SEVENTH EDITION

PRINCIPLES OF SURGERY

外科学原理（下）

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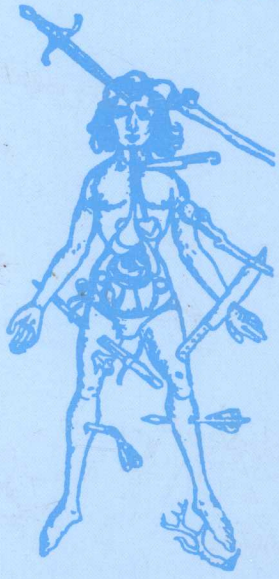

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PRINCIPLES OF SURGERY

SEVENTH EDITION

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Preface

The Seventh Edition of *Principles of Surgery* completes our participation in the surgical education of an entire generation of medical students and surgical residents throughout the world. We also were pleased to have played a role in the continuing education of practicing surgeons.

Many have regarded teachers as the noblest of people; others attach that designation to healers. As one who has been privileged to serve in both roles, namely, to have provided a vehicle for educating those who will perpetuate the healing profession in the realm of Surgery, and, at the same time, to have had the opportunity to participate in relieving patients of their disease, I consider myself twice blessed.

As a surgeon I have been satisfied by successes in patient care. As a teacher, I have been literally rewarded by expressions of appreciation by our readers who have indicated that we have enhanced their education.

Thirty-two years have passed since we accepted the publisher's and our own self-generated challenge to develop a "new and modern" textbook of Surgery. The favorable reception and the text's longevity suggest that we have succeeded. As the landmark of the Seventh Edition is completed, the frustrations and toils are erased and what remains is an immeasurable sense of gratification.

Seymour I. Schwartz, M.D.
June, 1998

ACKNOWLEDGMENT

We are particularly appreciative of the efforts of Andrea Weinstein, who had an integral role in each of the processes throughout the development of this edition. John Guardiano also contributed significantly to the technical editing of the manuscript.

Preface to the First Edition

The raison d'être for a new textbook in a discipline which has been served by standard works for many years was the Editorial Board's initial conviction that a distinct need for a modern approach in the dissemination of surgical knowledge existed. As incoming chapters were reviewed, both the need and satisfaction became increasingly apparent and, at the completion, we felt a sense of excitement at having the opportunity to contribute to the education of modern and future students concerned with the care of surgical patients.

The recent explosion of factual knowledge has emphasized the need for a presentation which would provide the student an opportunity to assimilate pertinent facts in a logical fashion. This would then permit correlation, synthesis of concepts, and eventual extrapolation to specific situations. The physiologic bases for diseases are therefore emphasized and the manifestations and diagnostic studies are considered as a reflection of pathophysiology. Therapy then becomes logical in this schema and the necessity to regurgitate facts is minimized. In appreciation of the impact which Harrison's PRINCIPLES OF INTERNAL MEDICINE has had, the clinical manifestations of the disease processes are considered in detail for each area. Since the operative procedure represents the one element in the therapeutic armamentarium unique to the surgeon, the indications, important technical considerations, and complications receive appropriate emphasis. While we appreciate that a textbook cannot hope to incorporate an atlas of surgical procedures, we have provided the student a single book which will satisfy the sequential demands in the care and considerations of surgical patients.

The ultimate goal of the Editorial Board has been to collate a book which is deserving of the adjective "modern." We have therefore selected as authors dynamic and active contributors to their particular fields. The au courant concept is hopefully apparent throughout the entire work and is exemplified by appropriate emphasis on diseases of modern surgical interest, such as trauma, transplantation, and the recently appreciated importance of rehabilitation. Cardiovascular surgery is presented in keeping with the exponential strides recently achieved.

There are two major subdivisions to the text. In the first twelve chapters, subjects that transcend several organ systems are presented. The second portion of the book represents a consideration of specific organ systems and surgical specialties.

Throughout the text, the authors have addressed themselves to a sophisticated audience, regarding the medical student as a graduate student, incorporating material generally sought after by the surgeon in training and presenting information appropriate for the continuing education of the practicing surgeon. The need for a text such as we have envisioned is great and the goal admittedly high. It is our hope that this effort fulfills the expressed demands.

Seymour I. Schwartz, M.D.

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CHAPTER 25

Small Intestine

B. Mark Evers, Courtney M. Townsend, Jr., and James C. Thompson

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INTRODUCTION

Considered teleologically, the small bowel is the raison d'être for the entire gut. The esophagus brings food to the stomach, which prepares it for digestion. The exocrine secretions of the liver and pancreas make digestion possible. Digestion is achieved in the lumen of the small bowel, and nutrients are absorbed through the small-bowel mucosa; the colon disposes of whatever is left.

In addition to its vital function in nutrition, the small bowel has other important roles. It is the largest endocrine organ in the body. It has tremendous defenses against infection and is one of the most important, if not the most important, organs in immune defense. It is a marvel of efficiency and works so well that, excepting the proximal 3 cm of the duodenum, it is seldom afflicted with disease. We are supplied with a great excess of small bowel and can exist on less than one-half of the absorptive surface provided.

Until recently, the small intestine had been relatively inaccessible for nonoperative diagnostic procedures, compared to the stomach or colon. Several diagnostic techniques are now avail-

able for specific diseases of the small bowel. Mucosal biopsy specimens obtained with the peroral biopsy capsule are often diagnostic in diffuse mucosal diseases. Enteroclysis is a more sensitive radiographic technique than the conventional barium follow-through examination of the small bowel. Selective mesenteric angiography often is helpful in cases of discrete lesions with abnormal vascular patterns, such as neoplasms, vascular malformations, or actively bleeding lesions. Scintigraphy may be helpful in localizing sites of bleeding. Fiberoptic endoscopy of the duodenum, proximal jejunum, and distal ileum are routinely employed.

Some diseases that affect the small intestine are discussed in other chapters: intestinal obstruction (Chap 22), mesenteric vascular disease (Chap 33), diseases of the intestine in infancy and childhood (Chap 37), the intestine in trauma (Chap 6), and duodenal and gastrojejunal peptic ulcer (Chap 24).

EMBRYOLOGY

The primitive gut forms during the fourth week of fetal development. The endoderm gives rise to most of the epithelium and glands of the digestive tract. The splanchnic mesodern surrounding the endoderm gives rise to muscular connective tissue and other layers comprising the wall of the digestive tract. Early in the fourth week the duodenum begins to develop from the caudal portion of the foregut and the cranial portion of the midgut. The remainder of the small bowel is derived from the midgut. During the fifth week, the fetal gut rapidly elongates and herniates through the umbilicus (Fig. 25-1). This midgut loop has both cranial and caudal limbs; the cephalic limb develops into the distal duodenum to proximal ileum, and the caudal limb becomes the distal ileum to proximal two-thirds of the transverse colon. The juncture of the cephalic and caudal limbs is where the vitelline duct joins to the yolk sac. This duct normally becomes obliterated before birth; however, it occasionally persists as a Meckel's diverticulum. This period of midgut herniation lasts until approximately 10 weeks' gestation, after which the intestines return to the abdomen. During this period of rapid elongation and herniation the midgut rotates 90 degrees counterclockwise around an axis formed by the superior mesenteric artery. As the gut reenters the abdomen at about the tenth week, it undergoes a further 180-degree rotation, thus completing 270 degrees of rotation from the starting point. The proximal jejunum is the first portion to reenter the abdomen and occupies the left side of the abdomen, with subsequent loops lying more to the right. The cecum enters last and is located temporarily in the right upper quadrant; during the third to fifth month of fetal gestation, it descends to its normal position in the right lower quadrant. Congenital anomalies of gut malrotation and fixation can occur during this process.

The small intestine is lined with simple cuboidal epithelium during the sixth and seventh weeks of fetal gestation. The rapid epithelial proliferation may lead to occlusion of the lumen, especially in the proximal gut (particularly in the duodenum). The lumen is reestablished during weeks 9 and 10 with the formation of coalescing vacuoles that restore the patency of the lumen. Villi begin to form first in the proximal intestine at 9 weeks, proceeding caudad and eventually lining the entire gut. Differentiated crypt cells appear between the ninth and twentieth weeks of gestation. Crypt formation begins in the tenth to twelfth weeks of gestation.

This complex and regimented pattern of gut differentiation appears to be mediated by sets of genes that are characteristically turned on and off with temporal and spatial specificity during developmental transitions as the gut evolves into its adult form. This developmental pattern also appears to be dependent on interaction with the mesoderm and lamina propria, which may provide "instructive" signals for developmental progression. The epithelial cells of the gut are generated from a fixed stem-cell population localized in the lower portion of the crypts that give rise to four main cell types: absorptive enterocytes, goblet cells, enteroendocrine cells, and Paneth cells. Until recently the analysis of the factors regulating these complex developmental processes in vivo was not possible; however, elegant studies by Gordon and colleagues, using transgenic mouse techniques, have described marker systems for inferring the biologic properties of intestinal stem cells and provided direct evidence of the transcriptional regulation of spatial patterns of intestinal gene expression. Other investigators have used the intestine-specific sucrase-isomaltase and neurotensin genes as molecular models to better understand the factors regulating gut development and eventual maturation.

ANATOMY

The most impressive thing about the small bowel is its immense mucosal surface area, which is responsible for the organ's tremendously efficient digestion of food. Several layers of muscle, combined with actin and myosin components in the microstructures, provide great motility, so that not only is there great surface area, but the interface between the surface and the luminal contents presented for absorption is in constant motion as well.

Gross Anatomy

The small bowel extends from the pylorus to the cecum. The length of the small intestine depends entirely on the state of bowel activity at the time of measurement. Careful estimates provide a duodenal length of 20 cm, a jejunal length of 100 to 110 cm, and an ileal length of 150 to 160 cm. The jejunoileum extends from the peritoneal fold that supports the duodenal-jejunal junction (the ligament of Treitz) downward to the ileocecal valve. The jejunoileum is estimated to constitute 60 percent of the entire length of the gut and to be approximately 160 percent of the body height, so that the small bowel is considerably longer in a 7-ft basketball player than in a 5-ft jockey.

Generally the jejunum occupies the upper abdomen, especially on the left, and is in contact with the pancreas, spleen, colon, and left kidney and adrenal gland. Affliction of these organs may affect the jejunum; pancreatitis, for example, may cause local ileus (the "sentinel loop") of the jejunum.

The jejunum has a larger circumference and is thicker than the ileum, and it may be identified at operation because of this and also because the mesenteric vessels usually form only one or two arcades and send out long, straight vasa recta to the mesenteric border of the jejunum. In contrast, the blood supply to the ileum may have four or five separate arcades; the vasa recta are shorter, and, most important, there is usually much more fat in the mesentery of the ileum than in that of the jejunum (Fig. 25-2). The jejunal mesentery may be transparent, but mesenteric fat usually reaches all the way to the bowel in the ileum. The ileum occupies the lower abdomen, especially on the right, and the pelvis. It is smaller in diameter and somewhat more mobile.

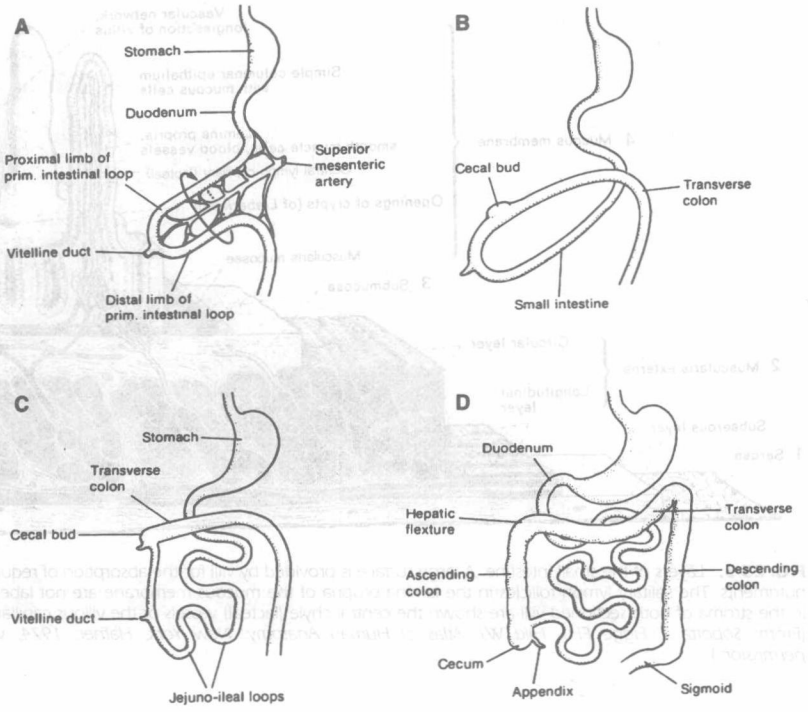

FIG. 25-1. Rotation of the intestine. *A.* The intestine after a 90-degree rotation around the axis of the superior mesenteric artery, the proximal loop on the right, and the distal loop on the left. *B.* The intestinal loop after a further 180-degree rotation. The transverse colon passes in front of the duodenum. *C.* Position of the intestinal loops after reentry into the abdominal cavity. Note the elongation of the small intestine, with formation of the small intestine loops. *D.* Final position of the intestines after descent of the cecum into the right iliac fossa. (From: *Podolsky DK, Babyatshky MW: Growth and development of the gastrointestinal tract, in Yamada T [ed]: Textbook of Gastroenterology, vol 2. Philadelphia, JB Lippincott, 1995, chap 23, with permission. [Adapted from Sadler TW (ed), Langman's Medical Embryology, 5th ed. Baltimore, Williams & Wilkins, 1985].*)

Except for the duodenum the small bowel is entirely covered with visceral peritoneum (the serosa) and is tethered only by its attachment to the mesentery, through which course arteries, veins, and lymphatics. The mesentery is obliquely attached to the posterior body wall, beginning superiorly well to the left of the second lumbar vertebra and ending obliquely downward and to the right to overlie the right sacroiliac joint. The mesentery normally is covered with glistening, nonadherent peritoneum, but after trauma (external, chemical, septic, or operative) it may become adherent to other surfaces (mesenteric, visceral, or parietal) and greatly limit bowel mobility.

Except for the proximal duodenum, which is supplied by branches of the celiac axis, the blood supply of the small bowel is entirely from the superior mesenteric artery, which is the second major branch of the infradiaphragmatic aorta. The superior mesenteric artery also supplies the appendix, cecum, and ascending and proximal transverse colon. There is an abundant collateral blood supply to the small bowel provided by the vas-

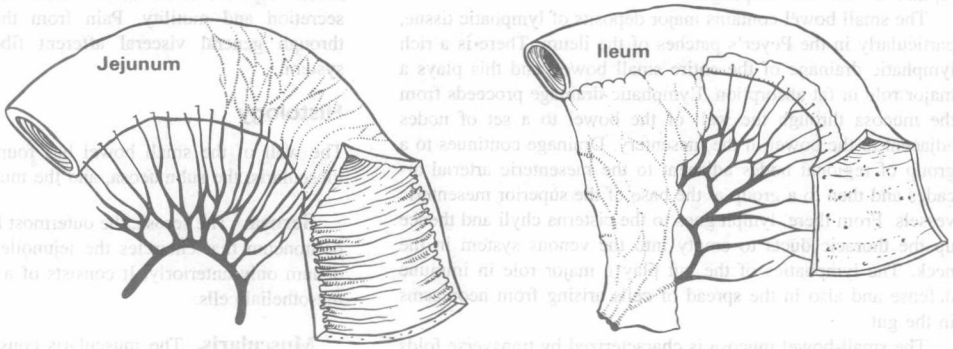

FIG. 25-2. Jejunum contrasted with ileum. In the jejunum, note the larger diameter, thicker wall, prominent plicae circulares, one or two arterial arcades, long vasa recta, and translucent (fat-free) areas at the mesenteric border. The ileum is smaller and thinner walled and has few plicae, multiple vascular arcades with short vasa recta, and abundant mesenteric fat.

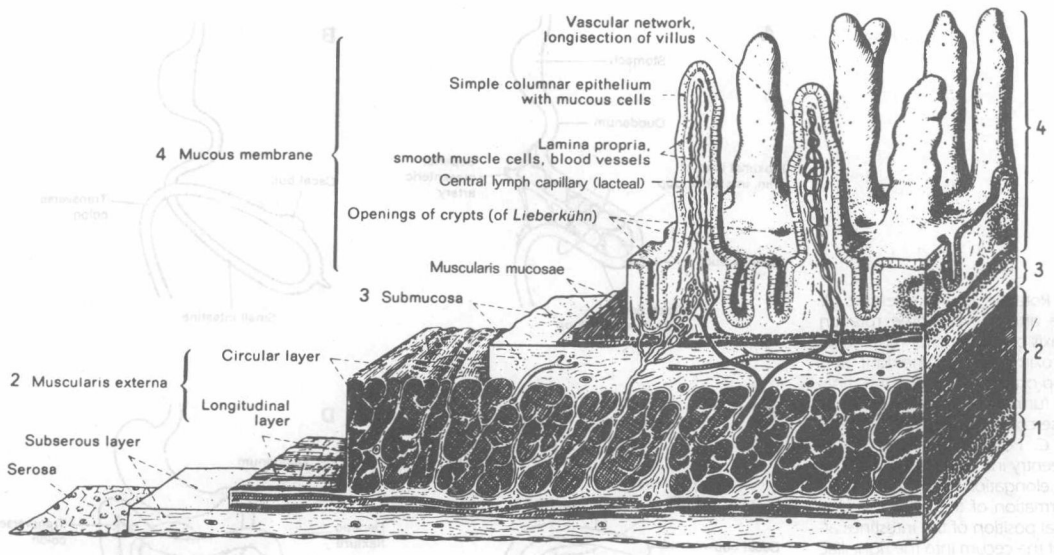

FIG. 25-3. Layers of the small intestine. A large surface is provided by villi for the absorption of required nutriments. The solitary lymph follicles in the lamina propria of the mucous membrane are not labeled. In the stroma of both sectioned villi are shown the central chyle (lacteal) vessels or the villous capillaries. (From: *Sobotta J, Figge FHJ, Hild WJ: Atlas of Human Anatomy. New York, Hafner, 1974, with permission.*)

cular arcades in the mesentery. In spite of this collateral supply, occlusion of a major branch of the superior mesenteric artery, or of the superior mesenteric artery itself, will lead to bowel death if not quickly corrected. Venous drainage of the segments of the small bowel is in parallel with the arterial supply. The superior mesenteric vein joins the splenic vein behind the neck of the pancreas to form the portal vein. The blood leaving the gut, with its relatively high oxygen content, provides a significant portion of the oxygen supply to the liver.

If the mesentery is not greatly infiltrated by fat and if there are no peritoneal adhesions, the bowel is extraordinarily mobile on its vascular tether. In some individuals jejunal segments may be sufficiently mobilized to allow anastomosis in the neck to replace the cervical esophagus.

The small bowel contains major deposits of lymphatic tissue, particularly in the Peyer's patches of the ileum. There is a rich lymphatic drainage of the entire small bowel, and this plays a major role in fat absorption. Lymphatic drainage proceeds from the mucosa through the wall of the bowel to a set of nodes adjacent to the bowel in the mesentery. Drainage continues to a group of regional nodes adjacent to the mesenteric arterial arcades and then to a group at the base of the superior mesenteric vessels. From there, lymph goes to the cisterna chyli and thence up the thoracic ducts to empty into the venous system in the neck. The lymphatics of the gut play a major role in immune defense and also in the spread of cells arising from neoplasms in the gut.

The small-bowel mucosa is characterized by transverse folds (plicae circulares, or valves or folds of Kerckring), though these are absent in the duodenal bulb and in the distal ileum. They are more prominent in the distal duodenum and the jejunum, where they may reach 1 cm in height and form interlocking transverse ridges (see Fig. 25-2). The small-bowel mucosa has a pink, velvety appearance with a glistening surface. It usually is thicker in the jejunum than in the ileum, where there may be no folds and the surface may be entirely smooth, except for small scattered lymphatic nodules.

The innervation of the small bowel comes from both sympathetic and parasympathetic systems. Parasympathetic fibers come from the vagus and traverse the celiac ganglia. They affect secretion and motility and probably all phases of bowel activity. Vagal afferent fibers are present but apparently do not carry pain impulses. The sympathetic fibers come from the three sets of splanchnic nerves and have their ganglion cells usually in a plexus around the base of the superior mesenteric artery. Their motor impulses affect blood vessel motility and probably gut secretion and motility. Pain from the intestine is mediated through general visceral afferent fibers in the sympathetic system.

Histology

The wall of the small bowel has four layers—the serosa, the muscularis, the submucosa, and the mucosa (Fig. 25-3).

Serosa. The serosa, the outermost layer, consists of visceral peritoneum that encircles the jejunoileum but covers the duodenum only anteriorly. It consists of a single layer of flattened mesothelial cells.

Muscularis. The muscularis consists of a thin outer longitudinal layer and a thicker inner circular layer of smooth muscle. Specialized gaps in the muscle-cell membranes permit cell-to-cell communication, which facilitates the ability of the muscle layer to function as an electrical syncytium. Ganglion cells from

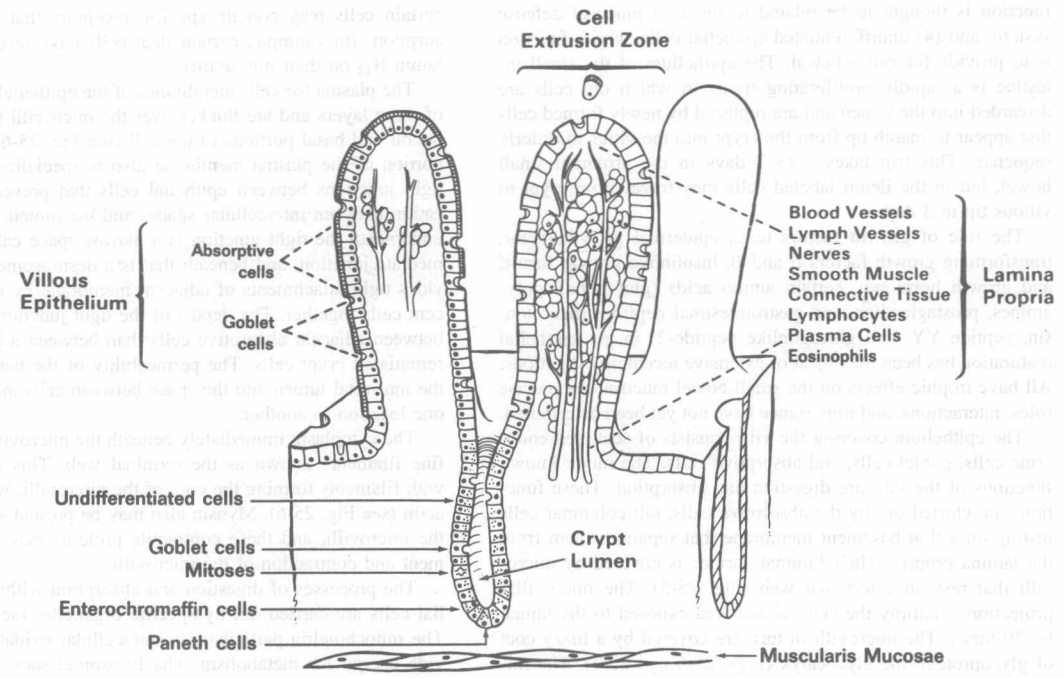

FIG. 25-4. Schematic diagram of two sectioned villi and a crypt of Lieberkühn illustrating the histologic organization of the small intestine mucosa. (Adapted and redrawn from: *Trier JS et al, in Sleisenger MH, Fordtran JS [eds]: Gastrointestinal Disease: Pathophysiology, Diagnosis, Management. Philadelphia, WB Saunders, 1983, chap 48, with permission.*)

the myenteric (Auerbach's) plexus are interposed between the two muscle layers and send fibers into both layers.

Submucosa. The submucosa is a layer of fibroelastic connective tissue containing blood vessels and nerves. It is the strongest component of the bowel wall and therefore must be included when sutures are placed through the bowel. It contains elaborate networks of lymphatics, arterioles, and venules and an extensive plexus of nerve fibers and ganglion cells (Meissner's plexus). Although frequently subdivided, the nerves from the mucosa, submucosa, and muscle layers are interconnected by small nerve fibers, and cross-connections between adrenergic and cholinergic elements have been described.

Mucosa. Looked on as a device to increase absorptive surface, the small-bowel mucosa is an architectural marvel. The gross transverse folds, the fingerlike villi protruding into the lumen of the bowel, the microvilli (brush border) covering the cells, and the glycocalyx fuzz covering the microvilli each tremendously increase the surface area exposed to luminal contents. Villi protrude 0.5 to 1 mm into the lumen; they are tallest in the distal duodenum and the proximal jejunum and become progressively shorter toward the terminal ileum.

The mucosa can be divided into three layers—the muscularis mucosae, the lamina propria, and the epithelium. The deepest of these, the muscularis mucosae, is a thin sheet of muscle separating mucosa from submucosa. The lamina propria is a continuous layer connective tissue between the epithelium and the muscularis mucosae. It extends into the villi and around the pit-like crypts of Lieberkühn (Fig. 25-4). The lamina propria contains, additionally, a variety of cells—plasma cells, lymphocytes, mast cells, eosinophils, macrophages, fibroblasts, smooth muscle cells—and noncellular connective tissue. The lamina propria is the architectural base on which the epithelium lies, but it also has important functions of its own and apparently serves protectively to combat microorganisms that penetrate the overlying epithelium. The plasma cells are an active site of synthesis of immunoglobulins. In addition, cells in the lamina propria release various mediators (for example, cytokines, arachidonic acid metabolites, and histamine) that can modulate various cellular functions of the overlying epithelium.

The innermost mucosal layer is a continual sheet, one layer thick, of epithelial cells covering the villi and lining the crypts of Lieberkühn (see Fig. 25-4). The main functions of the crypt epithelium are cell renewal and exocrine, endocrine, water, and ion secretion; the main function of the villous epithelium is digestion and absorption. The crypts contain at least four distinct cell types: (1) goblet cells, which secrete mucus; (2) enteroendocrine cells (often called enterochromaffin or argentaffin cells in the older literature), of which there are more than ten distinct populations that produce the gastrointestinal hormones, including gastrin, secretin, cholecystokinin, somatostatin, enteroglucagon, motilin, neurotensin, and gastric inhibitory peptide; (3) Paneth cells, which secrete lysozyme, tumor necrosis factor, and the cryptidins, homologues of leukocyte defensins, and whose

function is thought to be related to the host mucosal defense system; and (4) undifferentiated epithelial cells, whose function is to provide for cell renewal. The epithelium of the small intestine is a rapidly proliferating tissue in which old cells are discarded into the lumen and are replaced by newly formed cells that appear to march up from the crypt into the villus in orderly sequence. This trip takes 5 to 7 days in the proximal small bowel, but in the ileum labeled cells may travel from crypt to villous tip in 3 days.

The role of growth factors (e.g., epidermal growth factor, transforming growth factors α and β, insulinlike growth factor, and growth hormone), certain amino acids (glutamine), polyamines, prostaglandins, and gastrointestinal peptides (neurotensin, peptide YY, and glucagonlike peptide-2) in gut epithelial maturation has been the object of extensive recent investigations. All have trophic effects on the small-bowel mucosa; the precise roles, interactions, and importance have not yet been determined.

The epithelium covering the villi consists of scattered endocrine cells, goblet cells, and absorptive cells. The major known functions of the villi are digestion and absorption. These functions are carried out by the absorptive cells, tall columnar cells resting on a thin basement membrane that separates them from the lamina propria. Their luminal surface is covered by microvilli that rest on a terminal web (Fig. 25-5). The microvillar projections multiply the cell surface area exposed to the lumen by 30 times. The microvilli in turn are covered by a fuzzy coat of glycoprotein, the glycocalyx (Figs. 25-6 and 25-7). The microvilli participate actively in absorption and digestion. They contain enzymes for digestion of disaccharides and peptides, and certain cells may contain specific receptors that facilitate absorption (for example, certain ileal cells have receptors for vitamin B_{12} on their microvilli).

The plasma (or cell) membranes of the epithelial cells consist of three layers and are thicker over the microvilli than over the lateral and basal portions of the cell (see Fig. 25-6). The lateral portion of the plasma membrane also is specialized. There are tight junctions between epithelial cells that prevent communication between intercellular spaces and the lumen. Immediately underneath the tight junction is a narrow space called an intermediate junction, and beneath that is a desmosome, which provides tight attachments of adjacent membrane by binding adjacent cells together. The depths of the tight junctions are greater between adjacent absorptive cells than between adjacent undifferentiated crypt cells. The permeability of the barrier between the intestinal lumen and the space between cells may vary from one location to another.

The cytoplasm immediately beneath the microvilli consists of fine filaments known as the terminal web. This interconnects with filaments forming the core of the microvilli, which contain actin (see Fig. 25-6). Myosin also may be present at the base of the microvilli, and these contractile proteins may allow movement and contraction of the microvilli.

The processes of digestion and absorption within the epithelial cells are carried out by specific organelles (see Fig. 25-5). The mitochondria participate in intracellular oxidation and provide energy for metabolism. The lysosomal sacs contain cytotoxic substances and intracellular waste products. The endoplasmic reticulum is the main synthesizing element within the cell

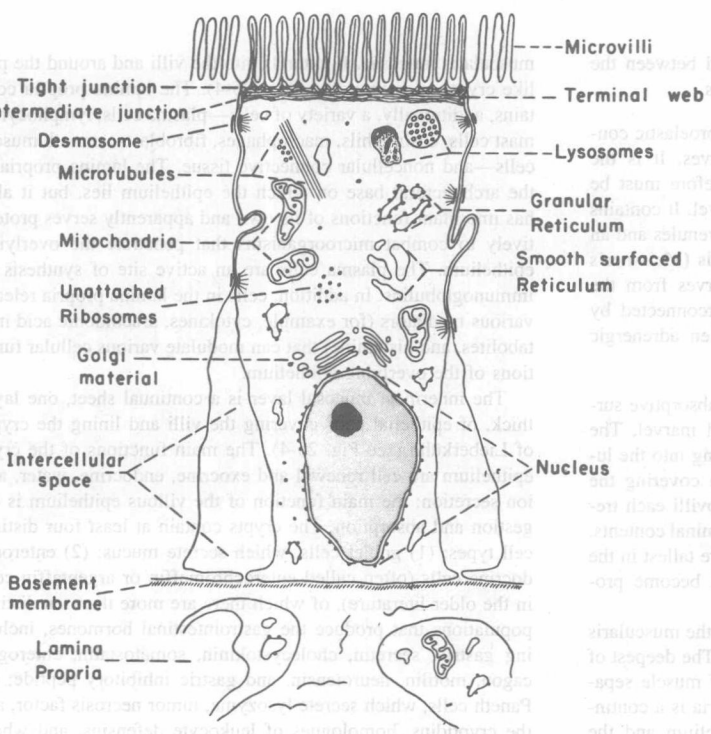

FIG. 25-5. Schematic diagram of an intestinal absorptive cell. (From: *Trier JS et al, in Sleisenger MH, Fordtran JE [eds]: Gastrointestinal Disease: Pathophysiology, Diagnosis, Management. Philadelphia, WB Saunders, 1983, chap 48, with permission.*)