Editorial/Van die Redaksie

Is there a role for surgery in the management of reflux oesophagitis?

Gastro-oesophageal reflux occurs for 3% of the measured time in normal people, but does not produce oesophagitis because of an effective acid-clearing mechanism and a mucosal resistance to hydrogen ions. A mechanically defective lower oesophageal sphincter is the primary disorder responsible for abnormal reflux, but poor acid-clearing resulting from defective motility, a high acid output, bile contamination of refluxing gastric content and delayed gastric emptying may contribute to the severity of the damage caused.

The potential for reflux is linearly related to lower oesophageal sphincter pressure.[1] Recently, Dent *et al.*[2] have shown that lower oesophageal sphincter relaxation, which occurs independently of swallow responses, is the commonest defect in the lower oesophageal sphincter mechanism in abnormal reflux. They suggest that this abnormality is related to a neural defect in the oesophagus. There is also evidence to support the concept that there is a progressive deterioration of oesophageal muscle power with increasing severity of reflux.[3] In particular, lower oesophageal sphincter pressure and the height of contraction responses in the body of the oesophagus are markedly decreased in advanced reflux disease with stricture or Barrett's syndrome (columnar-lined epithelium in the oesophagus). Non-propagating contraction responses to swallowing are more prevalent in advanced disease, again suggesting that reflux has caused neural damage to the intrinsic oesophageal nerve plexuses. We have confirmed this finding in an analysis of motility responses to swallowing in the columnar-lined oesophagus, which is considered to be end-stage reflux disease.

Ogilvie *et al.*[4] have demonstrated that although vagal impairment is common in reflux oesophagitis, it is not confined to the alimentary tract, suggesting that it is not reflux-induced but that it may be important in the pathogenesis of reflux oesophagitis. Transmural fibrosis has been found in resected specimens from such patients.[3]

Clinically, such abnormalities are associated with a variety of symptoms such as heartburn, dysphagia, chest pain, haemorrhage, acid laryngitis, vocal cord polyps and 'gastric asthma'. Reflux in infants may interfere with feeds and result in failure to thrive. The Sandifer syndrome in children is caused by gastro-oesophageal reflux, and presents with dystonic movements of the head and neck. Life-threatening aspiration and bronchial asthma can be associated with inhalation in infants with gastro-oesophageal reflux.

Conservative therapy has been the predominant treatment for the majority of patients presenting with reflux oesophagitis, and consists of weight control, postural treatment and antacids. With the advent of the H_2-receptor blocking drugs and prokinetic agents, responses to medical treatment have been infinitely better. More recently, omeprazole therapy has given remarkable results.

We now need to consider how effective medical therapy is in the management of gastro-oesophageal reflux. In one study on infants and children,[5] postural therapy together with antacids, bethanechol and cimetidine was effective by 18 months of age in 80% of patients, but surgery was necessary in 17% of children in the series. The remarkable results of Nissen fundoplication in children, and particularly in retarded children, have been reported by numerous authors in recent years. Fonkalsrud *et al.*[6] reported excellent results with low morbidity and mortality in 270 children, illustrating how frequently the operation is now performed in paediatric practice. Definite indications for surgery in infants and children with gastro-oesophageal reflux therefore include a failure to thrive, respiratory complications and failure to respond to medical treatment. The period of trial of medical therapy must be individualised according to the clinician's evaluation, but if this is too prolonged it could be detrimental to the patient and to oesophageal function. Similarly, not all adult patients respond to medical therapy.

Behar *et al.*[7] identified a group of patients with reflux oesophagitis who did not respond well to medical therapy — those with an incompetent lower sphincter, free-acid reflux and severe oesophagitis. More recently Lieberman and Keeffe[8] independently reported on poor results of intensive medical therapy, which included the use of H_2-receptor blockers and prokinetics in a group of patients with poor lower oesophageal sphincter pressure. Surgery was eventually necessary in 8% of their cases.

The above two reports from physician gastro-enterologists have been the first attempts to look at the problem realistically without condemning all categories of patients to medical therapy only, which is ineffective in a small number of cases. Mucosal protection with sucralfate has given results equal to those reported with the use of cimetidine,[9] and may have an added benefit in patients with alkaline reflux. Newer and more powerful acid blockers and prokinetics are being evaluated, and a combination of omeprazole and cisapride will surely give even better results. But these patients will be drug-dependent at great cost, and possible detriment to health. Furthermore, as yet there is no proof that the progressive deterioration in oesophageal function associated with reflux will be prevented by these drugs. The motility defects in the oesophagus are irreversible, even after

surgical therapy. Should these defects be allowed to progress to such a state of irreversibility?

In our experience, prokinetic drugs have the least effect where needed most, viz. poor lower oesophageal sphincter pressures and impaired oesophageal function.[10] The reason for failed medical therapy may be related to poor motility, resulting in inadequate acid clearance, and 'alkaline' reflux. There is no satisfactory medical treatment for 'alkaline' reflux, and bile-diverting procedures have often been the only solution for intractable reflux complications. The operation of antrectomy, vagotomy and Roux-en-Y diversion produces excellent results in 80-85% of cases,[11] and the newer 'duodenal switch' operation[12] (suprapapillary duodenojejunal anastomosis) may give superior results because the pylorus and vagus nerves are spared.

Advances in surgical technique are thus taking place concurrently with the introduction of newer and better drug therapy. These operations are aimed at restoring a mechanically defective lower sphincter, decreasing acid output or deviating irritant duodenal secretions. In extreme cases, oesophageal replacement is necessary, particularly in reflux strictures that have not responded to dilatation and medical therapy.

All clinicians will agree that the best treatment for other mechanical defects, such as inguinal hernia and rectal prolapse, is surgical. With a very low morbidity and mortality rate, a simple anti-reflux operation will give a success rate of 91% over 10 years[13] and is surely in the same category?

A stitch in time may prevent neural damage from long-continued reflux, and in many cases must be the correct therapy for serious reflux oesophagitis. The discomfort of wearing a truss for a hernia may be compared with the restricted living on medical therapy for serious reflux. The persistent use of conservative measures by some clinicians is very distressing to many patients, and the question should be asked whether it is ethical to withhold a better alternative in these selected cases.

Repeated endoscopies we have performed over a 6-year period in 18 patients with a columnar-lined oesophagus, treated vigorously with H_2-receptor blockers and prokinetics, have shown a progression of the columnar segment in 14 patients. This is evidence that reflux has not been adequately controlled on these medical measures. The H_2-receptor blockers have not given satisfactory results in all patients when used in a regular dosage schedule, and double-dosage regimens have now been recommended. From reported results of the medical management of reflux oesophagitis, it is obvious that not all patients will benefit adequately from such therapy and that surgery in these cases gives predictably good results. In many instances the failure of surgery has been the result of inadequate assessment of the physiological defect or poor technical surgery, and such operations by inexperienced surgeons will not equal the expected good results. Whether long-continued medical therapy will prevent deterioration of oesophageal function or not remains to be tested. Repeated physiological tests of oesophageal function over long periods of time will be necessary to answer this question.

The definite indications for surgery in adult patients therefore are a failure of medical therapy and pulmonary complications of reflux. It is our conviction that reflux strictures of the oesophagus and the columnar-lined oesophagus are further definite indications[14–16] for surgery, but such complications are frequently treated world-wide by medical measures, which include drug dependence, frequent consultations and a more restricted lifestyle than that offered by successful surgery. That there is no permanent cure by medical therapy is a fact infrequently explained to the patient, and compliance with long-continued drug taking, particularly from younger and middle-aged patients, is an unusual event.

Yes, there are definite indications for the surgical correction of gastro-oesophageal reflux. However, before surgery is undertaken, appropriate investigations to elucidate the pathophysiology of the defect are mandatory. Corrective surgery can then be planned to give optimal and long-lasting results.

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