The sons and daughters of Tagamet

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While clinical gastroenterologists and investigators continue to maintain the faith of 'no acid, no ulcer', this is unlikely to remain sine qua non, since the defence establishment is gaining more than a foothold in the door with the redirection of attention to the possibility of disturbed prostaglandin metabolism existing in patients with ulcer disease. The decline in the political agitation of the 70s has been replaced by the concern for the 'integrity' of the late 1980s, with mucus and bicarbonate secretion, unstirred layers, restitution and normal lipophobicity of the gastric epithelium. While we debate the maintenance of the term 'cytoprotection', there is a growing awareness of the importance of the integrity of Davenport's long respected gastric mucosal barrier. What have the past 10 years of therapeutic adventurism brought? First, second and third generation therapy will march forth between the pages of advertising copy. Certainly our understanding of the pathophysiology of ulcer disease and acid secretion itself has been greatly enhanced with our acquisition of tools for modifying the parietal and gastrin cell receptors, adenylylase activity, calcium entry and H⁺,K⁺-ATPase. Perhaps, in the end, the 'Goliath' of acid inhibition, omeprazole, may be replaced by the 'David' of understanding, with moderate acid inhibitory effects becoming the gold, or at least the silver standard. But what is the gold standard in this bull market, as we move two by two into the dark of many more than 40 days and 40 nights of inundation with the growing menu of newer therapeutic agents? We move from alpha to omega, from antacids to Zantac (ranitidine; Glaxo). But ulcer disease remains perplexing in its genesis and vexing to treat. With the slick invention of 'cost effectiveness', what has happened to the patient advocate? Has the proliferation of newer agents contributed to the welfare of patients with acid peptic ulcer? There is no doubt that the hospitalization rate, mortality rate and number of surgical procedures performed for peptic ulcer disease were already declining before the introduction of cimetidine. With the development of any new therapeutic agents, such as misoprostol or famotidine, have patients benefitted, or have the sins of the father simply been passed on to the son, unto the seventh generation of the sons of Tagamet (cimetidine; Smith Kline & French)? In this issue of The Canadian Journal of Gastroenterology, we are provided with new information on the use of an old friend, tried and true Tagamet. Clinically relevant material has been provided which focuses upon human disease and the needs of our patients. We remain with the impression that Tagamet, Zantac, and now Pepcid (famotidine; Merck Sharp & Dohme) do represent effective therapeutic modalities for the treatment of peptic ulcer disease. In this age of vices, what better than to have a further choice of safe, effective agents for what is rapidly becoming a disappearing disease? This research into clinically relevant material has also provided new insights into the physiology of acid secretion and recognition of stimulating and inhibiting receptors for prostaglandins on the parietal cell. Soon we will be inundated with the enthusiastic, if not occasionally fastidious, approach of the detailed person who will wish to convince us that in this era of perceived shrinkage of resources, the ravaged gastric and duodenal mucosa can be protected by reducing the aggressive and enhancing the defensive aspects of the alleged imbalance between the aggressive and defensive factors of ulcer disease. With its frequent recurrences, and with the entry of accountants into the healthcare team, what will be the cost of this new advance? Within recent memory medical texts suggested that the only effective therapy for ulcer disease was six weeks' rest in hospital and abstinence from smoking. In 50 years less than three score and 10, where do we stand? Certainly, smoking continues to be the major culprit in the development and recurrence of ulcer disease, but what would our accountant have to say about an average of less than $100.00 for a full course of effective anti-ulcer therapy versus the $25,000.00 1987 dollars (even Canadian dollars at that) required for the previously recommended six week confinement to hospital? Should the cytoprotective agent, that new therapeutic modality, enter the marketplace of our minds and the lumen of our patients' stomachs? Great promise is held out for the use of prostaglandins under circumstances in which the gastric mucosa may be damaged by agents such as alcohol and nonsteroidal anti-inflammatory agents, but the case remains to be proven.

And in the end, perhaps this is not an end but rather, a beginning. Perhaps only time will tell.