25 Cholelithiasis after Vagotomy

25.1 Incidence

Many observations in the past have suggested that interruption of the vagus should result in a higher incidence of cholelithiasis, possibly due to a decreased biliary flow [1–8], but whether a sequence of a higher incidence of gallstones after gastric surgery really exists remains to be proven [9–10]. A number of reports incriminated vagotomy as a causal factor in cholelithiasis. Clave and Gaspar [3] reported an increased frequency of cholelithiasis in 116 patients who had undergone truncal vagotomy. Unfortunately many of these studies are open to criticism. Thus in the study of Clave and Gaspar [3] no control group was analysed, and the significance of 22.8% for those developing gallstones after vagotomy may not be much greater than the frequency in the control population.


Clave and Gaspar [3] reported in 1969 another 21 cases. In their series of 116 patients who had a truncal vagotomy and pyloroplasty, 92 were submitted to routine postoperative roentgenography. Radiographic evidence of gallbladder disease was found in 22.8%, in contrast to the 8.22% probability determined by studies in the general population. More importantly, of 52 patients who had a normal cholecystogram preoperatively and a normal palpable gallbladder at the time of vagotomy and pyloroplasty, 23.5% had postoperative X-ray evidence of gallbladder disease. There has been reported to be no increased incidence of stone formation following selective vagotomy in which the vagal innervation of the biliary tree is preserved [7]. It is not clear in what manner operations on the stomach may affect the biliary tree and various suggestions have been put forward. Among the most prominent has been the idea that vagal denervation is likely to induce hypotonicity of the gallbladder and thus encourage biliary stasis [11, 12]. Johnson and Boyden [13] were among the first to implicate vagotomy as a cause of biliary dysfunction in man. Their studies on the motor activity of the gallbladder following vagotomy serve as a basis for the bile stasis theory of stone formation following this procedure.

25.2 Pathogenesis

Truncal vagotomy has also been shown, by others, to produce dilatation of the gallbladder in man [3, 13–17], whereas according to three reports selective vagotomy does not [16–18]. In addition, truncal vagotomy leads to a diminished
Pathogenesis

Fig. 82. Gall-bladder contraction after a meal in preoperative duodenal ulcer patients and patients after truncal vagotomy and pyloroplasty or proximal gastric vagotomy. Truncal vagotomy and pyloroplasty is followed by a significant dilatation of the gall-bladder (volume increased), but there is no impairment of gall-bladder contraction following a meal in truncal vagotomy and pyloroplasty (After Parkin et al., 1973 [21])

bile flow [9, 15, 19] and to the production of a potentially lithogenic bile [9, 15, 20] in dogs and may predispose to the formation of gallstones in man [3–6]. Parkin et al. [21] measured the gallbladder volume contractility in patients after truncal, selective and highly selective (parietal cell) vagotomy in man. The resting volume of the gallbladder was markedly increased in patients with truncal vagotomy and pyloroplasty compared to duodenal ulcer patients before the operation, but unaltered in highly selective and selective vagotomy (Fig. 82). Thus it may be concluded that the gallbladder is abnormally large after truncal vagotomy. It was to be expected that highly selective vagotomy should not increase the volume of the gallbladder, since in this kind of operation vagotomy is confined to the parietal cell mass. In selective vagotomy the hepatic and coeliac branches are preserved. The gallbladder has been shown to dilate progressively in the course of the first year after truncal vagotomy [13], which may explain why Glanville and Duthie [22], who measured the volume of the gallbladder less than 1 year after truncal vagotomy, did not find significant dilatation. That the vagally denervated gallbladder contracts vigorously in response to fat in the intestine has been known for many years [22, 23, 13, 24]. It has been shown that stimulation of the vagi leads to an increase in bile flow, both in man [25] and in dogs [15, 26]. Stimulation of bile flow thus cannot occur after truncal vagotomy. In dogs, truncal vagotomy leads to diminution