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THE CARBONATE AFFAIR — AND THE MAGNESIUM MYSTERY?

To the Editor: We were interested to read the recent articles in the *Journal*[1,2] supporting the argument for not using calcium carbonate as an antacid, because serum gastrin levels and gastric secretion were shown to be increased. Antacid therapy is effective not only in peptic ulceration, but in patients who have heartburn and reflux through an incompetent gastroesophageal sphincter. In the latter instance raising the gastrin levels should theoretically prevent reflux by increasing the resting sphincteric pressure.[3] Since it has been shown in duodenal ulceration that the epigastric pain can be reproduced by acid in the lower esophagus,[4,5] and that the sphincter is frequently displaced into the thorax and may be incompetent,[6] the same theoretical grounds for the continued use of calcium carbonate may apply to patients with duodenal ulcer as well.

Having defended calcium carbonate, how about an attack on magnesium salts? A full discussion of the actions of magnesium salts, whether in small doses such as found in the original Epsom waters or in the higher doses used as purgatives, is contained in a recent article discussing the hypothesis that magnesium ions act by releasing cholecystokinin from the duodenum.[7] The evidence is convincing that the duodenum is stimulated by magnesium to release cholecystokinin, which not only contracts the gallbladder but also has a direct action on the colon. Since the octapeptide of cholecystokinin reduces the gastroesophageal sphincteric pressure in man[8] and may increase gastric secretion, magnesium salts could theoretically be contraindicated whenver gastroesophageal reflux contributes to symptoms.

While waiting for more factual evidence, those with doubts about both these antacids should not forget the old English adage, "the proof of the pudding is in the eating."

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