Table 1 Sample sizes needed to demonstrate at least 90% success for different individual "true" success rates

Success rate	91%	92%	93%	94%	95%
95% confidence	2217	498	196	96	52
99% confidence	4428	995	392	191	103

Sample sizes were calculated for given true personal success rates (table 1, top row) of endoscopy in endoscopists aiming at demonstrating over 90% success in a test series of consecutive endoscopies. Sample sizes (n) were calculated to achieve confidence intervals not overlapping 90% success according to normally distributed Z statistics and n is given for 95% confidence ($z = -1.645$) as well as 99% confidence ($z = -2.325$). Table 1 can also be interpreted as sample size required with a given sample success rate (top row) to prove a true success rate of over 90% with the two different confidence levels.

might by chance demonstrate <90% success. For a confidence interval not crossing the 90% with a true success rate of 93%, over 196 procedures are needed (95% confidence, table 1 second row). A 5% risk of not proving competence (affecting about one trainee per region) might be seen as too high but 99% confidence (table 1, third row) roughly doubles sample size (table 1).

How about trainers signing trainees off as competent? Table 1 also demonstrates sample sizes with different success rates in test series and confidence intervals not overlapping 90% for the true success rate. Thus documentation of 95% success rate indicates a true success rate of $\geq 90\%$ (with 95% confidence) in a series of 52 (table 1, second row). Again, to avoid signing off incompetent trainees, 99% confidence might be safer.

How about the very good, the lucky, and the endoscopically challenged? Some may demonstrate 100% success in a series. Calculation of confidence intervals are more difficult, such as calculation of the probability of a complication that has not occurred before in a series. Everybody performing interventions should be aware of the size of this risk which can be calculated.³ A series of 30 successful colonoscopies in a row would demonstrate >90% success with 95% confidence.

Less competent endoscopists, with true personal caecal intubation rates <90%, might be lucky achieving by chance >90% success even in large series (table 1). Incompetence might come to light too late and who would be held responsible? The trainee or the trainer?

In the grey area of approximately 90% competency, a number of colonoscopists have to perform very high numbers of endoscopies (table 1, column of 91% competency) but fortunately, with the new cancer screening programme, there will be plenty of opportunity to prove competence in colonoscopy in the lifetime of a new consultant.

M Heydtmann

Correspondence to: Dr M Heydtmann, The Liver Research Laboratories, 5th Floor, Institute for Biomedical Research, Birmingham University, Edgbaston B15 2TH, UK; m.heydtmann@bham.ac.uk

doi: 10.1136/gut.2006.103903

Conflict of interest: declared (the declaration can be viewed on the Gut website at <http://www.gutjnl.com/supplemental>).

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Balloon gastrostomy migration leading to acute pancreatitis

A 57 year old woman with cerebrovascular disease had a 16 Fr Corflo percutaneous endoscopic gastrostomy (Merck Gastroenterology, West Drayton, Middlesex, UK) exchanged for a 20 Fr replacement Corflo balloon gastrostomy (volume 5 ml) as a bridge to eventual insertion of a permanent 20 Fr Corflo-cuBBY "button" gastrostomy. Four weeks later she presented with a seven day history of abdominal pain and vomiting. Initial investigations revealed a serum alanine transaminase level of 54 IU/l (normal range (NR) 0–35), serum alkaline phosphatase level of 491 IU/l (NR 70–300), serum bilirubin level of 7 $\mu\text{mol/l}$ (NR 5–21), and serum amylase level of 703 IU/l (NR <110). An abdominal ultrasound examination demonstrated a dilated common bile duct but no stones were seen in the gallbladder. A subsequent magnetic resonance (MR) cholangiopancreatogram revealed the gastrostomy tube traversing the stomach into the duodenum (fig 1), with the inflated balloon in the second part of the duodenum (figs 2, 3). No filling defects were visible in the biliary tree (fig 3). Forward viewing gastroscopy confirmed the location of the gastrostomy balloon in the duodenum. This was deflated and repositioned in the stomach. Side viewing duodenoscopy confirmed a small erythematous papilla adjacent to the site of the gastrostomy balloon. A diagnosis of acute pancreatitis secondary to obstruction by the gastrostomy balloon was made. Therefore, an endoscopic retrograde

Figure 1 True FISP (fast imaging with steady state precession) magnetic resonance transaxial slice, showing the gastrostomy tube (arrowhead) traversing the stomach into the duodenum.

Figure 2 True FISP (fast imaging with steady state precession) magnetic resonance transaxial slice, demonstrating the inflated balloon (arrowhead) lying in the second part of the duodenum.

Figure 3 Thick slab, single shot, fast spin echo sequence, demonstrating a dilated biliary tree. There were no calculi in the gallbladder or bile ducts. The gastrostomy balloon is visible at the bottom of the image.

cholangiopancreatogram was not performed. Her symptoms settled promptly and blood investigations all returned to normal over a period of six days.

This is the sixth reported case of acute pancreatitis or obstructive jaundice secondary to gastrostomy balloon migration.¹ However, this is the first description to include visualisation of the gastrostomy balloon in situ by MR imaging. We currently advise patients to rotate the replacement gastrostomy tube intermittently in order to maintain tract patency prior to "button" gastrostomy insertion. In our case, we believe that this instruction led to inadvertent distal migration of the gastrostomy balloon, despite the presence of a moveable external anchor device. In the light of this case, we now counsel patients/carers about this rare potential outcome and ensure that a visible reference mark on the external portion of the tube is recognised by the patient/carer in order to maintain an appropriate balloon position in the stomach.

J Periseleris

Department of Gastroenterology, St James's University Hospital, Leeds Teaching Hospitals NHS Trust, Leeds, UK

R England

Department of Radiology, St James's University Hospital, Leeds Teaching Hospitals NHS Trust, Leeds, UK

M Hull

Department of Gastroenterology, St James's University Hospital, Leeds Teaching Hospitals NHS Trust, Leeds, UK

Correspondence to: Professor M Hull, Section of Molecular Gastroenterology, Leeds Institute of Molecular Medicine, St James's University Hospital, Leeds LS9 7TF, UK; M.A.Hull@leeds.ac.uk

doi: 10.1136/gut.2006.105601

Conflict of interest: None declared.

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Does delayed gastric emptying really cause symptoms in functional dyspepsia? We still doubt it!

We enjoyed reading the provocative commentary by Camilleri (*Gut* 2006;55:909–10) on our large study of patients with functional dyspepsia (*Gut* 2006;55:933–9). We completely agree that delayed gastric emptying is a marker of disturbed pathophysiology in functional dyspepsia; indeed, we first reported this observation in 1989.¹ However, we remain unconvinced, based on the available data, that this abnormality alone is linked to upper gastrointestinal symptoms.^{1–3} Factor analysis studies from the general population, in the absence of objective testing, do not provide insights into this issue,^{4,5} although we have shown that non-consulters with dysmotility-like dyspepsia are more likely to have gastric emptying delay.⁶ Studies from selected tertiary care patients are of interest, but Camilleri has previously reported negative results.³ Importantly, in a previous study of 551 patients with functional dyspepsia and 247 patients with type I diabetes and postprandial dyspepsia, gastric emptying, as measured by C¹³ octanoic acid breath testing, was also not linked to any specific upper gastrointestinal tract symptomatology.²

Interpretation of the symptom associations identified does need clarification, as statistical significance in a large study does not equate with clinical relevance. Our primary goal was to determine whether delayed gastric emptying was inducing symptom disturbances that, in turn, impair quality of life. The results from the models clearly suggest that while symptoms are associated with impaired quality of life in functional dyspepsia, gastric emptying is, at best, a very minor contributor and arguably has little clinical relevance.

We therefore dispute the view that there is overwhelming evidence of an association between symptoms and delayed gastric emptying. Indeed, we would argue that there continues to be accumulating evidence that there is often a disconnection between symptoms and slow gastric emptying. This does not negate the fact that delayed gastric emptying is an important biomarker, but it does strongly suggest that the symptom experience is related to other key factors that remain to be accurately identified.

N J Talley, G R Locke III

Miles and Shirley Fiterman Center for Digestive Diseases, Dyspepsia Center, Mayo Clinic College of Medicine, Rochester, MN, USA

J Tack

Department of Gastroenterology, University of Leuven, Belgium

Correspondence to: Dr N J Talley, Mayo Clinic College of Medicine, 200 First Street SW, PL-6-56, Rochester, MN 55905, USA; talley.nicholas@mayo.edu

Conflict of interest: declared (the declaration can be viewed on the *Gut* website at <http://www.gutjnl.com/supplemental>).

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Author's reply

The first demonstration of impaired gastric emptying in functional dyspepsia should be attributed to Rees and colleagues.¹ Additional information provided does not specifically counter the solid arguments offered in the commentary.

In response to the points raised I would like to put forth the following.

Firstly, the reason for dismissing in the published study a positive and significant association between postprandial fullness and gastric emptying is still not provided and appears arbitrary.

Secondly, reference number 2 (Talley *et al* 2001) in the letter was also an observational study of patients with functional dyspepsia and postprandial diabetic dyspepsia (clearly different conditions) participating in a multi-centre drug study using a stable isotope gastric emptying test. The latter was subsequently shown to require more sophisticated mathematical analysis to provide accurate estimates of gastric emptying.^{2,3} As the gastric emptying data in reference number 2 (Talley *et al* 2001) are suspect, they cannot be used to support the claim that gastric emptying does not contribute to symptoms.

Thirdly, the strongest observational data in functional dyspepsia are in the approximately 800 consecutive patients diagnosed and studied in a uniform manner at a single centre by Tack and colleagues.⁴ In this study, a significant association between gastric emptying delay and symptoms of early satiety, nausea, vomiting, and fullness was convincingly demonstrated. It is possible that

discrepancies between the studies may reflect the wide spectrum of patients included under "epigastric pain and discomfort" in the Rome I and II criteria for dyspepsia. The reference to Bredenoord *et al* (reference No 3 in the letter) is quoted out of context as that study had a different aim, and symptoms were evaluated by patient history rather than in response to a standardised meal.

Fourthly, my commentary never claimed that there was overwhelming evidence that gastric emptying alone was responsible for symptoms. Rather, the literature (summarised in Tack's review⁴) shows that combinations of symptoms (typically those now grouped in the "postprandial distress syndrome" in the Rome III criteria for functional dyspepsia⁵) are associated with abnormal gastric functions, including abnormal gastric emptying.

Controlled perturbations of physiology provide a more meaningful appraisal of the causative relationship between pathophysiology and symptom generation. Abnormal gastric emptying is not just a biomarker. Our studies show that several physiological perturbations (for example, gastric emptying, sensitivity, and dysaccommodation) also contribute to the variance in symptoms.⁶ In studies to date, no single factor has explained more than 25% of the variance in symptoms of patients with dyspepsia.⁶ However, this does not justify dismissal of gastric emptying as a causative cofactor in dyspepsia.

The statement that "gastric emptying has arguably little clinical relevance" by Talley *et al* is not based on a fair global interpretation of the available literature.

M Camilleri

Correspondence to: Dr M Camilleri, Clinical Enteric Neuroscience Translational and Epidemiological Research (CENTER) Group, Mayo Clinic College of Medicine, Charlton 8-110, 200 First St SW, Rochester, MN 55905, USA; camilleri.michael@mayo.edu

Conflict of interest: None declared.

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Association of tumour necrosis factor α promoter haplotype with chronic pancreatitis

Genetic risk factors are attributed an important role in the pathogenesis of chronic pancreatitis.¹ The genetic basis of chronic pancreatitis is complex. The cationic trypsinogen gene,² serine