

Drugs used for the treatment of peptic ulcers

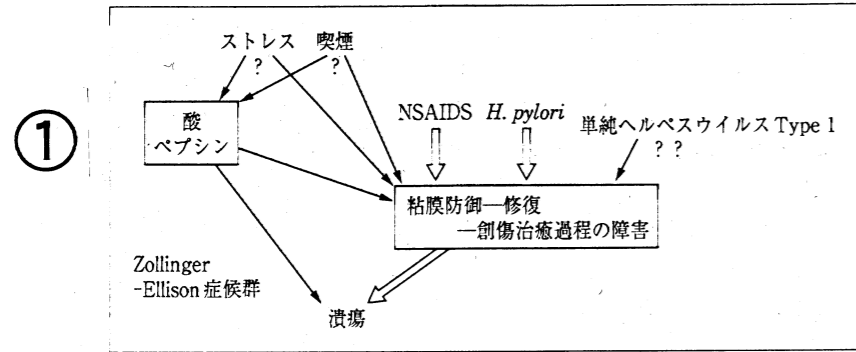


図1. 消化性潰瘍の病態生理 (Soll, 1998 を一部改変)

表1. *H. pylori* 感染と疾患の関係

疾患	有病率 (%)	<i>H. pylori</i> 感染率 (%)	<i>H. pylori</i> 感染者に占める有病率 (%)
慢性活動性胃炎	50	100	100
消化性潰瘍			
胃潰瘍	2	70~90	3
十二指腸潰瘍	1	90~95	2
胃癌	0.2	60~70	0.3
悪性リンパ腫	0.01	80~90	0.02

荒川ら, 1996 より一部改変

表3. *H. pylori* (-) 潰瘍

発表者	<i>Hp</i> (-) 潰瘍の割合	<i>Hp</i> (-) 潰瘍のなかで NSAIDS 潰瘍の占める割合
Nensey ら (1991)	DU 23% (12/52)	75% (9/12)
Borody ら (1991)	DU 6% (18/302)	44% (8/18)
Borody ら (1992)	GU 38% (44/115)	66% (29/44)
McColl ら (1993)	DU 2.8% (12/435)	33% (4/12)
桑山ら (1995)	DU, GU 1.5% (4/267)	38% (3/8)
加藤ら (1996)	DU, GU 3.7% (10/273)	40% (4/10)

DU は十二指腸潰瘍, GU は胃潰瘍

樋口ら, 1997 より一部改変

4 ●消化性潰瘍剤の分類

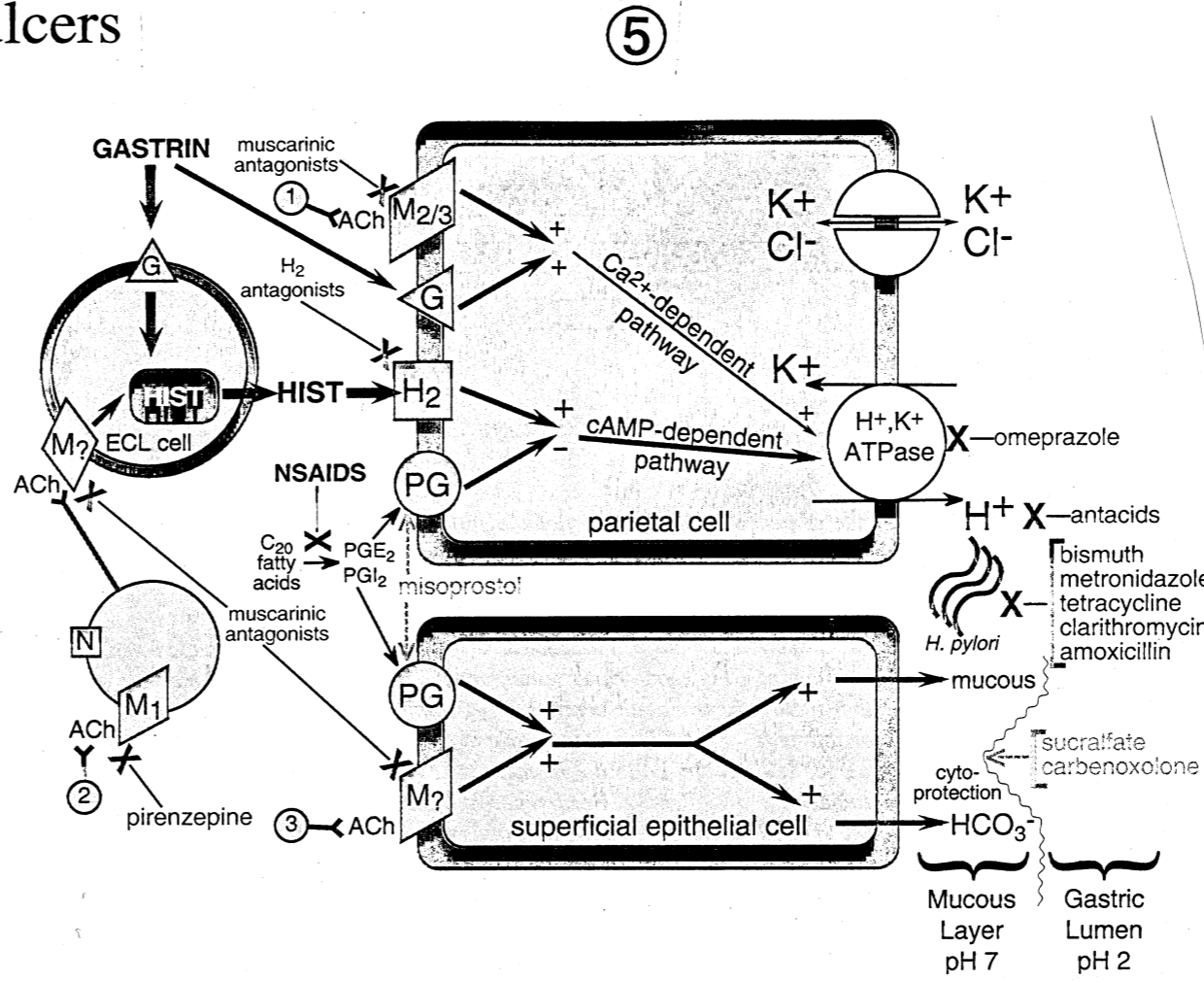
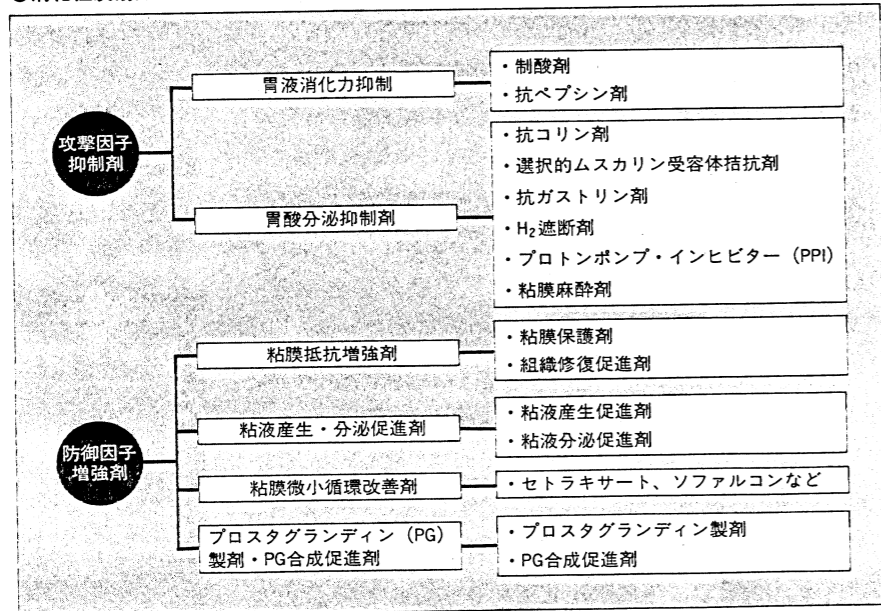
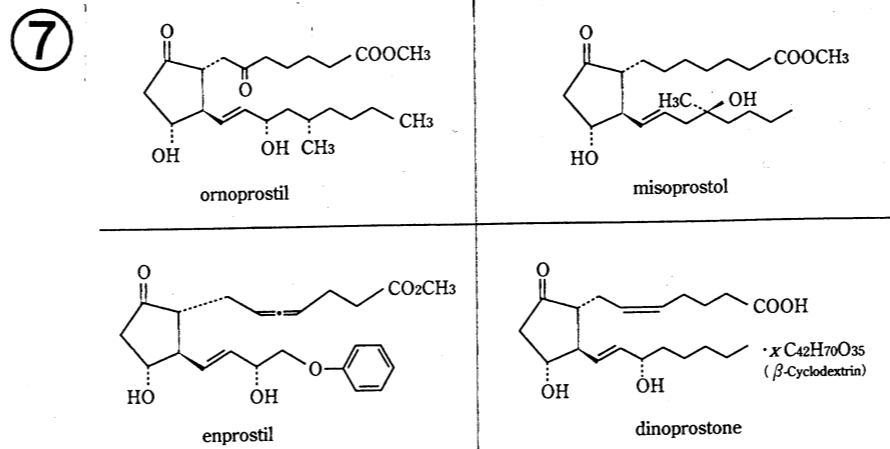
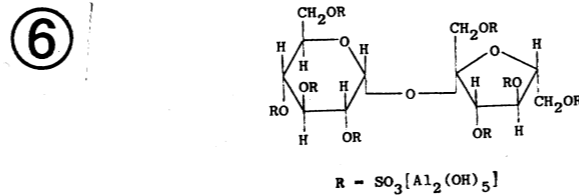


Figure 37-1. Physiological and pharmacological regulation of gastric secretions: the basis for therapy of peptic ulcer disease.



医薬品として使用されている PGE₁, PGE₂ 及びその誘導体

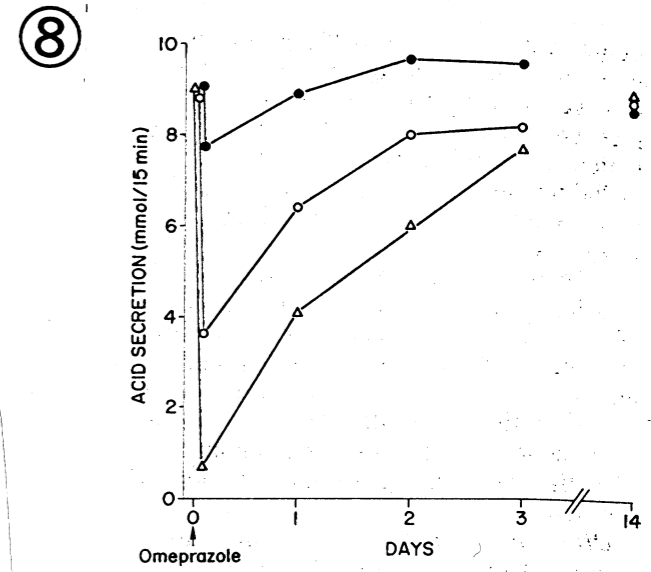


Figure 37-4. Inhibitory effect of omeprazole on secretion of gastric acid in man.

Maximal secretory responses were elicited in six healthy human subjects by infusing pentagastrin (91 µg) over a 1-hour period before and at various intervals after a single oral dose of omeprazole (○, 20 mg; △, 40 mg) or placebo (●). Note the profound and prolonged inhibition. (Modified from Lind *et al.*, 1983.)

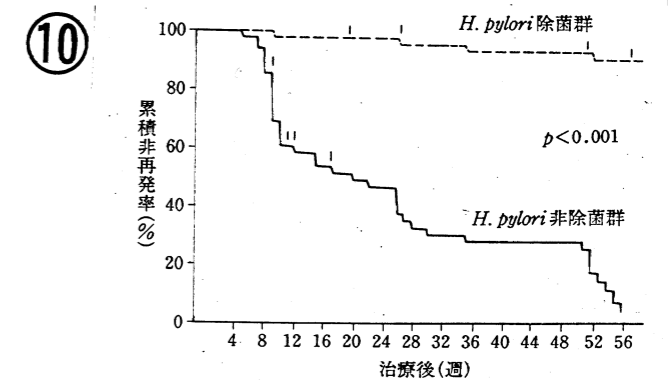


図 16-5 *H. pylori* 除菌の有無と十二指腸潰瘍再発 H. ヘンシェルら (1993)