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# Agenda



- 1. Ovid's Integrated Solution:  
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Editors: Alex M. Davison, J. Stewart Cameron, Jean-Pierre Gruunfeld, David N.S. Kerr, Eberhard Ritz, Christopher G. Winearls

Publisher: Oxford University Press, 1998

Chapter: The patient with diabetes mellitus

Passage: ... of patients diagnosed before 1942 the cumulative risk after 25 to 30 years of duration was approximately 41 per cent, but this has declined to between

Score: ★★★★★

□ 2. Source: *Oxford Textbook of Clinical Nephrology*

Editors: Alex M. Davison, J. Stewart Cameron, Jean-Pierre Grunfeld, David N.S. Kerr, Eberhard Ritz, Christopher G. Winearls

Publisher: Oxford University Press, 1998

Chapter: The patient with diabetes mellitus

Passage: ... the incidence of nephropathy. The time to development of microvascular complications is not influenced by prepubertal duration of the disease (Kostraba et al. 1989), and nephropathy develops more slowly in individuals who develop diabetes before the age of 10 than in those diagnosed after puberty (Krolewski et al. 1985). The highest incidence (44 per cent) is seen in subjects who develop diabetes between the ages of 11 and 20 years (Kofoed-Enevoldsen et al. 1987). Patients who develop diabetes after the age of 20 have a lower cumulative incidence of nephropathy, at around 35 per cent. Some authors (Krolewski et al. 1985), but not others (Kofoed-Enevoldsen et al. 1987), have found that current age influences the incidence of proteinuria, with maximum risk in the age ...

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□ 3. Source: *Oxford Textbook of Medicine*

Editors: David A. Warrell, Timothy M. Cox, John D. Firth, Edward J. Benz

Publisher: Oxford University Press, 2003

Chapter: The kidney in systemic disease

Passage: ... Diabetes mellitus and the kidney R. W. Bilous Introduction Diabetic nephropathy is the commonest single cause of endstage renal failure (ESRF) requiring renal replacement therapy in the United States, and the second most common in Europe and Japan. The incidence is increasing, largely because the incidence of diabetes itself is reaching what some have termed epidemic proportions, this growth being greatest in the developing world. Definition Nephropathy is a clinical diagnosis based upon the finding of proteinuria in a patient with diabetes and in whom there is no evidence of urinary infection. Conventionally, the level of proteinuria for a diagnosis of 'clinical nephropathy' or 'overt nephropathy' is 0.5 g/day, which is roughly equivalent to a urinary ...

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□ 4. Source: *Diseases of the Kidney and Urinary Tract*

Editors: Robert W. Schrier

Publisher: Lippincott Williams & Wilkins, 2001

Chapter: Diabetic Nephropathy

Passage: ... Diabetes Microalbuminuria predicts renal disease in diabetic Pima Indians (76), a group that tends to acquire type 2 diabetes at a relatively young age. However, in more elderly type 2 diabetic patients, the incidence of microalbuminuria and proteinuria (33,37,77,78,79 and 80) predicts an incidence of ESRD higher than the 8% to 10% of type 2 diabetic patients in whom uremia ultimately develops (7,32,33 and 34,37). This may have multiple explanations. First, microalbuminuria is a strong predictor of increased cardiovascular mortality in type 2 diabetes (33,35,36). Thus, many patients die before uremia supervenes. Second, because the nature of the underlying renal injury is more heterogeneous in type 2 diabetic patients (70) (see later), it is not surprising that the ...

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
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
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
Passage: ... and those with renal insufficiency, diabetes, or hypovolemia) if a radiocontrast media (RCM) study is planned. There is a higher incidence of RCM nephropathy in diabetics with serum creatinine levels above 1.5 mg/dL and in patients with chronic renal insufficiency and serum creatinine levels above 2.5 mg/dL.


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
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
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
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
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
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
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
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
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
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
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
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
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
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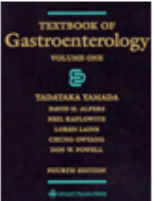

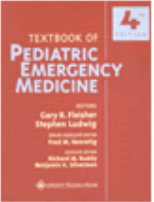






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## CHAPTER 1

# THE INTEGRATED RESPONSES OF THE GASTROINTESTINAL TRACT AND LIVER TO A MEAL

**Helen E. Raybould**

**Stephen J. Pandol**

**Hal Yee**

This chapter provides an overview of the mechanisms involved in the regulation of the various responses in the gastrointestinal (GI) tract and the liver to the ingestion of a meal. The coordination of these responses is essential to the overall function of the GI tract, which is to take in nutrients and eliminate wastes, and a key function of the liver, which is to coordinate the distribution and storage of vital nutrients. Each of the processes described in this introductory chapter is explored in greater depth and with extensive references to the literature elsewhere in the textbook.

The GI tract consists of the alimentary canal from the mouth to the anus and the associated glandular organs that empty their contents into the canal. In a general sense, the GI tract adds water, ions, and enzymes to a meal to convert it into an aqueous solution of molecules that can be transported into the blood. Importantly, most of the added substances are absorbed for reuse. The major physiological processes that occur in the GI tract are motility, secretion, digestion, absorption, and elimination. Food is taken into the mouth as large particles containing macromolecules that are not absorbable. The breaking down of food into absorbable material occurs by grinding and mixing the food (motility) with various secretions containing enzymes, ions, and water that enter the GI tract. The enzymes convert the macromolecules into absorbable molecules in a process termed digestion. The products of digestion, as well as the secretions from the upper parts of the GI tract, are then transported across the epithelium to enter the blood or lymph by a process termed absorption. Secretions and luminal contents are moved from the mouth to the anus and eliminated by GI motility. The coordination of GI function is regulated in a synchronized way to maximize digestion and absorption by means

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pylori**

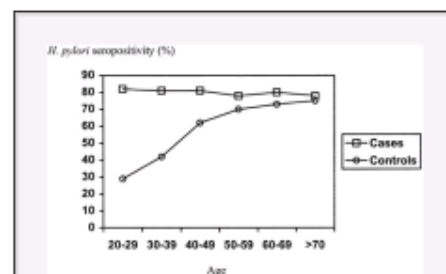
Epstein-  
Barr Virus

prospective cohort study in Japan that involved more than 26,000 residents.<sup>29</sup> The role of green tea in the prevention of gastric cancer needs further evaluation.

## Infectious Agents

### *Helicobacter pylori*

*H pylori* is a gram-negative, spiral-shaped organism found in the mucus layer of the human stomach. It was first successfully cultured by Marshall and Warren in 1984.<sup>30</sup> Since the discovery of this pathogen, numerous reports had been published to link this bacterium with various gastroduodenal diseases and even extraintestinal conditions. Based on several large-scale epidemiologic cohort studies published in the early 1990s,<sup>31,32,33</sup> and <sup>34</sup> the International Agency for Research on Cancer classified *H pylori* as a group 1 carcinogen in 1994.<sup>35</sup> Data from case control studies were summarized by a metaanalysis of 19 studies that included 2500 cases and 4000 controls.<sup>36</sup> The pooled data showed that the combined odds ratio for gastric cancer in *H pylori*-infected subjects is 1.92 (95% confidence interval, 1.32 to 2.78). Nonetheless, the attributable risk of *H pylori* infection to gastric cancer is still believed to be an underestimate. Because elderly patients with severe atrophic gastritis may have spontaneous remission of infection, the association of *H pylori* with gastric cancer is particularly strong in young persons with *H pylori* infection. In this regard, the odds ratio increased from 1.05 at age 70 years or older to 9.29 at age 29 years or younger (Fig. 69-3).



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FIGURE 69-3. Age distribution of *Helicobacter pylori* seropositivity in gastric cancer cases and controls. (Modified from ref. <sup>36</sup>.)

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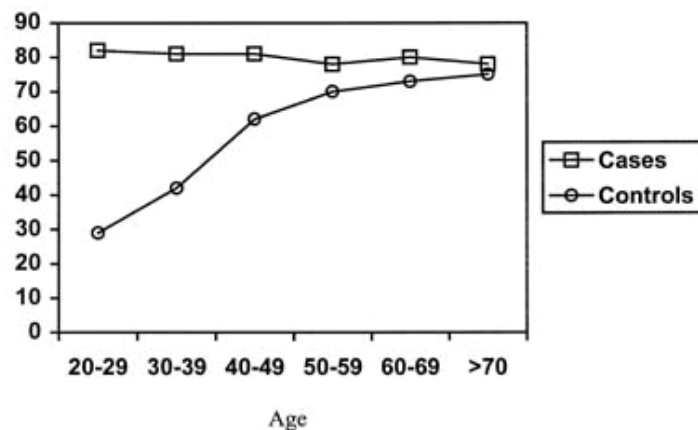


FIGURE 69-3. Age distribution of *Helicobacter pylori* seropositivity in gastric cancer cases and controls. (Modified from ref. [36](#).)



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## Risk Factors for *Helicobacter pylori* Resistance in the United States: The Surveillance of *H. pylori* Antimicrobial Resistance Partnership (SHARP) Study, 1993-1999

[Articles]

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**Disclaimer:** This article contains the professional views of the authors and does not necessarily represent the official position of the U.S. Food and Drug Administration.

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Drafting of the article: J.M. Meyer, R.J. Hopkins.

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Final approval of the article: J.M. Meyer, N.P. Silliman, N.Y. Siepman, E.C. King, H. Bhattacharyya, R.J. Hopkins.

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## Textbook of Gastroenterology

Editor(s): Yamada, Tadataka, Alpers, David H., Kaplowitz, Neil, Laine, Loren, Owyang, Chung, Powell, Don W.

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to the clinician caring for a given patient to decide when multiple cycles of evaluation and nonsurgical treatment may be excessively difficult, expensive, and burdensome to the patient. Nonhealing of any gastric ulcer raises the possibility of gastric carcinoma and warrants consideration of surgical treatment. Multiple endoscopic biopsies of the ulcer, if results are negative, reduce but do not eliminate the possibility of gastric carcinoma.<sup>1</sup> The clinical behavior of the ulcer as well as the results of endoscopic biopsy should influence the timing of operation, to avoid delaying treatment of a potentially early gastric cancer.

### “Giant” Ulcers

The question whether ulcer size should influence the decision to offer early operative treatment has long been a source of controversy. Data suggesting that gastric and duodenal ulcers larger than 3 cm are more likely to bleed, perforate, or prove intractable were acquired before the availability of proton pump inhibitor and anti-Helicobacter therapy.<sup>2,3</sup> Of 62 cases of giant gastric ulcers treated in Singapore between 1984 and 1989, 31% demonstrated penetration into adjacent structures (pancreas, liver, colon).<sup>3</sup> Malignant disease was found after histological examination in 13% of resected specimens, although it was not clear to what extent efforts were made preoperatively to obtain this information. Unexpected malignancy has not been a problem noted in more recent reports on the outcomes of medical therapy for large ulcers. These reports indicate that ulcer size has little bearing on response to appropriately aggressive treatment,<sup>4,5</sup> and <sup>6</sup> although surgery may be necessary for ulcer complications in as many as 16%.<sup>6</sup>

P.1442

## SURGERY FOR DUODENAL ULCER

### Vagotomy in the Treatment of Duodenal Ulcer

The reduction of acid secretion by gastric denervation serves as the physiological basis of all modern surgical treatments for duodenal ulcer. Truncal vagotomy reduces basal and maximal acid output by 85% and 50%, respectively.<sup>7</sup> This reduction in acid secretion results from the removal of the direct acetylcholine influences on the parietal cell, as well as reduction in sensitivity of the parietal cell to gastrin and histamine.<sup>8,9</sup> Although pepsin may play a role in mucosal injury in patients with duodenal ulcer, the clinical benefit of vagotomy in

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Editor(s): DerMarderosian, Ara, Beutler, John A.

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# Updates

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## Podophyllum

*Replaces Monograph Dated: JAN 1992*

### SCIENTIFIC NAME(S)

Podophyllum peltatum L., P. hexandrum Royle syn P. emodi Wall. Family: Berberidaceae<sup>1</sup>

### COMMON NAME(S)


Mayapple, mandrake, American podophyllum (P. peltatum), Indian podophyllum (P. hexandrum). Other common names include wild or American mandrake, devil's apple, vegetable mercury, duck's foot, racoonberry,<sup>2</sup> hog apple,<sup>2</sup> bajaolian,<sup>3</sup> and gwai-kou.<sup>4</sup> The plant should not be confused with the European mandrake (Mandragora officinarum L), which contains the anticholinergics hyoscamine, scopolamine, and mandragorine.

### BOTANY

P. peltatum is an herbaceous perennial found in damp, open woodlands and wet meadows in Canada and the eastern United States.<sup>2</sup> The plant grows to 0.3 to 0.6 m in height with a single stalk and 1 or 2 broad, deeply divided, shiny leaves about the size of a hand. Plants with 2 leaves bear a single, pendulous, small white flower in May (hence the name Mayapple); the odor has been described as "nauseous".<sup>2</sup> The fruit, which ripens in midsummer, is about 2.5 to 5 cm in length and is pulpy, lemon-yellow, and berry-like. It is the only

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