The Influence of Food, Beverages and NSAIDs on Gastric Acid Secretion and Mucosal Integrity

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Gastric acid secretion is stimulated by all foods, especially proteins, and many beverages, the most potent beverages are milk and fermented substances such as beer and wine. The effects of food on mucosal integrity have been little studied, whereas non-steroidal anti-inflammatory drugs are well known to induce tissue injury.

INTRODUCTION

The effects of orally ingested substances on gastric function has been the subject of human investigation almost since the “discovery” of hydrochloric acid in gastric juice. From chilis and peppers to vodka and cognac, a variety of substances have been tested. Furthermore, the recognition of their ulcerogenic potential has sparked an interest in the effects of NSAIDsb on acid secretion and the gastric mucosa. This review focuses on the effects of foods, beverages, and NSAIDs on gastric acid secretion and mucosal integrity. Cigarette smoking, ulcer therapeutic agents and other drugs will not be discussed.

GASTRIC ACID SECRETION

Foods and Beverages

All foods are capable of stimulating gastric acid secretion through distention of the stomach, but proteins are the the major stimulants. Digested protein in the form of peptides, peptones, and amino acids act primarily through the stimulation of gastrin from antral G cells [1]. The aromatic amino acids are the most potent of the amino acids. There is also evidence that amino acids absorbed into the circulation may directly stimulate parietal cells [2]. Protein is the most important physiologic stimulus to acid secretion in man.

The effects of various beverages on acid secretion have been assessed by McArthur and colleagues [3]. When compared to water control, soft drinks, coffee and tea, beer and milk were all substantial and significant stimulants of acid secretion. The highest outputs occurred with beer and milk, each producing acid output at or above the maximal acid output in response to pentagastrin. The authors found no correlation between pH, osmolality, caloric content, buffering capacity, ionized calcium levels or caffeine content and the degree of secretory response. For example, although 7-Up has no caffeine or protein and only minimal amounts of calcium, it is a significant stimulant of acid secretion.

Various investigators have studied individual classes of beverages in greater detail. Ippoliti found that low-fat or non-fat milk stimulated as much acid secretion as whole milk in both duodenal ulcer patients and normal subjects [4]. Low-calcium milk stimulated acid output in duodenal ulcer patients but not normal subjects. The most likely component of

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bAbbreviations: NSAID, non-steroidal anti-inflammatory drug.
milk responsible for acid secretion is protein. The observation that milk was a stimulant of acid secretion, as well as the atherogenic properties of fat and the potential for development of the milk-alkali syndrome, certainly hastened its demise as ulcer therapy.

Feldman and his collaborators at CURE found that decaffeinated coffee was more potent than a peptone meal in stimulating both gastrin release and acid secretion, with the coffee producing acid output equal to 70 percent of peak acid output to pentagastrin [5]. It remains unknown what component of decaffeinated coffee is the secretagogue. The amount of protein in coffee is only 12 percent of that in the peptone meal, and the amount of calcium is minimal.

The acid secretory response to intragastric ethanol is very weak, if at all [6, 7]. In one well controlled study, ethanol in concentrations of five percent, 12 percent, and 36 percent (concentrations found in popular beverages) produced no more acid than isovolumetric saline controls [6]. Similar results have been found for distilled spirits (e.g., whisky, cognac) [6]. On the other hand, beer and wine (red or white) are quite potent stimulants of acid secretion [6, 7]. In one study, the mean acid output in response to 300 ml of 12 percent red or white wine was higher than the peak acid output to pentagastrin [6]. In the latter study, it was noted that there was a highly significant correlation (r = .91) between the rise in serum gastrin concentrations and acid output in response to red or white wine. Beer and wine differ from whisky and cognac in that they are fermented rather than distilled. It seems reasonable that the stimulatory substances in fermented beverages are amino acids and amines, both of which are present in beer and wine. Histamine is one amine that does not play a role, however, since white wine has very low or absent levels of the amine and is still a potent stimulant of acid secretion.

NSAIDs

Only a few studies have been performed during the past 20 years measuring the effect of NSAIDS on gastric acid secretion or gastric acidity. In two studies involving healthy volunteers, indomethacin was shown to increase mean basal acid secretion [8, 9], increase histamine stimulated-acid secretion [9], but have no effect on meal-stimulated acid secretion [8]. Where acid secretion was increased, however, there was marked variation in the response from subject to subject. A more recent study found no significant differences in median 24 h pH in rheumatoid arthritis patients infected with *H. pylori* [10].

MUCOSAL INTEGRITY

Foods and Beverages

Literature on the effects of specific foods on gastric mucosal integrity in humans is sparse. The Singapore group has reported that chilis do not produce macroscopic gastric mucosal damage [11] and may even protect against aspirin-induced mucosal injury [12]. Graham and colleagues have shown that red or black peppers produce gastric mucosal injury comparable to that seen with aspirin [13], but that highly spiced meals (i.e., mexican food with jalapeno peppers or a pepperoni pizza) do not lead to endoscopically demonstrable gastroduodenal mucosal damage [14].

The effect of alcohol on human gastric mucosa was first described in 1833 by William Beaumont who observed erosions and hemorrhages through the gastric fistula of Alexis St. Martin. Others have confirmed that the acute ingestion of alcohol produces erythema, sub-epithelial hemorrhages and erosions [15-17]. Laine and Weinstein describe subepithelial hemorrhages ("blood under plastic wrap") in 20 of 125 actively drinking alcoholic patients undergoing endoscopy for upper gastrointestinal bleeding [18]. Histologically, they found extensive foveolar hemorrhage in the target lesions, but only edema in the surrounding mucosa. Inflammation was not a major component of alcohol-induced injury.
NSAIDs

NSAIDs are well-recognized to produce two types of lesions: acute gastric mucosal erosions and subepithelial hemorrhages or frank gastric ulceration. The first are most likely related to a topical effect of the NSAID on the gastric mucosa, whereas ulceration is related primarily to prostaglandin depletion. However, both mechanisms may well play roles in both situations. The incidence of new gastric ulcers in patients taking NSAIDs ranges from 10 percent to 20 percent and the incidence of duodenal ulcers ranges from four percent to 10 percent [19-22]. It should be noted, however, that only a small proportion of these are clinically important, producing either pain or complications. It appears that even very low doses of NSAIDs can lower prostaglandin levels and produce ulceration. Current investigation is ongoing to find compounds with relatively low ulcerogenicity or novel means of administering NSAIDs so to minimize gastric injury.

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