The important difference between these two studies is that Gaslander et al found a raised plasma cholecytokinin concentration in rats with duodenogastric reflux (at two weeks and six weeks), whereas we found a normal plasma cholecytokinin concentration (at six months). Cholecystokinin receptor antagonists decreased plasma levels of cholecystokinin, suggesting that the cholecystokinin concentrations found in the study of Gaslander et al were due to the interference of cholecystokinin receptor antagonist. However, we saw an increase in circulating cholecystokinin concentrations and they did not. In fact, baseline cholecystokinin concentrations in their study were 59% higher in animals with small gastrinogenostomy than controls, although this did not reach statistical significance. It is possible that a larger series is probable that this would have reached statistical significance. Furthermore, in their study of partial gastrectomy, baseline cholecystokinin concentrations were only 46% above control and lower than those seen in their split gastrunctomy group. While they concluded that partial gastricectomy increases plasma cholecystokinin, it is interesting to note that humans with longstanding gastric ulcer have increased concentrations of cholecystokinin postprandially, although their gastric concentrations are quite normal. Although gastrin concentrations were increased after gastrojejunostomy, we did not observe a corresponding rise in cholecystokinin concentration, which suggests that the increased gastrin concentrations are due to increased production of gastrin rather than decreased production of cholecystokinin. We have previously speculated that increased production of gastrin may be due to increased production of enteroglucagon, which is known to lower cholecystokinin concentration.

Expression of adhesion molecules in human Peyer's patches

EDITOR—We became aware of a publication in Gut by Fujimura and Khira on human Peyer's patches while reviewing published works (Gut 1994; 35: 46-50; the title of this paper was expressing because we had not previously speculated on the role of adhesion molecules in human Peyer's patches). The authors found that the follicle-associated epithelium (FAE) of Peyer's patches is characterized by the presence of a unique subset of lymphocytes that express high levels of adhesion molecules. This finding is of particular interest because it suggests that the FAE of Peyer's patches may play a role in the regulation of immune responses in the gut. The FAE is a specialized epithelial structure that lines the crypts of Lieberkühn and is thought to be the site of immune cell recruitment and activation in the gut. The authors found that the FAE of Peyer's patches is characterized by the presence of a unique subset of lymphocytes that express high levels of adhesion molecules. This finding is of particular interest because it suggests that the FAE of Peyer's patches may play a role in the regulation of immune responses in the gut. The FAE is a specialized epithelial structure that lines the crypts of Lieberkühn and is thought to be the site of immune cell recruitment and activation in the gut. The authors found that the FAE of Peyer's patches is characterized by the presence of a unique subset of lymphocytes that express high levels of adhesion molecules. This finding is of particular interest because it suggests that the FAE of Peyer's patches may play a role in the regulation of immune responses in the gut. The FAE is a specialized epithelial structure that lines the crypts of Lieberkühn and is thought to be the site of immune cell recruitment and activation in the gut. The authors found that the FAE of Peyer's patches is characterized by the presence of a unique subset of lymphocytes that express high levels of adhesion molecules. This finding is of particular interest because it suggests that the FAE of Peyer's patches may play a role in the regulation of immune responses in the gut. The FAE is a specialized epithelial structure that lines the crypts of Lieberkühn and is thought to be the site of immune cell recruitment and activation in the gut. The authors found that the FAE of Peyer's patches is characterized by the presence of a unique subset of lymphocytes that express high levels of adhesion molecules. This finding is of particular interest because it suggests that the FAE of Peyer's patches may play a role in the regulation of immune responses in the gut. The FAE is a specialized epithelial structure that lines the crypts of Lieberkühn and is thought to be the site of immune cell recruitment and activation in the gut.
to accept that the rather even distribution of ICAM-1 below the follicle associated epithelium, as reported by Fujimura and Kihara, should have anything to do with the numerically and phenotypically heterogeneous distribution of lymphocytes in this epithelium.

The authors furthermore discuss extensively the nature of membrane cells; without reservation it is claimed that these cells express MHC class II molecules and therefore may be present luminal antigens to T cells. This area is quite controversial, however, and the first study on class II expression in rat Peyer’s patches reported that the complete follicle associated epithelium is negative.13 This has been contradicted subsequently but we found human membrane cells to be negative for HLA-DR compared with the strongly positive remaining follicle associated epithelium.14,15 The antigen presenting capacity of membrane cells is therefore questionable although they are probably to some extent able to degrade foreign material as suggested by their lysosome like structures16 and cathepsin E expression.17 We have recently proposed that membrane cells might provide an opportunity for juxtaposed B cells to present partially processed luminal antigens to CD4+ memory T cells, thereby promoting diversification of mucosal immune responses.

In view of this immunobiological complexity of gut associated lymphoid tissue we feel that it is too speculative when Fujimura and Kihara on the basis of their findings in rat Peyer’s patches to blocking of ICAM-1 as a potential treatment for inflammatory bowel disease in the future.

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6 Kvale D, Brandtzæg P, Constitutive and cytokine-induced expression of HLA molecules, secretory component (SC), and ICAM-1 are modulated by butyrate in the colonic epithelial cell line HT-29. Gut 1995; 36: 114–21.

Ménétrier’s disease

EDITOR.—We read with interest the case report by Bayerdorfer et al showing that *Helicobacter pylori* is a potential cause of Ménétrier’s disease (Gut 1994; 35: 701–4). The findings in their patient showed clearly that *H pylori* gastritis can present as hypertrrophic gastritis combined with protein loss and that eradication of *H pylori* infection can lead to rapid disappearance of the hypertrrophic gastritis and restoration of normal gastric mucosa. We disagree, however, with the authors’ presumption that Ménétrier’s disease and hypertrrophic gastropathy are synonymous. There are many reasons for believing that equating the two terms is ill advised. (1) A variety of conditions can cause enlarged gastric folds. In addition to the true hypertrrophic gastropathies Ménétrier’s disease and Zollinger-Ellison syndrome the development of gastric folds are seen in hypertrrophic gastritis associated with various infections, including *H pylori*, cytomegalovirus, histoplasmosis, and syphilis, and in miscellaneous other conditions such as lymphoma, sarcoidosis, allergic (eosinophilic) gastritis, and Cronkhite-Canada syndrome. (2) While increased gastric protein loss can be found occasionally in many disorders that are associated with large gastric folds, protein loss is not a universal feature of any of these disorders. It is typically lacking in Zollinger-Ellison syndrome, and its reported occurrence in Ménétrier’s disease is variable. (3) Different authors’ diagnostic criteria for this gastropathy. (3) The cases described in 1888 by Ménétrier as ‘polyade-nome en nappe’ had as their cardinal feature exuberant proliferation of gastric mucous cells. This is a distinct entity that has been appropriately termed ‘massive foveolar hyperplasia’.1,2 Furthermore, Ménétrier’s descriptions and illustrations do not suggest that chronic gastritis, the hallmark of *H pylori* gastritis, was present. (4) The concept that massive foveolar hyperplasia is a definitive feature of true Ménétrier’s disease is greatly strengthened by studies showing that there is an accompanying change in the distribution and activity of transforming growth factor α in the gastric mucosa in Ménétrier’s disease. In addition, the experimental induction of an excess of gastric transforming growth factor α in transgenic mice results in similar mucous cell hyperplasia.4,5 It is highly probable that ‘hypertrrophic hypersecretory gastropathy’ (Schindler’s disease), another distinctive entity that is sometimes mistakenly designated as Ménétrier’s disease, is probably a manifestation of *H pylori* gastritis with large folds.5

We urge authors and editors not to use the term Ménétrier’s disease as a generic designation for any entity, or any combination of findings associated with enlarged rugae. The eponym should be limited to those rare cases that fulfil Ménétrier’s original description of massive foveolar hyperplasia without gastritis. This approach is essential from nosologic and patient treatment standpoint if the varied aetiologies and resulting treatment implications of hypertrrophic gastropathy are to be maintained. As Bayerdorfer et al we would have preferred to see it termed simply *H pylori* associated hypertrrophic gastritis.