared with the healthy subjects after the balanced meal (7 (2) mm Hg versus 11 (2) mm Hg; fig 3). However, whereas all

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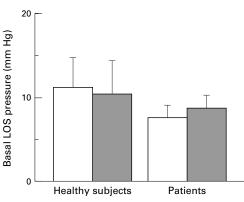


Figure 3 Basal LOS pressure after the balanced (open column) and the high fat meals (shaded column). Data are expressed as mean (SEM).

healthy subjects always had a pressure of greater than 3 mm Hg, basal LOS pressure was almost undetectable during the whole observation period in two patients and during the first 90 minutes in three others. Basal LOS pressure was unaffected by the high fat meal in either group (fig 3).

Discussion

In the present study a high fat meal did not promote gastro-oesophageal reflux or alter LOS motor function in either healthy subjects or patients with reflux disease, when compared with an equal energy balanced meal. This seems surprising in light of the currently held belief that fat worsens gastro-oesophageal reflux, but is in line with the few reports in the literature. The only previous study to look at the effect of a high fat compared with a low fat meal on oesophageal acid exposure over three postprandial hours in both the recumbent and upright posture² showed no difference in the patients with reflux disease and an increase after the high fat meal in the healthy subjects, in the upright posture only. However, whether the latter result depended on an increase in the rate of reflux episodes or not is unclear as this rate was not presented. A recent experimental study6 showed that, compared with saline infusion, intraduodenal infusion of a solution containing only fat causes a modest increase in the number of reflux episodes in patients with reflux oesophagitis by increasing the percentage of transient LOS relaxations associated with reflux. Although interesting, these observations cannot be extrapolated to the physiological postprandial situation.

In the design of our study we were careful to avoid possible bias which may occur when testing different meals. Firstly, we chose a type of meal which allowed us to use the same volume and osmolarity on the two occasions. Gastric distension is a potent stimulus for transient LOS relaxation, and hyperosmolarity delays gastric emptying, which prolongs the time acid material is available for reflux in the oesophagus. Secondly, as a control meal we used a balanced meal with the energy content of carbohydrates, lipids, and proteins typical of the Italian diet. Thirdly, we administered part of the meal directly in the stomach in order to

minimise the possibility that the subjects ate a different amount on the two days.

A lower LOS pressure has been observed in healthy subjects after a corn oil meal when compared with the fasting condition. This has been proposed as the possible cause of fatty food induced heartburn; subsequently Becker et al have suggested that in patients with reflux disease gastro-oesophageal reflux may not increase after fat ingestion if they already have a low basal LOS pressure. Our study does not support this notion. We found similar LOS pressure after meals containing different amounts of fat and our high fat meal did not affect gastro-oesophageal reflux in any of the subjects studied, irrespective of the level of LOS pressure after the balanced meal.

In light of our results, should it be concluded that, despite clinical beliefs, fat does not increase gastro-oesphageal reflux in patients with reflux disease? We do not think so. Our study is not definitive and at least one other hypothesis remains to be tested with a longer observation period. Although the gastric emptying of the two meals may not differ because their volume and the energy density was the same,11 the possibility exists that a higher fat content delays gastric emptying12 and consequently increases reflux in the late postprandial period by prolonging both gastric distension, which is a stimulus for transient LOS relaxation,7 and the presence of acid material in the stomach. Alternatively, fat reaching the small bowel could enhance perception of gastro-oesophageal reflux episodes without causing any increase in the acid load to the oesophagus. This possibility is supported by recent data showing that in dyspeptic patients intraduodenal lipid decreases the threshold gastric volume at which fullness is felt, suggesting that stimulation of duodenal chemoreceptors can influence sensation in other areas of the gut, possibly through neurohormonal mediation.

Finally, a comment on the rate of transient LOS relaxations and their association with reflux. We found a significantly higher percentage of transient LOS relaxations accompanied by reflux in the patients, as have other authors, ¹⁴ but only a trend towards a higher rate of transient LOS relaxations. The latter observation suggests that patients are heterogeneous in this respect, which may explain, at least in part, the conflicting findings in the literature. ¹⁴

In summary, our study did not provide evidence for an increase in gastro-oesophageal reflux after fatty foods, at least for the first three postprandial hours, suggesting that the relationship between fat and induction of heartburn in reflux disease is more complex than commonly thought.

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