GASTRO-OESOPHAGEAL REFLUX

Pages with reference to book, From 130 To 131

Gastro-oesophageal reflux is a common gastro intestinal disorder and consists of a retro-grade flow of gastric contents across an incompetent gastro-oesophageal junction into the oesophagus. It may be demonstrated in normal individuals (Fisher et al., 1976), and whether in a given instance it is pathological or physiological, requires a careful examination of the anti-reflux mechanism, the defence mechanism of the oesophageal mucosa and the quality and quantity of the refluxed material.

A positive pressure gradient exists across the gastro-oesophageal junction, necessitating an anti-reflux mechanism. A physiological lower oesophageal sphincter (Lipshutz and Cohen, 1973), which is a 2 to 5 cms zone of elevated pressure at the junction of the oesophagus and stomach, is generally regarded to be the major determinant of gastro-oesophageal competence, and correlates inversely with the quantity of reflux (Cohen and Harris, 1970). Hormonal, neural and mechanical factors have been proposed as regulating the resting sphincter tone, which may alter in response to food ingestion (Nebel and Castell, 1972), or to changes in intraabdominal pressure (Dodds et al., 1975).

Oesophagitis is a multifactorial disorder, diminished tissue resistance is one of them. Sensitivity of the epithelium to the digestive action of gastric juice, its regenerating capacity and its permeability to H^+IONS are factors affecting tissue resistance. Other factors include acid and pepsin which has maximal proteolytic activity at pH 2.0. Increased incidence of oesophagitis in patients with gastric acid hypersecretion has been reported (Goldman et al., 1967). The roles played by bile-salts (Gillison et al., 1972), pancreatic enzymes (Cross and Wangensteen, 1951), and the defensive action of saliva and oesophageal clearance mechanism (Booth et al., 1968) have been considered. Delayed gastric emptying has been suggested as a significant factor in the pathogenesis of reflux oesophagitis (Ippoliti et al., 1976).

Gastro-oesophageal reflux presents as heartburn aggravated by recumbency or by maneuvers which increases intra-abdominal pressure. Eructation of gastric contents and dysphagia due to lower oesophageal motor dysfunction and stricture formation may occur. Weight loss and anaemia may be due to decreased food intake and occult or massive blood loss. The intensity of symptoms correlate with the contact time of the refluxed material (Booth et al., 1968).

Diagnosis of gastro-oesophageal reflux may be aided, especially in atypical cases by Barium swallow, cine-oesophagography and endoscopy though in many instances symptomatic oesophagitis may show no mucosal abnormalities on endoscopy (Banatwala et al., 1980; Ismail-Beigi et al., 1970).

Oesophageal mucosal biopsy provides an indirect evidence of gastro-oesophageal reflux (Fisher and Cohen, 1978), which can also be inferred by detecting a lower oesophageal sphincter pressure of less than 10 mm Hg on oesophageal manometry (Haddad, 1970). Acid perfusion of the oesophagus gives a good correlation between the presence of oesophagitis and a positive test (Benz et al., 1972; Banatwala et al., 1980). Gastro-oesophageal reflux may also be detected by demonstrating a pH below 4, at 4 to 5 cms above the lower oesophageal sphincter and with the help of a radio-nucleotide marked on scintiscanning (Fisher et al., 1976).

Prevention of gastro-oesophageal reflux is achieved by weight reduction in the obese, and by avoiding maneuvers which increases intraabdominal pressure such as straining at defecation or lifting heavy objects, stooping and wearing tight garments. Elevating the head end of the bed on blocks decreases nocturnal reflux (Johnson and Demeester, 1974). Coffee and spicy foods which increase acid secretion and fatty meals, chocolates and smoking which decrease lower oesophageal sphincter pressure are best avoided (Fisher and Cohen, 1978). Antacids buffer gastric acid and increase the lower oesophageal sphincter pressure (Castell and Levine, 1971). The latter action is also shown by Bethanechol-a cholinergic agent (Farrel et al., 1974) and by Metoclopramide which also accelerates oesophageal

clearance and gastric emptying (McCallum et al., 1975). Failure of medical management, haemorrhage, stricture or ulcer formation, and repeated aspirations are all indications for surgical treatment, which aims at creating increased resistance at the gastro-oesophageal junction (Battle et al., 1973).

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