Introduction

Normal control of acid secretion depends on endocrine (gastrin), neural (vagal cholinergic nerves), and paracrine (histamine) limbs. Ingestion of meal causes increased acid and pepsin secretion due to an increase in gastrin release and vagally mediated cephalic stimulus to the parietal cell. After gastric and duodenal pH has been lowered, gastrin release is inhibited and acid secretion returns to baseline. Pepsinogen is secreted from the chief cell in response to gastrin and histamine. In the presence of acid, pepsinogen is elevated to pepsin, which is active at pH less than 4. It has been shown that acid is much more damaging to intestinal mucosa in the presence of Pepsinogen (Mertz, 1991).

Up until fairly recently it was usual for a lecturer of gastroenterology to say, “we know little more than we did one hundred years ago about the cause and treatment of peptic ulcer disease.” Actually there have been notable and significant advances with regard to etiology, pathogenesis, pathophysiology, and biochemistry of this disease, as well as what might prove to be dramatic advances in medical management (Chapman, 1978).

Modern therapy is extraordinarily effective in the management of gastric and duodenal ulcers, like Black’s Nobel Prize for creating H2 blockers. Such success, however, make doctors assume that every ulcer is caused by excess acid. But the bismuth compounds and antibiotics also speed the healing of ulcers; some remain unconvinced that every peptic ulcer is caused by Helicobacter pylori because of the very ubiquity of that alien invader. However, it may be equally simple to deem all ulcers the product of excess acid.

Indeed, different comments were on “heterogeneity” of peptic ulcer, to suggest that ulcer craters have different causes. Some, like those of the Zollinger-Ellison syndrome, are clearly erosions from a torrent of hydrochloric acid. In some patients with arthritis, are burned into a defenseless mucosa deprived of its protective prostaglandins by an excess of anti-inflammatory agents; the growing incidence of peptic ulcer in older women attest to that. The gastric ulcers turning up in young “crack” smokers tell of the ischemic origin of yet other ulcer. Some ulcer may even be the result of “stress”. In any event, growing agreement that “peptic ulcers” are multifactorial in origin means that there is no more reason to think that an ulcer crater signifies a specific disease (Spiro, 1991).
It is possible to divide peptic ulcers into three etiologic groups: those due to massive acid peptic hypersecretion in the Zollinger-Ellison syndrome; those due to non-steroidal anti-inflammatory drugs (NSAIDs); and ulcers associated with *Helicobacter pylori* infection. *H.pylori* related ulcers forms the largest and least well understood subset of ulcer disease. (Mertz,1991).