



Mitchell, D. R., Derakhshan, M. H., Wirz, A. A., Ballantyne, S. A., and McColl, K. E.L. (2017) Abdominal compression by waist belt aggravates gastroesophageal reflux, primarily by impairing esophageal clearance. *Gastroenterology*, (doi:[10.1053/j.gastro.2017.02.036](https://doi.org/10.1053/j.gastro.2017.02.036))

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Deposited on: 07 March 2017

Main Title

Abdominal Compression by Waist Belt Aggravates Gastroesophageal Reflux, primarily by Impairing Esophageal Clearance

Short Title:

Waist belt impairs esophageal clearance

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AUTHOR DISCLOSURES:

None to declare.

AUTHOR CONTRIBUTIONS:

DRM: Clinical investigations, manometry, pHmetry, data analysis and drafting manuscript.

MHD: Data analysis and drafting manuscript.

AAW: Recruitment of volunteers and assisting clinical investigations.

SAB: Radiological assessment.

KELM: Conception of original idea, drafting manuscript and overall supervision.

Financial support

The study was supported by a grant from the Western Infirmary Gastroenterology custodian fund.

Abbreviations used in this paper: WC, Waist circumference; BMI, Body mass index; LES, Lower esophageal sphincter; TLESRs, Transient lower esophageal sphincter relaxations; GERD, Gastroesophageal reflux disease; PPIs, Proton pump inhibitors; GEPPG, Gastroesophageal pressure gradient; IGP, Intra gastric pressure

Abstract:

Background & Aims: Central obesity promotes gastroesophageal reflux and this may be related to increased intra-abdominal pressure. We investigated the effect of increasing abdominal pressure by waist belt on reflux in patients with reflux disease.

Methods: We performed a prospective study of patients with esophagitis (n=8) or Barrett's esophagus (n=6); their median age was 56 years and their median body mass index, 26.8. Proton pump inhibitors were stopped at least 7 days before the study and H2 receptor antagonists were stopped for at least 24 hours before. The severity of upper gastrointestinal symptoms was assessed, and measurements of height, weight, waist and hip circumference taken. Combined high-resolution pH measurement and manometry were performed in fasted state for 20 minutes and for 90 minutes following a standardized meal. The squamocolumnar junction was marked by endoscopically placed radiopaque clips. The procedures were performed with and without a waist belt (a weight-lifter belt applied tightly and inflated to a constant cuff pressure of 50 mmHg). We compared variables between groups using the Wilcoxon Signed Rank test and tested for correlations using Spearman Rho bivariate analysis.

Results: Without the belt, intragastric pressure correlated with waist circumference ($r=0.682$; $P=.008$), with the range in pressure between smallest and largest waist circumference being 15mmHg. The belt increased intragastric pressure by a median of 6.9 mmHg during fasting ($P=.002$) and by 9.0 mmHg after the meal ($P=.001$). Gastroesophageal acid reflux at each of the pH sensors extending 5.5 cm proximal to the peak lower esophageal sphincter pressure point was increased by approximately 8-fold by the belt (all $P<.05$). Following the meal, the mean number of reflux events with the belt was 4, vs 2 without ($P=.008$). Transient lower esophageal sphincter relaxations were not increased by the belt but those associated with reflux were increased (2 vs 3.5; $P=.04$). The most marked effect of the belt was impaired esophageal clearance of refluxed acid (median values of 23.0 seconds without belt vs 81.1 seconds with belt) ($P=.008$). The pattern of impaired clearance was that of rapid re-reflux after peristaltic clearance.

Conclusions: In a prospective study of patients with esophagitis or Barrett's esophagus, we found belt compression increased acid reflux following a meal. The intragastric pressure rise inducing this effect is well within the range associated with differing waist circumference and likely to be relevant to the association between obesity and reflux disease.

KEY WORDS: Barrett's esophagus; LES; central obesity; TLESRs

BACKGROUND AND AIMS

Central obesity is strongly associated with gastroesophageal reflux and its complications of Barrett's esophagus and esophageal adenocarcinoma (1,2). The nature of this association is incompletely understood and both mechanical and humoral effects of central obesity may be important.

Both BMI and waist circumference (WC) show a strong positive correlation with intra-gastric pressure (IGP) and the gastroesophageal pressure gradient (GEPG) (3,4,5,6). Abdominal compression by a waist belt also increases these pressures and thus reproduces the manometric characteristics associated with central obesity (5,7). Previous investigators have examined the effect of waist belt compression on the manometric characteristics of the lower esophageal sphincter (LES) in both healthy volunteers and patients with reflux disease. The rise in IGP caused by the waist belt is accompanied by a rise in LES pressure though sometimes of a lesser magnitude (5,7,8,9,10,11). Waist belt compression in short term studies does not result in the development of, or aggravation of, hiatus hernia or in increased separation of the intrinsic and extrinsic components of the LES (9,12,13).

Surprisingly, there is a paucity of information on the effects of waist belt compression on gastroesophageal acid reflux itself despite this being the main mediator of esophageal damage. We recently examined the effect of waist belt compression on gastroesophageal pH in healthy volunteers without reflux disease (7). The belt caused the location of the pH transition point where the pH changes from gastric to esophageal pH to migrate 2cm more proximally within the LES and this was most apparent after a meal. The belt did not cause the pH transition point to extend above the squamocolumnar junction (SCJ) onto esophageal mucosa. There was an increase in short segment reflux detected 1.3cm above the SCJ but none detected at any of the 7 pH sensors spaced at 1 cm increments proximal to this. In these subjects with a normal LES there was, therefore, little evidence that the waist belt significantly increased esophageal acid exposure.

In our current study, we investigate the effects of waist belt compression in patients known to have reflux disease and find that it induces a marked increase in their esophageal

acid exposure. We believe our findings are relevant to the understanding of the relationship between central obesity and reflux diseases, and also relevant to potential adverse effects of tight waist belts or clothing in reflux patients.

METHODS & MATERIALS

Subjects

Study subjects were patients with typical symptoms of gastroesophageal reflux disease (GERD) and at least Los Angeles (LA) Grade B reflux esophagitis or Barrett's esophagus on upper GI endoscopy. Proton pump inhibitors (PPIs) were stopped at least 7 days prior to the study and H₂ receptor antagonists were stopped for at least 24 hours.

Study design

Study Day 1: Clinical measurements

The severity of upper gastrointestinal symptoms was assessed using the Short-Form Leeds Dyspepsia Questionnaire (14). Medication history was recorded. Measurements of height, weight, waist and hip circumference were taken.

Study day 2: Upper gastrointestinal endoscopy

Volunteers attended after an overnight fast for upper gastrointestinal endoscopy. They were offered topical lidocaine throat spray or conscious sedation with midazolam 1-3mg. The upper gastrointestinal tract was inspected. The distance from incisors to SCJ was measured. If a hiatus hernia was present, the distance to the diaphragmatic impression was also noted. Two small metal radio-opaque clips were attached to the SCJ using a single use rotatable clip fixing device (QuickClip 2™; Olympus, Southend-on-Sea, UK). In subjects with Barrett's esophagus the clips were attached to the most proximal margin of the gastric folds.

Study Days 3 and 4: Combined manometry and pH study with and without waist belt

The volunteers attended fasted for a further two study days. On both days, a combined high

resolution manometer and pH probe was passed pernasally and positioned so that the pH sensors were lying across the LES and extending at least 5.5 cm above the LES. The relative positions of the 12-sensor pH catheter, 36-sensor manometer and SCJ is shown in Figure 1.

One of the study days was performed without the application of the waist belt. Manometry and pH data were recorded concurrently for a 20 minute fasting period with the subjects sitting upright at a 60 degree angle. They then consumed a standardised meal over ten minutes [400g Waitrose spaghetti bolognese ready meal and 100ml water (500kcal; 55.2g carbohydrate, 27.8g protein, 17.6g fat)]. Following this, manometry and pH recordings were continued for a further 90 minutes. An X-ray was taken before and after the meal to visualise the metal clips at the SCJ.

On the other study day, the above procedure was repeated but with the application of a waist belt throughout the whole recording period. A weight-lifter belt (Nike, USA) was applied tightly with a blood pressure cuff placed under the belt. This was inflated to a constant cuff pressure of 50mmHg. The order of the study days with and without the waist belt was alternated in random fashion. Any upper GI symptoms experienced during the tests were recorded with respect to time, location, duration and character.

Equipment

High-resolution pHmetry

pH recordings were taken using a high-resolution pH catheter (Synectics Medical Ltd, Enfield, UK). This was a custom-made pH probe composed of 12 antimony pH electrodes with the most distal electrode situated 5mm from the tip of the catheter, with the other eleven electrodes 35, 46, 57, 68, 79, 90, 101, 112, 123, 134 and 169mm proximal to the tip. The probe was calibrated prior to each study using pH buffer solution (Synmed Ltd, Enfield, UK) at pH 7.01 and pH 1.07. Recordings were captured using Polygram Net software (Synectics Medical Ltd, Enfield, UK).

High-resolution manometry

Manometry was performed using a high resolution solid-state catheter with 7.5mm spacing between 36 circumferential sensors (Given Imaging, Hamburg, Germany). Calibration was performed prior to each study and In vivo calibration was carried out weekly and applied to each study to compensate for thermal drift. Recordings were captured with ManoScan 360 high-resolution Manometry System and analysed with ManoView ESO v3.0.1 software (Given Imaging, Hamburg, Germany).

Combined probe

The manometry and pH catheters were combined using two thin strips of Leukoplast Sleek waterproof tape (BSN Medical, Pinetown, SA) such that manometry sensor 21 was immediately adjacent to pH sensor 7.

Data analysis

Acid exposure

Acid exposure was examined by calculating the percentage of time pH was less than 4 for each sensor across the LES and up to 5.5cm proximal to LES in the 20 minute fasting period and the 90 minute postprandial period. Location of the pH transition point was defined by the position of the pH sensor recording a drop in median pH of at least one unit from proximal to distal and correcting for 1.1 cm spacing as previously described.⁽⁶⁾ Reflux events were defined as a drop in pH to below 4 and lasting at least 1 second. The total number of reflux events were counted within the 20 minute fasting period and 90 minute postprandial period.

Manometric parameters

Manometric characteristics were analysed in detail during fasting and after the meal. For each two minute period, one inspiratory point and one expiratory point was chosen from the

longest period without interference from swallowing, coughing or transient lower esophageal sphincter relaxations (TLESRs). The mean pressure in inspiration and expiration was calculated for each of the 36 sensors in the fasting period and postprandial period. The peak LES pressure was taken as the sensor showing the highest mean pressure. IGP was also calculated on inspiration and expiration and was defined as the mean pressure of the first three sensors immediately distal to the LES. Intra-esophageal pressure (IEP) was defined as the mean pressure of three consecutive sensors located 6, 6.75 and 7.5 cm proximal to the peak LES pressure.

Measurement of manometric locations

All measurements were made using data collected in the expiratory phase of respiration. The upper border of the LES was defined as the most proximal sensor where the pressure was at least 2mmHg above IGP. The lower border of the LES was defined as the most distal sensor where the pressure was at least 2mm Hg above IGP. The pressure inversion point (PIP) was defined as the transition point from the abdominal pressure compartment (positive wave deflection) into the thoracic pressure compartment (negative wave deflection). The position of the SCJ was derived from the position of the metal clips relative to the combined manometry and pH sensors seen on X-ray. In the event of clips being visible at different levels, the mid-point between the two clips was used as the position of the SCJ. All measurements (in cm) were determined from the nares.

Statistical analysis

All continuous data are expressed as medians and interquartile ranges unless otherwise stated. Comparison of variables between related groups was made using the Wilcoxon Signed Rank test. For all correlations between two continuous variables, the Spearman Rho bivariate correlations were used. Significance for all statistical tests was set as p value <0.05.

Ethics

The study protocol was approved by the West of Scotland Ethics Committee and all volunteers provided informed written consent.

RESULTS

Fifteen subjects completed the study protocol but one had to be excluded due to a technical issue resulting in loss of the manometry data for one study day. Thus 14 subjects were included in the final analysis. The median age of the group was 56 years (range 24-76) and all subjects were male. The median BMI was 26.8 (range 22-42) and the median WC was 101cm (range 79-142cm). At endoscopy, 11/14 had evidence of a hiatus hernia (length 2-4cm). 8/14 had reflux esophagitis (either LA grade B or C) and 6/14 had Barrett's esophagus (median length 3.5cm, range 1-9cm).

Effect of belt on Intra-gastric Pressure and GEPG

During fasting the belt increased IGP and GEPG during both inspiration and expiration (Table 1). On inspiration, the median IGP was 13.5mmHg without the belt versus 19.9mmHg with the belt ($p=0.005$) and the GEPG was 13.7mmHg versus 18.6 mmHg ($p=0.041$). On expiration, the median IGP was 9.8mmHg without the belt compared to 16.7mmHg with the belt ($p=0.002$) and the GEPG was 5.0mmHg versus 9.1mmHg ($p=0.035$).

Following the meal, the belt also increased IGP on both inspiration and expiration (Table 1). On inspiration, the IGP without the belt was 13.5mmHg versus 23.3mmHg with the belt ($p=0.001$) and the GEPG was 16.2 versus 22.5mmHg ($p=0.008$). On expiration, the IGP was 10.8mmHg without the belt compared to 19.8mmHg with the belt on ($p=0.001$) and the GEPG was 8.0mmHg versus 11.9mmHg ($p=0.016$). The greater increase in the IGP than GEPG was due to the belt also causing an increase in intra-esophageal pressure.

Without the belt there was no difference in IGP fasting versus after the meal [9.8mmHg (IQR 8.9) versus 10.8mmHg (IQR 7.2); $p=0.084$]. With the belt the IGP was greater after the

meal compared with under fasting conditions [19.8mmHg (IQR 7.6) versus 16.7mmHg (IQR 9.5); $p=0.002$].

Effect of belt on LES

During the fasting period the belt increased median peak LES pressure on expiration relative to atmospheric pressure, being 23.9mmHg with the belt off versus 27.5mmHg with the belt on ($p=0.030$) (Table 1). However, there was a fall in the median peak LES pressure relative to the IGP on inspiration apparent after the meal, being 27.1mmHg with the belt off and 17.8mmHg with the belt on ($p=0.041$).

The belt did not cause any significant changes in the LES with respect to the distance between its upper border and nares, its length, or the position of the PIP, peak LES pressure or SCJ relative to upper border of the LES (Table 2). In addition, the belt did not influence the number of subjects with a double peak manometric pattern. When fasted, 5 subjects had a double peak pattern without the belt and 7 with the belt and after the meal, 6 without the belt and 7 with it.

Effect of belt on gastroesophageal reflux

The waist belt caused a marked increase in gastroesophageal reflux during the 90 minutes following the meal (Table 3). Acid exposure at each of the 5 pH sensors extending 5.5cm proximal to the peak LES pressure point was significantly increased with versus without the belt with the percentage time $pH < 4$ being increased by approximately 8 times at each position. Both with and without the belt acid exposure progressively increased with proximity to the peak LES pressure point so that with the belt the pH was less than 4 at 1.1cm above the peak LES pressure point for 49.7% of the time following the meal compared to 7.3% without the belt ($p=0.03$). The waist belt also increased the acid exposure at the peak LES pressure point (66.1% versus 18.4%, $p=0.056$) and 1.1 cm distal to it (89.6% versus 59.4%, $p=0.026$).

The waist belt also increased acid reflux after the meal relative to the clip marking the SCJ or in the case of the 6 patients with Barrett's, the proximal extent of the gastric folds. At 1.1cm proximal to the clip, the percentage time pH<4 was 41.4% (IQR 61.1) with the belt versus 7.0% (IQR 18.9) without it ($p<0.05$); at 2.2cm proximal 12.5% (IQR 44.0) versus 1.3% (IQR 8.4; $p=0.01$); at 3.3cm proximal 11.3% (IQR 21.2) versus 0.7% (IQR 6.5; $p<0.02$) and at 4.4cm proximal 4.5% (IQR 9.9) versus 0.3% (IQR 2.8; $p<0.01$).

Following the meal, the median number of reflux events with the belt was twice that without the belt [2 (IQR 2) vs 4 (IQR 6); $p=0.008$] (Table 4). The median number of TLESRs was not different but the number accompanied by acid reflux was increased with the belt [2 (IQR 2) vs 3.5 (IQR 5); $p=0.041$]. The median time from onset of TLESR until return of the LES to stable tone and original position was not different with the belt off versus on [46.0s (IQR 10.4) vs 44.8s (IQR 14.4); $p=0.279$]. The most marked effect of the belt was to reduce the rate of esophageal clearance of refluxed acid with the median time being 23.0 seconds without the belt versus 81.1 seconds with the waist belt ($p=0.008$). Examining the pH plots of the long reflux events occurring after the meal with the belt revealed evidence of attempted clearance of acid followed by immediate re-reflux of acid (Figure 2). There was no difference in the median amplitude of distal esophageal contractions with or without the waist belt [85.8mmHg (IQR 32.8) vs 79.5mmHg (IQR 48.1); $p=0.387$] (Table 4)

During the fasting period there was no difference in esophageal acid exposure with versus without the belt. However, the acidity at the peak LES pressure point and at the intragastric sensors located 1.1cm, 2.2cm, and 3.3cm distal to it was greater with versus without belt. ($p< 0.02$ for each). (Table 3)

Effect of Belt on Gastroesophageal pH Step-Up Point

The belt caused the location of the point where acidity changes from gastric pH to esophageal pH (pH transition point) to move proximally after the meal with respect to the LES upper border and peak pressure as well as the SCJ (Table 2). Without the belt the pH

transition point was 0.78cm distal to the upper border LES but with the belt it was 0.64cm proximal to it ($p=0.003$). Likewise, without the belt the pH transition point was precisely at the level of the clip marking the squamocolumnar junction (or in the 6 subjects with circumferential Barrett's the proximal extent of the gastric folds) but with the belt it was 1.17cm proximal to it ($p=0.016$). This meant that with the belt on the distal esophagus was constantly exposed to the level of acidity normally only seen in the stomach.

There was no significant difference in the position of the pH step up with and without the belt during the 20 minute fasting period (Table 2).

Correlation of WC with both the intragastric pressure and GEPG.

Without the belt, there was a strong correlation between the WC of the 14 patients included in the study and their fasting IGP both on expiration ($r=0.682$, $p=0.008$) and inspiration ($r=0.581$, $p=0.029$). The range in IGP between smallest and largest WC was 15.1mmHg on expiration and 15.7mmHg on inspiration. There was also a positive correlation with fasting GEPG on inspiration ($r=0.640$, $p=0.014$) but this was not seen on expiration. No significant correlations were apparent in the 90 minute period following the meal.

DISCUSSION

Our study indicates that in reflux patients, waist belt constriction causes a marked increase in gastroesophageal reflux most evident after a meal. The effect of the belt was most marked close to the gastroesophageal junction where the pH of the distal esophagus lined, or normally lined, by squamous mucosa became like that of the proximal stomach.

As previously reported the belt caused a rise in the IGP, which in the empty stomach is equivalent to intra-abdominal pressure, and also an increase in GEPG (5,7). The rise in GEPG was less than in IGP and this can be explained by the fact that the belt also caused an increase in intra-esophageal pressure.

The belt also raised peak LES pressure which has previously been observed both in healthy volunteers and reflux patients (5, 7-11). Mittal et al observed that the rise in LES pressure with abdominal compression was associated with tonic contraction of crural diaphragm EMG activity (10). In our current study after the meal the belt caused a greater rise in the IGP than in LES pressure causing a significant fall in LES pressure relative to the IGP which is the pressure gradient preventing reflux. This fall in LES pressure relative to the IGP has been reported by some but not all investigators (5,7,8,9,10,11). The fall in LES pressure relative to the IGP in our current study was only apparent after the meal and involved patients with reflux disease and in these respects differed from previous studies. Consistent with previous reports we found no evidence that the belt, at least in the short term of our study, caused any increased separation of the two components of the LES which would be indicative of promoting hiatus hernia formation (9, 12).

We extended previous work by monitoring the effect of the belt on actual acid reflux. We found that the belt markedly increased acid exposure following the meal at each of the pH sensors placed at 1.1cm increments and extending 5.5cm proximal to the peak LES pressure point. At each of these locations the belt increased esophageal acid exposure by approximately 8-fold relative to that without the belt. Without the belt the amount of acid increased with proximity to the LES and the 8-fold increase with the belt caused the pH of the most distal esophagus to be < 4 for 49.7% of the time following the meal. The belt also caused a marked increase in acid exposure after the meal when measured relative to the clip marking the SCJ or proximal extent of gastric folds.

Our combined high resolution pH and manometry system allowed us to examine the mechanism of the increased esophageal acid exposure induced by the belt. After the meal the belt doubled the number of reflux episodes. There was no increase in the number of TLESRs but there was an increase in those associated with reflux. The most marked effect of the belt was impairment of esophageal acid clearance which was approximately 4 times longer than without the belt. This impaired clearance was often related to re-reflux of acid occurring immediately after an esophageal peristaltic clearance wave.

The pH pattern of the impaired esophageal clearance with the belt in our study is similar to that previously reported in patients with hiatus hernia. Mittal et al in 1987 observed that in hiatus hernia patients esophageal acid clearance by a swallow was often followed by rapid re-reflux due to retrograde flow of contents from the hiatal sac during the swallow induced relaxation of the LES (15). Jones et al also found that impaired esophageal clearance was strongly correlated with esophagitis and hiatus hernia (16). In hiatus hernia patients reflux of barium trapped in the hiatal sac following a swallow has also been observed and shown to be most marked in non-reducing hernias (17,18). The vast majority of the reflux patients in our study had hiatus hernias and the belt is thus aggravating the impaired esophageal clearance associated with hiatus hernia.

The waist belt also caused the pH step up point (where the pH changes from gastric to esophageal) to move proximally by 1-2cm within and even above the LES and again this was most marked following the meal. We were also able to see the effect of the belt on the location of the pH step-up point relative to the location of the SCJ or in the case of the 6 patients with circumferential Barrett's the proximal extent of the gastric folds. Without the belt the pH step-up was at the level of the SCJ (or proximal gastric folds in Barrett's patients) but with the belt was displaced 1-2cm above it. The cause of this proximal displacement of the pH step-up point is unclear but might be due to marked impaired distal esophageal acid clearance. In hiatus hernia patients, the impaired clearance is most marked near to the gastroesophageal junction (15).

We considered the possibility that the belt might cause some artefactual evidence of distal esophageal acid reflux by increasing the duration and/or magnitude of proximal migration of the gastroesophageal junction during TLESRs. This could increase acid detected by the distal esophageal sensors due to their contact with the acidic gastric mucosa. However, our analysis indicated that the time for restitution of normal tone and position of the LES following TLESRs was the same with versus without the belt. This excludes the prolonged acid clearance, which was the main effect of the belt, from being attributed to prolonged proximal migration of the gastroesophageal junction during TLESRs.

Although we could not measure the amplitude of migration of the gastroesophageal junction during TLESRs, a previous study by Kahrilas *et al* showed that abdominal compression did not influence the proximal movement of the gastroesophageal junction during peristalsis in healthy volunteers or subjects with hiatus hernia (12).

The increase in esophageal acid exposure produced by the belt in our current study is substantially more than observed in our earlier study in healthy volunteers without reflux disease or hiatus hernia (7). This indicates that the reflux promoting effect of the belt is much more significant in patients with impaired LES function.

The increase in esophageal acid exposure induced by the belt was confined to the 90 minute period after the meal and several factors may explain this. The actual IGP with the belt on was higher after the meal than fasted despite these pressures being similar without the belt. In addition most of the increase in reflux occurred during TLESRs and its subsequent impaired clearance and TLESRs mainly occur after meals. Though there was no increased esophageal acid exposure with the belt during the fasting period the acidity of the most proximal stomach close to the gastroesophageal junction was increased and the reason for this is not clear.

The acid exposure of the distal esophagus in our reflux subjects with the belt was equivalent to that of the proximal stomach. The proximal region of the stomach escapes the buffering effect of food and remains highly acidic after a meal. If this degree of acid exposure of the distal esophagus were prolonged it would be likely to result in columnar metaplasia as the squamous mucosa transforms to a type more suited to a gastric rather than esophageal luminal environment. Six of our patients did have Barrett's esophagus.

Our findings are likely to be relevant to the mechanism of the association between central obesity and reflux disease. Increasing WC is accompanied by an increase in intra-abdominal and intra-gastric pressure (3,4,5,6). Even with the relatively small number of subjects in our current study there was a strong and highly significant correlation between WC and IGP. The range in IGP between the smallest and largest WC was 15mmHg and this is greater than the average rise in intragastric pressure produced by the waist belt of 6.9mmHg fasted and

9.0mmHg after the meal. It would appear, therefore, that much of the association between WC and reflux could be explained by the effects on intra-abdominal pressure.

Our findings are also relevant to potential adverse effects of tight waist bands or clothing in subjects with impaired LES function. As both central obesity and tight waist band increase intra-abdominal pressure it would seem appropriate to advise reflux patients to both lose weight and avoid such clothing. Our findings suggest that it will be particularly important to avoid tight waist belts after meals when their reflux promoting effects are most pronounced. However, caution needs to be taken in extrapolating the findings of our short-term study to long-term use of waist constricting clothing.

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Legend for Figure 1

Diagrammatic representation of relative positions of the pH probe, manometer probe, squamocolumnar junction marked by radio-opaque clip and crural diaphragm. The marks on the probes indicate the sensor numbering of each probe.

Legend for Figure 2

An example of an esophageal pH recording at sensor 5.5cm above peak LES pressure from one of the study subjects wearing a belt during the postprandial period. Following the initial reflux event (marked by arrow) there is clearance of acid by a peristaltic wave but this is followed immediately by re-reflux.

Table 1: Effect of waist belt on manometric parameters in fasting and postprandial states in expiration and inspiration

Expiration	Fasting			Postprandial		
	Belt Off	Belt On	<i>p</i> value	Belt Off	Belt On	<i>p</i> value
Median Peak LES pressure (IQR)	23.9 (8.4)	27.5 (11.9)	0.030	25.3 (9.6)	30.9 (12.8)	0.177
Median LESP vs IGP (IQR), mm Hg	12.6 (7.2)	11.3 (9.4)	0.826	14.2 (12.7)	10.7 (13.7)	0.158
Median IEP (IQR), mm Hg	5.2 (5.0)	6.3 (4.9)	0.124	4.3 (4.7)	6.8 (5.5)	0.004
Median IGP (IQR), mm Hg	9.8 (8.9)	16.7 (9.5)	0.002	10.8 (7.2)	19.8 (7.6)	0.001
Median GEPG (IQR)	5.0 (4.8)	9.1 (5.9)	0.035	8.0 (3.2)	11.9 (7.4)	0.016
Inspiration						
Median Peak LES pressure (IQR)	33.2 (12.9)	39.5 (17.6)	0.433	41.3 (21.5)	41.2 (4.5)	0.778
Median LESP vs IGP (IQR), mm Hg	20.2 (17.5)	20.0 (15.4)	0.124	27.1 (18.8)	17.8 (16.2)	0.041
Median IEP (IQR), mm Hg	-0.9 (3.7)	0.5 (2.0)	0.158	-0.2 (4.8)	0.9 (4.0)	0.022
Median IGP (IQR), mm Hg	13.5 (7.6)	19.9 (11.5)	0.005	13.5 (5.8)	23.3 (8.1)	0.001
Median GEPG (IQR)	13.7 (7.7)	18.6 (11.5)	0.041	16.2 (6.6)	22.5 (4.9)	0.008

LES = Lower esophageal sphincter, LESP = Lower esophageal sphincter pressure, IGP = Intra-gastric pressure, IEP = Intra-esophageal pressure, GEPG = Gastroesophageal sphincter pressure, IQR = Interquartile range.

Table 2: Effect of waist belt on relative locations of anatomical structures of the gastroesophageal junction.

	Fasting			Postprandial		
	Belt Off	Belt On	<i>p</i> value	Belt Off	Belt On	<i>p</i> value
Upper border LES (cm from nares)	43.38(4.81)	43.38 (4.00)	0.271	41.75 (3.63)	42.21 (2.40)	0.330
LES length, cm	3.75 (1.50)	3.38 (1.88)	0.218	3.00 (2.06)	2.88 (1.38)	0.636
PIP (cm from upper border LES)	0.43 (0.93)	0.43 (1.80)	0.801	0.60 (2.01)	0.54 (0.88)	0.245
Peak LESP (cm from upper border LES)	1.13 (0.75)	1.13 (0.75)	0.809	1.25 (0.69)	1.13 (0.56)	0.598
SCJ (cm from upper border LES)	1.12 (1.80)	1.10 (1.90)	0.241	0.88 (1.40)	0.48 (1.79)	0.124
pH transition point (cm from upper border LES)	2.18 (1.55)	1.53 (2.80)	0.220	0.78 (1.51)	-0.64 (3.37)	0.003
pH transition point (cm from SCJ)	0.83 (2.61)	1.05 (2.51)	0.444	0.00 (1.02)	-1.17 (2.89)	0.016

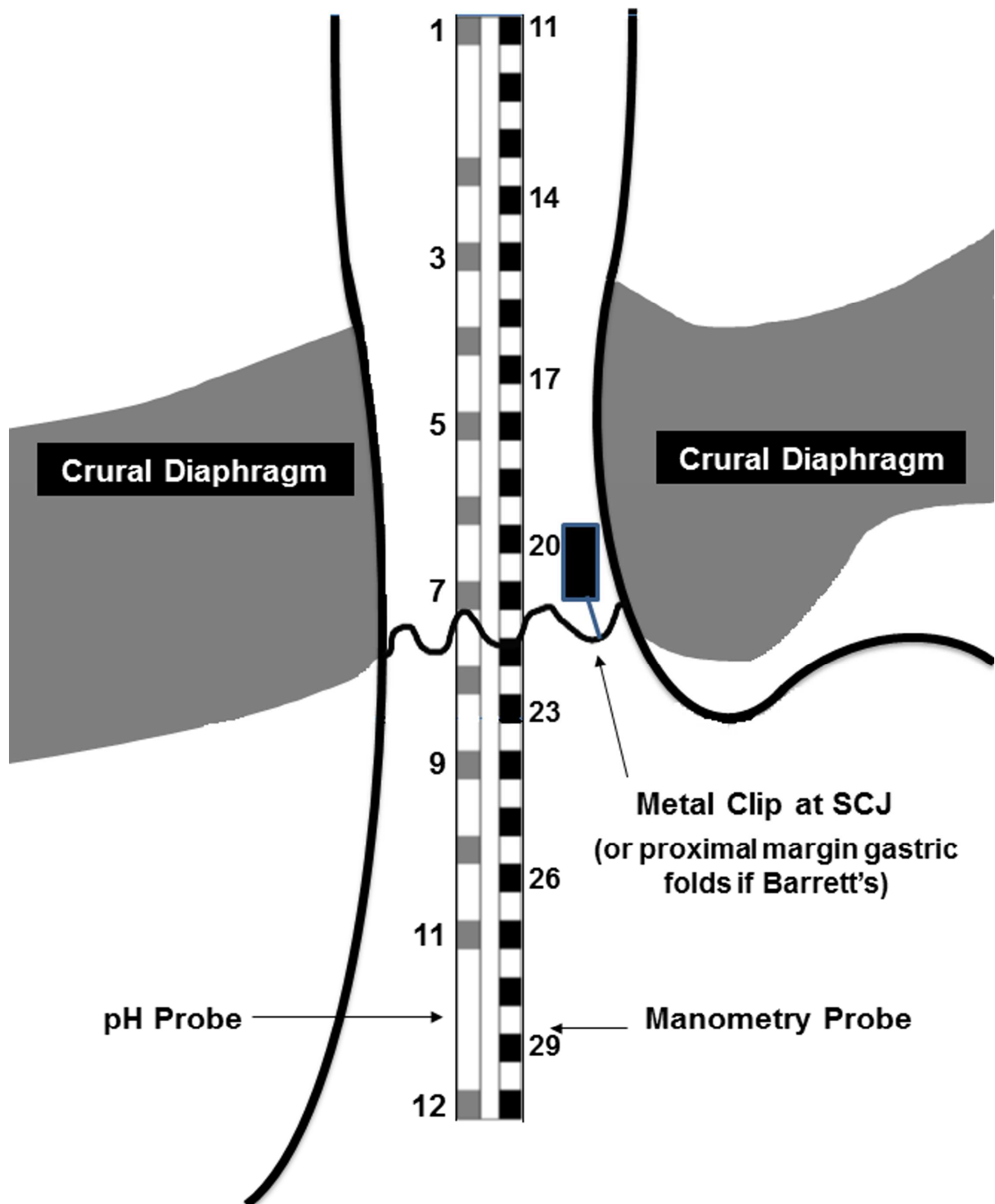
LES = Lower esophageal sphincter, PIP = Pressure inversion point, LESP = Lower esophageal sphincter pressure, SCJ = Squamocolumnar junction.

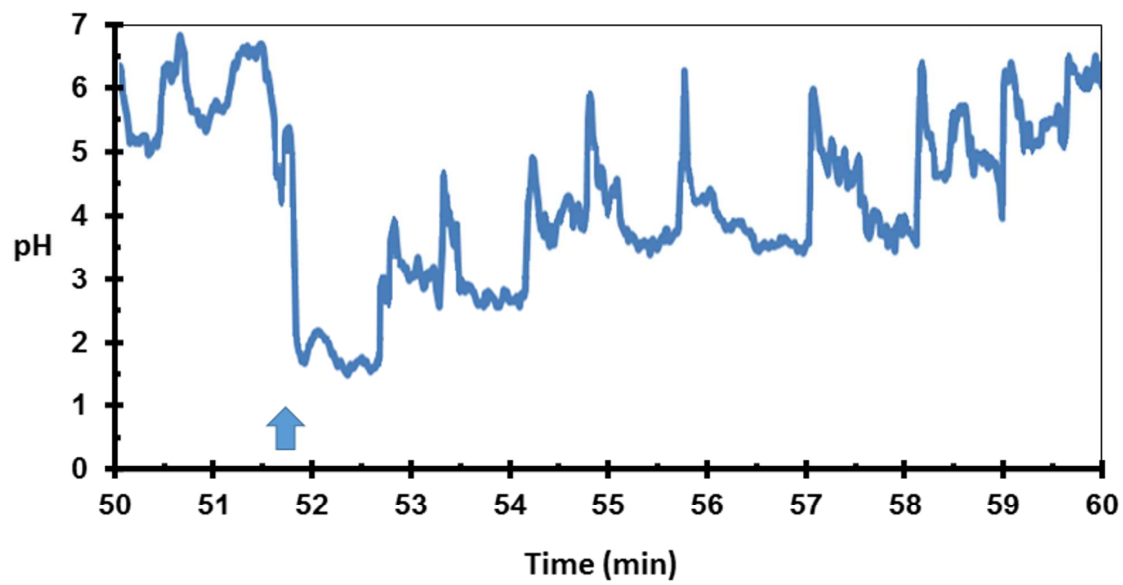
Table 3: Median (IQR) percentage time pH<4 at sensors relative to peak LES pressure comparing subjects with and without waist belt.

Sensor Location	Fasting			Postprandial		
	Belt Off	Belt On	<i>p</i> value	Belt Off	Belt On	<i>p</i> value
5.5cm proximal	0 (0)	0 (0)	0.285	0.2 (1.4)	2.5 (9.6)	0.038
4.4cm proximal	0 (0.1)	0 (0)	1.000	0.5 (2.6)	4.7 (14.3)	0.002
3.3cm proximal	0 (0.1)	0 (0.6)	0.341	1.0 (5.0)	8.0 (31.4)	0.013
2.2cm proximal	0.1 (1.5)	0.1 (1.3)	0.415	3.5 (10.1)	12.4 (41.3)	0.009
1.1cm proximal	0.3 (3.8)	0.4 (9.0)	0.286	7.3 (15.0)	49.7 (52.0)	0.030
Peak LES pressure	2.6 (6.9)	5.2 (41.5)	0.016	18.4 (38.6)	66.1 (42.3)	0.056
1.1cm distal	18.3 (48.2)	55.6 (79.3)	0.019	59.4 (48.5)	89.6 (14.2)	0.026
2.2cm distal	53.4 (57.4)	95.1 (15.8)	0.005	86.7 (19.9)	85.3 (26.2)	0.701
3.3cm distal	88.6 (66.4)	99.8 (5.0)	0.016	88.3 (36.2)	89.8 (31.9)	0.722

Table 4: Effect of waist belt on mechanism of acid exposure across the LES during 90 minute postprandial period

	Belt Off	Belt On	<i>p</i> value
Median no. of reflux events (IQR)	2 (2)	4 (6)	0.008
Median no. TLESRs (IQR)	7 (3.3)	6 (5.3)	0.279
Median no. TLESRs associated with reflux (IQR)	2 (2)	3.5 (5)	0.041
Average clearance time (IQR), seconds	23.0 (63.4)	81.1 (110.6)	0.008
Median no. peristalsis to clear acid (IQR)	1.0 (1)	1.5 (2)	0.074
Median peristaltic distal esophageal pressure (IQR), mmHg	79.5 (48.1)	85.8 (32.8)	0.387





ACCEPTED