
# Malabsorption*

**Dr. Julius E. Linn:** Today's topic on the clinician's approach to the diagnosis of malabsorption will be presented by Dr. Thomas W. Sheehy, Professor of Medicine, and Chief of the VA Medical Service.

**Dr. Thomas W. Sheehy:** Today's world traveler must beware of a number of tropical diseases that may plague him, and one of these is tropical sprue. A patient, in the tropics for two months, developed diarrhea and steatorrhea. The stool contained no parasites, or pathogenic bacteria. She had no history of gastrointestinal disease, so it was reasonable to suspect that she had tropical sprue; a trial of folic acid led to marked clinical improvement.

This patient's sudden onset of symptoms after a brief sojourn in the tropics is not unusual. During World War II, entire units developed tropical sprue after only three to four weeks in an endemic area. The shortage of supplies of folic acid often resulted in prolonged convalescence and sometimes death.

Tropical sprue and celiac disease have taught us much about the normal functions of the intestine and its absorptive mechanisms. Today, I would like to discuss with you some of the physiologic aspects of absorption, some findings observed in patients with intestinal malabsorption, and certain tests which can be used in the office or hospital to evaluate patients suspected of malabsorption.

*Digestion* is the process by which large, complex nutrient molecules are reduced to immunologically inactive particles that can be transferred across the intestinal mucosa. *Absorption* is the process by which these digested products are transferred across the intestinal mucosal cells into the blood or lymph.

Digestion is initiated by salivary enzymes, is continued within the bowel lumen by gastric, biliary, and pancreatic secretions, and is completed by the brush border enzymes of the small intestinal epithelium (Fig 1). The prior concept that the "succus entericus" was responsible for the final stages of digestion is no longer tenable; neither is the belief that

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FIG 1

Tips of normal jejunal villi. (US Army photograph)

products of digestion are absorbed unchanged by the intestinal mucosa by simple diffusion, osmosis, or filtration. The intestinal mucosal epithelium has distinct roles in both digestion and absorption. Carbohydrates, proteins, and fats are profoundly altered by enzymatic activities of the epithelial cell's brush border. For example, brush border dipeptidases are specifically responsible for the hydrolysis of peptides composed of neutral amino acids. Their presence makes it possible for patients with pancreatic deficiency to digest and to absorb the bulk of dietary protein.

Our concepts of intestinal absorption also have changed, and we now know it entails: (1) penetration of the lipoprotein membrane surrounding the epithelial cell; (2) transport of substances through intracellular canals and organelles; (3) metabolic or biochemical transformation of materials within the cell; (4) extrusion at the bottom or sides of the cell; and (5) passage through the basement membrane to lymph or blood channels.

*Passive* absorption plays a significant role in the intestine's remarkable ability to render the luminal contents of the proximal bowel "isotonic." After a hypertonic meal, the osmolality of gastric contents may reach 400 mOsm/kg, but by the time the chyme travels from the pylorus to the ligament of Treitz, a mere 12 inches, it is rendered isotonic with plasma. This is accomplished mainly through bulk transfer of water from blood to lumen in response to an osmotic gradient. In contrast, bulk flow of water occurs from the lumen to blood when a hypotonic meal is ingested. Passive absorption, however, primarily is responsive to osmotic gradients, and when the luminal contents are isotonic, passive movement stops.

*Active* absorption is responsible for the absorption of most of our nutrients. It is an energy-dependent process wherein energy derived from cellular metabolism is coupled to carrier mediated ion transport. This permits the transport of ions against an electrochemical or concentration gradient. The absorption of glucose is an excellent example of active transport abetting passive diffusion. The glucose molecule is transported actively across the intestinal mucosa against a high concentration gradient. Once across, the glucose molecule is too large to diffuse back, creating an osmotic gradient within the cell that draws water and sodium through the pores in the direction of glucose flow. Specific carrier systems for active transport are most effective in the proximal jejunum. However, transport systems can be saturated. Spillover from the jejunum can be be accommodated by active carrier systems throughout the small bowel. Thus, fats, sugars, and amino acids are all absorbed beyond the jejunum. Certain carrier systems are localized to specific segments of the gut. These include: vitamin $B_{12}$, which is transported only in the ileum; iron transport, which is restricted to the duodenum and proximal jejunum; and active sodium and water absorption, which is most effective in the distal ileum and colon.

These physiologic concepts have improved our understanding of intestinal absorption, and have allowed clinicians to group various

TABLE 1

DISORDERS OF MALDIGESTION

| |
|---|
| **Inadequate Digestion** |
| Pancreatic insufficiency |
| Pancreatic carcinoma |
| Chronic pancreatitis |
| Cystic fibrosis |
| Acid hypersecretion |
| Zollinger-Ellison syndrome |
| Carcinoid syndrome |
| Gastric resection |
| Shortened transit time |
| Gastrocolic fistula |
| Diarrheal states |
| **Altered Bile Salt Metabolism** |
| Hepatobiliary disease |
| Intestinal resection, regional ileitis |
| Bacterial overgrowth |
| Blind loop syndrome |
| Scleroderma |
| Diabetic enteropathy |

TABLE 2

DISORDERS OF INTESTINAL MALABSORPTION

| | |
|---|---|
| Tropical and nontropical sprue | Ischemic and regional enteritis |
| Lymphoma | Short bowel syndrome |
| Whipple's disease | Lymphangiectasia |
| Strongyloidiasis, giardiasis | Drugs |
| Scleroderma, amyloidosis | Dysgammaglobulinemia |
| Hyperthyroidism | Radiation enteritis |
| Adrenal insufficiency | Congestive heart failure |
| Kwashiorkor | |

small intestinal diseases into disorders of maldigestion, malabsorption, and specific impairment of selective transport systems (Tables 1-3).

*Disorders of Maldigestion.* Here the basic problem is inability to reduce luminal nutrients to small, absorbable compounds. In pancreatic deficiency, defective lipase secretion impairs fat hydrolysis. In liver or biliary tract disease, not enough bile salts are present to solubilize fatty acids and monoglycerides. In the Zollinger-Ellison syndrome, excess jejunal acid inhibits pancreatic enzymatic activity. In the blind-loop syndrome, bacterial overgrowth of the upper small bowel leads to deconjugation of bile acids. As a result, bile salt micellar formation is impeded and monoglycerides and fatty acids are not solubilized into hydrotropic complexes. Vitamin $B_{12}$ also is absorbed poorly because of bacterial competition for the vitamin.

Ordinarily, active peristalsis restricts enterobacteria to the distal ileum. Any abnormality that causes stasis in the upper bowel, such as a stricture, an afferent loop after Billroth 2 procedure, scleroderma, diverticulosis, or diabetic enteropathy can lead to bacterial overgrowth and to impaired fat and vitamin $B_{12}$ absorption.

Except for the Zollinger-Ellison syndrome, the intestinal mucosa is normal by light microscopy in disorders of maldigestion.

*Disorders of Malabsorption.* Generalized malabsorption may result from mucosal surface loss, epithelial cell injury, or damage to subepithelial strictures and collecting systems (Table 2). These disorders all cause steatorrhea, and many are associated with mucosal lesions. Gluten is so toxic to some treated celiac patients that morphologic changes occur in the surface epithelium as early as 4½ hours after its ingestion, and functional changes, such as decreased disaccharidase activity, may occur within an hour. Striking morphologic changes also occur in the small intestinal mucosa of patients with tropical sprue, Whipple's disease, severe protein deficiency, and intestinal lymphangiectasia (Fig 2).

TABLE 3

DISORDERS INVOLVING MALABSORPTION OF SPECIFIC SUBSTANCES

| | |
|---|---|
| Disaccharidase deficiency | — loss of brush-border enzymes |
| Monosaccharide malabsorption | — transport failure |
| Vitamin $B_{12}$ deficiency | — lack of intrinsic factor (pernicious anemia), impaired separation of intrinsic factor-$B_{12}$ complex (Immerlund's syndrome) |
| α-β-lipoproteinemia | — lipoprotein lipase abnormality |
| Cystinuria, Hartnup's disease | — transport defect for certain amino acids |

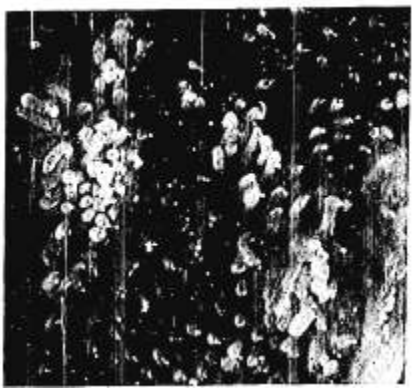
FIG 2

Abnormal jejunal mucosa taken from a patient with nontropical sprue. (US Army photograph)

Most physicians are unaware that the mucosal surface of the small intestine is 100 times greater than the skin surface and exceeds 200 sq m. Its surface area is increased threefold by the plica circularis, eightfold by the villi, and 30-fold by the 300 to 500 microvilli perched atop each individual epithelial cell (Fig 3). These surface structures are altered strikingly in sprue syndromes. In celiac disease and tropical sprue, the villus-crypt ratio is changed, the intestinal epithelium is damaged, the brush border is altered, and enzymes located in the brush border zone are depleted or absent.

Villous appearance in celiac disease is related to epithelial cell turnover time: finger-shaped villi have an epithelial cell turnover time of four days, leaf-shaped villi two days, convoluted villi 6 to 12 hours, and a flat mucosa less than six hours. These villous changes result from increased epithelial cell loss or decreased

FIG 3



Normal villi as seen with dissecting microscope.

cell production. In celiac disease, epithelial cell production is increased greatly, but the rate of cell shedding from the surface exceeds production. If cell loss greatly exceeds cell production, a totally flat mucosa results.

Cell loss also contributes to clinical weight loss in celiac disease. Normally, 0.25 to 0.50 lb of cells are lost from the gut lining daily, but most cell constituents are reabsorbed. With severe celiac disease, however, cell loss may be six times the normal rate, and the shed cellular constituents are not absorbed readily. Villous changes also occur in protein deficiency and starvation, but cell production is decreased instead of increased and failure of production leads to villous changes and thinning of the gut wall.

*Selective Malabsorption.* The third group of disorders consists of malabsorption of specific substances due to failure at the mucosal cell level (Table 3). This group includes: disaccharidase deficiency, monosaccharide malabsorption, malabsorption of vitamin $B_{12}$ with proteinuria in the young, Hartnup's disease, and $\alpha$-$\beta$-lipoproteinemia.

Disaccharidase deficiency may be single or multiple and may be congenital or acquired; lactase deficiency is probably the most common type of selective malabsorption.

In Hartnup's disease, the transport of neutral amino acids (alanine, serine, asparagine, trypsin, tryptophan, glutamine, etc), is defective in both the kidney and small bowel. Similarly, in cystinuria, the transport system for dibasic amino acids (lysine, ornithine, and arginine) is defective. Selective malabsorption of vitamin $B_{12}$ proteinuria is primarily a childhood disease and its cause is unknown. Gastric secretion and intrinsic factor production are formal in this entity.

## Clinical Approach

*History.* Over 30 disorders of maldigestion or malabsorption are listed (Tables 1-3), and these lists are not complete. Such an impressive group of diseases taxes the clinician's diagnostic ability and often the patient's pocketbook. Fortunately, a good history is invaluable in identifying the cause of malabsorption and permitting a rational approach to diagnosis. For example, childhood illness characterized by delayed growth and diarrhea suggests celiac disease or milk intolerance. The onset of diarrhea in a diabetic with neurologic complications serves as a clue to diabetic enteropathy. Arthritis or recurrent pleuritis followed years later by onset of steatorrhea suggests Whipple's disease. Gastric or other intestinal surgery may set the scene for a bacterial overgrowth syndrome, while an ileal resection may cause anemia or bile salt deficiency. Onset of diarrhea after a visit to the tropics should suggest tropical sprue, dysentery, or parasitic infestation. The presence of aphthous ulcers suggests sprue or ulcerative colitis. Repeated bacterial infections in the child may suggest dysgammaglobulinemia. Raynaud's phenomenon and skin changes may imply scleroderma. Extensive pelvic irradiation may lead to an irradiation enteritis.

*Physical Findings.* Skin disease has recently received a great deal of attention as a clue to diagnosis. (1) Malabsorption of any type may cause the skin lesion seen in tropical and nontropical sprue: increased pigmentation, acquired ichthyosis, psoriasis, eczematous lesions, or pellagra-like rashes. Ten to 20 percent of adults with malabsorption have rashes, but malabsorption is found in only about 2% of patients with eczema or psoriasis.

(2) Malabsorption may be caused by the disease which produces the skin lesion (dermatogenic enteropathy). Patients with severe generalized exfoliative dermatitis have mal-

absorption, which regresses with healing of the skin lesion; the severity of malabsorption is related to the extent of the dermatosis. Histologically, the jejunum mucosa is normal in appearance in dermatogenic enteropathy in contrast to its abnormal appearance in many patients with dermatitis herpetiformis.

(3) In dermatitis herpetiformis, the dermatitis and malabsorption are not related etiologically. Dermatitis herpetiformis causes a purpuric, vesicular rash that leads to formation of subepidermal blisters on the elbows, back, and buttocks (Fig 4). These lesions are bilateral and symmetrically distributed. Two thirds of these patients have a flat intestinal mucosa that resembles the lesion of celiac disease, and the lesion is made worse by gluten ingestion. The intestinal mucosa heals with a gluten-free diet, but dapsone is necessary to treat the skin disorder. Finally, other disorders, such as ischemia and scleroderma, may affect both skin and gut, and cause malabsorption.

The patient with severe malabsorption suffers from malnutrition, and, like the starved patient, may have a thin face and extremities, a potbelly due to gaseous distention, muscle wasting, and edema due to protein deprivation or loss. Invariably, the patient suffers weight loss, and with time may develop clinical signs of fat soluble vitamin deficiency, such as hemorrhagic tendencies due to lack of vitamin K. If severe enough, the condition may lead to osteomalacia, hypokalemic tetany, bone fractures, megaloblastic anemia, or refractory iron deficiency anemia, and even fever of unknown origin. Signs, such as exophthalmos, jaundice, uremic breath, macroglossia, generalized lymphadenopathy, and erythema nodosum, may quickly suggest an underlying disease that is the cause of diarrhea or malabsorption.

FIG 4

Dermatitis herpetiformis with typical lesions on the buttocks. This patient had an abnormal mucosa and mild malabsorption.

A skin flush and hepatomegaly may reflect a metastatic carcinoid tumor. Fever is a key finding, although it may be absent with infection, present with some neoplasms, and usually absent in ulcerative colitis. Rectal and pelvic examinations permit bimanual palpation of diseased loops of large and small bowel. Proctosigmoidoscopic examination also may reveal evidence of localized disease. Gaseous distention that grows worse as the day progresses suggests malabsorption, intestinal obstruction, or simple aerophagia.

*Laboratory Studies.* The appearance of a stool has much to tell the clinician, for it often reflects the level of disease. Certainly, this is true of the pale, porridge-like, foul smelling, greasy stool of classical steatorrhea. But only about two thirds of patients with steatorrhea have such a classical stool; some are even constipated!

Stool aroma also is helpful. In lactase deficiency, fermentation of the sugar often gives the stool an aroma of sour milk, and ammoniac odor suggests proteus infection, while no odor implies an overgrowth of staphylococcus. Stool pH also should be checked: low pH suggests fermentation diarrhea due to production of short-chain organic acids (butyric, proprionic, etc). The pH is checked easily with a dip stick, while a Dextrostix can be used to detect excess stool sugars.

Of all the diseases listed in Tables 1-3, only primary glucose malabsorption, the amino acid deficiencies, and the vitamin $B_{12}$ transport defects are not associated with steatorrhea. Normally, almost all ingested fat is absorbed: on a diet containing 100 to 150 gm fat daily, 3 to 5 gm of fat is excreted every 24 hours. This stool fat comes from endog-

enous fat production or from fat sloughed in epithelial cells. Increasing dietary fat to 200 gm daily doubles the fecal fat content, and a daily diet containing 300 gm may lead to excretion of 20 or more gm of fat. In most hospitals, 72 hr fecal fat measurements are difficult to obtain, and often they are done imprecisely.

*Office Tests.* The following tests are easy, simple maneuvers that can be done in the doctor's office.

(1) *Microscopic Fecal Fat Test*: An old but useful test for detecting steatorrhea is microscopic examination of a Sudan III stained fecal smear. Increased microscopic stool fat is present in 75% of patients with mild chemical steatorrhea (6 to 10 gm), and in 94% of patients with 15 gm of fecal fat.

Another technic for studying microscopic stool fat is very simple. A fresh or frozen stool sample is stained with Sudan III (50% alcohol), a coverslip is added, and 20 randomly selected fields are microscopically examined under low power. With this technic, the upper limit of normal is six globules of fat per low-power field. The minimum diameter of the globules should range from 15$\mu$ to 25$\mu$ (8$\mu$ to 10$\mu$ = diameter of RBC). Care should be taken to stay away from the edge of the coverslip, for fat globules, like WBCs, tend to concentrate at the edge and give a false count.

Large amounts of neutral fat are found in the stools of patients ingesting large amounts of mineral oil, castor oil, and diabetic mayonnaise (which contains large amounts of mineral oil), or using rectal suppositories. An old dictum implies that few, if any, neutral fat globules are found in normal stool, and if they are present, chronic pancreatitis should be suspected. However, the presence of neutral fat in a Sudan III preparation is neither characteristic nor diagnostic of chronic pancreatitis, but it is suggestive of malabsorption. The absence of fat globules in a plain Sudan III preparation, and their appearance after heating and acidifying the fecal material, also has been considered diagnostic of intrinsic malabsorption. This too is false. Heat and acid increase the number of fat globules per low-power field, because they precipitate fatty acid crystals and globules that are easily identified with Sudan III stain.

Among 150 patients with chemical steatorrhea and a mean stool fat of 12.1 gm/24 hr, we found 124 had microscopic evidence of steatorrhea (83%).

(2) *Meat-Fiber Tests*: The presence of meat fibers in the stool also correlates well with chemical fat determinations (76%), and the test seldom requires more than five minutes. One can examine a fresh stool directly or mix the stool with 1.0% eosin for three minutes, and then examine it under a coverslip for rectangular fibers with coarse striations. Textbooks disagree on how many meat fibers are found in a normal stool preparation. The older literature implies one meat fiber per high-power field is abnormal. We believe that ten per coverslip preparation is abnormal in patients eating a normal diet with two portions of meat. I wish to destroy another old fable: in the adult, increased numbers of meat fibers in the stool do not distinguish pancreatic disease from other causes of malabsorption. However, the presence of large numbers of meat fibers does indicate malabsorption.

(3) *Serum Turbidity Test*: This test is performed easily in the office. The patient is given 30 gm of butter on a cracker, or an ice cream shake prepared with 40% cream, and is checked for alimentary lipemia. A fasting serum sample is compared with serum samples drawn two, three, and four hours after ingestion of the meal. If the postprandial serum is lactescent, the patient probably has normal fat absorption. If a distinct change is not discernible to the naked eye, simply compare the fasting and postprandial serum samples in a Klett photometer set at 620 m$\mu$ wave length. This measures turbidity or particulate fat droplets, and an optical density rise of 0.1 unit above the fasting level is considered a normal lipemic response. This is an easy, inexpensive test that correlates well with chemical steatorrhea.

(4) *Water Tests*: Other old, useful, and inexpensive tests that can be done in the office or hospital are the water tests. In one, the fasting patient is asked to urinate, and then is given 20 ml of water per kilogram of body weight. Urine is then collected for four hours. Ordinarily, 75% or more of the water load administered is recovered within four hours. Low values are found with malabsorption,

Addison's disease, congestive heart failure, myxedema, and cirrhosis.

Wollaeger and Scribner,[4] in 1951, first called attention to increased nocturnal diuresis in patients with nontropical sprue and attributed it to prolonged postprandial retention of water in an intestine packed with unabsorbed nutrients. We found a striking reversal of the day-night ratio of diuresis in most of our tropical sprue patients. In sprue, nocturnal diuresis probably stems from changes in mucosal permeability to water and electrolytes. The effective pore size in the jejunal mucosa of celiac sprue is decreased below that normally found in the ileum. This impairs water flow in response to osmotic gradient pressures (solvent drag), and decreases passive absorption of sodium chloride and other water soluble solutes in the jejunum. Hence, these patients have an increased luminal osmotic load (due to malabsorption of digested food), exposed to an impermeable jejunal mucosa. Nocturnal diuresis was observed in 93% of 70 tropical sprue patients with steatorrhea. The ratio of night-to-day urine volumes varies from 0.85 to 2.71 (mean 1.70) in comparison to a normal ratio of 0.28 to 0.54.

(5) *Sugar Tests*: The glucose tolerance test is seldom used today as a screening test for primary malabsorption, because 10% to 25% of normal individuals have a flat glucose tolerance curve after ingestion of a standard glucose meal. Lactose and sucrose tolerance tests are easily accomplished if a disaccharidase deficiency is under consideration. One merely gives 1.0 gm of lactose or sucrose per kilogram of body weight, not to exceed 100 gm, and obtains fasting 15, 30, 60, and 120 minute blood samples for blood glucose levels. Onset of intestinal cramps, or diarrhea, or a flat glucose curve, ie, with less than a 15 mg% rise in blood glucose, suggests an enzyme deficiency (Fig 5).

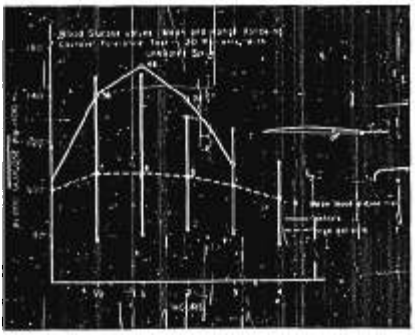
While these simple tests are useful, they are not always diagnostic. Table 4 shows a battery of laboratory tests that will establish the presence or absence of malabsorption in most patients, and usually will allow identification of the disorder causing it. In a study of 150 patients with tropical sprue, we found that when two tests were abnormal, steatorrhea was present in 85% of our patients. If three tests were abnormal, 90% of patients had steatorrhea. Such batteries are very useful in detecting malabsorption.

TABLE 4

LABORATORY TESTS USED IN DIAGNOSING MALABSORPTION

| |
|---|
| Xylose absorption |
| Serum carotene |
| Vitamin A tolerance |
| Jejunal biopsy |
| Secretin-pancreozyme test |
| Therapeutic trial |
| Schilling test |
| Gastrointestinal roentgenographic studies |

FIG 5



Broken line indicates the flat glucose curve found in sprue after a lactose tolerance test.

If none of the clinical clues, office tests, and screening tests provide a provisional diagnosis, the following sequence of laboratory procedures is useful.

(1) A gastrointestinal roentgenogram of the entire gastrointestinal tract, including the small bowel. Routine chest films also should be searched at this time for evidence of tuberculosis or lymphoma.

(2) A jejunal biopsy beyond the ligament of Treitz. There are a number of diseases which may have late mucosal lesions and look the same under the dissecting microscope, but have distinctive and specific histologic features. In hypogammaglobulinemic sprue, there is an absence of plasma cells in the lamina propria. Whipple's disease is characterized by the typical PAS staining macrophages. A multiplicity of eosinophilic leukocytes in the lamina propria is seen in eosinophilic gastroenteritis. In primary lymphoma, the mucosa may be infiltrated by malignant lymphoid cells, and in the Zollinger-Ellison syndrome, patchy flat

areas contain many neutrophils. Measurement of mucosal disaccharidase activity can be obtained if a primary or secondary deficiency of these enzymes is suspected.

There is also a group of diseases that share the same flat mucosal lesion as celiac disease. In these, therapeutic trials may be necessary for diagnostic differentiation. They are: tropical sprue, dermatitis herpetiformis, kwashiorkor, giardiasis, and collagenous sprue. Recently it was found that treatment of giardiasis in patients with hypogammaglobulinemic sprue led to clinical remission and healing of the mucosal lesion. Treatment of dermatitis herpetiformis has been discussed. Tropical sprue responds to combination therapy with folic acid and broad-spectrum antibiotics. We still are not certain if collagenous sprue is a distinct entity. We have observed this entity in a child who died of evident starvation. Her sisters died with a similar illness and the parents were told she had celiac disease.

(3) A Schilling's test with intrinsic factor is useful if bacterial overgrowth is suspected. Failure of vitamin $B_{12}$ absorption in this situation results either from massive steatorrhea or from significant bacterial overgrowth. The latter can be tested by aspiration of the upper reaches of the jejunum with a simple, open-end polyethylene tube with a mercury bag attached; complicated instruments are not required. Relatively few organisms are present in the upper bowel and these usually consist of enterococci, lactobacilli, and diphtheroids. Bacterial concentrations seldom exceed more than $10^3$/ml or $10^4$/ml of intestinal fluid. Coliform organisms are found occasionally in the upper small intestine in small titers. With stasis, however, gram-negative bacteria such as enterobacteria and *Bacteroides* appear in the upper bowel and may reach concentrations of $10^9$/ml or $10^{12}$/ml of intestinal fluid.

(4) The secretin-pancreozyme test is a useful procedure for determination of pancreatic exocrine function. In this test the volume, pH, bicarbonate, and enzyme content of duodenal fluid are measured before and after administration of these hormones.

(5) *Therapeutic Trial*: Finally, recourse may be necessary to a diagnostic-therapeutic trial. If gluten enteropathy is suspected, a trial of gluten-free diet may prove worthwhile. On this diet, patients with celiac disease often improve within a few days, but it may be necessary to continue the diet for several weeks or even months. Progress of the patient is ascertained by measuring body and stool weight, as well as the stool fat content before and after treatment. Similarly, use of the lactose-free diet is beneficial in those who have a deficiency of lactase. Broad-spectrum antibiotics, particularly tetracycline, may be useful diagnostically in Whipple's disease, in bacterial overgrowth syndromes, and in tropical sprue.

**Dr. Linn:** Thank you Dr. Sheehy, for an interesting Grand Rounds.

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