Evaluation of Chronic Diarrhea: Case Studies;
Dizziness: Four Case Studies

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Chapter 1—Evaluation of Chronic Diarrhea: Case Studies

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I. INTRODUCTION

Diarrhea is a common cause of patient visits to their internist. Although definitions vary, diarrhea is often defined as more than 200 g of stool production per day with higher than normal water content. Normal stool weight is 100 to 200 g per day with a water content of 60% to 80%. Although definitions vary, acute diarrhea is typically characterized as lasting less than 2 weeks, whereas chronic diarrhea lasts more than 2 to 3 weeks. Chronic diarrhea is often recurrent and has a much broader differential diagnosis. This review will discuss the clinical presentation and appropriate diagnostic evaluation for patients presenting with chronic diarrhea. Three case patients are presented to highlight features of evaluation for patients with chronic diarrhea.

II. CASE PATIENT 1

PRESENTATION

Patient 1 is a 35-year-old woman with a 6-month history of diarrhea. She reports having soft bowel movements approximately 7 times a day and notes she has abdominal pain that is relieved with defecation. She is taking no medication and has no medical history except for 2 uncomplicated vaginal deliveries.

- The most appropriate next step in evaluating patient 1 is which of the following?
  A) Complete blood count, measurement of serum electrolyte levels, and stool culture
  B) Empiric treatment with antibiotics
  C) Further questioning and measuring the amount of stool
  D) Referral to a psychiatrist

- Which of the following additional findings is most indicative of the need for further testing?
  A) History of blood in her stool
  B) Weight loss
  C) Fever
  D) All of the above

HISTORY AND PHYSICAL EXAMINATION

The correct answers for the 2 previous questions are C and D, respectively. The first step in the evaluation of diarrhea is quantifying the stool in addition to obtaining a detailed history. Diarrhea can be broadly classified as inflammatory, osmotic, or secretory or resulting from altered intestinal motility (ie, rapid transit). Table 1 shows clinical features that are helpful in narrowing the differential diagnosis. Motility-related diarrhea, primarily caused by irritable bowel syndrome, is often manifested by increased stool frequency out of proportion to the daily stool weight and can be accompanied by abdominal pain that is relieved by bowel movements. Patients with this condition rarely have a change in weight or evidence...
Chapter 1—Evaluation of Chronic Diarrhea: Case Studies

Table 1. Classification of Chronic Diarrhea

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Clinical Features</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory</td>
<td>Mucosal and submucosal inflammation</td>
<td>Ulcerative colitis</td>
</tr>
<tr>
<td></td>
<td>Damaged epithelium</td>
<td>Crohn’s disease</td>
</tr>
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<td></td>
<td>In some cases, impaired intestinal absorption and excessive secretion</td>
<td>Radiation enterocolitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Eosinophilic gastroenteritis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infections associated with AIDS</td>
</tr>
<tr>
<td></td>
<td>Fever; abdominal pain, blood and/or leukocytes in stool</td>
<td></td>
</tr>
<tr>
<td>Osmotic</td>
<td>Nonabsorbed or nondigested intraluminal solute</td>
<td>Pancreatic insufficiency</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bacterial overgrowth</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Celiac disease</td>
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<tr>
<td></td>
<td></td>
<td>Lactase deficiency</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Whipple’s disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abetalipoproteinemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Short bowel syndrome</td>
</tr>
<tr>
<td>Secretory</td>
<td>Excessive secretion of electrolytes</td>
<td>Carcinoid syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zollinger-Ellison syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vasoactive intestinal peptide—secreting pancreatic adenomas</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medullary carcinoma of thyroid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Villous adenoma of rectum</td>
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<tr>
<td></td>
<td></td>
<td>Microscopic colitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cholerrheic diarrhea</td>
</tr>
<tr>
<td>Altered intestinal motility</td>
<td>Alternating diarrhea and constipation</td>
<td>Irritable bowel syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fecal impaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neurologic diseases</td>
</tr>
<tr>
<td>Factitious</td>
<td>Self-induced</td>
<td>Laxative abuse</td>
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</tbody>
</table>

FURTHER PRESENTATION OF PATIENT 1

A 24-hour stool collection yields a stool weight of 500 g for patient 1. Further questioning reveals that bowel movements are occasionally bloody and painful. Patient 1 has had episodes of arthralgias and fever as well as a 10-pound weight loss during the past 2 months.

- The most likely cause of patient 1’s diarrhea is which of the following?
  A) Irritable bowel syndrome
  B) IBD
  C) Laxative abuse
  D) Dumping syndrome

The correct answer is B. Patient 1 most likely has IBD. Her systemic complaints make irritable bowel syndrome unlikely, and laxative abuse is less likely given her history. She probably does not have dumping syndrome because she has not had previous surgeries. Given the likelihood of IBD, a referral to a gastroenterologist is the next appropriate step because colonoscopy is often needed for diagnosis.

The 2 major categories of IBD are ulcerative colitis (UC) and Crohn’s disease (CD). UC is more common, with an incidence of 6 to 8 per 100,000 persons compared with 2 per 100,000 for CD. Diagnosis is made by biopsy. Early in the course of CD of the small bowel, diagnosis is difficult. Lesions are sometimes not accessible by endoscopy or routine radiologic studies. In these cases, enteroclysis is indicated. This study involves infusion of barium via a nasoduodenal tube with fluoroscopic observation of the mucosa. Often, biopsy is the only option when the mucosa appears normal on radiologic and endoscopic studies. Collagenous and microscopic colitis (IBD subcategories that are far less frequently encountered) both have normal appearing mucosa but show characteristic microscopic changes on biopsy.

Microscopic colitis is characterized by secretory diarrhea in the presence of mucosa that appears normal on colonoscopy. Biopsy samples reveal intraepithelial lymphocytes and inflammatory infiltrates in the lamina propria. Collagenous colitis is very similar except that in addition to inflammatory infiltrates, subepithelial collagen bands are present on mucosal biopsy.

In cases for which the diagnosis is not apparent from the history and physical examination, a stepwise approach is warranted (Table 3).
### Table 3. Evaluation of Chronic Diarrhea

<table>
<thead>
<tr>
<th>Stage</th>
<th>Test</th>
<th>Assessment for</th>
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<tbody>
<tr>
<td><strong>Stage 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stool studies</strong></td>
<td>Evaluation of stool weight in g/24 hr</td>
<td>To confirm and assess severity of diarrhea</td>
</tr>
<tr>
<td></td>
<td>Evaluation for fecal leukocytes, ova, and parasites (3 times)</td>
<td>Infectious diarrhea</td>
</tr>
<tr>
<td></td>
<td>Clostridium difficile toxin</td>
<td>C. difficile infection</td>
</tr>
<tr>
<td></td>
<td>Evaluation of fat in 72-hr stool sample while patient consuming 100 g fat/24 hr (Sudan black B fat stain can be the initial screen)</td>
<td>Steatorrhea</td>
</tr>
<tr>
<td><strong>Blood studies</strong></td>
<td>Complete blood count and differential</td>
<td>Screen for infection</td>
</tr>
<tr>
<td></td>
<td>Measurement of erythrocyte sedimentation rate, electrolytes, blood urea nitrogen, creatinine, thyroid-stimulating hormone, thyroxine, gastrin</td>
<td>Screen for systemic illness</td>
</tr>
<tr>
<td></td>
<td>Measurement of vasoactive intestinal polypeptide, substance P, calcitonin, and histamine* in patients with diarrhea &gt; 1 L/day, especially if hypokalemia is present</td>
<td>Secretory diarrhea</td>
</tr>
<tr>
<td><strong>Radiologic studies</strong></td>
<td>Plain abdominal radiography</td>
<td>Pancreatic calcification</td>
</tr>
<tr>
<td></td>
<td>High-quality barium studies of the upper gastrointestinal tract, small bowel, and colon</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td><strong>Endoscopic studies</strong></td>
<td>Sigmoidoscopy and biopsy (before a barium study and without hyperosmotic preparation)</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Nutritionist-supervised trial of lactose-free diet</td>
<td>Lactose intolerance</td>
</tr>
<tr>
<td></td>
<td>Urine 5-hydroxyindoleacetic acid assay in patients with skin flushing</td>
<td>Carcinoid tumors</td>
</tr>
<tr>
<td><strong>Stage 2 (if stage 1 does not reveal etiology)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stool studies</strong></td>
<td>Enzyme-linked immunosorbent assay for <em>Giardia</em> antigen</td>
<td>Giardiasis</td>
</tr>
<tr>
<td></td>
<td>Alkalization assay for phenolphthalein</td>
<td>Laxative abuse</td>
</tr>
<tr>
<td></td>
<td>Measurement of fecal sodium, potassium, sulfate, osmolality</td>
<td>Secretory diarrhea</td>
</tr>
<tr>
<td><strong>Urine studies</strong></td>
<td>Thin-layer chromatography for bisacodyl, phenolphthalein, anthraquinones</td>
<td>Laxative abuse</td>
</tr>
<tr>
<td><strong>Radiologic studies</strong></td>
<td>Enteroclysis</td>
<td>Crohn’s disease</td>
</tr>
<tr>
<td></td>
<td>Abdominal computed tomography</td>
<td>Numerous conditions (note that this is mostly a last resort test)</td>
</tr>
<tr>
<td><strong>Endoscopic studies</strong></td>
<td>Colonoscopy and ileoscopy with biopsy</td>
<td>Right-sided colitis, amebiasis, Crohn’s disease, and microscopic/collagenous colitis</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Test of bile acid or other breath test</td>
<td>Bacterial overgrowth</td>
</tr>
</tbody>
</table>

*Assay usually done in a commercial laboratory.

FOLLOW-UP OF PATIENT 1

Patient 1 is diagnosed with Crohn’s disease by colonoscopy and biopsy. She has a moderate response to oral prednisone therapy and undergoes small bowel resection 3 times during the next 15 years because of stricture formation. Eventually, her diarrhea symptoms improve; stool output decrease to 200 g per day and frequency decreases to 4 times per day. At age 60, 25 years after initial presentation, she reports worsening diarrhea. Stool weight is now 750 g per day, decreasing to 200 g after a 24-hour fast.

- Which of the following conditions is LEAST likely to be the cause of the current episode of diarrhea in patient 1?
  A) Recurrence of Crohn’s disease
  B) Short bowel syndrome
  C) Pancreatic insufficiency
  D) Bacterial overgrowth

CROHN’S DISEASE

The correct answer is A. Diarrhea associated with Crohn’s disease is not responsive to fasting, whereas the other conditions listed may improve with a fast. A 24-hour fast is helpful in distinguishing between osmotic and nonosmotic diarrhea. Appropriate intravenous fluids are given to maintain hydration and stool volume is measured. Diarrhea is unaffected by fasting when the etiology is inflammatory or secretory (Table 4).

OSMOTIC DIARRHEA

Osmotic diarrhea occurs when excess intraluminal solutes result in transport of water into the lumen down the osmotic gradient. The excess solutes may be secondary to ingestion of poorly absorbed osmoles (some laxatives) or malabsorption (pancreatic insufficiency, ileal disease, and other conditions; see Table 1 for causes of osmotic diarrhea). The presence of these substances can be confirmed by measuring the osmolar gap. The stool osmolality is measured by standard techniques. The predicted osmolality is estimated as twice the sum of the stool sodium and potassium (2 × [Na + K]). If the difference between the measured and calculated osmolality is greater than 40 mOsm, then the presence of excess osmoles is assumed and the diagnosis of an osmotic diarrhea is made.

III. CASE PATIENT 2

PRESENTATION

Patient 2 is a 65-year-old man with a long history of diabetes, alcohol abuse, and chronic pancreatitis. He presents to his primary care physician with diarrhea and bone pain. Stool is of large volume and malodorous. He has lost 10 lb during the past 3 months despite a good appetite and no alcohol use.

- The most appropriate initial test to confirm malabsorption is evaluation using which of the following?
  A) D-Xylose absorption test
  B) Bentiromide urinary excretion test
  C) Endoscopy with small bowel biopsy
  D) Sudan black B fat stain

STEATORRHEA

The correct answer is D. The most common clinical presentation of malabsorption is steatorrhea, and the most appropriate initial step to confirm suspected malabsorption is a test for stool fat. In steatorrhea, stool is bulky and foul smelling. Contrary to common belief, it rarely floats, but it is difficult to flush. Although the ideal diagnostic test is a 72-hour stool collection for fat while the patient is on a high-fat diet, this is very cumbersome and is rarely performed. More commonly, a 24-hour collection is done. Normal stool fat content is less than 6 g/day, although up to 14 g/day is nonspecific, given the North American diet. A qualitative fat content test, such as the Sudan black B fat stain is very useful because of its simplicity. This test has the advantage of not needing a 24-hour collection and has a rapid turnaround time, but the disadvantage is that it is less sensitive. Steatorrhea is confirmed in patient 2 using the Sudan black B fat stain test.

- On routine laboratory testing, which of the following studies is most likely to be abnormal in patient 2?
  A) Complete blood count
  B) Serum albumin level
  C) Serum calcium level
  D) Prothrombin time
  E) All of the above

MALABSORPTION

The correct answer is E. Clinical manifestations of malabsorption are variable and are caused by deficiencies of various substances. Fat-soluble vitamin deficiency can lead to night-blindness (vitamin A deficiency), osteoporosis and bone pain (vitamin D deficiency), and easy bruising (vitamin K deficiency). Iron, folate, and B-vitamin deficiencies lead to anemia and paresthesias. Protein malabsorption leads to edema and fatigue.

When malabsorption is present, it is first necessary to distinguish between small bowel and pancreatic disorders. The first step is the D-xylose absorption test. The patient is given 25 g of D-xylose, an easily absorbed sugar
requiring no digestion. The serum xylose level is checked in 2 hours (normal value > 30 mg/dL) and 5-hour urine collection is obtained (normal value > 5 g). If the D-xylose test is normal, pancreatic disease is suspected.5

**PANCREATIC INSUFFICIENCY**

Inadequate exocrine function is a hallmark of pancreatic insufficiency, which can result from recurrent episodes of pancreatitis or pancreatic duct obstruction. Affected patients often have malnutrition with preservation of appetite. An abdominal radiograph may show pancreatic calcification. A simple test for pancreatic insufficiency is the bentiromide urinary excretion test. Bentiromide is a synthetic peptide attached to para-aminobenzoic acid (PABA). Chymotrypsin is required to break the bond, and PABA is then absorbed in the small intestine. After ingestion of 500 mg of bentiromide, a 6-hour urinary excretion of arylamine (PABA metabolite) is measured. An excretion of less than 50% is diagnostic of pancreatic insufficiency.5 Because this test requires a functional small bowel, it should be done after a D-xylose test yields normal results.

**BACTERIAL OVERGROWTH**

If the D-xylose test is abnormal, a small bowel etiology for the diarrhea is suspected. Bacterial overgrowth is the most common pathology in this situation. When intestinal motility is affected, bacteria can colonize the normally sterile proximal jejunum. The bacteria affect bile salt metabolism, resulting in diarrhea.6 Common causes of decreased motility include advanced age and diabetes. Many drugs (including opiates and anticholinergics, as well as others) can decrease motility especially in elderly persons. The initial noninvasive screening test is a breath test. In a positive glucose breath hydrogen test, a 50-g load of glucose results in an increase of 12 parts per million in breath hydrogen in 2 hours. The definitive diagnostic test is culturing of polymicrobial flora (> 10^5/mL) from a jejunal aspirate. Alternatively, if bacterial overgrowth is strongly suspected, a 14-day empiric course of antibiotics is a reasonable next step. Three commonly used regimens are (1) amoxicillin-clavulanate monotherapy (875 mg twice daily), (2) cephalexin (250 mg 4 times daily) with metronidazole (250 mg 3 times daily), or (3) trimethoprim-sulfamethoxazole (1 double-strength dose twice daily) with metronidazole (250 mg 3 times daily).9 Note that patients who are allergic to penicillin may also be allergic to cephalexin.

**OTHER CAUSES OF DIARRHEA**

If the cause of a chronic osmotic diarrhea is not made by these studies, more invasive tests are required.

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**Table 4. Functional Classification of Types of Chronic Diarrhea Based on the Response to Fasting**

<table>
<thead>
<tr>
<th>Types or causes responsive to fasting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incontinence</td>
</tr>
<tr>
<td>Bile acid diarrhea</td>
</tr>
<tr>
<td>After cholecystectomy</td>
</tr>
<tr>
<td>After ileal resection</td>
</tr>
<tr>
<td>Steatorrhea</td>
</tr>
<tr>
<td>Osmotic diarrhea</td>
</tr>
<tr>
<td>Carbohydrate malabsorption</td>
</tr>
<tr>
<td>Excessive carbohydrate ingestion</td>
</tr>
<tr>
<td>Laxatives (containing poorly absorbable anions: sodium sulfate, sodium phosphate, or sodium citrate), magnesium</td>
</tr>
<tr>
<td>Food allergy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Types or causes not responsive or only partly responsive to fasting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory bowel diseases</td>
</tr>
<tr>
<td>Celiac disease</td>
</tr>
<tr>
<td>Intestinal lymphoma</td>
</tr>
<tr>
<td>Villous adenoma of the rectosigmoid</td>
</tr>
<tr>
<td>Chronic infections (eg, <em>Mycobacterium tuberculosis, Giardia, amebe</em>)</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
</tr>
<tr>
<td>Congenital diarrhea</td>
</tr>
<tr>
<td>Chloride-bicarbonate exchange deficiency</td>
</tr>
<tr>
<td>Sodium-hydrogen exchange deficiency</td>
</tr>
<tr>
<td>Microvillus inclusion disease</td>
</tr>
<tr>
<td>Bacterial overgrowth</td>
</tr>
<tr>
<td>Neuroendocrine tumors</td>
</tr>
<tr>
<td>Zollinger-Ellison syndrome</td>
</tr>
<tr>
<td>Pancreatic cholera</td>
</tr>
<tr>
<td>Carcinoid tumor</td>
</tr>
<tr>
<td>Medullary carcinoma of the thyroid</td>
</tr>
<tr>
<td>Systemic mastocytosis</td>
</tr>
<tr>
<td>Laxative or diuretic abuse</td>
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</tbody>
</table>


An upper gastrointestinal series with small bowel follow-through may demonstrate mucosal abnormalities, and rapid transit can easily diagnose short bowel syndrome. Endoscopy with biopsy of the abnormal mucosa is necessary to diagnose small bowel disorders (ie, Whipple’s disease and celiac disease).
Celiac disease, or gluten-sensitive enteropathy, is characterized by nonspecific manifestations of abdominal pain, bloating, cramps, and weight loss. Steatorrhea is almost always present. It is not clear whether gluten and its related compound, gliadin, play a toxic or an immunogenic role in the disease. Diagnosis of celiac disease requires (1) the presence of malabsorption and (2) the detection of blunting and flattening of jejunal villi on biopsy. In addition, patients should show improvement on biopsy evaluation or in immunologic parameters when they switch to a gluten-free diet. Immunologic tests such as immunoglobulin A (IgA) anti-gliadin antibody (sensitivity, 80% to 90%; specificity, 85% to 95%) and IgA anti-endomysial antibody (sensitivity, 85% to 98%; specificity, 97% to 100%) are helpful in equivocal cases.

IV. CASE PATIENT 3

PRESENTATION

Patient 3 is a 43-year-old woman who presents with a 9-month history of diarrhea. She reports having bowel movements 3 to 4 times a day that are either watery or formed. The need to defecate sometimes awakens her at night. She has not experienced fevers or weight loss. Laboratory evaluation of serum electrolyte, albumin, and calcium levels as well as prothrombin time are normal. Stool osmolal gap is normal.

- Which of the following conditions is the LEAST likely cause of patient 3’s diarrhea?
  A) Giardiasis
  B) Pancreatic insufficiency
  C) Laxative abuse
  D) Clostridium difficile infection

The correct answer is B. Pancreatic insufficiency is unlikely in a patient with a normal osmolar gap. In patients with normal stool osmolar gap, secretory diarrhea, such as that caused by Giardia or C. difficile, should be suspected. Patients who are abusing laxatives can present with either a normal or an elevated osmolar gap, depending on the agent used.

SECRETORY DIARRHEA

Secretory diarrhea is suspected when fasting does not result in a decreased stool volume (Table 4). In patients with this condition, the osmolar gap is generally absent. The predominant causes of secretory diarrhea are infectious and factitious. Less commonly, celiac disease and bacterial overgrowth are unresponsive to fasting. If chronic secretory diarrhea is of large volume (> 1 L/day) and is associated with electrolyte abnormalities (especially hypokalemia), more esoteric causes such as rare neuroendocrine tumors should be suspected. These tumors can be diagnosed by measuring serum vasoactive intestinal peptides, substance P, histamine, and calcitonin.

FACTITIOUS DIARRHEA

Factitious diarrhea refers to diarrhea that is self-induced. It occurs when patients use laxatives and diuretics inappropriately or excessively, which can increase stool volume and water content. Patients may not realize that they are abusing these agents and provoking their diarrhea. A careful history is required to elicit the appropriate information; the physician must be considerate and sensitive when asking potentially embarrassing questions. Factitious diarrhea can be extremely difficult to diagnose. It accounts for at least 33% of the cases of diarrhea of undetermined origin referred to tertiary care centers. No pathonomic stool appearance or specific laboratory/radiologic study is diagnostic.

Diarrhea can be osmotic or secretory depending on the agent used. Patients can be asymptomatic or present with evidence of dehydration, malabsorption, or electrolyte abnormalities. Magnesium-containing laxatives can be detected by an abnormal osmolar gap (> 40 mOsm/kg) and by increased stool magnesium (> 30 mEq/day). Although some older laxatives containing phenolphthalein would turn red in an alkali environment, most laxatives sold over the counter today will not be detected by stool alkalization. Unfortunately, the only way to confirm factitious diarrhea is to send the stool to a referral laboratory for a laxative survey, which involves examination of stool for specific agents using various methods (eg, liquid chromatography and spectrophotometry).

INFECTIOUS DIARRHEA

Although infectious diarrhea is the most common cause of acute diarrhea, it is an uncommon cause of chronic diarrhea in an immunocompetent host. However, Giardiasis and amebic diarrhea are chronic in nature. Diarrhea caused by C. difficile can have a prolonged course in some patients.

Giardia can be detected by routine examination of the stool, although direct observation has been supplanted by enzyme-linked immunosorbent assay (ELISA) detection of Giardia antigen in the specimen. The sensitivity and specificity of the ELISA method exceed 90%, whereas microscopic detection at best has a sensitivity of 85%.

Amebic diarrhea is more difficult to diagnose, mostly
because of difficulties in collecting the specimen. Stool must be directly collected in a container with fixative. Excess water and urine will decrease the yield. Even with 3 well-collected samples, the sensitivity is only 60% to 90%.

In some cases, the organisms are fortuitously detected during radiologic and endoscopic studies. *C. difficile*-induced diarrhea can be more easily excluded by 3 negative samples for the toxin. In patients who are HIV positive, diarrhea is almost always infectious, and pathogens can be identified in more than 85% of the cases. Pathogens may include bacteria, viruses, protozoa, and others agents. Cryptosporidium, Microsporidia, and *Mycobacterium avium* complex are the most frequently identified organisms in AIDS-related chronic diarrhea.11

**V. SUMMARY**

Although chronic diarrhea may be associated with numerous conditions, diagnosis can be made rapidly and in a cost-effective manner in most cases. The first step is always confirmation of the diarrhea with quantitative measurement of stool mass and water content. If diarrhea is confirmed, the stool should be examined for blood, leukocytes, ova and parasites, and *C. difficile* toxin (Table 3). In the presence of extraintestinal clues, specific tests can rapidly lead to a diagnosis (Table 2). The presence of blood warrants referral to a gastroenterologist for colonoscopy to evaluate for IBD or cancer. Presence of steatorrhea can be detected by the Sudan black B fat stain. Steatorrhea suggests malabsorption; the next step in confirming this diagnosis is the D-xylose test to distinguish pancreatic (negative result) versus small bowel (positive result) pathology. In patients with a positive D-xylose test, the hydrogen breath test should be performed to exclude bacterial overgrowth. The remaining etiologies can be diagnosed using an upper gastrointestinal series with a small bowel follow-through; any abnormalities noted should be further evaluated with endoscopy and biopsy.

**REFERENCES**

8. Talal AH, Murray JA. Acute and chronic diarrhea. How to keep laboratory testing to a minimum. Postgrad Med 1994;96:2–8,35–8,43 passim.
Chapter 2—Dizziness: Four Case Studies

I. INTRODUCTION

Dizziness is one of the most common problems presenting to primary care physicians. This disorder accounts for approximately 1% of visits to U.S. physicians or 7 million office visits per year. Of these patients, 70% are initially seen by general internists or family practitioners. Dizziness can result in considerable disability, restricting the ability of patients to drive or walk and putting them at risk for falls or accidents. It may also be the presenting symptom of a serious or life-threatening disease.

The differential diagnosis of dizziness is quite broad, ranging from fairly benign conditions to life-threatening ones. Kroenke and colleagues did a meta-analysis of studies on the causes of dizziness, quality-adjusted to reflect patients who present to primary care offices (Table 5). According to this study, nearly 50% all cases of dizziness involve the peripheral vestibular system. However, 10% of cases involve the central vestibular system (e.g., stroke or tumor); these cases are the most dangerous and therefore the most important to identify quickly. Psychiatric causes are common particularly among younger patients. Another 25% of cases are caused by “nonvestibular nonpsychiatric” conditions, which involve other organ systems and are often multifactorial. Approximately 13% of all cases go undiagnosed even after a full workup.

Most causes of dizziness can be diagnosed using the history and physical examination alone. However, some caution needs to be used when taking the history because the phrase “I feel dizzy” can mean many different things. Daroff and Martin note that the term “dizziness” usually refers to 1 of 4 basic sensations: (1) faintness (the feeling that you are on the verge of losing consciousness), (2) vertigo (a feeling of moving or spinning), (3) miscellaneous head sensations that are neither faintness nor vertigo, and (4) gait disturbances. Patients themselves may have difficulty describing the sensation, and so they are at risk of allowing the investigator to put words in their mouths even when those words are not entirely accurate. This difficulty is particularly true with elderly patients and can lead to an incorrect diagnosis.

For this reason, Professor Marty Samuels of Harvard, a renowned neurologist, recommends first asking an open-ended question: “What do you mean when you say you feel dizzy?” Only after patients first use their own words to describe the sensation should the investigator then use leading questions to narrow the differential diagnosis. If the diagnosis is still uncertain, several simple provocative maneuvers can be performed in the office to clarify the diagnosis. For example, if a patient’s symptoms are reproduced by spinning in a swivel chair and then rapidly stopping, vertigo is a more likely diagnosis. On the other hand, if symptoms are reproduced by standing, orthostatic hypotension is likely. Symptoms occurring after a period of hyperventilation suggest a psychological cause. Finally, if the patient begins to feel dizzy only when walking across the room, then a gait disturbance is likely.

II. CASE PATIENT 4

PRESENTATION

Patient 4 is a 62-year-old man with type 2 diabetes, hypertension, and benign prostatic hypertrophy (BPH) who presents with dizziness. When asked to describe the sensation, he says, “It’s like the blood rushes to my head, and I feel like I might pass out.” There is a mild sensation of spinning, but mainly he feels “real light-headed.” Although he has never actually lost consciousness, there have been 3 occasions in the past 2 weeks...
where he has felt so faint while walking that he had to sit down. Symptoms are particularly troublesome in the hour or 2 after a meal. He never feels dizzy while lying or sitting, only when standing or walking. His medicines include glipizide, terazosin, hydrochlorothiazide, and long-acting nifedipine.

On physical examination, he is an obese man. His mucous membranes and axillae are moist, and his tongue has no furrows. He has severely reduced sensation in his feet extending to just below his knees. Lying flat, his pulse is 75 bpm and his blood pressure is 142/95 mm Hg. After standing for 1 minute, his pulse is 78 bpm, his blood pressure is 112/80 mm Hg, and his symptoms of dizziness are reproduced. During the episode of dizziness, he has no nystagmus; when he sits down, his symptoms abate.

**Discussion**

Patient 4 has orthostatic hypotension, which by definition is diagnosed when any one of the following occurs with standing: (1) drop in systolic BP of 20 mm Hg or more; (2) drop in diastolic BP of 10 mm Hg or more; (3) symptoms of cerebral hypoperfusion. In addition to causing dizziness, orthostatic hypotension can result in more severe conditions such as syncope, stroke, or myocardial infarction.

In the normal process of standing, up to 1 L of blood can suddenly pool in the veins of the abdominal compartment and lower extremities. This pooling leads to decreased venous return to the heart and decreased left ventricular filling, which in turn leads to decreased cardiac output and decreased blood pressure. Arterial baroreceptors then discharge, leading to compensatory reflexes that increase sympathetic outflow and vasopressin release, and decrease vagal tone. As a result, a normal individual who stands up may undergo a decrease in systolic blood pressure of 5 to 10 mm Hg, an increase in diastolic blood pressure of 5 to 10 mm Hg, and an increase in pulse of 10 to 25 bpm.5

An interruption anywhere in this pathway can lead to orthostatic hypotension. Patient 4, for example, has evidence of peripheral neuropathy (eg, decreased sensation in his feet). It is common for diabetics to have an associated autonomic neuropathy as well, which impairs sympathetic outflow from the central nervous system (CNS) to compensate for standing. Many other medical conditions are associated with autonomic insufficiency, such as Parkinson’s disease, Shy-Drager syndrome, or amyloidosis. In addition, elderly patients, particularly those with hypertension, often experience failure of their arterial baroreceptors, which results in an inadequate baroreceptor-mediated heart rate response to standing.6

| Table 5. Causes of Dizziness in Patients Presenting to Primary Care Clinics |
|---------------------|----------------------|
| Cause               | Prevalence, % |
| Peripheral vestibular | 44 |
| BPPV                | 16 |
| Vestibular neuritis  | 9 |
| Ménière’s disease    | 5 |
| Other               | 14 |
| Central vestibular   | 10 |
| Cerebrovascular      | 6 |
| Tumor               | 0.7 |
| Other               | 3 |
| Psychiatric          | 16 |
| Psychiatric disorder | 11 |
| Hyperventilation     | 5 |
| Nonvestibular nonsymp | 24 |
| Presyncope (eg, OH, arrhythmia) | 6 |
| Dysequilibrium       | 5 |
| Other               | 13 |
| Unknown             | 13 |

BPPV = benign paroxysmal positional vertigo; OH = orthostatic hypotension.


The most common causes of orthostatic hypotension are hypovolemia (secondary to overdiuresis, hemorrhage, hypoaldosteronism, adrenal failure, etc.) and autonomic failure (either primary or secondary to diseases such as diabetes, parkinsonism, or baroreceptor failure).7 The postprandial state can cause increased blood pooling in the splanchnic circulation and can therefore exacerbate orthostatic hypotension. Postprandial hypotension should always be considered in older patients who become dizzy or faint after meals. In one study, 12% of functionally independent elderly patients had symptomatic orthostatic hypotension by tilt table testing, but the percentage increased to 22% after ingestion of a standardized meal.8

Orthostatic hypotension is diagnosed by physical examination, specifically by the orthostatic vital signs; however, these signs must be taken properly. Vital signs should be taken in the supine and standing (not sitting) positions. When the patient becomes supine, the investigator should wait for 2 minutes before taking the vital
signs, and, after the patient stands, the investigator should wait 1 minute. If taken sooner the patient’s vital signs may not have stabilized after the change in position, and may turn out to be falsely high or low.

The first condition that should be considered is hypovolemia; assessment can be accomplished through the physical examination. In a meta-analysis of the physical signs of hypovolemia, McGee and colleagues concluded that the most helpful physical signs for hypovolemia caused by acute blood loss are severe postural dizziness (preventing measurement of upright vital signs) and an increase in the pulse rate of 30 bpm or more with standing (97% sensitivity, 98% specificity). For hypovolemia caused by vomiting, diarrhea, or decreased oral intake, the most helpful sign is a dry axilla (positive likelihood ratio, 2.8). Moist mucous membranes and a tongue without furrows are the most helpful negative signs (negative likelihood ratio, 0.3 for both). The commonly used signs of capillary refill time and poor skin turgor have no proven diagnostic value.9

Laboratory values that could be used to confirm the physical examination include sodium and blood urine nitrogen (BUN)/creatinine ratio.

Once hypovolemia has been excluded, all medications should be reviewed. Classes of drugs that commonly cause orthostatic hypotension include antihypertensives, antianginals, antidepressants, antiparkinsonians, diuretics, oral antihypoglycemics, and alcohol. If drugs in these classes can be discontinued, the patient’s orthostatic hypotension may be easily reversible.

Antihypertensive drugs are one of the most common causes of orthostatic hypotension and one of the most difficult to discontinue outright. Some changes may be possible within a patient’s regimen, however. α-Blockers, which are used to treat BPH and hypertension simultaneously in elderly men, are a common cause of orthostatic hypotension. Although all α-blockers appear to have equal efficacy in improving symptoms and flow in BPH, tamsulosin (Flomax) and alfuzosin appear to cause less orthostatic hypotension and dizziness than doxazosin (Cardura), terazosin (Hytrin), and prazosin.10 This difference may be because the latter are nonspecific α-1 antagonists, whereas the former have more specificity for α-receptors in the prostate and therefore fewer systemic side effects. Switching to a drug in an entirely different class such as finasteride (or to proven herbal medicines such as saw palmetto) is another option for patients with BPH and orthostatic hypotension.

Although any antihypertensive drug has the potential to cause orthostatic hypotension, some are more consistently associated with hypotension than others. Luukinen and colleagues11 found that only diuretics and calcium channel blockers were associated with orthostatic hypotension in elderly persons. In a double-blinded crossover study of enalapril versus long-acting nifedipine, Slavachevsky and colleagues12 found that although both drugs were equipotent in decreasing supine blood pressure, only nifedipine increased the orthostatic decline in systolic blood pressure. Enalapril actually decreased the number of orthostatic episodes relative to placebo and thus is a better choice for patients with orthostatic hypotension.12 Adjusting an individual patient’s antihypertensive regimen in the face of orthostatic hypotension may be a matter of trial and error. Based on these studies, however, a good initial strategy might be to switch away from diuretics and calcium channel blockers (especially nifedipine) and toward angiotensin-converting enzyme inhibitors.

**FURTHER PRESENTATION OF CASE PATIENT 4**

Patient 4 does not appear hypovolemic by physical examination. The decision is made to discontinue his hydrochlorothiazide, nifedipine, and terazosin and to begin enalapril and finasteride. Patient 4 returns 4 weeks later reporting that his symptoms have improved somewhat, but he still experiences dizziness with standing and walking. When vital signs are taken in the office, he is still found to be orthostatic with no change in pulse rate.

**Discussion**

Patient 4 likely has autonomic insufficiency because of his diabetes, which continues to cause orthostatic hypotension even after his medical regimen has been adjusted. At this point, it is time to consider specific treatment for his orthostatic hypotension. Several nonpharmacologic methods can be attempted first. For example, the patient should be educated not to stand suddenly after waking or eating. Custom-fitted elastic stockings can decrease venous pooling in the lower extremities. A program of daily exercise, too, can increase tone in the lower extremities and aid venous return to the heart. Raising the head of the bed at night by 10 to 20 degrees can decrease renal perfusion, thus increasing the renin-angiotensin system and decreasing nocturnal diuresis.

The first-line drug for treating orthostatic hypotension is fludrocortisone, an adrenal corticosteroid with predominantly mineralocorticoid activity. Its efficacy in orthostatic hypotension is believed to come through an increase in blood volume and possible increased norepinephrine release from sympathetic neurons. Common
side effects include exacerbation of hypertension, diabetes, and congestive heart failure, and thus it must be used with caution in patients with these conditions. The starting dose of fludrocortisone is 0.1 mg once daily, and it can be titrated upward to a maximum of 1 mg daily. Electrolytes should be monitored regularly because fludrocortisone can cause hypokalemia.

If fludrocortisone is contraindicated or ineffective, the second-line agent is midodrine, an \( \alpha \)-1 agonist. Its side effects include exacerbation of hypertension and urinary retention, as well as dry mouth and bradycardia. The usual dose is 10 mg three times a day (TID). Other drugs that have been shown to be effective include other \( \alpha \)-1 agonists (such as phenylephrine), nonspecific sympathomimetics (such as ephedrine and pseudoephedrine), and amphetamines (such as methylphenidate). Third-line agents include nonsteroidal anti-inflammatory drugs (NSAIDs), caffeine, somatostatin, yohimbine, clonidine, and vasopressin.5

**CASE PATIENT 4 FOLLOW-UP**

The decision is made to avoid adding further pharmaceutical agents to patient 4’s regimen because of the concern of aggravating his other medical problems. Patient 4 is educated on the value of rising slowly (ie, slowly moving into a standing position) after eating and waking to avoid orthostatic hypotension. He is fitted for elastic stockings and begins a program of exercise that does not involve standing or walking (eg, stationary bicycle, rowing machine, weight training). His enalapril is titrated upward to control his hypertension and to protect his kidneys. Four weeks later he returns to the office and states that his symptoms are improved.

**III. CASE PATIENT 5**

**PRESENTATION**

Patient 5 has no pertinent medical history and takes no medicines beyond an occasional aspirin. On examination, she is fit. Blood pressure is 135/85 mm Hg and pulse is 80 bpm, with no substantial orthostatic changes. A careful neurologic examination—including cranial nerves, motor, sensory, cerebellar, and deep tendon reflexes—is normal.

A Hallpike maneuver is performed. In the head-hanging right position, patient 5 feels normal. After about 5 seconds in the head-hanging left position, however, she begins to feel the same severe dizziness she felt previously. During this episode she is observed to have torsional, mainly horizontal nystagmus with the slow phase toward the left ear and the fast phase toward the right (so that it appears to be “beating” toward the right). When she looks to the right side, the nystagmus decreases. When she looks to the left, the nystagmus decreases but does not reverse direction. Her dizziness and nystagmus subside after 30 seconds. The Hallpike maneuver is repeated 3 times. The nystagmus and dizziness recur each time but become progressively less intense and shorter in duration.

**Discussion**

Patient 5 has benign paroxysmal positional vertigo (BPPV). This condition is the most common cause of dizziness presenting to primary care offices (Table 5). Women are affected slightly more than men are by a 1.6:1 ratio. Peak age of presentation is in the sixth decade (54 years). In most cases, there is no precipitating factor, although in some cases the episode of BPPV is preceded by head trauma, viral labyrinthitis, or another form of injury. The classic symptoms of BPPV are intense vertigo and nystagmus occurring in response to head motion, such as turning over in bed, getting out of bed, or reaching for something on a high shelf. There is typically a short latency period of several seconds between the head motion and the onset of vertigo. When vertigo does occur, it lasts 30 seconds or less.

The key to diagnosis is the Hallpike maneuver. To perform this, the patient should be seated upright on an examining table. With the physician behind, the patient is then moved quickly to a supine position, with her head hanging over the end of the examination table. The patient’s head is then rotated 45 degrees to the right (the “head-hanging right” position). Recall that there is an average of 5 seconds latency to the onset of nystagmus; therefore, the patient should be held in that position for 20 or 30 seconds with the physician observing her eyes. The patient should be asked whether or not her vertigo symptoms are reproduced. If there is no
vertigo or nystagmus in the head-hanging right position, the maneuver should be repeated with the head rotated 45 degrees to the left (“head-hanging left” position). When the symptoms are elicited, the ear that is closest to the floor is the one that is affected. Knowing which ear is affected is critical to the success of treatment. The nystagmus is typically torsional, beating upward and away from the undermost ear. With repeated Hallpike maneuvers, there is a lessening of the response. This is known as “fatiguing.” When the patient sits up again, she will often experience a recurrence of vertigo, with nystagmus in the opposite direction. Baloh and colleagues performed a study of 240 patients with BPPV, and found this stereotypical pattern in 81% of patients. BPPV is an example of the form of dizziness known as vertigo, which is a sensation of movement associated with a dysfunction of the vestibular system. Vertigo is commonly thought of as a “spinning” sensation; however, it can also present as a feeling of motion, tilting, or even a nonspecific lightheadedness. Other forms of dizziness may also be associated with a feeling of spinning; therefore, spinning and vertigo should not be considered synonymous. More reliable for diagnosis are other elements of the history: the time course, exacerbating factors, and risk factors. Table 6 shows a classification of various causes based on the duration of the sensation of vertigo.

To understand the various causes of vertigo, it is necessary to understand the basics of the vestibular system. The peripheral vestibular system consists of the vestibular labyrinth, which is made up of the otolithic organs (the saccule and utricle) and the semicircular canals. The otolithic organs contain small granules or “otoliths,” moving in a watery fluid called endolymph. The positions of these otoliths produce the sensations of linear acceleration (in the saccule) and gravity (in the utricle). Movement of endolymph in the 3 semicircular canals produces the sensation of angular acceleration. These sensations are carried via the vestibular nerve (a component of cranial nerve VIII) to the CNS where they are routed to the cerebellum and the vestibular nuclei in the brainstem. The vestibular neurons have a high tonic spontaneous firing rate, with the semicircular canals on the right and left sides of the head firing in opposite polarities. When the head turns in one plane, it causes increased firing from one semicircular canal and decreased firing from the opposite one. This input is integrated in the CNS to produce the sensation of the head turning. It also controls the vestibulo-ocular reflex, which permits the eyes to remain fixed smoothly on an object as the head turns.

A lesion anywhere along the path of the vestibular system produces a loss of the baseline nerve impulses from that side of the head; however, the undamaged side continues to fire at its normal rate. This asymmetry will be interpreted by the brain as motion, producing the sensation of vertigo. The vestibulo-ocular reflex attempts to compensate for the perceived motion, resulting in nystagmus. The current leading theory for BPPV is that it is caused by displaced otoliths or otolithic debris, which has made its way from the utricle into the semicircular canals. Such debris deposits in the most dependent portion of the labyrinth, which is the long arm of the posterior semicircular canal. This debris is then stirred up by any motion that moves the posterior semicircular canal.
canal with respect to gravity, such as looking up, rising from bed, or bending forward. The motion of the debris produces an abnormal movement of the endolymph in the posterior semicircular canal on that side of the head, resulting in asymmetric neural firing and causing the abnormal sensation of vertigo and nystagmus. Autopsy evidence supports this model of BPPV. Researchers have found basophilic deposits on the cupulae of the posterior semicircular canals of patients with BPPV who died from other causes. These deposits were present only on the side that was undermost when the BPPV was induced. Other evidence to support this model is the fact that the torsional nystagmus observed in BPPV is always in the plane of the posterior semicircular canal.13

TREATMENT

This understanding of BPPV has led to an ingenious and very effective treatment that can be performed at the bedside or in the primary care office and that can easily produce a cure. It is known as “the particle repositioning (or “Epley”) maneuver,” after its inventor, Dr. J. M. Epley.16 Using knowledge of the anatomy of the semicircular canals, the goal of the maneuver is to move the debris out of the posterior semicircular canal and back into the utricle where it belongs.

The patient should be pretreated with a scopolamine patch on the night before the repositioning maneuver is to occur. This pretreatment minimizes the patient’s subjective sense of vertigo. During the procedure, the physician should hold an 80 Hz oscillating device in one hand, and place this hand against the patient’s mastoid process ipsilateral to the affected ear. The vibrations from this oscillator prevent the otolithic debris from becoming hung up on the walls of the semicircular canal.

**Figure 1** shows the series of maneuvers that must be performed when the left ear is affected. The head should be positioned on the opposite side for the patient whose right ear is affected, as indicated. The patient’s head should be held in each position for at least 30 seconds, to allow the otolithic debris to settle out of that section of canal. As the maneuvers are being done, the patient’s eyes should be observed for nystagmus. The presence of nystagmus indicates that the debris is still in motion, and thus the patient’s head should not be moved to the next position until nystagmus ceases.

When the series of positions is completed, they should be repeated until there is no nystagmus in any position or until there is no progress in 2 successive cycles. The patient should avoid head-back and head-forward positions for 48 hours. If the BPPV is not cured, the repositioning maneuver can be repeated at weekly intervals until it is successful. Using this maneuver on a series of 400 patients at the Portland Otologic Clinic, Dr. Epley reports complete resolution of symptoms in 95% of patients, with 80% success after a single session.16

The particle repositioning maneuver, performed by a properly trained physician, is now the standard of care for treating BPPV. Vestibular suppressing drugs, such as meclizine, have not been shown to be effective on such short-lasting episodes of vertigo, and should not be used in patients with BPPV. Even without any treatment, BPPV is considered a self-limited condition and typically resolves within 3 months.1 For the rare patients in whom the particle repositioning maneuver is ineffective and in whom symptoms are incapacitating, surgical options exist for effecting a cure.17

### IV. CASE PATIENT 6

**PRESENTATION**

Patient 6 is a 64-year-old man with a long-standing history of poorly controlled hypertension and hyperlipidemia who presents with severe dizziness, nausea, and vomiting that began abruptly about 3 hours ago. He is leaning heavily against his son as he walks. He describes the sensation as “like I’m spinning around and around,” and he feels that if he tried to walk on his own he would fall. He has had 3 similar episodes during the past 6 months; however, those resolved after 20 to 30 minutes, and he neglected to seek medical treatment because “I didn’t want to bother you.”

His medications include metoprolol and simvastatin, but his prescriptions ran out 2 months ago and he has not yet refilled them. He has smoked a pack of cigarettes per day for 50 years.

His blood pressure currently is 190/100 mm Hg. He has a left-sided facial droop and an asymmetric smile. Hearing is markedly decreased in his right ear. He has diminished pain and temperature sensation on the right side of his face. He has nystagmus with the slow phase toward the right and the fast phase toward the left (“beating left”). When he looks to the left side, the nystagmus increases; however, when he looks toward the right the nystagmus reverses direction and appears to be beating right. When he attempts to fix his vision on one of the lights in the room, the nystagmus is unaffected. Strength and sensation are intact in his arms and legs. When he attempts to touch his finger to his
Figure 1. The particle repositioning (or “Epley”) maneuver for treating benign paroxysmal positional vertigo. Positioning sequence for presumed debris in the left posterior semicircular canal as viewed by the operator who is behind the patient. (Box) Exposed view of the labyrinth, showing migration of particles (arrow). (A) Patient is seated and oscillator is applied. The operator should pause at each position (B–F) as shown until the induced nystagmus approaches termination or for at least 30 seconds if there is no nystagmus. (B) Place head over end of table, positioned 45 degrees to the left. (For debris in the right canal, the head should be on the right). (C) Keeping the head tilted downward, rotate the head 45 degrees to the right. (For debris in the right canal, the head should be on the left). (D) Rotate the head and body until facing downward at 135 degrees from the supine position. (E) Keeping the head turned to the right, bring the patient into a sitting position. (F) Turn the head forward, with the chin down 20 degrees. Keep repeating the entire series (A–F) until there is no nystagmus in any position. Lat = lateral; Post = posterior; Sup = superior. Adapted with permission from Epley JM. Particle repositioning for benign paroxysmal positional vertigo. Otolaryngol Clin North Am 1996;29:323–32.
nose, however, he is able to do so only with great difficulty. Romberg and gait cannot be tested because he cannot stand on his own. Patient 6 is sent to the hospital for emergent magnetic resonance imaging (MRI) of his head.

**DISCUSSION**

Patient 6 has an acute vertebrobasilar stroke. The vertebrobasilar system accounts for 20% of cerebral blood flow, and reduced perfusion in this system is the major cause of central vestibular disorders. These disorders are typically seen in patients in the fifth to ninth decades of life who have risk factors such as hypertension, diabetes, hyperlipidemia, smoking, and known cerebrovascular or cardiovascular disease. The stroke is often preceded months or weeks beforehand by transient ischemic attacks (TIAs), lasting less than 30 minutes. Vertigo is abrupt in onset and is accompanied by postural instability, nausea/vomiting, and nystagmus. The intense vertigo accompanying a vertebrobasilar vascular event can last from days to weeks.

Another condition that can present with a similar time course and onset is vestibular neuronitis, also called labyrinthitis (Table 6). Vestibular neuronitis is a peripheral cause of vertigo that in most cases is probably precipitated by viral infection of the vestibular nerve. Vestibular neuronitis is responsible for 9% of cases of dizziness presenting in the primary care setting, whereas vertebrobasilar cerebrovascular accident accounts for about 6% (Table 5). It is important to differentiate these 2 conditions because a vertebrobasilar stroke can result in edema of the cerebellum and brainstem (a potentially life-threatening state), whereas vestibular neuronitis is commonly self-limited.

**DIAGNOSIS**

Clues in the history and physical examination can help with the diagnosis. A patient with vertebrobasilar insufficiency usually has risk factors for stroke, and may report having previous intermittent TIAs. Typically, other abnormalities are apparent in the neurologic examination, such as facial weakness, sensory defects, diplopia, Horner’s syndrome, or ataxia. In patients with vestibular neuronitis, these neurologic defects are not present. Rather, vestibular neuronitis is preceded in 50% of cases by an upper respiratory infection. It frequently occurs in epidemics (affecting multiple members of the same family) and is seen most commonly in the spring and summer months.

The pattern of nystagmus also differs in these 2 conditions because vertebrobasilar insufficiency affects the CNS, whereas vestibular neuronitis affects the peripheral nervous system (Table 7). With vestibular neuronitis, the nystagmus is always in the same direction, no matter what the direction of gaze. It is never vertical, and it is inhibited if the patient fixes his or her gaze on an object in the office. The nystagmus associated with vertebrobasilar insufficiency, on the other hand, may change direction when the direction of gaze is altered, it may be vertical, and it will not be suppressed by visual fixation.

If the clinical picture is consistent with a stroke, some form of head imaging should be ordered immediately to rule out intracerebral hemorrhage. Laboratory tests should include complete blood count, electrolytes, BUN/creatinine, glucose, prothrombin time/partial thromboplastin time, liver function tests, toxicity screen, and human chorionic gonadotropin (in women of childbearing age).
**TREATMENT**

If the patient presents within 3 hours of the onset of symptoms and no contraindications are present, administration of thrombolytic agents (eg, tissue–plasminogen activator) can be considered for thrombotic strokes. Unfractionated heparin or low-molecular-weight heparin are options if the patient presents outside the 3-hour window for use of thrombolytics. However, their benefit is questionable, and their use increases the risk of intracerebral bleeding.

Aspirin, on the other hand, has solid evidence showing a benefit if given within 48 hours of the onset of a stroke. The Fifth American College of Chest Physicians (ACCP) consensus conference recommended that aspirin (160 or 325 mg/day) be given to all patients with ischemic stroke who are not receiving tissue–plasminogen activator, intravenous heparin, or oral anticoagulation. Aspirin can be given together with low-molecular-weight heparin. Ticlopidine (250 mg twice daily) and clopidogrel (75 mg/day) are alternatives for patients who cannot tolerate aspirin.19

Blood pressure is typically high in patients presenting with an acute stroke. This increased pressure is necessary to maintain cerebral perfusion; therefore, blood pressure should not be lowered unless systolic BP is more than 220 mm Hg or diastolic BP is more than 120 mm Hg. Often, the hypertension spontaneously improves with no treatment.

Intense vertigo, nausea, and vomiting typically accompany an acute vertebrobasilar stroke. This occurs because, as with BPPV, there is an imbalance in the vestibular input to the CNS and the brain interprets any imbalance as angular motion. Although damage to the brain may be permanent, the sensation of vertigo is not permanent. During the process of recovery, the brain goes through 2 distinct phases where it learns to compensate for the unilateral lesion by setting a new baseline for perception of motion.

The first phase is known as acute compensation, and it occurs in the first week or so after the event. The cerebellum and the vestibular nuclei undergo subtle neurochemical changes to minimize side-to-side discrepancies. Until this is accomplished, the patient may experience days or weeks of continuous severe vertigo.20 It is during this phase that agents to suppress the vestibular system are most helpful to minimize dizziness. Effective vestibular suppressants include meclizine (25 mg TID), diazepam (2 to 5 mg TID), or promethazine (25 to 50 mg every 8 hours). When nausea and vomiting are so severe that oral agents are not tolerated, options include intramuscular injections of diazepam or droperidol, as well as prochlorperazine per rectum.21

The second phase of recovery from vertebrobasilar stroke is known as chronic compensation. During this period, the worst of the vertigo is gone; however, the patient continues to have considerable movement-induced dizziness because the damaged system is not able to respond properly to head motion. To avoid this feeling, patients in this phase tend to avoid moving the head and want to continue using vestibular suppressing agents. However, these tendencies should be discouraged because both inactivity and vestibular suppressants tend to delay the process of chronic compensation. Vestibular suppressing agents should only be used in the first week or 2 after the cerebrovascular accident, then they should be tapered off. Furthermore, the patient should actually be encouraged to do exercises that exacerbate the vertigo, such as walking, turning, and bending over. Such head motions allow the CNS to reestablish its baseline in the face of the new lesion, and thus they enhance the central compensation process.

If the patient is still experiencing substantial motion-provoked vertigo after several weeks of such an active program, he or she should be referred to a physical therapist for vestibular rehabilitation. Studies have shown substantial advantages for vestibular rehabilitation compared with meclizine in terms of improving balance in patients who have had a unilateral vestibular lesion.20

**V. CASE PATIENT 7**

**PRESENTATION**

Patient 7 is a 72-year-old woman with Parkinson’s disease and bilateral cataracts who presents with dizziness that she says has been getting worse for several months. She describes this as a “lightheaded” feeling, occurring mainly when she walks. She often feels as if she is on the verge of losing her balance. However, she does not feel like she is close to losing consciousness, and she has no sense that the room is spinning. Symptoms are worse in the evenings. Her biggest worry is that she may fall as a result of her unsteadiness. This fear is so great that she rarely goes out of her house.

Other medical conditions include mild hypertension, glaucoma, arthritis, hypothyroidism, and depression. Her medications include trihexyphenidyl for Parkinson’s disease, an estrogen/progesterone combination, citalopram, celecoxib, thryoxine, betaxolol eye drops, hydrochlorothiazide, a multivitamin, ranitidine, amiodipine, loratadine, atorvastatin, and aspirin.

On examination, she is a thin, elderly woman with a mild resting “pill rolling” tremor in her hands.
When she stands, her blood pressure decreases from 132/85 mm Hg with a pulse of 75 bpm down to 120/90 mm Hg with a pulse of 80 bpm. Cataracts are visible in her eyes. Her visual acuity is 20/100 in the right eye and 20/200 in the left. She has cogwheel rigidity in her arms, and she has no vibration sense in her feet. When she walks, she takes short shuffling steps and requires 5 steps to make a 180-degree turn. She loses balance during the Romberg test. When her unsteady feeling occurs during walking, she has no nystagmus.

Discussion

Patient 7 has dysequilibrium, a disorder characterized by a feeling of imbalance while walking, without a feeling of movement or of impending loss of consciousness. Patients typically do not report dizziness while sitting or lying down. It is not caused by a single clinical entity but is caused by a combination of problems such as peripheral neuropathy, visual impairment, musculoskeletal problems, gait difficulties, cervical spondylosis, and vestibular dysfunction. Aging is significantly associated with onset of dysequilibrium because the ability of the nervous system to process sensory inputs and to control postural reflexes declines with age.

Many elderly patients lose proprioception in their feet as a result of vitamin B12 deficiency or other means. This loss makes them less aware of where their feet are in space, which impairs balance and puts them at risk for tripping. A good question to ask to assess this loss is, “Do you have to look at your feet when you walk or when you go down stairs?” Testing vibration sense in the feet can give an idea of a patient’s adequacy of proprioception because vibration and position sense are carried together in the same neurons.

Over the years, many patients have learned to compensate for decreased proprioception by using their eyes. When asked to stand with their feet together and close their eyes (the Romberg test), these patients may suddenly lose balance as their visual compensation is removed. Conditions that cause visual impairment (ie, cataracts, glaucoma, and macular degeneration) can have a similar effect. Many patients have dysequilibrium symptoms, particularly at night, because dim light further reduces their ability to employ visual compensation.

Parkinson’s disease is an especially common contributor to dysequilibrium for several reasons. Parkinson’s patients typically have impaired postural reflexes. The short, shuffling steps characteristic of Parkinson’s disease make patients prone to tripping and do not provide a stable base for standing. Patients tend to have an unpredictable gait, with sudden speeding up and freezing episodes. As a result of these factors, approximately 50% of Parkinson’s patients experience falls.

As if the problems of aging were not enough, elderly patients are typically taking many medications, with poorly understood side effects and interactions. Taking more than 5 medicines is an independent risk factor for dizziness, as are other common problems of elderly persons such as anxiety, depression, and impaired hearing. Antidepressants and anticholinergic drugs in particular can exacerbate the feeling of imbalance. As a result, when treating dysequilibrium, it is likely to be more helpful to discontinue medicines from the patient’s list rather than add to them.

TREATMENT

The diagnosis of dysequilibrium is made chiefly on the basis of the history, a careful observation of gait, and the neurologic examination. The causes of dysequilibrium are multiple; therefore, the treatment for it must focus on multiple interacting factors.

Because visual impairment can be a major contributor to dysequilibrium, every effort should be made to improve the patient’s vision. Proper treatment of glaucoma, removal of cataracts, and even something as simple as an updated eyeglass prescription can make a big difference. Patients should be encouraged to increase the lighting in their houses and to leave night lights on while they sleep. Bifocal glasses should not be worn by patients while they are walking. Use of hearing aids when necessary can also increase sensory input and help patients compensate for other deficiencies.

Physical therapy to improve balance, posture, and gait can also be helpful. Many elderly patients, especially those with dizziness, become deconditioned. Weakness in the ankles and lower extremities can exacerbate imbalance and increase the risk of falls. Physical therapy can address this issue as well as providing the patient with exercises to improve proprioception. Patients should be encouraged to wear supportive but thin-soled shoes to improve sensory input.

A home visit by a younger relative or by a physical or occupational therapist can identify risk factors for falls such as loose rugs or electrical cords. Most patients with dysequilibrium can walk smoothly when holding onto furniture or bannisters; therefore, the home should be modified when necessary.

The role of the physician is to trim unnecessary medications from the patient’s list while maximizing treatment of those conditions that are amenable to it. For example, if Parkinson’s disease symptoms are increasing enough to exacerbate dysequilibrium, switching to carbidopa/levodopa may be advisable.
rather than continuing with less effective initial medications. In addition to treating all the conditions that can lead to falls, it is important not to forget to diagnose and treat osteoporosis. Many elderly patients experience falls despite the best efforts of their caretakers; however, the stronger their bones are, the less likely it is that they will sustain a crippling fracture.

**CASE PATIENT 7 FOLLOW-UP**

The treatment regimen for Parkinson’s disease in patient 7 is revised by substituting carbidopa/levodopa for trihexyphenidyl. Ranitidine, loratadine, and amlodipine are discontinued. After a DEXA scan reveals osteoporosis, patient 7 is started on calcium with vitamin D and alendronate. She also is referred for an ophthalmology consultation, and her cataracts are removed. A physical therapist designs an exercise program for her and gives patient 7 suggestions for safety improvements after a home visit. After 3 months, patient 7 returns for a follow-up visit and reports that her symptoms have greatly improved.

**VI. CONCLUSION**

Dizziness is one of the most common symptoms of patients presenting to internal medicine clinics and emergency departments. It is important to quickly identify those causes of dizziness that present an immediate threat to the patient’s life or health. By allowing patients to describe the dizziness first in their own words and then narrowing the differential diagnosis with a rational history and physical examination, most cases of dizziness can be diagnosed and effectively treated without recourse to costly tests or specialist referrals.

**VII. SUMMARY POINTS**

- Allow patients to define dizziness in their own words before asking specific questions.

**ORTHOSTATIC HYPOTENSION**

- Orthostasis is diagnosed when any one of the following occurs with standing: (1) decrease in systolic blood pressure of 20 mm Hg or more, (2) decrease in diastolic blood pressure of 10 mm Hg or more, or (3) symptoms of cerebral hypoperfusion.
- The most common causes are hypovolemia, autonomic failure, and pharmaceutical agents.
- The first-line therapy is fludrocortisone; midodrine is second-line therapy.

**BENIGN PAROXYSMAL POSITIONAL VERTIGO**

- Symptoms include transient (< 30 s) vertigo occurring when changing position of the head with respect to gravity.
- Diagnosis is by the Hallpike maneuver.
- Current understanding is that this condition is caused by otolithic debris in the posterior semicircular canal.
- First-line treatment is the particle repositioning (“Epley”) maneuver.

**VERTEBROBASILAR STROKE**

- Symptoms include abrupt onset of vertigo, nausea, vomiting, nystagmus, and positional instability.
- It can be differentiated from vestibular neuronitis by the pattern of nystagmus and the presence of other neurologic defects.
- Treatment includes thrombolytic agents if patients present within 3 hours of their stroke or antiplatelet agents otherwise.
- Meclizine is helpful in the first week or two after the stroke but should be discontinued thereafter to avoid delaying chronic compensation.

**DYSEQUILIBRIUM**

- It is characterized by a sensation of imbalance while walking.
- It is caused by a combination of problems associated with aging, such as peripheral neuropathy, visual impairment, musculoskeletal problems, gait difficulties, and vestibular dysfunction.
- Treatment includes discontinuing unnecessary medicines, improving the patient’s vision, and addressing the individual conditions contributing to the gait disturbance.

**REFERENCES**

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