Chairman's Introduction

J.		Dent
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Gastroenterology Unit, Royal Adelaide Hospital, Adelaide, Australia

J. Dent, Gastroenterology Unit, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000 (Australia)

For many years, pharmacological inhibition of gastric acid secretion was associated with such troublesome side-effects that it was not a viable therapeutic option. The scene was transformed following the development of H₂-receptor antagonists – the first agents capable of substantial inhibition of gastric acid secretion, in the virtual absence of side-effects. Consequently, dramatic progress was made in the medical therapy of chronic peptic ulcer. The results were, however, less impressive in patients with reflux oesophagitis, and inconclusive in those with acute upper gastrointestinal bleeding from a chronic peptic ulcer.

Shortly after the introduction of the H₂-receptor antagonist cimetidine, clinical trials were started with omeprazole, the first acid pump inhibitor. It was already well known that omeprazole could inhibit gastric acid secretion more reliably than H₂-receptor antagonists. The practical value of the greater effect of omeprazole on acid secretion was then far from clear, there being a quite generally held belief that the level of acid inhibition achieved by H₂-receptor antagonists was such that it would

be difficult for any new drug to improve upon the results obtained with these agents. This belief has proved to be incorrect. Omeprazole has now been well documented to provide very significant gains over H₂-receptor antagonists, through a higher level of gastric acid inhibition, particularly in patients with reflux oesophagitis.

There are still many intriguing questions concerning the therapeutic use of levels of gastric acid inhibition that are achieved with omeprazole. Of particular importance is the question regarding the efficacy and safety of long-term omeprazole as maintenance therapy. In addition, promising new therapeutic applications for omeprazole have emerged recently.

The programme for this symposium in Vienna, November 1991, was designed to examine the leading edge of knowledge regarding the therapeutic use of omeprazole, rather than to summarize what is already known. I very much hope that these Proceedings capture the liveliness and interest of the symposium, and provide a stimulus for thinking about the growing edge of the therapeutic uses for gastric acid inhibition.

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Karger AG, Basel

0012-2823/92/

0517-0001 \$2.75/0