

Fifth Edition

First Principles of Gastroenterology

The Basis of Disease and an Approach to Management

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JANSSEN-ORTHO

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Common Symptoms and Signs in Gastroenterology

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1. INTRODUCTION / W.G. Thompson and C. Dubé

The key to accurate diagnosis and effective management of gastrointestinal problems is flawless history-taking. Since up to 50% of gastrointestinal disorders are associated with no anatomical change, no physical findings and no positive test result, diagnosis and therapy must often be based on the medical interview. The gastrointestinal history must include an accurate description of the symptom itself, of its relationship with food ingestion and defecation, a personal and familial history of gastrointestinal disorders, treatment or surgery and a meticulous search for symptoms that might suggest organic disease. Finally, the physician should assess the patient's psychosocial state, with particular attention directed toward traumatic events or concerns associated with the onset of his or her complaints.

The physician should determine the time of onset of the symptom, its occurrence in the past, its periodicity, its location and radiation if appropriate, its aggravating and relieving factors, and its relationship to dietary as well as other symptoms. A review of past history should include not only any previous gastrointestinal surgery or diseases, but also systemic illnesses (such as diabetes or severe cardiovascular disease) that might affect the gut. One should pay particular attention to "alarm" symptoms such as gastrointestinal hemorrhage, profound weight loss, voluminous diarrhea or episodes of extreme abdominal pain, which might indicate organic disease. Similarly,

such phenomena as anemia, fever or incapacity to work may indicate a more serious gastrointestinal disorder demanding investigation, treatment and follow-up. A family history of peptic ulcer disease, gastric or colonic cancer, celiac disease, or liver disease may provide clues to the diagnosis as well as indicate the need to screen for such entities as *H. pylori* infection, celiac disease, colonic neoplasm or hemochromatosis

When considering a gastrointestinal complaint, the astute physician cannot ignore the patient's psyche. Many studies establish that those who bring gastrointestinal complaints to a physician, even if they are organic in nature, frequently have psychosocial disabilities. Failure to identify and manage the patient's reaction to his or her psychosocial environment (whether it be hostility toward a spouse, an abnormal fear of cancer or a profound loss) may lead to an unsatisfactory therapeutic outcome.

The following is a synopsis of the common gastrointestinal symptoms. These notes include a description of the symptom itself, a word about how the symptom is generated, the important historical features and associated physical findings, and a brief approach to diagnosis and management. These serve as introductory comments; greater detail can be found throughout the text in discussions of specific diseases. The final section of this chapter presents a sequential approach to the examination of the abdomen.

2. GLOBUS / C. Dubé and W.G. Thompson

2.1 Synonyms

Globus hystericus; globus pharyngeus; lump in the throat.

2.2 Description

Globus sensation is defined as the persistent or intermittent sensation of a lump or foreign body in the throat. This symptom is not associated with food intake: patients with globus can usually swallow meals normally but feel an inability to swallow their saliva between meals.

2.3 Epidemiology

Globus sensation is found to occur at least once in up to half of the general population, usually during an emotional event.

2.4 Etiology

The etiology of globus is unknown but it has been associated with stress, psychologic and psychiatric disorders; an association with either upper esophageal sphincter dysfunction, esophageal dysmotility and gastroesophageal reflux disease has also been suggested, although inconsistently observed.

2.5 Differential Diagnosis and Management

It is important to distinguish globus sensation from dysphagia or odynophagia by careful history-taking. Investigations of patients with globus should be geared at ruling out a pathology of the ear, nose and throat (ENT) spherules (e.g., oropharyngeal carcinoma), pathologic gastroesophageal reflux or esophageal motility disorder, especially achalasia. In this regard, a complete physical and otolaryngological examination should be performed, as well as a barium swallow with a solid bolus (such as bread or a barium tablet) to exclude a mechanical problem and to look for an obvious, underlying motility disorder. Once confirmed by the absence of any organic pathology, globus sensation is best managed by simple reassurance.

2.6 Management

There is no treatment beyond reassurance. No diagnostic tests are indicated. If deep-seated emotional features exist, they may warrant a psychiatric opinion.

3. HEARTBURN AND REGURGITATION / C. Dubé and W.G. Thompson

3.1 Description

Heartburn is a burning sensation experienced behind the sternum, which may radiate toward the neck. It is most commonly experienced in the postprandial period, or when the subject is bending over, lying down or straining. Unlike angina, it is not usually worsened by exercise.

Regurgitation is the effortless return of gastric or esophageal contents into the pharynx without nausea, retching, or abdominal contractions. Patients typically regurgitate acidic material mixed with small amounts of undigested food.

Heartburn and regurgitation often occur concomitantly and may also be associated with chest pain, waterbrash, globus sensation, odynophagia, and nausea. Waterbrash is defined as the spontaneous flooding of the mouth with a clear, slightly salty fluid, which may be of sufficient quantity to require expectoration. It is believed to result from a vagal cholinergic reflex, with afferences originating in the upper gastrointestinal tract and efferences destined to the salivary glands.

3.2 Epidemiology

Heartburn and/or acid regurgitation is common and occurs at least weekly in up to 20% of the general population; only a minority of heartburn sufferers will eventually consult a physician for such symptoms, and those that seek medical advice may be characterized by a higher anxiety level, recent death or morbidity in the family as well as associated chronic MSK complaints or pain.

3.3 History

When heartburn is suspected, one should determine the effect of position, food, stress and exercise on the symptom. A careful cardiac history should be taken to rule out the possibility of angina. It is important to inquire about dysphagia, odynophagia, weight loss, symptoms suggestive of bleeding or anemia, as well as chronic cough or respiratory symptoms that could suggest aspiration.

The presence of heartburn and regurgitation implies that the subject has gastroesophageal reflux disease (GERD). The approach to investigation and management of GERD will be reviewed in its designated chapter.

4. DYSPHAGIA / A.S.C. Sekar

4.1 Description

Dysphagia means difficulty in swallowing. Some patients describe food sticking in the throat or retrosternally.

4.2 Important Historical Points and Differential Diagnosis

A careful history is important. Mechanical narrowing is a common cause; an inflammatory stricture must be distinguished from a carcinoma. If the dysphagia is relatively short in duration (e.g., only a few months) and is worsening, this suggests a progressive mechanical narrowing of the lumen such as may occur with an esophageal carcinoma. With benign disease, symptoms are often present for a longer period of time than with carcinoma. A previous history of heartburn or acid regurgitation in a patient with progressive dysphagia might point to an esophageal stricture secondary to gastroesophageal reflux disease. Not all patients with a benign esophageal stricture have a clear history of preceding heartburn or acid regurgitation. This is particularly true in the elderly patient. A history of ingestion of caustic agents such as lye suggests an esophageal stricture secondary to severe chemical esophagitis.

Infections of the esophagus can also cause difficult swallowing. Infections, usually due to *Candida albicans* or herpes virus, are often accompanied by significant pain on swallowing, termed *odynophagia*. Often the odynophagia is so severe that the patient even has difficulty swallowing his or her saliva. Although herpes esophagitis can occur in relatively healthy patients, *Candida* esophagitis is associated with diabetes, an underlying malignancy or immunosuppression.

The patient may point to the site of obstruction, but this is not always reliable. A stricture of the lower esophagus may be experienced at the xiphoid area or as high as the throat. Upper esophageal obstruction is experienced high in the throat region, not low in the chest.

Dysphagia can also occur with motor disorders of the esophagus. These conditions include esophageal spasm and achalasia. With motor disorders of the

esophagus, the dysphagia may be for both solids and liquids. The dysphagia is intermittent and may have a long history. Sometimes with esophageal spasm the dysphagia may be accompanied by pain (odynophagia), especially with extremely cold or hot liquids. These patients are usually able to wash down impacted particles of food, whereas patients with a mechanical cause (such as a stricture) may need to regurgitate impacted particles of food to obtain relief.

A common cause of intermittent dysphagia is a mucosal ring at the gastro-esophageal junction (lower esophageal or Schatzki's ring). On occasion when a relatively large bolus of food is swallowed the ring can cause mechanical obstruction, producing a dramatic onset of acute dysphagia (sometimes associated with pain). Often such patients will have to leave the table and regurgitate. Patients with a Schatzki's ring usually have symptoms for many years before they seek medical attention.

A rare cause of upper esophageal dysphagia is the Paterson-Kelly syndrome or Plummer-Vinson syndrome. Here, a chronic iron deficiency anemia is associated with narrowing of the upper esophagus due to a web.

Cricopharyngeal dysphagia may be due to a cricopharyngeal or Zenker's diverticulum, which develops from an abnormality of the cricopharyngeal sphincter. Patients with a diverticulum often complain of regurgitating food that they swallowed a day or so earlier.

There are non-esophageal causes of dysphagia. Underlying neuromuscular disease may cause cricopharyngeal dysphagia, where patients have difficulty initiating a swallow. A large goiter or mediastinal tumor can cause extrinsic compression of the upper esophagus.

4.3 Approach to Diagnosis and Management

A barium swallow is the most important initial investigation in the diagnosis of dysphagia. It might reveal a Zenker's diverticulum, an esophageal stricture (benign or malignant) or a Schatzki's ring. If inflammation of the esophagus is suspected, endoscopy with biopsies is indicated. If a stricture is identified on a barium swallow, endoscopy with biopsies is necessary to determine whether this stricture is benign or malignant. Also, benign strictures can be dilated following the endoscopic diagnosis. A barium swallow may help diagnose motility disturbances such as esophageal spasm and achalasia. Esophageal manometry is often required to confirm such motility disturbances.

Once a cause of dysphagia has been established, management will depend on the cause. Strictures secondary to gastroesophageal reflux disease are managed with periodic esophageal dilations and long-term proton pump inhibitors (e.g., omeprazole). Esophageal strictures can be dilated following endoscopy. Esophageal carcinoma requires either surgery, radiation or palliative insertion of prosthesis. Esophageal motility disturbances can sometimes be managed

medically with nitroglycerin or calcium channel blocking agents. Achalasia and esophageal spasm sometimes require surgical myotomy or pneumatic dilation.

5. DYSPEPSIA / C. Dubé

5.1 Description

Dyspepsia refers to chronic or recurrent pain or discomfort centered in the upper abdomen. Individuals might refer to this symptom as “indigestion.” Bloating, early satiety, nausea, and vomiting are other symptoms that may also be reported in association with dyspepsia. Dyspepsia can be intermittent or continuous, and, importantly, may or may not be related to meals.

5.2 Etiology

The major organic diseases causing dyspepsia are gastroduodenal ulcer, atypical gastroesophageal reflux, and gastric cancer. Up to 60 % of patients with dyspepsia have no definite explanation and are classified as having functional (idiopathic) dyspepsia, also referred to as nonulcer dyspepsia. The pathophysiology of functional dyspepsia is unclear. Factors such as gastric motor dysfunction, visceral hypersensitivity, psychosocial factors and *Helicobacter pylori* infection might play a role in its pathophysiology.

5.3 History and Physical Examination

It is impossible to differentiate between organic and functional dyspepsia on the basis of the patient’s description of the symptom alone. Similarly, the presence of epigastric pain on physical examination is not discriminating. It is therefore important to look for clues to an organic etiology of the dyspepsia, so-called alarm features, such as gastrointestinal bleeding, symptoms of anemia, a history of weight loss, or the presence of an epigastric mass or hepatomegaly on physical examination. Peritoneal signs or a succession splash would be suggestive of free ulcer perforation and gastric outlet obstruction respectively.

Careful history will also help differentiate between dyspepsia and gastroesophageal reflux disease (GERD), irritable bowel syndrome (IBS), and biliary colic. Up to a third of GERD sufferers do experience epigastric pain or discomfort that is centered in the epigastrium. However, in such cases, symptoms of heartburn and regurgitation, often recumbency-aggravated, will usually be associated. Bloating and epigastric pain or discomfort may also occur in IBS. However, this is generally distinguished from dyspepsia by its association with altered bowel habits, as well as the relief of the pain with defecation. There should be no confusion between the episodic nature and severity of pain due to biliary colic or pancreatic disease and the more predictable and regular

occurrence of dyspepsia. Indeed, dyspeptic symptoms are equally common in those who have and in those who do not have gallstones.

5.4 Approach to Diagnosis and Management

In younger patients with no alarm features who have not been investigated previously, it is recommended that a noninvasive *H. pylori* test (e.g., serology or urea breath test) is undertaken to determine if the patient is infected. If there is documented *H. pylori* infection, then an empiric trial of anti-*H. pylori* therapy is recommended. The rationale is that ulcer disease will heal and the ulcer diathesis will be abolished. If symptoms fail to respond or rapidly recur or alarm features develop, then prompt upper endoscopy is indicated. In younger patients with no alarm features who are *H. pylori* negative, and who have persistent symptoms, one option is to opt for a therapeutic trial of antisecretory therapy (e.g., H₂-blocker or proton pump inhibitor) or of a prokinetic agent. If symptoms persist or rapidly recur on stopping treatment, then endoscopy is recommended. It is worth noting that endoscopy is the test of choice to exclude gastroduodenal ulceration, reflux esophagitis, and upper gastrointestinal tract malignancy. Although upper gastrointestinal radiographs have inferior diagnostic accuracy to upper endoscopy, they are more accessible and cheaper. Another option is to refer all dyspepsia sufferers to prompt endoscopy, since investigations for dyspepsia in the setting of low clinical suspicion for organic disease have the added advantage of providing reassurance to the patient, a strategy with proven therapeutic efficacy.

Referral for early upper endoscopy is always indicated in older patients presenting with new-onset dyspepsia. This is because the incidence of gastric cancer increases with advancing age; a threshold of 45 years is recommended. As mentioned above, patients with alarm symptoms (e.g., weight loss, recurrent vomiting, dysphagia, evidence of bleeding, or anemia) and patients whose symptoms have failed to respond to empiric therapeutic approaches should undergo endoscopy.

6. NAUSEA AND VOMITING / M.C. Champion

6.1 Synonyms

Barf, upchuck, bring up.

6.2 Description

Nausea is a psychic as well as physical experience and defies precise definition. Vomiting is evacuation of the stomach contents through the mouth. Nausea normally precedes vomiting. There can be associated tachycardia, hypersalivation, waterbrash and excessive perspiration.

6.3 Mechanism (Figure 1)

A variety of stimuli may produce nausea (labyrinthine stimulation, pain, unpleasant memories). The neural pathways mediating nausea are not known, but evidence suggests that they are the same pathways that mediate vomiting. During nausea, gastric tone and peristalsis are reduced. The tone of the duodenum and proximal jejunum tends to be increased, with frequent reflux of duodenal contents into the stomach.

Vomiting occurs as the gastric contents are forcefully brought up to and out of the mouth. This occurs by forceful sustained contraction of the abdominal muscles at a time when the cardia of the stomach is raised and open and the pylorus is contracted. Elevation of the cardia eliminates the intra-abdominal portion of the esophagus and relaxes the lower esophageal sphincter. This allows the stomach contents to enter the esophagus. The act of vomiting is completed with rapid upward displacement of the diaphragm and reversal of thoracic pressure from negative to positive. The glottis closes, the soft palate rises, the mouth opens and the stomach contents are expelled. The control of vomiting consists of two anatomically and functionally separate units, a vomiting center and a chemoreceptor trigger zone. The vomiting center is in the reticular formation of the medulla and is excited directly by visceral afferent impulses (sympathetic and vagal) arising from the gastrointestinal tract and other peripheral trigger areas. These trigger areas are found in the pharynx, cardiac vessels, peritoneum, bile ducts, cortex and stomach. The chemoreceptor trigger zone is on the floor of the fourth ventricle, on the blood side of the blood–brain barrier. The chemoreceptor trigger zone is unable to cause vomiting without an intact vomiting center.

6.4 History and Physical

Patients may complain of nausea and hypersalivation. With gastrointestinal causes of the nausea (and vomiting) there may be associated symptoms of heartburn or epigastric pain. Prior to vomiting, patients may retch (spasmodic, abortive respiratory movements with the glottis closed).

History-taking should probe for precipitating factors, other symptoms that suggest the underlying cause, drug use and dietary habits. The history should also explore psychological trauma or disturbances of body image suggestive of anorexia nervosa.

Physical examination is often normal. An abdominal mass may point to an underlying cause (e.g., gastric carcinoma). Prolonged vomiting may cause dehydration.

6.5 Differential Diagnosis

Prolonged nausea, by itself, rarely has an organic origin. There are many causes

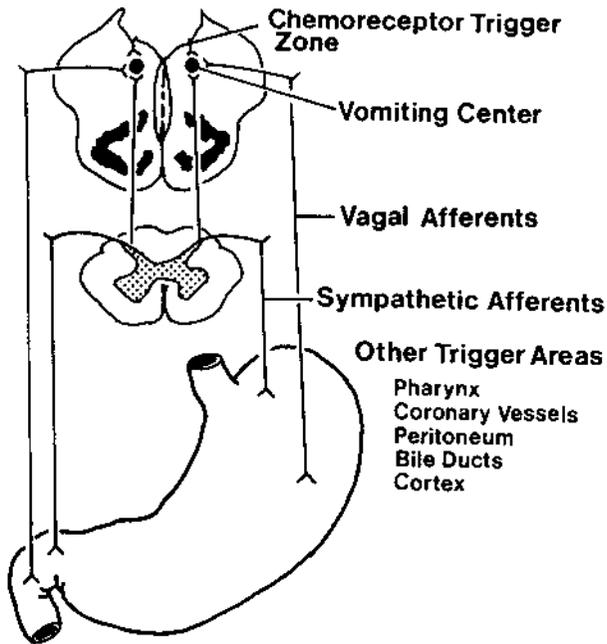


FIGURE 1. The vomiting center and chemoreceptor trigger zone control vomiting. Peripheral trigger areas send visceral afferent impulses, which excite the vomiting center into action.

of nausea and vomiting, including intracerebral problems (e.g., hydrocephalus, brain tumor), stimulation of the peripheral trigger areas (e.g., severe chest pain, pain from kidney stones), systemic disease (malignancy), medications and pregnancy. Upper gastrointestinal diseases (esophagitis, peptic ulcer disease, gastric carcinoma) are common. Early morning nausea and vomiting suggest pregnancy, gastroesophageal reflux disease, alcohol withdrawal, a metabolic cause (e.g., uremia) or a psychogenic origin.

6.6 Approach to Diagnosis and Management

In approaching a patient with nausea and vomiting, one should look for and correct any underlying causes. Prolonged vomiting may cause dehydration and the patient may need to be rehydrated intravenously. Medications should be discontinued.

There are many drugs that have anti-emetic actions. Antihistamines act on the vestibular apparatus as well as on the chemoreceptor trigger zone.

Phenothiazines also exert their action on the chemoreceptor trigger zone. Metoclopramide and domperidone are both anti-emetics and gastric prokinetics (which stimulate the stomach to empty). Domperidone exerts its action on the chemoreceptor trigger zone, whereas metoclopramide also crosses the blood-brain barrier and affects the vomiting center. Cisapride, a newer gastric prokinetic, has no effect on the chemoreceptor trigger zone or vomiting center. Like the other prokinetic agents, it may improve nausea and vomiting if they are due to gastric stasis or gastroparesis.

7. ANOREXIA / M.C. Champion

7.1 Description

Anorexia is the lack (or loss) of appetite. Anorexia is a common and important, but nonspecific, symptom. It can be a presenting feature in patients with organic or psychological disease. Anorexia and weight loss may be the early signs of malignancy.

7.2 Mechanism

The hypothalamus plays a major role in regulating the intake of food. At one time it was generally held that a “satiety center” and a “feeding center” in the hypothalamus exerted the fundamental control over food intake. Stimulation of the satiety center was believed to inhibit the feeding center and gastric hunger contractions. The feeding center was considered to be an integrative station that coordinates complex reflexes associated with food intake. However, it is now believed that control of appetite is best considered as multiple neuropharmacologic interactions in the hypothalamus rather than the effect of a distinct satiety center and feeding center.

7.3 History and Physical

The history should detect other symptoms that may suggest underlying organic or psychological disease. A calorie count is also helpful to assess the actual intake of food. The amount and duration of weight loss should also be documented.

Physical examination may be normal except for evidence of weight loss. It may point to the underlying organic problem, such as cardiac failure or malignancy.

7.4 Differential Diagnosis

Many (and perhaps most) illnesses feature a loss of appetite. These range from gastrointestinal disease to malignancy, chronic renal failure, congestive heart failure and many psychiatric diseases, such as depression and anorexia nervosa.

7.5 Approach to Investigation and Management

Investigation should exclude organic disease. The approach depends upon the patient's symptoms and signs. If no physical ailment is discovered, careful screening may be necessary to exclude psychiatric disease.

8. GAS AND BLOATING / W.G. Thompson

8.1 Synonyms and Related Terms

Borbulence, flatulence, burp, belch, borborygmi, gaseous distention, wind, flatus, fart.

Gas and bloating embrace three unrelated phenomena. *Farting* is a physiologic phenomenon due to the production of gas by colon bacteria. Excessive belching or burping is associated with *aerophagia* (air swallowing). This is also partly physiological, but it may become exaggerated through habit. The mechanism of *bloating* is obscure. These phenomena are unrelated, yet they often occur together.

8.2 Farting, Gas, Wind, Flatus

8.2.1 MECHANISM

Farting is a physiologic excretory process. Normally, the gut contains 100 to 200 mL of gas. An average person on a normal diet emits about 1 L per day. We pass 50 to 500 mL a mean of 13.6 times per day, although there is great variation from person to person and from time to time. Those prone to produce greater amounts of gas or who are unduly sensitive may suffer socially. Most emitted gas originates in the colon. Some carbohydrates such as cellulose, glycoproteins and other ingested materials, not assimilated in the small intestine, arrive intact in the colon where resident bacteria digest them to produce hydrogen, carbon dioxide, methane and trace gases.

Intestinal floras differ from person to person. Some bacteria produce hydrogen, while others consume it. In one person out of three, an organism called *Methanobrevibacter smithii* converts hydrogen to methane. The presence of this organism and the methane-producing trait are a result of early environment. Spouses do not share the trait with one another. Another product of fermentation, carbon dioxide, is also released when hydrochloric acid reacts with bicarbonate in the intestines. However, this gas is quickly absorbed. Hydrogen, carbon dioxide, methane and swallowed nitrogen comprise 99% of colon gas. The remaining 1% consists of trace gases that compensate for their small quantities by their strong odors. Smelly gases include hydrogen sulfide ammonia, skatole, indole and volatile fatty acids.

Borborygmi is the name given to the noises generated as air and fluid gurgle through the gut. Bloating is not due to excessive gas.

8.3 Aerophagia

8.3.1 MECHANISM

During inspiration, the normally negative intraesophageal pressure draws in ambient air. Forced inspiration against a closed glottis (intentionally closed windpipe) draws in even more air. The air may be forced out again as intraesophageal pressure increases with expiration. Adolescents love to shock their elders with voluntary belching. As a practical application, those who have lost their larynx because of cancer put this learnable skill to use in generating esophageal speech. More commonly, aerophagia is an unwanted but learned habit in those who repeatedly belch in response to other gut symptoms.

Some air is ingested with each swallow, perhaps more with food. Nervous patients undergoing abdominal x-rays accumulate more intestinal gas than those who are relaxed. Other mechanisms of aerophagia include thumb sucking, gum chewing, drinking carbonated drinks, rapid eating and wearing poor dentures. Stomach gas has the same composition as the atmosphere.

In achalasia, where the lower esophageal sphincter cannot relax, the stomach is gasless. In bowel obstruction or a gastrocolic fistula colon gases reach the stomach. Sometimes gastric stasis permits bacteria to grow and produce hydrogen in the stomach. Normally, gastric gas is swallowed air.

8.3.2 CLINICAL MANIFESTATIONS OF AEROPHAGIA

Belching is to bring forth wind noisily from the stomach. The word *burp* means to “cause to belch,” as one would burp a baby, but colloquially, the terms are used interchangeably. A belch after a large meal is a physiologic venting of air from the stomach. A meal stretching the muscle of the stomach, which can stretch to accommodate food, causes distress with little increase in intragastric pressure. A satisfying belch eases the discomfort. Some individuals seem unduly sensitive to intragastric pressure. People with gastroenteritis, heartburn or ulcers swallow more frequently. If release of gas transiently relieves the distended feeling, a cycle of air swallowing and belching may be established. The swallow-belch cycle may continue long after the original discomfort is forgotten.

Of course, venting gas is important, as those unable to do so will attest. When the lower esophageal sphincter is reinforced by antireflux surgery, belching may be impossible. Bedridden patients such as those recovering from surgery may trap air in the stomach. In the supine position gastric contents seal the gastroesophageal junction so that air cannot escape until the subject assumes the prone position.

While a patient may insist that his or her stomach is producing prodigious amounts of gas, in reality air is drawn into the esophagus and released. A little may even reach the stomach. Some can belch on command, and the inspiration against a closed glottis is demonstrable. Most sufferers are relieved to have their habit pointed out, but some are incredulous. Quitting the habit is often difficult. Repeated and intractable belching is termed *eructio nervosa*.

8.4 Functional Abdominal Bloating

8.4.1 MECHANISM

Those complaining of bloating and distention are often convinced that it is due to excess intestinal gas. Although the sensation may induce aerophagia, it seldom results from it. Farting may temporarily relieve bloating, but intestinal gas production does not cause it. Research has demonstrated that gas volume in bloaters is not abnormal. Despite visible distention, x-rays and computerized tomography (CT) show no large collections of intestinal gas. The distention disappears with sleep and general anesthesia.

Gut hypersensitivity may explain the sensation of abdominal bloating. The hypersensitive gut feels full at lower than normal filling, and abdominal muscles relax to accommodate the perceived distention. The stomach is and feels distended with normal amounts of air.

Abdominal girth of female irritable bowel syndrome (IBS) patients complaining of distention may increase 3–4 cm over an eight-hour day. CT has demonstrated the change in profile despite unchanged gas content or distribution. There were no corresponding changes in control subjects. Lumbar lordosis (arching of the spine) is sometimes increased. When women deliberately protrude their abdomens, the configuration is different from when they are bloated, so a conscious mechanism poorly explains increased abdominal girth. Perhaps abdominal muscles are weakened. The reality of the phenomenon is indisputable; the mechanism remains a mystery.

8.4.2 CLINICAL FEATURES

Bloating occurs in 30% of adults and is frequent in 10%. Amongst those with the irritable bowel syndrome and dyspepsia the figures are much higher. It is often the most troublesome feature of these conditions. Typically, the abdomen is flat upon awakening, but distends progressively during the day, only for the distention to disappear with sleep. Women complain of the need to let out their clothing and sometimes volunteer “It’s as if I’m six months pregnant.” Many report that bloating occurs quickly, in some cases within a minute. It is often aggravated by eating and relieved by lying down. Menstrual periods and stress affect a few cases. Usually, it is most

obvious in the lower abdomen, but many report it near the umbilicus or all over the belly.

8.4.3 DIFFERENTIAL DIAGNOSIS

Observable bloating has been called *hysterical nongaseous bloating*, *pseudo-tumor* or *pseudocyesis* (false pregnancy). If distention is present at the time of the examination (more likely late in the day) and absent on other occasions, the phenomenon is likely functional. There is no abdominal tympany to suggest gaseous intestines, and sometimes the distended abdomen can be mistaken for ascites or a tumor.

Bloating is often associated with dyspepsia or IBS. On its own, it is not a symptom of organic disease and should prompt no investigation. In intestinal obstruction or postoperative ileus (paralyzed intestines), gas accumulates and distends the gut to cause discomfort and pain. In such a case, there are other symptoms and signs with which to make a diagnosis.

9. CONSTIPATION / C. Dubé and W.G. Thompson

9.1 Synonyms

Costiveness, obstipation.

9.2 Description

Constipation defies accurate definition. What is “normal” frequency? Ninety- five percent or more of the population have between three movements per day and three movements per week. Some people consider that fewer than three movements a week without discomfort or dissatisfaction is normal. The effort needed to pass the stool and the consistency of the stool are probably of greater importance. Most would agree that hard bowel movements that are difficult to pass constitute constipation even if they occur as often as daily. One definition of constipation is the need to strain at stool on more than 25% of occasions. Thus constipation is defined as persistent symptoms of difficult evacuation, including straining, stools that are excessively hard, unproductive urges, infrequency, and a feeling of incomplete evacuation.

9.3 Mechanism

Constipation may be due to primary colonic conditions, such as obstructing lesions of the colon, irritable bowel syndrome and idiopathic slow-transit constipation. It may also be caused by systemic afflictions, either endocrine (diabetes mellitus, hypothyroidism), metabolic (hypo- or hypercalcemia),

TABLE 1. Some causes of chronic constipation

<i>Primary diseases of the colon</i>	<i>Pharmacologic</i>
Stricture	Opiates
Cancer	Antidepressants
Anal fissure	Antispasmodics
Proctitis	Calcium channel blockers
Irritable bowel syndrome	Iron
Idiopathic slow-transit constipation	Laxative abuse
<i>Pelvic floor dyssynergia</i>	<i>Neurologic and muscular disorders</i>
<i>Metabolic disturbances</i>	Parkinson's disease
Hypercalcemia	Spinal cord lesion
Hypocalcemia	Multiple sclerosis
Hypothyroidism	Autonomic neuropathy
Diabetes mellitus	Hirschsprung's disease
Pregnancy	Systemic sclerosis
	Myotonic dystrophy

neurologic (multiple sclerosis, Parkinson's disease), muscular (systemic sclerosis, myotonic dystrophy), or medications (opiates, anticholinergic agents, antihypertensives). The most common kind of constipation is that associated with the irritable bowel syndrome (Table 1).

Proper defecation requires normal transit through the proximal colon, an intact gastrocolonic response to a meal, and normal mechanisms of defecation, involving: (1) the defecation reflex (i.e., the presence of stool in the rectum initiating the evacuating response of the internal sphincter); (2) the coordinated relaxation of the puborectalis and external anal sphincter muscles; (3) adequate functional anatomy of the rectal outlet together with (4) increased intra-abdominal pressure; and (5) inhibition of colonic segmenting activity. Any failure at any level of colorectal function may therefore lead to constipation.

9.4 Important Points on History and Physical

A detailed dietary history with respect to the daily intake of fibre and liquids as well as eating patterns is important: the majority of constipated patients do not eat breakfast, which is an important trigger of the gastro-colonic response. Physical impairments and bedridden states will also contribute to constipation. The list of medications should be reviewed and a history of prolonged intake of cathartics, often in the form of herbal remedies or teas, should be sought. Symptoms suggestive of IBS, such as bloating, abdominal pain, and

alternation of diarrhea with constipation should be enquired about. History also includes symptoms related to pelvic descent, such as stress incontinence. A history of weight loss, of a recent onset of constipation, of the presence of blood around the stools, as well as the presence of risk factors for colorectal cancer, should raise the possibility of an obstructing carcinoma.

On physical examination, one should note the presence of abdominal distension or of palpable stools, either in the left lower quadrant or in the more proximal colon. Rectal examination is useful to identify fissures or hemorrhoids which may either cause or be caused by constipation. A lax or asymmetric anal opening may suggest a neurologic disorder with impaired sphincter function, and the presence of stools in the rectum may suggest an impaired defecation reflex.

9.5 Approach to Diagnosis

Sigmoidoscopic examination using either the rigid or flexible instrument is necessary to rule out local diseases such as fissures, fistulas or distal proctitis. Many cancers are within the range of the sigmoidoscope. One might also detect melanosis coli, a pigment in the rectal mucosa that indicates chronic laxative use.

If the constipated patient is over 40, has blood or pus in the stool, or has had significant weight loss, a colonoscopy is indicated to rule out cancer, polyps or Crohn's disease of the colon. Barium enema may be useful to assess for megarectum or megacolon, but distal rectal lesions may be missed, so sigmoidoscopic examination should not be foregone.

A gut transit study may be revealing. Twenty radiopaque markers are ingested and daily plain abdominal x-rays are taken. If 80% of the markers have disappeared in five days, the transit time is said to be normal. In cases of longer transit, the position of the markers may help distinguish colonic inertia from anorectal disorder. More sophisticated studies, such as anorectal manometry and defecography, are then required.

9.6 Approach to Management

Management of constipation includes education as to the great variability of bowel habits among the general population, reassurance as to the benign nature of the condition once appropriate investigations have been carried out, dietary changes, and judicious use of laxatives. Dietary changes include the intake of at least three meals a day, the first meal of the day being taken upon arousal. Adequate amounts of liquids, such as 6 to 8 cups per day, should be ingested, and a high fibre diet, either with the intake of roughage and cereals or with the addition of bulk-forming agents, should be achieved. The recommended amount of dietary fiber is 20 to 35 g/day.

Chronic severe constipation may require the daily use of osmotic agents such as lactulose or sorbitol, of polyethylene glycol solution, or of pharmacologic agents, such as tegaserod. The long-term use of stimulant laxatives such as bisacodyl or senna should be avoided.

10. DIARRHEA / W.G. Thompson

10.1 Synonyms

Lax bowels, the flux.

10.2 Description

Diarrhea is best described as too frequent passage of too loose (unformed) stools. Diarrhea is frequently accompanied by urgency, and occasionally incontinence. When considering a patient with diarrhea the following must be considered: frequency (> 3 movements/day), consistency (loose/watery), urgency, volume (> 200 g/day) and whether the condition is continuous. Persistent, frequent, loose, urgent, large-volume stools are most likely to have a pathology. Lesser and intermittent symptoms are more likely to be functional.

10.3 Mechanism

Diarrhea is due to one or more of four mechanisms: osmotic attraction of excess water into the lumen of the gut, secretion of excess fluid into the gut (or decreased absorption), exudation of fluid from the inflamed surface of the gut, and rapid gastrointestinal transit.

Osmotic diarrhea results if the osmotic pressure of intestinal contents is higher than that of the serum. This may result from malabsorption of fat (e.g., in celiac disease) or of lactose (e.g., in intestinal lactase deficiency). Certain laxatives, such as lactulose and magnesium hydroxide, exert their cathartic effect largely through osmosis. Certain artificial sweeteners, such as sorbitol and mannitol, have a similar effect. Characteristically, osmotic diarrhea ceases when the patient fasts.

Secretory diarrhea occurs when there is a net secretion of water into the lumen. This may occur with bacterial toxins, such as those produced by *E. coli* or *Vibrio cholerae*, or with hormones, such as vasoactive intestinal polypeptide (VIP), which is produced by rare islet cell tumors (pancreatic cholera). These provoke adenylate cyclase activity in the enterocyte (intestinal epithelial cell), increase cyclic AMP and turn on intestinal secretion. A similar effect may occur as a result of excess bile salts in the colon (choleric enteropathy) and from the cathartic effect of hydroxylated fatty acids resulting from the bacterial action on

malabsorbed fat. Such a diarrhea does not diminish with fasting. Osmotic and secretory diarrhea result from abnormalities in the small intestine such that the flow of water through the ileocecal area overcomes the absorptive capacity of the colon.

Exudative diarrhea results from direct damage to the small or large intestinal mucosa. This interferes with the absorption of sodium salts and water and is complicated by exudation of serum proteins, blood and pus. Infectious or inflammatory disorders of the gut cause this kind of diarrhea.

Acceleration of intestinal transit may result in diarrhea (e.g., as a result of hyperthyroidism). The rapid flow-through impairs the ability of the gut to absorb water, resulting in diarrhea.

In most instances of diarrhea two or more of these four mechanisms are at work, so these pathogenetic concepts are seldom of great help in diagnosis.

10.4 Important Historical Points and Physical Examination Features

It is important to establish the frequency of defecation, the duration of the diarrhea, the nature of the stool and its volume. If diarrhea has been present for less than two weeks, it is most likely a result of an infection or toxin. A history of many previous attacks, on the other hand, may indicate a recurrence of inflammatory bowel disease. The frequency of the stool gives some idea of severity; one should establish whether incontinence is also present. To elicit the latter history may require direct questions. Stool from malabsorption is often foul-smelling and contains oil droplets. A history of nutrient deficiency, anemia or weight loss also suggests malabsorption. Watery diarrhea, particularly when large in volume, supports a diagnosis of small bowel disease. However, a large villous adenoma of the distal colon may produce a watery diarrhea. The presence of blood or pus in the stool suggests an exudative diarrhea, a type of diarrhea that is often relatively small in volume and indicative of colitis. Loose bowel movements interspersed with normal or even constipated ones are evidence of the irritable bowel syndrome.

There are many causes of diarrhea, some of which are summarized in Table 2. The presence of profound weight loss and malnutrition in a young person points to a malabsorption syndrome due to small bowel or pancreatic disease or to inflammatory bowel disease. Metabolic conditions such as hyperthyroidism or the overuse of (magnesium-containing) antacids or laxatives might also be responsible for chronic diarrhea.

Travel to tropical countries can be marred by an attack of so-called traveler's diarrhea. The most common cause is toxigenic *E. coli* (it is known as toxigenic because a toxin is produced). However, a large variety of intestinal infestations can occur with travel. Pseudomembranous colitis may occur within weeks of the use of antibiotics. *Campylobacter* or cryptosporidiosis may be acquired from

TABLE 2. Anatomic approach to the causes of chronic diarrhea

Gastric

Dumping syndrome

Small intestine

Celiac disease

Lymphoma

Whipple's disease

Parasitic infection (*Giardia lamblia*)

Abnormal intestinal tract motility with bacterial overgrowth (scleroderma, amyloidosis, diabetes, hyperthyroidism)

Large bowel

Villous adenoma (adenocarcinoma)

Inflammatory bowel disease (ulcerative colitis, Crohn's disease)

Irritable bowel (diarrhea phase)

Functional diarrhea

AIDS-related infections

Pancreatic

Chronic pancreatitis

Islet cell tumors

Gastrin secretions

VIP secretions

Drugs

Antacids

Antibiotics

Alcohol

Antimetabolites

Laxatives

Digitalis

Colchicine

Sorbitol, fructose

Many others

Metabolic

Hyperthyroidism

Hypoparathyroidism

Addison's disease

Diabetes

Carcinoid syndrome

pets. Contaminated water may result in giardiasis, amebiasis or cryptococcosis. Chronic use of alcohol may damage the small intestinal mucosa. Diabetics frequently have diarrhea because of autonomic neuropathy, perhaps with bacterial overgrowth.

Finally, it is essential to establish if the patient is homosexual. Almost any of the usual gastrointestinal pathogens can be spread by homosexual activity, including lymphogranuloma venereum and gonococcus. In addition to the “gay bowel syndrome,” homosexuals are liable to the gastrointestinal complications of AIDS.

10.5 Differential Diagnosis and Management

The recent onset of acute diarrhea requires careful examination of the stool for pus cells and culture for bacterial pathogens, or a study for ova and parasites in the case of suspected protozoa. Viral studies are important in infants, and special studies are required in AIDS.

The differential diagnosis of chronic diarrhea is very complex. A careful history is often the most important diagnostic tool. Patients examined for the first time deserve at least a sigmoidoscopy to rule out local colon disease. If a small intestinal diarrhea is suspected, a three-day collection of stool to determine daily weight and fat content is necessary. If there is steatorrhea, or if stool weight exceeds 500 g per day, there is likely to be small intestinal or pancreatic disease. Smaller volumes, particularly if accompanied by blood, point to inflammation of the colon.

11. MALNUTRITION / D.G. Patel

11.1 Description

Nutrition may be defined as the process by which an organism utilizes food. This complex process involves ingestion, digestion, absorption, transport, utilization and excretion. Any alteration in one or many of these factors can produce malnutrition. Globally, primary malnutrition due to lack of food is the most common cause of malnutrition. Malnutrition in the Western world is mainly due to inadequate intake of nutrients, malabsorption and/or the hypercatabolism accompanying a critical illness. Protein-energy undernutrition is increasingly recognized in eating disorders such as anorexia nervosa.

11.2 Mechanism

The malnutrition associated with gastrointestinal disorders is usually multifactorial and varies with the nature and activity of the disease.

1. Lack of food intake due to anorexia or food-related symptoms such as dysphagia, pain or vomiting.
2. Maldigestion due to pancreatic disease. Deficiency of bile salts due to cholestatic hepatobiliary disease or to ileal disease leads to maldigestion of triglyceride and lipid-soluble vitamins. Steatorrhea (fat malabsorption) produces negative caloric balance and deficiency of fat-soluble vitamins.
3. Malabsorption due to mucosal disease of the small intestine or loss of surface area due to intestinal bypass, fistula or resection.
4. Excessive loss of nutrients, as in protein-losing enteropathy and loss of zinc in diarrheal illness.
5. Therapeutic agents that may selectively affect nutrient utilization – e.g., cholestyramine use for bile acid-induced diarrhea can worsen steatorrhea.
6. Alcoholism – an extremely common cause of malnutrition in the Western world. Social and economic status, behavior problems, isolation and depression cause reduced intake of nutrients. Alcoholics rarely consume a well-balanced diet and depend very heavily on “empty” calories from alcohol. Protein and vitamin deficiencies, particularly of the B-complex group, are extremely common. Alcohol is a toxic agent that even in the presence of adequate nutritional intake can produce damage to the pancreas, liver and small bowel mucosa, aggravating malnutrition.

11.3 Signs of Malnutrition

1. Weight loss in the absence of edema is a good indicator of energy deficiency.
2. Muscle wasting, particularly in the temporal area and dorsum of the hand between thumb and index finger, suggests protein-calorie deficiency.
3. Dry, scaly skin with pigmentation results from vitamin and trace metal deficiency.
4. Angular mouth fissure (cheilosis) is due to riboflavin deficiency.
5. Glossitis and depapillation of the tongue are due to B₁₂, folate or iron deficiency.
6. Hepatomegaly may be due to fatty liver, a common finding in protein malnutrition or alcoholism.
7. Peripheral neuropathy (decreased position sense), decreased vibration sense or ataxia may result from B₁₂ deficiency.
8. Weakness and paresthesia of the legs are signs of nutritional polyneuropathy, especially in alcoholics (due to thiamine or pyridoxine deficiency).
9. Anemia due to iron, folate or B₁₂ deficiency or chronic disorders.
10. Peripheral edema.
11. Hypoalbuminemia.

12. ACUTE ABDOMEN / J.M. Watters

12.1 Description

The term *acute abdomen* is best used to describe abdominal pain and related symptoms and signs that are sufficiently severe as to suggest a serious intra-abdominal condition. Pain has usually been present for 72 hours or less and sometimes for only a few hours. Since some patients with an acute abdomen require resuscitation and early surgical treatment, it is important to assess the patient and establish a plan of management as soon as possible. The initial goal is not necessarily to make a definitive diagnosis, but rather to identify those patients who require prompt surgical intervention.

12.2 Mechanism

Acute abdominal pain may be referred to the abdominal wall from intra-abdominal organs (visceral pain) or may involve direct stimulation of the somatic nerves in the abdominal wall (somatic pain). The nerve supply to the viscera is bilateral, and visceral pain is not usually lateralized. Foregut pain is typically epigastric in location, midgut pain is central, and hindgut pain is felt in the lower abdomen. Organs that are bilateral give rise to pain that is predominantly felt on one or the other side of the body. Visceral pain arises from tension in the bowel wall (e.g., distension or vigorous contraction), mesenteric traction, or irritation of the mucosa or serosa of the bowel (e.g., chemical irritation, bacterial contamination, ischemia).

Somatic pain is more precise in location than visceral pain and corresponds more directly to the anatomic site of the underlying pathology. Occasionally, pain is referred to the abdomen from extra-abdominal sites (e.g., lower lobe pneumonia). Unusually, acute abdominal pain is a feature of systemic disease (e.g., diabetic ketoacidosis). Somatic pain occurs with stimulation of pain receptors in the peritoneum and abdominal wall.

12.3 History

The initial location and character of acute abdominal pain and their subsequent evolution often give useful clues to the site and nature of the underlying pathology. A history of pain with movement (e.g., riding in a car or walking) suggests the presence of peritonitis if it is not otherwise obvious. Steady, severe pain is more ominous than colicky pain. Severe pain of sudden onset suggests a catastrophic event (e.g., perforation of an ulcer, intestinal ischemia, or rupture of aortic aneurysm). Colicky pain corresponds to peristaltic waves and eases or disappears between waves. Examples are the intermittent, mid-abdominal pain of uncomplicated small bowel obstruction and the intermittent flank pain radiating anteriorly to

the groin accompanying ureteric obstruction. However, when the viability of obstructed small bowel is comprised, for example, the now-ischemic segment of bowel causes unremitting pain that is localized to the area of the involved loop. Biliary “colic” is a misnomer in that the pain is typically steady. It is usually epigastric or upper abdominal and relatively diffuse, becoming more localized in the right upper quadrant if the process evolves into acute cholecystitis.

Radiation of pain may provide important clues to diagnosis. Irritation of the diaphragm, from blood in the peritoneal cavity, for example, may cause shoulder tip pain. Biliary tract pain may radiate to the right scapular region. Pain arising from retroperitoneal structures may be perceived in the back (e.g., pancreatitis, leaking abdominal aortic aneurysm).

12.4 Associated Symptoms

Anorexia, nausea and vomiting are more common in diseases of the gastrointestinal tract and not specific in terms of diagnosis. Abdominal distention and obstipation accompanying acute abdominal pain suggest intestinal obstruction. In a patient with colonic obstruction and/or perforation, a recent change in bowel habit or blood in the stool (prior to the onset of pain) suggests the possibility of a colon cancer. Bloody diarrhea may arise from severely inflamed, ulcerated or infarcted bowel. In women an accurate menstrual history aids the diagnosis of ovarian disease, ectopic pregnancy and pelvic inflammatory disease. Urinary symptoms may suggest a genito-urinary diagnosis (e.g., pyelonephritis, renal stones).

12.5 Physical Examination

Examination of the abdomen is normally carried out with the patient supine. Analgesia may impair the sensitivity of physical examination when signs are subtle, but should be given promptly once assessment has been completed or when it will be unavoidably delayed. When the presenting pain is severe, alleviating it may well permit a more useful history and subtle examination to be obtained.

Inspection of the abdomen should note any distention or masses. The patient with peritonitis typically lies immobile, since any movement increases peritoneal irritation and pain. With ureteral colic, the patient may appear restless, seeking a more comfortable position.

Auscultation may reveal a range of bowel sounds, from the silent abdomen of peritonitis to the hyperactive sounds of bowel obstruction. Bruits suggest vascular disease, but an epigastric bruit may also be found normally.

Gentleness is the key to palpation. Palpation detects and localizes tenderness, muscle guarding, rigidity and masses. *Guarding* refers to the involuntary

contraction of initially relaxed muscles of the abdominal wall in response to the pain stimulated or exacerbated by palpation. Guarding may be localized (e.g., uncomplicated appendicitis) or generalized throughout the abdomen (e.g., perforated diverticulitis with diffuse contamination of the peritoneal cavity). In some instances (e.g., perforated duodenal ulcer), the muscles are in a state of continuous contraction and are rigid or “board-like” even without palpation. In subtle situations, peritonitis is suggested by the triggering of pain in the area of suspected pathology (e.g., appendicitis) through palpation elsewhere on the abdominal wall, by having the patient cough or by gently shaking the pelvis. Gentle percussion is also a very useful way to assess peritoneal irritation, as well as to assess the nature of abdominal distention. Testing for “rebound” tenderness by deeply palpating the area of concern and then suddenly releasing the abdominal wall is very distressing to the patient with peritonitis, may be misleading in the patient without peritonitis, and does not contribute to diagnosis.

Rectal and pelvic examination should be carried out and recorded by at least one examiner. The sites for inguinal and femoral hernias should be specifically examined. Femoral pulses should be palpated. A careful physical examination will identify pertinent extra-abdominal findings (e.g., jaundice, lymphadenopathy), systemic effects of an acute abdominal condition (e.g., hypotension, tachycardia, tachypnea), and significant coexisting conditions.

12.6 Differential Diagnosis

Intra-abdominal conditions requiring surgery (open or laparoscopic) are the most common causes of an acute abdomen. Some conditions require immediate surgery (e.g., ruptured abdominal aneurysm). They must always be included in the differential diagnosis, therefore, and confirmed or excluded promptly. In other instances, the specific diagnosis and the need for surgery may take some time to establish. The likelihood of specific diagnoses varies to an extent with the age of the patient. Clinical presentations are more likely to be atypical in the elderly and in patients with coexisting conditions (such as diabetes or stroke). Particular care must be taken to not overlook an important intra-abdominal process in such patients.

One must always consider in the differential diagnosis: (1) intra-abdominal conditions for which surgery is not indicated (e.g., acute pancreatitis, primary bacterial peritonitis); and (2) extra-abdominal (e.g., pneumonia) or systemic conditions (e.g., diabetic ketoacidosis) that can be accompanied by acute abdominal pain.

12.7 Investigations

In many instances, a careful history and physical examination provide the clinical diagnosis. Complete blood count (CBC) and urinalysis are routine.

Serum amylase or lipases, electrolytes, creatinine and glucose are frequently obtained. Other blood work is obtained as indicated. Chest and plain abdominal x-rays are obtained routinely unless the diagnosis is clear (e.g., appendicitis). Free intraperitoneal air, suggesting a perforated viscus, may be apparent on either. Abdominal x-rays can also provide information about the pattern of bowel gas (e.g., intestinal obstruction), edema and pneumatosis of the bowel wall, retroperitoneal structures (e.g., pancreatic calcification), bony structures (e.g., fractures, bone metastases).

More sophisticated diagnostic imaging is often valuable. Ultrasound is very useful in the diagnosis of biliary tract, abdominal aortic, and gynecologic disease and is often used in suspected appendicitis. Increasingly, abdominal CT scanning is being used for diagnosis of the acute abdomen, often obviating the need for more invasive or uncomfortable studies. A contrast enema may be obtained to show the level of a large bowel obstruction and to exclude pseudo-obstruction. Intravenous pyelography can demonstrate kidney nonfunction or hydronephrosis in suspected renal pain. An opaque calculus may be seen on plain abdominal x-rays. In suspected bowel ischemia, mesenteric angiography may be used to confirm the diagnosis and evaluate therapeutic options. In suspected diverticulitis, ultrasound and CT scanning will demonstrate diverticula and thickening of the wall of the sigmoid colon, and evaluate the presence of an associated abscess or gross perforation. The choice of investigation should be discussed with a radiologist.

Laparoscopy has an important diagnostic role, as well as allowing definitive surgical therapy (e.g., appendectomy, omental patch of a perforated duodenal ulcer).

12.8 Approach to Management

A reasonably specific diagnosis or focused differential can usually be established early on and is the ideal basis for determining further management. In some instances (e.g., possible appendicitis), careful observation with repeated examination and selected imaging studies (e.g., ultrasound) allow a diagnosis to be reached. In many individuals, acute abdominal pain of mild to moderate severity resolves, at least in the short term, without a confirmed diagnosis.

In patients with more serious conditions, intravenous fluid administration, other supportive measures and monitoring must be instituted following rapid initial assessment, even before a specific diagnosis can be made. In such individuals, diagnostic and therapeutic maneuvers must proceed in a coordinated and efficient manner. Occasionally, patients with an acute abdomen, typically those who are unstable despite resuscitation or who have obvious generalized peritonitis, require urgent laparotomy without a definitive preoperative diagnosis.

13. CHRONIC ABDOMEN / W.G. Thompson

13.1 Synonyms

Recurrent abdominal pain; recurrent abdominal pain in children.

13.2 Description

Ten percent of children suffer recurrent abdominal pain and approximately 20% of adults have abdominal pain at least six times per year unrelated to menstruation. The pain is chronic when it is continuous and has been present for six months or more, unrelated to gastrointestinal functions such as eating and defecation. It is often a feature of dyspepsia or the irritable bowel syndrome. Characteristically, the pain has no relationship to bodily functions, and no gastrointestinal, hepatobiliary, genital or renal cause for the pain can be found.

13.3 Causes and Mechanism

The mechanisms of abdominal pain are discussed above, in Section 12 (“Acute Abdomen”). Of course, chronic abdominal pain may be caused by many organic diseases. *Peptic ulcer* generally produces pain after meals or on an empty stomach and is relieved by food or antacid. Abdominal pain awakening the patient at night is a particularly discriminating feature. Peptic ulcers are now more common in the elderly, especially women on NSAIDs. In them the pain may be atypical.

Biliary colic may be due to cystic or common bile duct obstruction by a stone. Characteristically this pain is significant enough to awaken the patient at night or require a visit to the emergency room for analgesia. It lasts from 1 hour to 12 hours; beyond that time consider acute cholecystitis or pancreatitis. Attacks are sporadic and at intervals, not continuous. Biliary pain is located in the epigastrium, the right upper quadrant and/or the right scapula. It leaves the patient shaken but well. Should the gallbladder become inflamed, cholecystitis results. Obstruction of the common bile duct with a stone results in pain, jaundice and sometimes fever (cholangitis).

Pancreatitis is a devastating illness, with steady epigastric pain radiating to the back and sometimes accompanied by shock. It almost always requires admission to hospital.

Ischemic bowel disease, subacute bowel obstruction caused by Crohn’s disease, *neoplasm* or *volvulus* may present with recurrent bouts of abdominal pain, often related to eating. These conditions are usually progressive and accompanied by physical signs.

In a patient with *diverticular disease*, a peridiverticular abscess may develop, causing recurrent bouts of severe left lower quadrant abdominal pain and

fever. Usually, diverticula are asymptomatic and symptoms that do occur are those of coincident irritable bowel syndrome.

Renal colic due to a stone in the ureter is rarely chronic but may be recurrent. It consists of severe flank pain radiating to the groin and testicle, and may be accompanied by hematuria. Typically, a patient smitten with renal colic is unable to lie still.

Gynecologic conditions ranging from mittelschmerz (ruptured ovarian cyst) to pelvic inflammatory disease may account for recurrent abdominal pain. Menstruation-related pain in a young woman suggests endometriosis. Chronic pelvic pain often relates to the irritable bowel syndrome.

Chronic appendicitis probably does not exist.

The chronic abdomen is seldom explained by the above mechanisms. *Functional abdominal pain* may originate in any part of the gastrointestinal tract or biliary tree. It is unrelated to bodily function and may be continuous. The commonest cause of recurrent abdominal pain is the irritable bowel syndrome, in which there is a relationship to disordered defecation. It is uncertain whether such pain is due to a normal perception of abnormal gut motility or an abnormal perception of normal motility, or indeed if it is due to the gut at all; there are frequently accompanying psychosocial difficulties.

13.4 Important Historical Points and Physical Examination Features

Pain, when related to a bodily function – defecation, eating, micturition or menstruation – should focus the investigation upon the involved system. Certain physical findings (such as an abdominal mass, or blood or mass upon rectal examination) point to specific organic diseases. Fever, weight loss, rectal bleeding and/or anemia indicate further tests. These features are absent in chronic functional abdominal pain.

13.5 Differential Diagnosis, Diagnosis and Management

Management of the organic causes of the chronic abdomen can be directed at the underlying disease process. In many instances, however, there is no organic basis. Here, the physician's responsibility is to reassure the patient that no serious disease exists, and help the patient coexist with the symptoms in the light of the patient's social background. One might improve digestion through regular and better eating habits, and treat bowel dysfunction, particularly constipation, with increased dietary bulk.

13.6 Pain and Emotion

There are patients who have severe recurrent abdominal pain unrelated to bodily function or organic disease. Such patients see many doctors without satisfaction;

the genesis of the symptom is thought to be psychogenic. This pain is often given such descriptors as “illness behavior” and “pain proneness.” Some have hypochondriasis and do not improve when organic disease has been disproved. An extreme example is the Münchausen syndrome, where the patient deliberately relates a tall tale of medical duress in order to precipitate treatment, perhaps even surgery.

Functional pain is frequent in those who have recent conflicts, have experienced a death in the family, or have become overly concerned with fatal illness. Depression and anxiety are frequent. Here, it is important not to carry out extensive investigation in a fruitless search for an elusive cause. This only reinforces the patient’s belief that something is wrong and undermines the patient’s confidence in the benign diagnosis.

Such pain may be an emotional expression, in which case regular visits are necessary to allow the patient to vent his or her problems. Drugs, especially narcotics, should be used with restraint, and the physician should strive to develop a strong doctor–patient relationship while dealing with the patient’s depression, anxiety, frustration and often hostility. Some individuals benefit from low-dose antidepressants, as in other chronic pain syndromes. These patients test our skill in the art rather than the science of medicine.

14. JAUNDICE / L.J. Scully

14.1 Definition

A state characterized by increased serum bilirubin levels and a yellow appearance due to deposition of bile pigment in the skin and mucus membranes.

14.2 Mechanism

Bilirubin is a waste product of hemoglobin metabolism. Interruption of the breakdown pathway at any of a number of steps, or a marked increase in load due to red cell destruction, results in an increase in serum bilirubin and (if high enough) clinical jaundice.

Under normal circumstances senescent red blood cells are taken up and destroyed in the reticuloendothelial system. Through a number of steps the heme molecule of hemoglobin is converted to bilirubin and, tightly bound to albumin, is transported in the plasma to the liver cells. Hepatocytes take up bilirubin, conjugate it to glucuronide and excrete the bilirubin diglucuronide in bile into the duodenum. In the bowel, bacteria break down bilirubin to urobilinogen, 80% of which is excreted in the feces, contributing to the normal stool color. The remaining 20% is reabsorbed and excreted in bile and urine (enterohepatic circulation of urobilinogen).

Functional or anatomic obstruction at almost any level in this pathway (from hemoglobin breakdown to uptake by the hepatocellular membrane to excretion into the biliary system) will result in jaundice, with an increase in serum bilirubin. A large increase in the breakdown products of hemoglobin alone (e.g., hemolytic anemia) will cause an increase in serum unconjugated bilirubin. If the problem lies after the uptake and conjugation step, the increase is in serum conjugated bilirubin. Causes of jaundice are usually classified as: (1) hemolysis; (2) genetic defects in bilirubin handling; (3) hepatocellular disease; and (4) obstruction.

14.3 Clinical Presentation

Clinical jaundice is detected when the serum bilirubin level reaches 2–4 mg/dL (40–80 $\mu\text{mol/L}$). It is usually preceded by a few days of pale stools (as excretion of bilirubin into the intestine is decreased) and dark urine (due to increased glomerular filtration of conjugated bilirubin). Jaundice is usually first detected in the sclera, although the bilirubin is actually deposited in the overlying conjunctival membranes. Yellow skin without scleral icterus should suggest carotenemia or the ingestion of such drugs as quinacrine.

Most patients with jaundice, excluding those in whom it is secondary to hemolysis, have nausea, anorexia and discomfort over the liver. There may be hepatomegaly, masses in the epigastrium or pancreas or a dilated gallbladder. Signs of chronic liver disease such as spider nevi or palmar erythema are important. Pruritus may result, presumably from the deposition of bile salts (or a retained pruritogen normally excreted in bile) in the skin.

Several genetic defects in the conjugation or excretion of bilirubin may cause long-standing unconjugated or conjugated hyperbilirubinemia.

14.4 Approach to Diagnosis

Initially the most important information is whether the jaundice is due to conjugated or unconjugated hyperbilirubinemia (Figure 2). Serum bilirubin can be fractionated from “total” into conjugated and unconjugated, but the presence of bile in the urine determined by a test strip at the bedside confirms that the bilirubin rise is predominantly in the conjugated form. If the bilirubin is unconjugated, hemolysis or genetic defects are implicated. If the bilirubin is conjugated, “liver biochemical tests” (AST, ALT, GGT and alkaline phosphatase) will help determine if the jaundice is primarily due to obstruction/cholestasis (high GGT and alkaline phosphatase) or hepatocellular damage (high AST and ALT). Cholestatic jaundice requires ultrasound as the best, first test to detect biliary tract disease. If the jaundice is cholestatic, then an ultrasound of the abdomen is required to determine if there is obstruction of the ducts or intrahepatic bile duct dilation.

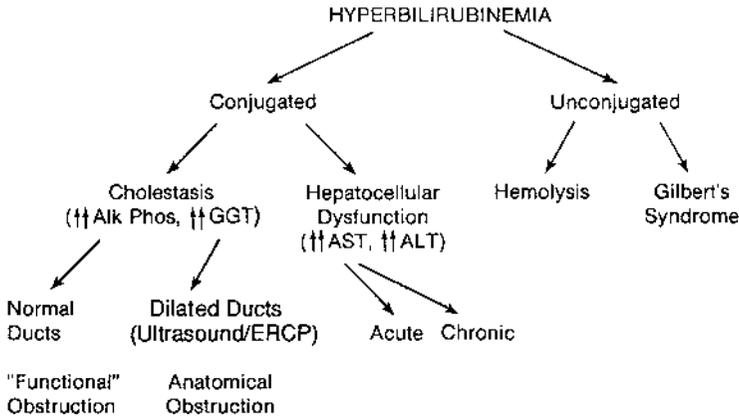


FIGURE 2. Causes of hyperbilirubinemia.

14.5 Management

The management of obstructive jaundice is directed toward the cause where possible (e.g., removal of obstructing gallstone). Jaundice secondary to hepatocellular disease, such as viral hepatitis, does not require any specific treatment. Jaundice due to alcohol, toxin or drug requires withdrawal of the offending agent.

15. ASCITES IN CHRONIC LIVER DISEASE / L.J. Scully

15.1 Definition

Ascites is the accumulation of nonsanguinous fluid in the peritoneal cavity.

15.2 Mechanisms

With significant liver disease, albumin synthesis is reduced. Low serum albumin results in a decrease in intravascular osmotic pressure. This causes renal blood flow changes, resulting in sodium and water retention. Increased aldosterone levels, possibly due to decreased catabolism of this hormone by the liver, also contribute. There is a generalized salt and water retention, but the fluid accumulation may be confined to the peritoneal cavity or may be associated with peripheral edema. Ascites develops because of increased portal pressure and the transudation of fluid from the capillaries in the portal system to the peritoneal cavity. Hepatic lymph production also increases and extravasates directly into the peritoneal cavity.

15.3 Signs and Symptoms

Ascites most commonly presents with increasing abdominal girth, often associated with an uncomfortable feeling of distention, and sometimes nausea and anorexia. Shortness of breath may develop, resulting from either elevation of the diaphragm or pleural effusion. Ankle edema may accompany ascites.

Clinical examination reveals flank fullness on inspection. “Shifting dullness” or a “fluid thrill” may be elicited. Smaller amounts of fluid may be detected on ultrasound when clinical signs are absent. One should look for other signs of portal hypertension, such as dilated abdominal wall veins or an enlarged spleen.

15.4 Differential Diagnosis

Newly developed ascites must have a diagnostic aspiration to determine the albumin level, cell count and cytology. The fluid should be clear and straw-colored. Occasionally, lymph can accumulate in the peritoneal cavity, causing “chylous ascites,” which requires different management. Ascitic fluid may become infected, in which case the white blood cell count will be elevated in the fluid. If the fluid is sanguinous, other causes – such as infection or malignancy – must be sought. The serum ascites albumin gradient is the best way of confirming if the ascitic fluid is secondary to portal hypertension. In this situation the gradient is high (i.e., > 11 g/L) whereas it is low if the ascites is due to peritoneal carcinomatosis. This is far more accurate than our previous assessment of transudative versus exudative ascites.

15.5 Approach to Management

Management initially includes bed rest and salt restriction. Most cases also require adding a diuretic such as spironolactone. Careful aspiration of large quantities (up to 8 L) of ascitic fluid may be necessary in some resistant cases; this can be safely performed, and if the serum albumin level is very low an intravenous infusion of albumin is given before the paracentesis.

16. GASTROINTESTINAL BLEEDING / A. Rostom and C. Dubé

16.1 Description

Gastrointestinal (GI) bleeding may be referred to as upper, lower, obscure or occult.

Upper GI bleeding commonly presents with hematemesis (vomiting of blood or coffee-ground like material) and/or melena (black, tarry stools). The physical aspect of melena is the result of degradation of blood by intestinal bacteria. In comparison, hematochezia (bright red or maroon colored blood or fresh clots per rectum) is usually a sign of lower GI bleeding. Lower GI bleeding is usually defined as bleeding distal to the ligament

TABLE 3. The important causes of upper GI bleeding

Acute bleeding

Peptic ulcer disease: duodenal ulcer, gastric ulcer, stress erosions

Esophagitis: peptic esophagitis, pill esophagitis, infectious

Portal hypertension-related: gastro-esophageal varices, portal hypertensive gastropathy

Neoplastic: esophageal cancer, gastric cancer, lymphoma, metastatic cancer

Vascular: angiodysplasia, Dieulafoy lesion, radiation-induced

Traumatic: Mallory-Weiss tear, aorto-enteric fistula, foreign body ingestion

Miscellaneous: hemobilia, hemosuccus pancreaticus

Chronic bleeding

Esophagitis

Portal hypertensive gastropathy

Malignancies

Angiodysplasia

Radiation

Inflammatory bowel disease

of Treitz. However this definition can cause confusion since proximal small bowel bleeds tend to be associated with elevated levels of blood urea nitrogen (BUN) which is classically associated with upper GI bleeding. Alternatively the location of GI bleeding can be defined as upper – above ligament of Treitz, small bowel – ligament of Treitz to distal ileum, and lower – terminal ileum and colon.

Occult bleeding is defined as the initial presentation of a positive fecal occult blood test (FOBT) result and/or iron-deficiency anemia (IDA), when there is no evidence of visible blood loss to the patient or physician. Obscure bleeding is defined as bleeding of unknown origin that persists or recurs after a negative initial or primary endoscopy (colonoscopy and/or upper endoscopy) result. Obscure bleeding may be so-called “obscure-overt,” (i.e., clinically manifest such as melena or hematochezia), or may be “obscure-occult,” such as persistent IDA.

The important causes of upper and lower GI bleeding are presented in Tables 3 and 4 respectively.

16.2 Approach to Diagnosis and Management

The initial evaluation of the patient with upper GI bleeding involves early assessment of the “ABCs.” Patients with upper GI bleeding are at risk of airway compromise from aspiration of vomited blood and/or from reduced level

TABLE 4. The important causes of lower GI bleeding

<i>Acute bleeding</i>
Diverticulosis
Angiodysplasia
Ischemic colitis
Inflammatory bowel disease
Colonic malignancy
Radiation-induced
Infectious enteritis or colitis
Solitary rectal ulcer syndrome
Post-polypectomy
Hemorrhoids
<i>Chronic bleeding</i>
Angiodysplasia
Colonic malignancy
Radiation-induced
Inflammatory bowel disease
Hemorrhoids

of consciousness due to shock or hepatic encephalopathy. Early assessment of the airway and breathing is crucial; some patients may require supplemental oxygen or even intubation for airway protection and/or assisted breathing. During the early assessment of the hemodynamic status, two large bore peripheral IVs (18 gauge or greater) are placed for fluid and blood product restoration, and their early placement allows for the simultaneous drawing of blood for urgent blood typing and cross-matching, and measurement of the blood count, coagulation parameters, electrolyte and renal function as well as albumin and liver enzymes as clinically indicated.

In chronic or recurrent bleeding, either occult or clinically manifest, symptoms are most often related to iron deficiency and anemia: pallor, fatigue, dyspnea and, in a predisposed individual, congestive heart failure or angina.

In acute GI bleeding, the symptoms associated with blood loss, such as weakness, diaphoresis, pre-syncope, and syncope, may occur before any blood appears externally. Blood passage through the intestines acts as a cathartic, so that hyperperistalsis may be present and bowel frequency is increased. The pigmentation of the stool will depend on the rate of bleeding as well as the length of time in transit along the bowel: stools can vary from a deep black tarry aspect to a dark burgundy or to a bright red color. In determining the likely source of bleeding, the clinician needs to interpret the patient's manifestations of bleeding in conjunction

with the hemodynamic status. Blood originating from the left colon typically is bright red. However, hematochezia associated with resting tachycardia and/or hypotension raises the suspicion of a brisk upper GI bleed; similarly, while the passage of black tarry stools is most commonly associated with an upper GI source, dark burgundy or black stools can sometimes be encountered in proximal colonic bleeds. In the absence of spontaneous passage of stools, a digital rectal examination to determine the stool color will be most informative.

Hematemesis, when present, may be apparent as bright red blood, with or without clots, or of a dark brown and granular appearance, so-called coffee-grounds. Bright red emesis is suggestive of an esophageal source of bleed or of a brisk upper GI source. Bleeding into the duodenum may or may not reflux into the stomach, therefore the absence of hematemesis or of a bloody aspirate from nasogastric suction does not rule out that condition.

Under certain circumstances it may be difficult to determine if the GI bleed, particularly if it is significant, is of upper or lower origin. It is then safest to initially proceed on the assumption of an upper GI bleed, and to arrange for early upper endoscopy after initial resuscitation. Upper endoscopy is quick to perform, allows relatively easier endoscopic treatment of potentially serious causes of bleeding, and if negative, little time has been wasted. Furthermore, a negative upper endoscopy in the setting of ongoing rapid GI bleeding assists the angiographer, by clearing the celiac axis and allowing them to concentrate on the superior and inferior mesenteric arteries.

On history, information should be gathered about medication use, in particular the intake of NSAIDs or anticoagulants, prior history of peptic ulcer disease, history of abdominal surgery (e.g., gastric surgery raises the suspicion of prior peptic ulcer disease or carcinoma, bowel resection may reduce the transit time of blood through the gut leading to atypical presentations, vascular grafts raise the suspicion of aorto-enteric fistulas), history of chronic liver disease or alcohol abuse which might also be supported by suggestive physical signs.

The hemodynamic status should be interpreted in light of the patient's abilities to compensate for hypovolemia: in a young and fit adult, the presence of a resting or orthostatic tachycardia should be interpreted as a sign of significant volume loss, while the loss of an equivalent blood volume in an elderly or debilitated subject would more likely be manifested by hypotension or shock.

Initial investigations include a complete blood count (CBC), red cell indices, partial thromboplastin time (PTT) and international normalized ratio (INR), as well as urea and creatinine. It is important to remember that, acutely, the hemoglobin (Hb) and hematocrit (Ht) may not be reduced from normal, since these measures reflect the red blood cell (RBC) concentration in the blood. Over the ensuing 36–48 hours, most of the volume deficit will be repaired by the movement of fluid from the extravascular into the intravascular space. Only at these

later times will the Hb and Ht reflect the true degree of blood loss. Furthermore, if a patient presents with an acute GI bleed and the initial Hb is low, one should expect that the actual Hb is even lower than that measured, so that early blood transfusion would be advisable in such cases. Some patients, in particular those with GI malignancies, may have had chronic occult bleeding prior to their acute presentation, in which cases hypochromia and microcytosis from iron deficiency may be observed. Coagulopathy, either iatrogenic or secondary to liver failure, should be addressed and corrected as clinically indicated. An elevated blood urea nitrogen (BUN) value in the presence of a normal creatinine may result from an upper GI bleed with hypovolemia.

Patients should be categorized as either low or high risk for complications based upon their clinical presentation and hemodynamic status.

In upper GI bleeding, clinical predictors of mortality are: age older than 60 years, shock, poor overall health status, comorbid illnesses (coronary artery disease, renal failure, sepsis and/or onset of bleeding while hospitalized for another reason), presence of fresh red blood on rectal examination, in the emesis or in the nasogastric aspirate, as well as continued bleeding or rebleeding. In such cases, early endoscopy with risk classification by clinical and endoscopic criteria allows for safe and prompt discharge of patients classified as low risk, improves outcomes of patients classified as high risk and reduces resource utilization for patients classified as either low or high risk. In peptic ulcer bleeding, endoscopic criteria of rebleeding include active bleeding, a visible vessel, or adherent clot. Early administration of intravenous proton pump inhibitors, in conjunction with endoscopic therapy, is beneficial in such cases. In variceal bleeding, intravenous administration of somatostatin analogs and endoscopic therapy are also beneficial.

In lower GI bleeding, patients who have been successfully resuscitated should undergo bowel cleansing followed by colonoscopy. If these procedures are not feasible due to ongoing hemodynamic instability, arteriography and surgical consultation should be obtained.

17. ABDOMINAL MASS / S. Grégoire

17.1 Description

When an abdominal mass is discovered on physical examination, one must define its nature. Using a systematic approach often permits the identification of the mass before the use of sophisticated tests.

17.2 Important Points in History and Physical Examination

Important clues in the history and general physical examination may help to identify the enlarged viscus. For example, in a young patient presenting with

diarrhea, weight loss and abdominal pain, finding a right lower quadrant mass would suggest inflammatory bowel disease. However, an abdominal mass may be discovered during physical examination of an asymptomatic individual. Certain observations made during the abdominal examination may be helpful (See also Section 19).

17.2.1 INSPECTION

Where is the mass located? A practical approach is to divide the abdomen into four quadrants (See Section 19.1). Starting from the principle that an abdominal mass originates from an organ, surface anatomy may suggest which one is enlarged. A mass seen in the left lower quadrant, for example, could be of colonic or ovarian origin but, unless there is situs inversus, one would not consider an appendiceal abscess.

Does the mass move with respiration? In the upper abdomen a mobile intra-abdominal mass will move downward with inspiration, while a more fixed organ (e.g., aorta, pancreas) or an abdominal wall mass (e.g., hematoma of rectus muscle) will not.

Is there visible peristalsis?

17.2.2 AUSCULTATION

Careful auscultation for bowel sounds, bruit or rub over an abdominal mass is part of the systematic approach.

17.2.3 DEFINING THE CONTOUR AND SURFACE OF THE MASS

This is achieved by inspection, percussion and palpation. Is the organ air-filled (e.g., stomach) or fluid-filled? Is it a well-defined mass (e.g., liver, spleen) or are its borders difficult to define (matted loops of small bowel)? Is the surface regular? An enlarged liver due to fatty infiltration may have a smooth surface, while a cirrhotic organ is usually irregular and nodular. What is the consistency of the mass? Firm? Hard or soft? Is it pulsatile? In the absence of ascites, ballotement of an organ situated in either upper quadrant more likely identifies an enlarged kidney (more posterior structure) than hepatomegaly or splenomegaly.

17.3 Differential Diagnosis

The following suggests an approach to the differential diagnosis of an abdominal mass located in each quadrant:

17.3.1 RIGHT UPPER QUADRANT

This location suggests liver, right kidney, gallbladder and, less commonly, a colon or gastroduodenal mass. A pancreatic mass is rarely palpable.

17.3.1.1 Liver

As a subdiaphragmatic organ, the liver moves downward with inspiration. This anterior organ has an easily palpable lower border, which permits assessment of its consistency. A bruit or venous hum can be heard in certain conditions. An enlarged left lobe can usually be felt in the epigastric area.

17.3.1.2 Right kidney

The kidney may protrude anteriorly when enlarged and be difficult to differentiate from a Riedel's lobe of the liver. It may be balloted.

17.3.1.3 Gallbladder

This oval-shaped organ moves downward with inspiration and is usually smooth and regular.

17.3.1.4 Colon

Colon masses are deep and ill-defined, and do not move with respiration. High-pitched bowel sounds suggest obstruction.

17.3.2 LEFT UPPER QUADRANT

Location in the left upper quadrant suggests spleen or left kidney. Less commonly, a colonic (splenic flexure) or gastric mass can be felt. A pancreatic mass is rarely palpable.

17.3.2.1 Spleen

This anterior organ moves downward with inspiration. Since it has an oblique longitudinal axis, it extends toward the right lower quadrant when enlarged. It has a medial notch and the edge is sharp.

17.3.2.2 Left kidney

Its more posterior position and the presence of ballottement helps distinguish the left kidney from the spleen.

17.3.2.3 Colon, pancreas, stomach

It is practically impossible to differentiate masses in these organs by physical examination. The history helps but often one must resort to radiology or endoscopy.

17.3.3 RIGHT LOWER QUADRANT

A mass in this area has its origin either in the lower GI tract (colon, distal small bowel, appendix) or in a pelvic structure (ovary, uterus, fallopian tube).

17.3.3.1 Lower GI tract

These deeper organs are usually ill-defined. Clinical context is important. Inflammatory bowel disease usually would be associated with pain on palpation but carcinoma of the cecum would be painless.

17.3.3.2 Pelvic organs

Bimanual palpation is the preferred method.

17.3.4 LEFT LOWER QUADRANT

As with a right lower quadrant mass, the differential diagnosis here is between lower GI (in this quadrant the sigmoid colon) and pelvic origin. The shape of the organ and pelvic examination should help differentiate the two.

17.4 Approach to Diagnosis

To complete the assessment of an abdominal mass, one may choose among several different investigational tools. The use of specific tests depends on availability and on the organ studied.

Generally, ultrasound is useful. This noninvasive, safe, cheap and widely available method identifies the mass and provides information on its origin and nature. Ultrasound may also be used to direct a biopsy. Other noninvasive modalities are nuclear imaging and CT scan. Hollow organs may be demonstrated radiographically through the use of contrast media (e.g., barium enema, GI series, ultrasound, intravenous pyelogram, endoscopic retrograde cholangiopancreatography, etc.). Sometimes, laparotomy or laparoscopy will be necessary to make the diagnosis.

18. PROCTALGIA FUGAX / W.G. Thompson

18.1 Description

Proctalgia fugax is a sudden severe pain in the anus lasting several seconds or minutes and then disappearing completely.

18.2 Mechanism

The pathophysiology of proctalgia fugax is uncertain. Although some observations (under obviously fortuitous circumstances) suggest a rectal motility disorder, the symptom appears more likely to result from spasm of the skeletal muscle of the pelvic floor (specifically, the puborectalis).

18.3 History and Physical Examination

Proctalgia fugax occurs in about 14% of adults and is somewhat more common in females than males. The pain may be excruciating, but since it is so

short-lived patients seldom report it to their physician. In 90% of instances it lasts less than five minutes and in many cases less than a minute. About one-third of patients suffer attacks following defecation. A small minority report attacks following sexual activity. There are no physical signs.

18.4 Differential Diagnosis

Perianal disease may cause pain but it usually accompanies, rather than follows, defecation. One should be particularly careful to exclude the presence of an anal fissure, which may be difficult to see on anal inspection. Pain originating from the coccyx may be accompanied by coccygeal tenderness both externally and from within the rectum. An acute attack of anal pain lasting several hours may indicate a thrombosed hemorrhoid. However, constant pain for many months or years is not likely to be proctalgia fugax or to have an organic explanation.

18.5 Management

Beyond reassurance there is no treatment.

19. EXAMINATION OF THE ABDOMEN / R.F. Burseley, J.M. Fardy, D.G. MacIntosh and N. Saloojee

Examination of the abdomen is an important component of the clinical assessment of anyone presenting with suspected disease of the gastrointestinal tract. As in all other parts of the examination, care must be taken to show respect and concern for the patient while ensuring an appropriate and thorough examination.

While performing the examination it is useful to keep in mind the concepts of sensitivity and specificity. How confident can we be that a suspected physical finding is in fact present and has clinical significance? For example, how sensitive and specific is our bedside examination for hepatomegaly? What is the clinical significance of an epigastric bruit heard in a thin 20-year-old female versus a 55-year-old hypertensive, obese male?

In the following sections we will describe an appropriate sequential examination of the abdomen and highlight some of the potential pitfalls of this process.

19.1 Inspection

Start from the usual position to the right side of the patient. Ensure that the abdomen is exposed from the costal margin to symphysis pubis. When describing the location of an abnormality it is useful to divide the abdomen into four quadrants with a perpendicular line through the umbilicus from the xiphoid process to the symphysis pubis. A horizontal line through the umbilicus

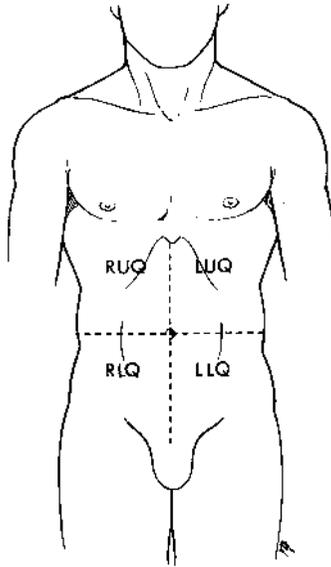


FIGURE 3. Division of the abdomen into four quadrants: the left upper quadrant, right upper quadrant, left lower quadrant and right lower quadrant.

then allows the abdomen to be divided into 4 areas: the left upper, right upper, left lower and right lower quadrants (Figure 3). On occasion it may be helpful to divide the abdomen into 9 regions with the spaces marked by vertical lines through the left and right mid-clavicular lines and horizontal lines passing through the subcostal margins and anterior iliac crests (Figure 4).

The overall appearance such as scaphoid, protruberant, or obese should be described, and the location of any surgical scars noted. One should look for any abnormal surface markings, including cutaneous lesions as well as vascular markings. Note any pulsation. A comment should also be made about the apparent ease of movement of the abdominal wall with respiration and change in body position. Normally the abdominal wall will rise with inspiration. Occasionally organomegaly or a mass will be visible. It is helpful to look at the abdomen from the foot of the bed as well.

19.2 Auscultation

It is useful to auscultate the abdomen prior to palpation or percussion, as bowel sounds induced by further examination may mask vascular bruits or pleural rubs. When listening for vascular bruits it is useful to keep in mind

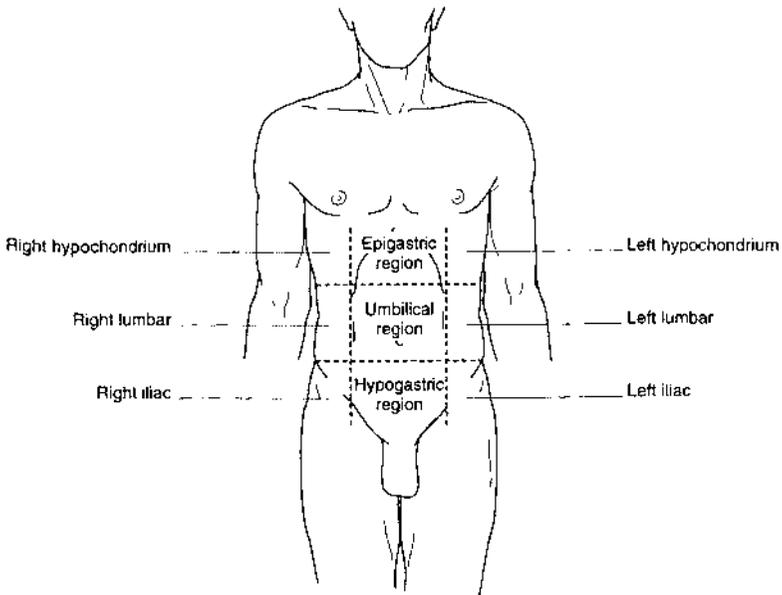


FIGURE 4. Division of the abdomen into nine regions.

the surface markings. The aorta enters the abdomen at or just to the left of the xiphoid process and bifurcates to the left and right common iliac arteries at the level of the umbilicus. The renal arteries are found approximately one-half the distance between the xiphoid process and the umbilicus, and auscultation is best performed within 2 cm of the midline. Bruits are often best heard at the bifurcation of major vessels; therefore it is also appropriate to listen at the bifurcation of the common iliacs into the internal and external iliacs, approximately halfway between the umbilicus and the inguinal ligament. One should listen over the inguinal ligament for femoral bruits as well.

A venous hum is best heard overlying the portal vein, which is found in an area approximated by an elliptical shape between the umbilicus and the mid-clavicular line where it crosses the right subcostal margin. Arterial bruits are usually heard only during systole and best heard with the diaphragm of the stethoscope, as they are high pitched. A venous hum is more likely to be continuous and best heard with the bell of the stethoscope, as this is a low-pitched sound. There are, however, no studies to suggest these findings are helpful or reliable in routine examination. Venous hum can occur in portal venous hypertension of any cause. Undifferentiated liver patients in one study

had a prevalence of bruits reported as less than 3%. The ability of clinicians to distinguish hepatic arterial bruits from other arterial bruits such as a renal artery bruit has not been studied.

Friction rubs may occur overlying the liver or spleen and are always abnormal, though rare. Even with careful auscultation of patients with known liver tumors, fewer than 10% are found to have a rub.

19.2.1 BOWEL SOUNDS

Auscultation for bowel sounds is a rather controversial subject. Bowel sounds should be listened for prior to palpation or percussion, but the yield of this examination is low. The diaphragm of the stethoscope should be placed on the abdomen, as least initially in the right lower quadrant near the ileocecal valve. The particular characteristics of the bowel sounds or even absence of them is not diagnostic of a particular condition, perhaps except for the very high pitched noises of acute small bowel obstruction. Complete absence of bowel sounds may indicate an ileus or peritonitis.

19.3 Palpation

Palpation of the abdomen should be done in an orderly sequence with the patient in the supine position. Light palpation should be done first in all four quadrants assessing for areas of potential tenderness. Light palpation is a one-handed technique. With the pads of the fingertips, palpate in a gentle, circular motion. If no areas of obvious tenderness are elicited, then deep palpation is performed, again in all four quadrants using a two-handed technique. Pressure is applied with one hand over the other hand, which is placed on the abdominal wall, as it is thought that deep palpation with one hand may lead to the inadvertent nonrecognition of subtle fullness or mass if the hand applying deep pressure is also responsible for detecting the abnormality. The accuracy of this is untested. It is stated that if a patient has difficulty relaxing the abdominal wall musculature, then placing the soles of the patient's feet on the bed with hips and knees flexed will aid relaxation; in all likelihood, however, a calm, organized approach with verbal reassurance by the examiner will be just as effective. Assess for peritoneal irritation in each quadrant by checking for rebound tenderness. Press the fingertips in slowly and firmly. Quickly withdraw them. If withdrawal elicits pain, this suggests peritoneal irritation.

The techniques of palpation of liver and spleen are discussed in Sections 19.5 and 19.6.

19.4 Percussion

Percussion of the abdomen will detect the presence of bowel gas. The technique as it relates to defining organomegaly and the presence of fluid is discussed in later sections.

19.5 Examination of the Liver

Examination of the liver consists mainly of palpation for the lower edge of the liver and percussion to determine the span. This examination is performed after inspection for right upper quadrant swelling and extrahepatic signs of liver disease. To palpate the lower edge of the liver the examiner starts with gentle pressure in the right lower quadrant of the abdomen. The edge of an enlarged liver may be missed by starting too high in the abdomen. The patient is asked to breathe gently and slowly, in order to bring the liver edge down to the examining fingertips of the right hand. The examiner moves the right hand in a cephalad direction about 2 cm with each breath. If the edge is not felt, no further examination is required. If liver disease is suspected the lower liver edge can be located by percussion.

If the edge is located, mark the lower border in the mid-clavicular line. Percuss for the upper border starting in the third intercostal space with a finger that is held flat and lies within the space. Move down one interspace at a time until the percussion note changes from resonant to dull. To confirm the change of percussion note strike the third and fourth fingers laid in adjacent interspaces. The note on the top finger should be resonant and on the lower dull. Measure the distance between the upper and lower percussion edges in the mid-clavicular line. Determination of the liver span can be done with firm or gentle percussion to locate the lower border. Gentle percussion is the recommended technique, as this method appears to better estimate liver span as judged by ultrasound. Remember that the upper edge of the liver is dome shaped and not straight across.

The scratch test has been used to find the lower liver margin. The diaphragm of the stethoscope is placed at the right costal margin in the mid-clavicular line. A finger moves up the abdomen in the mid-clavicular line, scratching gently and with consistent pressure. When the liver edge is reached, there is a sudden increase in the scratching sound heard through the stethoscope. In one comparative study the scratch test was not felt to offer any advantage over the techniques of palpation and percussion.

When the liver edge is palpable, trace the edge working laterally to medially. Try to determine the characteristics of its surface – for example, soft, firm or nodular. These characteristics may help in the assessment of patients with liver disease; however, agreement about the characteristics is poor, even among experts. Auscultation is rarely helpful. An attempt should be made to assess the left lobe in the epigastrium using these techniques.

What is the significance of a palpable liver edge? A recent review suggested that a palpable liver is not necessarily enlarged or diseased. When clinical examination is compared to nuclear medicine scanning, about one-half of palpable livers are not enlarged. The inability to feel a liver edge does not rule out hepatomegaly, but does reduce its likelihood.

What is the normal percussion span? Only one study has been done to establish the normal span. Castell examined 116 healthy subjects using firm percussion. The mean span in the mid-clavicular line was 7 cm in women and 10.5 cm in men. The following nomograms were developed to predict estimated liver dullness in a normal population using firm percussion technique: Male liver dullness equals $(0.032 \times \text{weight in pounds}) + (0.183 \times \text{height in inches}) - 7.86$. The female liver dullness equals $(0.027 \times \text{weight in pounds}) + (0.22 \times \text{height in inches}) - 10.75$. The 95% confidence intervals were ± 2.64 cm. Therefore a 5 ft. 10 in., 175 lb. male would have an estimated liver span of 10.2 cm (range 7.6–12.8) and a 5 ft. 5 in., 130 lb. female would have an estimated liver span of 7.1 cm (4.5–9.7 cm) by this formula.

19.6 Examination of the Spleen

The normal spleen is a curved, wedge-shaped organ located beneath the rib cage in the upper left quadrant. The spleen lies beneath the left tenth rib and normally weighs about 150 g, measuring approximately 12 cm in length, 7 cm in width and 3 cm in thickness. The normal spleen usually cannot be palpated, but as it enlarges it descends below the rib cage, across the abdomen toward the right lower quadrant. An enlarged spleen may have a palpable notch along its medial edge.

Examination of the spleen should begin with observation of the left upper quadrant for an obvious mass, though such a mass is quite uncommon. The examiner should then proceed with percussion over the area of the spleen to look for evidence of dullness, implying splenic enlargement. The two most useful methods are percussion over Traube's space and Castell's sign.

The surface markings for Traube's space are the left sixth rib, the left mid-axillary line and the left costal margin. An enlarged spleen may cause dullness over Traube's space. Percussion should be carried out at one or more levels of Traube's space from medial to lateral. This maneuver has a sensitivity and specificity between 60 and 70% for splenic enlargement; however, the sensitivity and specificity increases to approximately 80% in non-obese patients who are fasting.

Castell's method involves percussion in the lowest intercostal space in the left anterior axillary line. In normal individuals this area is resonant on percussion and remains resonant on inspiration. In patients with mild splenic enlargement this area will be resonant on percussion and become dull on maximal inspiration. This method has a sensitivity and specificity of approximately 80% for detection of splenic enlargement and would seem particularly suited for detection of a minimally enlarged spleen that may not be palpable.

Palpation of the spleen should begin in the right lower quadrant and proceed toward the left upper quadrant in order to follow the path of splenic

enlargement. Palpation should initially be carried out in the supine position with a bimanual technique using the left hand to gently lift the lowermost portion of the left rib cage anteriorly. The fingertips of the right hand are used to palpate gently for the spleen tip on inspiration. The hand is moved from the right lower quadrant, advancing toward the left upper quadrant. If the spleen is not palpated in the supine position the patient should be moved into the right lateral decubitus position and again with bimanual technique the spleen tip should be sought using the fingertips of the right hand on inspiration. This technique has a sensitivity of about 70% and specificity of 90% for splenic enlargement.

19.7 Examination for Suspected Ascites

The presence of ascites – free fluid within the abdominal cavity – is always due to an underlying pathological process. Most often the underlying etiology is cirrhosis of any type. Other potential causes include severe right-sided heart failure, lymphatic obstruction, primary intra-abdominal malignancy and peritoneal metastases. It is easy to identify large-volume ascites clinically, but the sensitivity of the examination techniques falls with lower volumes of fluid. Ultrasound, which can detect as little as 100 mL of free fluid, is the gold standard against which the clinical diagnostic maneuvers are compared.

An approach involves inspection for bulging flanks, followed by palpation for the presence or absence of fluid waves combined with percussion to demonstrate flank dullness as well as shifting dullness. One has to be aware that adipose tissue in the flanks may be occasionally mistaken for free fluid. To demonstrate a fluid wave it is necessary to enlist the aid of the patient or another individual. With the patient in the supine position, place one hand on the patient's flank. With the other hand briskly tap the other flank. A third hand is placed in the mid-abdomen with sufficient pressure applied to dampen any wave that may pass through adipose tissue in the anterior abdominal wall. If fluid is present a shock wave will be felt with the palpating hand. The sensitivity of this technique is approximately 50% but it has a specificity of greater than 80%.

When percussing for free fluid one should place the finger parallel to the expected edge and percuss from resonance in the mid-abdomen to dullness in the flanks. This area is then marked and the patient rolled to the opposite side. For example, if flank dullness is demonstrated on the left then the patient should be rolled onto the right side. One should allow approximately 30 seconds for the fluid to move between the mesentery and loops of bowel into the inferior portion of the abdomen. The previous area of dullness in the left flank should now be resonant. It does not matter which side one chooses to start with. In three separate studies shifting dullness had a sensitivity that ranged from 60–88% and a specificity that ranged from 56–90%.

In one study involving six gastroenterologists and 50 hospitalized alcoholic patients, the overall agreement was 75% for the presence or absence of ascites and reached 95% among senior physicians.

Interestingly, symptoms are often as useful as physical examination techniques for the clinical diagnosis of ascites. The most useful findings to make a diagnosis of ascites are a positive fluid wave, shifting dullness or peripheral edema. The absence of these findings is useful in ruling out ascites, as is a negative history of ankle swelling or increasing abdominal girth.

SUGGESTED READING LIST

Section 19 Examination of the Abdomen

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Practice points

- Gastrointestinal complaints are common in the general population.
- Stress factors and fears of underlying malignancies are important triggers for complaints to be brought to medical attention.
- Thorough and careful history-taking is crucial in gastroenterology and no amount of endoscopic and radiologic investigations can replace it as a diagnostic tool.
- Interpretation of the clinical manifestations of gastrointestinal disorders requires a sound knowledge and understanding of gastrointestinal physiology.
- Gastrointestinal symptoms should be interpreted in light of their relation to diet, food ingestion, digestion and defecation.
- Symptoms and signs such as weight loss, gastrointestinal bleeding, jaundice or anemia should alert the clinician to the possibility of underlying organic conditions and warrant investigations.
- Family history is an important part of the anamnesis; in particular, a family history of bowel cancer, gastric cancer, celiac disease or hemochromatosis should prompt screening tests as clinically appropriate.