
### 23 Vagal Denervation Syndrome

#### 23.1 Pathophysiology

The physiology and pathophysiological changes of the motor activity of the stomach are very complex. Besides nervous factors, gastro-intestinal hormones are mainly involved, as is demonstrated in Table 44. Furthermore, receptors in the duodenum represent important regulatory mechanisms; these receptors respond to distention, changes in pH and osmolarity, etc. Three complexes have to be considered separately: (1) motility of the stomach, (2) motor activity of the pylorus, and (3) gastric emptying.

The classical concept included cholinergic vagal stimulation and adrenergic sympathetic inhibition of the smooth muscles of the stomach [2]. Besides these, non-cholinergic, non-adrenergic nerve fibres exist, which are now named peptidergic. At least some of these fibres can be activated by reflexes from oesophagus or stomach.

The anatomical distribution of the stomach in corpus, fundus and antrum cannot be maintained by the motoric physiology of the stomach [3]. Food intake induces a receptive relaxation of the proximal stomach, so that this part acts as a reservoir for solid and liquid food particles [2, 4]. While liquids reach the duodenum mainly by a tonus increase of the middle portion of the stomach, solid food particles are transported to the duodenum above all by contractions of the distal part. The peristaltic activities of the distal stomach originate from a so-called pacemaker, which is located in the upper third of the stomach, close to the greater curvature [5, 6].
Each type of vagotomy considerably alters the motor activity of the stomach, influencing both peristalsis and receptive relaxation. After total denervation of the stomach, the regular rhythm of the pacesetter potential is replaced by multiple ectopic pacemakers with ineffective propagation of gastric peristalsis [7, 8]. Whereas in selective gastric and truncal vagotomy all vagal fibres to the stomach are transected, selective proximal vagotomy results only in vagal denervation of the proximal stomach.

While all vagotomy procedures which result in total denervation of the stomach produce a delayed gastric emptying, to some extent of liquids, but mainly of solid food, the findings after selective proximal vagotomy are more complex: gastric emptying of fluids seems to be accelerated during the initial phase, but normal in the later phase [8–10]. Emptying of solid food after selective proximal vagotomy (SPV) is either unchanged [8, 10] or delayed [9]. When SPV is combined with a pyloroplasty, gastric emptying of solid and liquid food is more rapid [10].

Adaptive relaxation of the stomach is abolished after all types of vagotomy, since the reflex areas to the gastric fundus are interrupted. This phenomenon seems to be responsible for the complaints of epigastric fullness observed by most patients; furthermore, it is possible that the initial rapid gastric emptying of fluids is caused by the lack of receptive relaxation of the gastric corpus and fundus.

To what extent the function of the pylorus is altered by vagal denervation is unknown at the moment, since no reliable methods for the measurement of pyloric function are available. After vagal denervation the pylorus is adjusted to a certain diameter, which will not open in co-ordination with antral motor activity; it will thus act as a relative stenosis. This lack of co-ordination causes a further delay of gastric emptying.

However, delayed gastric emptying is also observed during the early post-operative phase in patients with simple gastro-enterostomy, pyloroplasty and partial gastrectomy without vagotomy. The pathophysiological mechanisms are unknown. This phenomenon is particularly observed in patients with organic pyloric stenosis and dilatation of the stomach.