Human small intestinal contractions and aboral traction forces during fasting and after feeding

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Abstract

Small intestinal intraluminal pressure activity and aboral traction forces were explored in 19 healthy volunteers using a combined manometry and traction force detecting assembly sited in the upper small intestine. Each aboral traction event was classified as being associated with either a propagating or a stationary contraction and its force measured. During phase I no contractions or traction events were seen. During phase II, traction events related to propagating contractions mean (SEM) (2.2 (0.2)/min) and to stationary contractions (0.3 (0.1)/min) generated similar force/event (7.5 (0.9 g v 8.7 (1.4) g, $p > 0.05$). During phase III, all traction events were related to propagating contractions and generated 9.3 (2.4) g force/event ($p > 0.05$ v phase II). After feeding, traction events related to propagating contractions generated similar force/event to those related to stationary contractions (5.9 (1.0) g v 9.3 (2.7) g, $p > 0.05$ v each other and v fasting). No consistent pattern was seen in the temporal distribution of the traction events or in the pattern of the amplitude of the force of successive traction events.

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It has also been suggested that the length of propagation of contractions is responsible for the speed of transit.³⁶ In humans, however, the average distance travelled by a propagating contraction remains unchanged by a meal,¹ so that the postprandial transit acceleration cannot be explained on the basis of propagation distance alone. Another possible explanation for the meal induced transit changes is that the propulsive forces generated by contractions are greater after a meal. So far, however, propulsive forces generated remain unexplored.

The aim of our study was to test the hypothesis that the propulsive forces generated in the small intestine would be modified by food ingestion in a manner consistent with the observed transit effects. We therefore measured the aboral forces generated within the small bowel during fasting and after a meal.

Subjects

Nineteen healthy volunteers (nine men; mean age 27 years; range 21-39) without past or current history of gastrointestinal disease participated in the study after providing informed consent.

Methods

THE TRACTION FORCE DETECTOR

This device (Fig 1), which has been previously described in detail,⁸ consisted of a miniature electronic strain gauge force transducer (Gaeltec Ltd, Dunvegan, Isle of Skye, UK) mounted on a catheter to which a silicone extension was attached by Kevlar threads. Because of their inelasticity, the Kevlar threads ensured that aboral traction forces acting on the extension were transferred directly to the force transducer without damping.

The force transducer was calibrated before and after each study by suspending the catheter vertically and applying known weights to the extension piece. The voltage output from the transducer amplifier was recorded on a polygraph (Grass Instruments Co, Quincy, Mass, USA) calibrated so that a 10 mm pen deflection was equivalent to 10 g force.

BALLOON

A roughly cylindrical, latex balloon, 1.5 cm in length (HSC4 Precision Dippings Marketing, Stover Trading Estate, Yate, UK) was attached to the silicone extension 3 cm proximal to the end of the catheter. During each experiment the balloon was inflated with 1.5 ml air through a channel running through the catheter, this

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Figure 1: Diagram of the components of the distal end of the traction catheter assembly.

volume being chosen from the results of pilot studies because it permitted the detection of both pressure changes and aboral forces without itself inducing any distension related contractile responses.

INTRALUMINAL MANOMETRY

Intraluminal pressure activity was measured, both proximal and distal to the balloon, by attaching two polyvinyl chloride perfusion catheters (internal diameter 0.63 mm, Portex Ltd, Hythe, Kent, UK) to the traction catheter. The perfusion port of one catheter was sited 2 cm proximal to the mid point of the balloon while the other was 2 cm distal (Fig 1). Two additional perfusion catheters with ports 7 and 17 cm proximal to the balloon were also attached to the catheter to aid the placement of the device in the duodenum. All the perfusion catheters were bonded to the traction catheter using tetrahydrofuran. The external diameter of the entire catheter assembly was 4.5 mm.

Each of the four channels was perfused with degassed water at a rate of 0.5 ml/min using a low compliance pneumohydraulic perfusion system (Arndorfer Medical Specialties Inc, Greendale, Wisconsin, USA) run at a pressure of 15 pounds per square inch. A transducer (Statham P23-ID, Gould Inc, Oxnard, Ca 93030, USA) was attached to the proximal end of each of the four perfused channels, the outputs of each transducer amplifier being recorded on the polygraph, run at a paper speed of 50 mm/min and calibrated so that a pen deflection of 10 mm on the chart recorder corresponded to a pressure change of 10 mm Hg. Sudden complete occlusion of each channel gave a pressure rise of >400 mm Hg/s.

To record the intraluminal pressure at the site of the balloon, the intraballoon pressure was also recorded continuously on the chart recorder using a similar Statham P23 transducer connected to the balloon inflation channel.

TEST MEAL

A mixed nutrient test meal was used for all studies (H J Heinz and Co Ltd, Cream of Chicken Soup 360 ml; 425 g; 200 kcal; fat = 11.5 g; protein = 5.5 g). Before ingestion, the meal was warmed to a temperature of 37°C in a microwave oven.

STUDY PROTOCOL

The study protocol was submitted to, and approved by, the Salford District ethics committee.

All studies were conducted in the morning after an overnight fast of at least 11 hours, the subjects reclining supine with their trunk at an angle of 30° to the horizontal throughout the study. The catheter assembly was first passed through the nose and the tip advanced into the stomach. The catheter was then passed through the pylorus and into the duodenum, its position being monitored by the pattern of the manometric events recorded from the perfusion channels. The identification of a typical duodenal

manometric pattern from the three distal perfusion channels, and an antral pattern from the most proximal port, was taken to show that the balloon was sited in the duodenum, 12–15 cm beyond the pylorus. After correct placement, the catheter was tethered to the cheek with adhesive tape to prevent aboral movement, and the balloon was inflated.

A continuous recording of fasting manometric and aboral traction activity was then made for 60 minutes, after which the subject ingested the test meal. After a further five minutes to permit a fed pattern to develop, a 30 minute recording of postprandial activity was made.

Definition of terms

The criteria used for the classification of small intestinal contractions in this study are identical to those previously reported for humans, and are based on the assumption that the aboral propagation velocity of a contraction would be approximately 1 cm/s.¹

Contraction was defined as the occurrence of a pressure change either at a perfusion port or at the balloon.

Propagating contraction was defined as a contraction at the balloon that occurred between 1–2 seconds after a contraction in the proximal channel or between 1–2 seconds before a contraction in the distal channel, or both.

Stationary contraction was defined as a contraction at the balloon that did not occur either 1–2 seconds after a proximal contraction or 1–2 seconds before a distal contraction. The simultaneous occurrence of pressure activity in all channels was considered to be a result of an extraintestinal artifact and was not included in data analysis.

Traction events were defined as all events recorded by the traction force detector, other than those associated with artifacts as described above. These were subdivided as being either: (a) Related to propagating contractions – that is, those that occurred synchronously with a propagating contraction; or (b) related to stationary contractions – that is, those that occurred synchronously with a stationary contraction.

Traction force – This was defined as the force in grams (g) exerted by a traction event.

Data collection

CONTRACTIONS

During fasting, the number of contractions at the proximal and distal perfusion ports and at the balloon were counted during a 30 minute segment of phase II activity (excluding the first 10 minutes after phase I) and in addition during phase III and during phase I.

During the 30 minute fed state, all contractions at the balloon and at the proximal and distal perfusion ports, were counted.

The perfusion/balloon contraction ratio was calculated as:

$$\frac{\text{Proximal perfusion port contractions} + \text{distal perfusion port contractions}}{2 \times \text{balloon contractions}}$$

TRACTION EVENTS

All traction events occurring during the study periods were identified and the force generated by each was calculated from the amplitude of the deflection on the chart recorder.

TRACTION FORCE/MINUTE

The total force generated in each period was calculated by dividing the total force by the duration of the recording period.

AVERAGE TRACTION FORCE/EVENT

The average traction force/event was calculated by dividing the total force generated by the number of events. This was calculated both for traction events related to propagating contractions and those related to stationary contractions.

PATTERN OF TRACTION FORCE ACTIVITY

To investigate in detail the temporal dispersion of the occurrence and the amplitude of successive traction events, data from three randomly selected individual studies were taken and both the time interval between successive traction events and their amplitude was noted.

Statistical methods

The distribution of the data was first tested by applying the Shapiro-Wilk test,⁹ which showed that all data were normally distributed. The results are therefore expressed in the text as mean (standard error of the mean).

Student's paired *t* test¹⁰ was used for comparison of paired data. Because three comparisons of the data were made (between phases II, III, and the fed state), a *p* value of <0.017 (0.05 divided by the number of comparisons) was taken to show a biologically relevant difference.

The temporal dispersion of the occurrence and the amplitude of traction events was analysed by applying the method of autocorrelation. In this method, all findings in a time series are correlated successively with the same series, with a lag of 1, 2, 3 . . . , *n*, where *n* is 10 + √*n*, *n* being the number of findings in the series.¹¹

The temporal relation between traction events related to propagating and those related to stationary contractions was tested by applying the non-parametric Runs test.¹¹ This method assesses whether a sequence of consecutive find-

ings of more than one category in a time series are randomly occurring.

Results

The Table and Figures 2-4 show the traction data during fasting and after feeding.

FASTING STATE

The characteristically irregular pattern of phase

Figure 2: The frequency of traction events/min during phases II, III, and after food is shown. Note that a greater proportion of traction events are related to stationary contractions in the fed state than during fasting.

Figure 3: The traction force generated/minute is shown during phases II, III, and after food.

Figure 4: The traction force in g/event generated by traction events related to propagating and stationary contractions during phases II and III and after food.

Traction data during fasting and after feeding

	Phase II	Phase III	Fed state
Traction events/min related to:			
Propagating contractions	2.2 (0.2)*	10.5 (0.6)	2.2 (0.3)*
Stationary contractions	0.3 (0.1)		1.1 (0.2)†
Traction force g/min exerted by traction events related to:			
Propagating contractions	18.3 (2.4)*	98.3 (26.9)	14.2 (2.1)*
Stationary contractions	2.5 (0.7)		9.4 (2.1)†
Traction force g/event related to:			
Propagating contractions	7.5 (0.9)	9.3 (2.4)	5.9 (1.0)
Stationary contractions	8.7 (1.4)		9.3 (2.7)
Contractions/min			
Proximal port	3.7 (0.4)	11.3 (0.5)	4.8 (0.4)
Balloon	2.5 (0.2)‡	10.9 (0.5)	3.3 (0.3)‡
Distal port	3.5 (0.3)	10.5 (0.6)	5.1 (0.5)

**p*<0.001 *v* phase III; †*p*<0.01 *v* phase II; ‡*p*<0.001 *v* proximal and distal perfusion ports. Data shown as mean (SEM).

Figure 5: Randomly irregular pattern of successive traction events. The continuous lines represent traction events related to propagating contractions, while dotted lines represent traction events related to stationary contractions, in three randomly selected individual studies, A, B, C.

II activity was seen in all 19 subjects. In seven subjects, phase III and phase I activity were also seen.

Phase I

No traction events or contractions were observed.

Phase II

Contractions – The number of contractions recorded at the proximal and distal perfusion ports were similar ($p=0.3$), and both were consistently greater than the contractions recorded at the balloon ($p<0.001$), with a perfusion/balloon contraction ratio of 1.5 (0.01).

Traction events related to propagating contractions were greater in number and generated greater force/min than those related to stationary contractions (87% *v* 13%) (Figs 2 and 3). The average force, however, generated by both types of traction events was similar ($p=0.1$, Fig 4). No consistent temporal dispersion pattern was seen, for either the occurrence or the amplitude of successive traction events (Fig 5). In addition the occurrence of successive traction events related to propagating contractions *v* those related to stationary contractions were randomly irregular.

Phase III

The duration of this phase was 4.3 (0.6) minutes.

Contractions – The number of contractions recorded at the proximal and distal perfusion ports and at the balloon were similar ($p>0.07$ for all comparisons) with a perfusion/balloon contraction ratio of 1.05 (0.02) ($p<0.01$ *v* phase II).

Traction events – All traction events were related to propagating contractions and although the force generated/minute was greater than phase II (Figs 2 and 3), the force generated/traction event was similar in both phases (Fig 4).

FED STATE

Contractions

The number of contractions recorded at the proximal and distal perfusion ports were similar ($p=0.6$), and both were greater than the number of contractions recorded at the balloon ($p<0.001$), with a perfusion/balloon contraction ratio similar to that of phase II (1.52 (0.04), $p=0.72$).

Traction events

Traction events related to propagating contractions were similar in number to phase II, and generated similar force/event. In contrast, more traction events related to stationary contractions occurred than in phase II, $p<0.001$, and greater force was thus generated/minute.

No consistent pattern was seen in either the temporal dispersion of the occurrence or the amplitude of successive traction events (Fig 5). In addition, the occurrence of traction events related to propagating contractions and stationary contractions seemed randomly irregular.

Discussion

Our study has shown that, in the human small intestine, aboral traction forces of similar magnitude are generated both during fasting and after meal ingestion in a randomly irregular pattern.

Because stationary contractions do not seem to be associated with movement of intraluminal contents,^{3,6,12} it might have been predicted that they would have been associated with less traction force than propagating contractions. Our data are inconsistent with this prediction as both types of contraction were associated with aboral forces of similar magnitude.

We also found, as have others,^{1,3,6} that after feeding, the number of propagating contractions remains unchanged, while the number of stationary contractions increases. As it has been shown that intraluminal transit is faster after a meal compared with phase II⁷ and as we have shown that similar forces are associated with stationary and propagating contractions, it is possible to speculate that the increased number of stationary contractions is responsible for transit acceleration after the meal. Against this suggestion, however, is the finding that transit is slower after a nutrient, compared with a non-nutrient, meal even though stationary contractions increase.^{3,6} It is therefore difficult to explain why a nutrient induced increase in stationary contractions does not generate greater aboral traction force and thus accelerate transit, unless the aboral forces are in some way balanced by oral forces of similar magnitude.

In considering this possibility, it is relevant to note that both during phase II and after food, the number of contractions recorded at the balloon was less than the number recorded by either of the perfusion ports, although it was expected that the number would have been similar.^{13–16} This discrepancy does not seem to be explained by a difference between the ability of the perfusion ports and the balloon to detect contractions, as we have shown the ratio of perfusion to

balloon contractions varied with the phase of motility studied. The most probable explanation, therefore, seems to be orad movement of the catheter, induced by stationary contractions. Although the catheter was tethered to prevent aboral movement, it was, however, free to move in an orad direction, in response to any forces generated immediately below the balloon, which would then have been missed both by the traction detector, and by the balloon pressure transducer. The suggestion that all these 'missed' contractions were indeed stationary contractions is supported by our finding that during phase III, when all contractions were propagating, the number of contractions at the balloon were equal to the number of contractions recorded from the perfusion ports. On the assumption therefore that most of these 'missed' contractions at the balloon were associated with orad forces, it is probable that the net propulsive force associated with stationary contractions in the fed state would have been small, as the number of missed contractions equalled those recorded.

If, as seems probable, the overall effect of stationary contractions is non-propulsive and there is no contribution to transit,^{13 12} then the transit acceleration seen after meal ingestion could be attributed to a change in propulsive force associated with propagating contractions. This does not seem probable, however, as both the number, and strength, of the traction force associated with propagating contractions were similar during fasting and after feeding. Moreover, the distance travelled by a propagating contraction is known to be similar under both conditions.¹

It therefore seems from our data that neither the number or the type of traction event explain the change seen previously in transit between fasting and feeding and further exploration of the problem is required, in particular study of the relation between aboral force, and factors, such as viscosity of luminal contents, intestinal wall tone, and luminal diameter, all of which could also independently influence flow and traction force.

During fasting the catheter is situated in an empty lumen whereas after feeding it is sited in a lumen containing nutrients, which could have provided a resistance to aboral movement of the device. If this was a relevant factor in our experiments, then the traction forces detected would have been lower than those actually generated by gut contractions. It remains to be determined, however, how such an effect would actually explain the discrepancies seen between force and transit. A further factor that has to be considered is that forces of the same magnitude could influence the magnitude of luminal flow differently during fasting and after feeding. For example, a decrease in wall tone and a resulting increase in luminal diameter after feeding may favour greater aboral flow for a given luminal force generated. Further studies are needed to explore these possibilities.

Our data also contribute to the debate about the comparative contributions made by phases II and III to the maintenance of fasting intraluminal clearance.^{17 18} Because all the propagat-

ing contractions in a segment of fasting small intestine are associated with aboral forces of equal magnitude, the efficiency of aboral movement is probably related both to the number of propagating contractions and to the duration of that phase. Thus although there is a four to fivefold increase in the traction force generated/minute during phase III, the greater duration of phase II (4–10 times longer than phase III)^{13–16} suggests that both phase II and III may be equally important for the maintenance of clearance of the fasting small intestine. It must be accepted, however, that such a speculation presumes that both the pattern of contractions and the forces generated remains similar throughout the length of the entire small bowel.

It is of further note that both the pattern and the amplitude of the traction events occurred in a randomly irregular manner, both during fasting phase II and after feeding. Although it is also known that the pattern of small intestinal contractions is randomly irregular,¹⁹ it cannot be assumed that the irregular pattern of traction events was a result of the irregular pattern of contractions. Both the ambient intraluminal pressure and traction force depend upon several independent factors, such as wall tension, wall diameter, the nature of intraluminal contents, and the pressure gradient across the sensing site²⁰ and it is not known how these factors interact.

In conclusion, the measure of aboral traction forces in the small bowel, show the presence of a randomly irregular pattern of traction events in the human proximal small intestine, which are similar during phase II and after feeding and are independent of the type of contraction associated.

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- 1 Sarna SK, Soergel KH, Harig JM, *et al.* Spatial and temporal patterns of human jejunal contractions. *Am J Physiol* 1989; 257: G423–32.
- 2 Schemann M, Eheim H-J. Mechanical characteristics of phase II and phase III of the interdigestive migrating motor complex in dogs. *Gastroenterology* 1986; 91: 117–23.
- 3 Schemann M, Ehrlein H-J. Postprandial patterns of canine jejunal motility and transit of luminal content. *Gastroenterology* 1986; 90: 991–1000.
- 4 Siegle M-L, Ehrlein H-J. Interdigestive contractile patterns of the ileum in dogs. *Am J Physiol* 1987; 253: G452–60.
- 5 Ehrlein H-J, Schemann M, Siegle M-L. Motor patterns of small intestine determined by closely spaced extraluminal transducers and videofluoroscopy. *Am J Physiol* 1987; 253: G259–67.
- 6 Siegle M-L, Ehrlein H-J. Digestive motor patterns and transit of luminal contents in canine ileum. *Am J Physiol* 1988; 254: G552–9.
- 7 Kerlin P, Zinsmeister A, Phillips S. Relationship of motility to flow of contents in the human small intestine. *Gastroenterology* 1982; 82: 701–6.
- 8 Williams D, Thompson DG, Marples M, Heggie L, O'Hanrahan T, Mani V, *et al.* Identification of an abnormal esophageal clearance response to intraluminal distension in patients with esophagitis. *Gastroenterology* 1992; 103: 943–53.
- 9 Minitab. Minitab Statistical Software, DOS microcomputer version, release 7, 1989. Minitab Inc, 3081 Enterprise Drive, State College PA 16801-2756, USA.
- 10 Armitage P, Berry G. *Statistical methods in medical research*. Oxford: Blackwell Scientific, 1987: 141–59.
- 11 SPSS/PC+. SPSS/PC+ V2.0 Statistical Software, 1988. SPSS Inc, 444 N Michigan Avenue, Chicago, Illinois 60611, USA.
- 12 Ramorino MA, Colagrande C. Intestinal motility: preliminary studies with telemetry capsules and synchronized fluorocinematography. *American Journal of Digestive Diseases* 1964; 9: 64–71.
- 13 Szurszewski JH. A migrating electric complex of the canine small intestine. *Am J Physiol* 1969; 217: 1757–63.

- 14 Code CF, Marlett JA. The interdigestive myo-electrical complex of the stomach and small bowel of dogs. *J Physiol (Lond)* 1975; **246**: 289–309.
- 15 Sarna SK. Cyclic motor activity; migrating motor complex. *Gastroenterology* 1985; **89**: 894–913.
- 16 Thompson DG, Wingate DL, Archer L, Benson MJ, Green WJ, Hardy RJ. Normal patterns of human upper small bowel motor activity recorded by prolonged radiotelemetry. *Gut* 1980; **21**: 500–6.
- 17 Sarr MG, Kelly KA. Patterns of movement of liquids and solids through the canine jejunum. *Am J Physiol* 1980; **239**: G497–503.
- 18 Summers RW, Helm J, Christensen J. Intestinal propulsion in the dog. Its relation to food intake and the migratory myoelectric complex. *Gastroenterology* 1976; **70**: 753–8.
- 19 Ehrlein HJ, Schmid HR, Fienle C. Characteristic motor patterns of phase II and behaviour of phase III in the fed state. *Journal of Gastrointestinal Motility* 1992; **4**: 317–27.
- 20 Macagno EO, Christensen J. Fluid mechanics of the duodenum. *Annual Reviews of Fluid Mechanics* 1980; **12**: 139–58.