

***VAN DIE REDAKSIE***

***EDITORIAL***

## Stiptheid

Daar is sekere mense wat kronies laat is vir enige afspraak. Hulle het iedere keer 'n skynbaar aanvaarbare verskoning, maar telkens is dit maar weer dieselfde persoon of persone wat nie 'n vooraf opgestelde tydtabel kan handhaaf nie. Hulle is diegene wat, tot steurnis van die res van die gehoor, in die donker tastend die teater binnekom nadat die program reeds 'n aanvang geneem het. Hulle daag 'n halfuur of meer laat by sosiale funksies soos dinees op, tot ergernis van die gasvrou wie se kos staan en bederf. Naderhand ken hul kollegas en vriende hulle so en word dit maar skouerophalend aanvaar dat stiptheid skynbaar nie binne die bestek van hul organisatoriese vermoë val nie.

Daar is ook sulke medici. Dit is hulle spreekkamers wat dag na dag oorloop van pasiënte wat met moedelose uitdrukings op hul gesigte sit en wag om geholpe te raak. Ook hulle het altyd 'n onomseilbare verskoning: 'n onverwagte bevalling, 'n skielike noodgeval of iets dergeliks. Maar dit is

telkens dieselfde dokter wie se skedule so omvergewerp word. En dit is ook hierdie kollegas wat 'n tikkie laat opdaag vir 'n operasie, of by vergaderings ingestap kom wanneer 'n deel van die besprekings reeds afgehandel is.

Soms is die gebrek aan stiptheid ook 'n massaverskynsel. Wanneer 'n vergadering van 50 of meer mense 'n blaaskans neem om tee te drink, moet die voorsitter twee of drie keer vra dat die lieue vriende asseblief weer moet kom sit sodat die vergadering kan voortgaan, al was die teeskinkery ook hoe doeltreffend. Mense is eenvoudig net nie gebore in staat om sonder onderskraging aan 'n vooraf opgestelde tydtabel te hou nie. Aan hierdie massaverskynsel kan mens seker niks doen nie, maar soos ondervinding geleer het dat dit telkens dieselfde pasiënte in 'n huisartspraktyk is wat na-ure pla, is dit keer op keer ook dieselfde dokters wat laat is. Is dit nodig? En is dit nie ook hulle wat vergoed vir laat kom deur vroeg te loop nie?

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## Heartburn and Oesophagitis

Acid reflux is a major factor in the causing of oesophagitis, and measures to reduce or neutralise gastric acidity are of value in the treatment of this condition. Antacids remain the mainstay of therapy. Their success in relieving symptoms may be due to

increased release of gastrin, which tightens the gastro-oesophageal sphincter rather than by simply neutralising gastric acid.<sup>1</sup> The major pharmacological approach in the management of active peptic ulcer is neutralisation of the acid gastric content



with antacids, but their efficiency is limited by several factors which include gastric emptying, the continuous secretion of acid (often in excessive amounts), and the intrinsic limitations of the antacids themselves. Various commercial antacid agents are available, of which tablets are less effective than liquid or powder preparations. The objective of medication should be continual maintenance of the pH of the gastric contents between 4 and 5 or higher. Three important reasons for failure are insufficient frequency of administration, inadequate dose, and poor choice of preparation.

While developing a low-density contrast medium for radiological study of hiatus hernia, Sandmark<sup>2</sup> observed that alginate floated on gastric contents, and patients with heartburn reported relief of this symptom. This led to the production and marketing of an alginate/antacid compound, Gaviscon, for the medical management of reflux oesophagitis. It contains alginic acid, sodium alginate, sodium bicarbonate, aluminium hydroxide, and magnesium trisilicate, which forms a soft viscous layer of floating antacid foam that precedes the reflux of gastric contents into the oesophagus.

Alginates have been used safely for many years in food for human consumption. Alginic acid and its salts have also been used for medical purposes, and available scientific evidence indicates that ingestion of even considerably larger amounts than would be used in normal food manufacturing practice, is harmless. They do not reduce the nutritive value of food and do not themselves have nutritive value. Sodium alginate is not decomposed or absorbed appreciably, and therefore it would not be expected to be harmful during pregnancy.<sup>3</sup>

There have been several publications on the efficacy of this alginate/antacid preparation. Andrup and Jakobsen<sup>4</sup> reported complete or almost complete relief of symptoms in many patients with

hiatus hernia. Hiatus hernia is not necessarily associated with gastro-oesophageal reflux, but when it occurs there is a need for symptomatic relief and for treatment that will prevent oesophagitis and aspiration of gastric contents into the mouth and lungs. Beeley and Warner<sup>1</sup> reported that the compound relieves symptoms of regurgitation into the mouth and also the heartburn produced by oesophageal reflux in hiatus hernia. Hansky<sup>5</sup> indicated that the preparation is effective in the treatment of heartburn and oesophagitis. Recently, Stanciu and Bennett<sup>6</sup> studied patients with gastro-oesophageal reflux, using 15-hour recordings of lower oesophageal pH before and after two weeks' treatment. Significant reduction of reflux episodes and percentage time during which the lower oesophageal pH was acid was noted in the group that received Gaviscon, whereas no significant changes occurred in two groups receiving antacids alone, or a placebo tablet. Symptoms were fewer after Gaviscon than after antacid alone.

The success of the alginate/antacid treatment is apparently not due to neutralisation of gastric contents; the quantity of antacid present in the compound is far less than that contained in standard antacid preparations. One explanation of its success is that it enters the oesophagus, and, being almost neutral, it is non-irritating to the mucosa. It breaks the vicious cycle of increased gastric secretion produced by acid in the oesophagus. Also, the presence of a near-neutral foam in the upper part of the stomach may stimulate the secretion of gastrin, thereby closing the sphincter. The alginate may also act as a physical barrier at the cardia.

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3. Gleason, M. N., Gosselin, R. E., Hodge, H. C. and Smith, R. D. (1969): *Clinical Toxicology of Commercial Products*. Baltimore: Williams & Wilkins.
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6. Stanciu, C. and Bennett, J. R. (1974): *Lancet*, **1**, 109.