Molloy and David Kingsmore of a near-fatal complication of stapled haemorrhoidectomy. How the pelvic sepsis arose is not clear. A possibility is that the firing of the stapler enabled gas-producing organisms in the rectal lumen to enter the pararectal space. Other invasive methods of treatment of haemorrhoids have rarely been complicated in a similar way. Molloy and Kingsmore's comment that antibiotics be given prophylactically seems reasonable.

Yet another complication, which may arise if the purse-string suture is placed too close to the dentate line, is partial excision or injury of the internal sphincter, with perhaps short-term or long-term sphincter dysfunction. If the purse-string suture is placed too high, residual external haemorrhoidal skin tags may remain (as occurred in five of 20 cases in the Hull study) and later cause symptoms. A suggested prophylactic technique is submucosal injection of saline at the planned site of the purse-string suture, to reduce the risk of incorporation of rectal muscularis propria into the stapler housing.

Even if the sphincter is not incorporated into the tissue ring that is excised, the internal sphincter can be damaged when it is stretched by the stapler head (33 mm diameter[1]) or by the anoscope used during placement of the purse-string suture. That the anoscope can damage the sphincter was shown in a study using a stapled ileopouch anal-anastomosis model.[2] This study showed that resting sphincter pressure was lower with a transanally placed than with transabdominally placed purse-string suture, for which no anoscope was used. Impaired sphincter function may occur with conventional haemorrhoidectomy. Some surgeons have noted problems with the stapled technique when there is a very large internal haemorrhoidal component. The tissue can be simply too bulky to be incorporated into the stapler housing. The Leicester group point out that, with the stapling procedure, not all of the haemorrhoids have to be excised, which lessens the chance of inclusion of pain-sensitive anoderm where the caudad extent of the haemorrhoids lie. Other advantages are that haemorrhoids in the lower anal canal are preserved (not unlike the case with elastic-band ligation), and that the prolapsed haemorrhoidal tissue is drawn back into a more physiological position.

Despite these concerns, there is no getting around the fact that the evolution of and advances in the treatment of haemorrhoids have occurred because of the deserved reputation that haemorrhoidectomy has of being a painful procedure. Cost, and to an extent long-term effectiveness of treatment, are lesser considerations. The two reports published today provide evidence of the advantage of stapled haemorrhoidectomy in terms of a lessened degree of pain and restoration of normal activity in the short term. The data on length of stay in hospital favours the stapled technique in the Leicester but not in the Hull study. This variable is notoriously difficult to assess because of the difficulties in defining exact criteria for discharge, in standardisation of assessment of postoperative analgesic consumption, in masking those who decide on date and time of discharge to type of procedure, and in the reporting of readmission rates.

How should the value of stapled haemorrhoidectomy be investigated further?

- Doing multicentre trials, ideally without sponsorship by stapler manufacturers, will strengthen conclusions. A point to note is that the power calculation for the Leicester trial was based on length of stay rather than on pain scores.
- Pain-assessment data should include amounts of intravenous and oral narcotic drugs used postoperatively.
- Prospective assessment of the test and control groups should include objective (ie, manometric and sonographic) measures of sphincter function, which should be followed up long term with validated continence-scoring systems.
- Long-term complications, especially stricture and recurrence of haemorrhoid symptoms, should be investigated.

Meanwhile, the data available on stapled haemorrhoidectomy indicate that the procedure looks promising.

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Treatment of chronic bulimic symptoms: new answers, more questions

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Bulimia nervosa has been recognised for only about 30 years.[1] Its essential features are binge eating and inappropriate methods of preventing weight gain. The self-esteem of these individuals is excessively influenced by body shape and weight.[2] Treatment consists of psychotherapy, antidepressant drugs, or both. A recent review reports on efficacy of cognitive-behavioural psychotherapy,[3] but other forms of psychotherapy, such as interpersonal psychotherapy, can also be effective.[4] Most placebo-controlled trials of antidepressants have confirmed the efficacy of the active agent in reducing binge-eating and purging. In clinical practice, serotonin-reuptake inhibitors are the most commonly used drugs because they do not produce many side-effects. The combination of psychotherapy and antidepressants seems to be more effective than either alone.[5]

Although the short-term effect of therapy is impressive, the long-term outcome is not. 5 to 10 years after initial presentation, there is no difference in recovery rates between treated and untreated individuals: 20% of individuals continue to meet full criteria for bulimia nervosa and another 30% experience relapse of minor bulimic symptoms.[6] Because there is so little to offer chronic bulimic patients, the report in today's *Lancet* by P L Faris and colleagues on the effect of ondansetron on bulimic symptoms is of interest.

The use of ondansetron, a selective serotonin ($5\text{-}HT_3$) antagonist, is well established for patients with nausea and vomiting associated with cancer chemotherapy, radiotherapy, or anaesthesia.[7] Faris and colleagues show, in their randomised double-blind trial, that ondansetron reduces binge-eating and self-induced vomiting. They suggest that ondansetron might be a treatment option for chronic bulimia. They hypothesise that increased afferent vagal activity underlies the perpetuation of bulimia nervosa and that ondansetron decreases this activity.

There is no doubt that ondansetron reduces bulimic symptoms, but some questions remain about how the drug works and whether it is really a treatment option for patients with bulimia.

The first question is whether the effect of ondansetron is

restricted to the gut. Faris and colleagues suggest that ondansetron acts predominantly on the peripheral nervous system, where it reduces afferent vagal neurotransmission. However, novel applications of ondansetron, such as for the treatment of psychosis in advanced Parkinson's disease, indicate that ondansetron exerts a direct effect on the central nervous system.[8] Furthermore, prevention of vomiting induced by anticancer drugs involves blockade of 5-HT_3 receptors at central sites, such as the area postrema and nucleus tractus solitarius.[7] Thus the possibility remains that the improvement in bulimic symptoms by ondansetron is in part due to its central action.

The second question is whether there is really evidence of vagal pathophysiology in bulimia, as suggested in the title of the paper. Faris and colleagues base their hypothesis on the facts that in bulimic patients the process of satiation is altered and that the pain thresholds are raised. However, satiation is the result of a complex process involving not only afferent vagal activity, but also hormonal and metabolic systems. Altered pain thresholds remain an indirect indicator of abnormal intestinal vagal physiology. Since there is no suitable direct way of measuring intestinal afferent vagal activity in human beings, the evidence for vagal dysfunction in bulimia will be difficult to prove.

Does the reduction in binge-eating and vomiting influence other bulimic symptoms? Suppose the binge-eating and self-induced vomiting are perpetuated by raised afferent vagal activity. If this pathophysiological mechanism is of such central importance in perpetuating the whole disorder, other essential features of bulimia nervosa, such as distorted body image and drive for thinness, should be affected by ondansetron. There are no data on these issues.

The fourth question is what does it mean to chronic bulimic patients to vomit once a day instead of twice. Only the patients can provide this answer. In other areas of medicine, such as oncology, the relation between treatment and quality of life is assessed in part by asking the patient what the improvement in symptoms really means for him or her in daily life.[9] If chronic bulimic patients were to perceive symptom reduction as an enhancement of their quality of life, the drug would be a valuable novel treatment option.

Faris and colleagues' findings are promising, but at least some of the questions raised above should be answered before ondansetron is recommended for the treatment of chronic bulimia. Finally, a temptation to give ondansetron in addition to a serotonin-reuptake inhibitor should be resisted because there has been a report that a patient, whose major depression was well controlled with fluoxetine, had severe dysphoria when given ondansetron.[10]

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Renal conservation for gas-forming infections

A century has past since the recognition of pneumaturia,[1] a sign possibly due to gas-forming bacterial infections of the kidney. Such infections are unusual, severe, and life threatening. Emphysematous pyelitis (gas in the collecting system) occurs when an obstructed kidney is infected by a fermenting organism. Management consists of relief of obstruction and antibiotics, as with any pyonephrosis. A more severe disorder is emphysematous pyelonephritis, in which there is a necrotising infection, with microabscesses and gas bubbles in the renal parenchyma and sometimes in the perirenal tissues as well. Gas can also be found in localised renal or perirenal abscesses. Emphysematous pyelonephritis occurs predominantly in diabetic patients, who account for 90% or more of cases, but also in debilitated (eg, alcoholic) or immunocompromised patients.[2] Most of the infections are caused by *Escherichia coli*, *Klebsiella*, or a mixture of organisms. Gas formation is believed to require pathogenic bacteria capable of mixed-acid fermentation, a hyperglycaemic milieu, and local tissue ischaemia, which exacerbates tissue destruction, encourages purulent infection, and inhibits the removal of locally produced gas.[2,3]

Emphysematous pyelonephritis can usefully be categorised according to appearances on computed-tomography (CT) scans.[4] Type I emphysematous pyelonephritis runs a fulminant course, with a mortality of about 70%, and is characterised by renal parenchymal destruction with a streaky or mottled picture of gas distributed radially along the pyramids. In most series more than 95% of type I patients have diabetes. Type II emphysematous pyelonephritis runs a more protracted course and carries a lower mortality, of about 20%.[4] This type gives a CT-scan picture of renal or perirenal fluid with bubbles or locules of gas. In some cases, there may be progression from type I through type II to the formation of gas-filled abscesses.

Because of the considerable potential for morbidity and mortality, early diagnosis of gas-forming renal infections is important. However, in most series there is an average interval of about 18 days between onset of symptoms and diagnosis.[5] The delay may be due to the non-specificity of symptoms or the use of inappropriate radiological techniques for investigation. The gas may be obscured by overlying bowel or be below the limit of resolution of plain radiography or intravenous urography. Emphysematous pyelonephritis should be suspected when a patient with diabetes, especially a woman, has a urinary-tract infection and signs of sepsis, loss of glycaemic control, and raised serum creatinine concentrations.[2,3,6] Such patients require not only hospital admission and vigorous antibiotic therapy and resuscitation but also prompt investigation, initially by renal ultrasonography, followed by CT scanning if gas is detected or if the patient does not significantly and rapidly respond to therapy. A management algorithm is proposed in the figure.

The accepted teaching is that nephrectomy is the