Acute upper gastrointestinal bleeding

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بیمارستان ۴۷۹۰۱۱۱۸ (۰۲۱)

ESSENTIALS OF DIAGNOSIS

- Symptoms: Coffee ground vomiting, hematemesis, melena, hematochezia, anemic symptoms
- Past medical history: Liver cirrhosis, use of non-steroidal anti-inflammatory drugs
- Signs: Hypotension, tachycardia, pallor, altered mental status, melena or blood per rectum, decreased urine output
- Bloods: Anemia, raised urea, high urea to creatinine ratio
- Endoscopy: Ulcers, varices, Mallory-Weiss tear, erosive disease, neoplasms, vascular ectasia, and vascular malformations

Causes

- Peptic ulcer (35–50%).
- Mallory–Weiss tear (5–15%).
- Gastroduodenal erosions (8–15%).
- Oesophagitis (5–15%).
- Gastro-oesophageal varices (7–10%).
- Vascular malformations (5%):
  - Hereditary haemorrhagic telangiectasia.
  - Gastric antral vascular ectasia (GAVE).
  - Portal hypertensive gastropathy.
  - Angiodysplasia.
  - Dieulafoy lesion.
- Rare miscellaneous causes (5%):
  - Hiatus hernia

tment

- monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place)
- T give patient anything by mouth
- sh two large bore IV lines (16 gauge or larger)
- e supplemental oxygen
- ypotension initially with rapid, bolus infusions of isotonic crystalloid
- use for:
  - dynamic instability despite crystalloid resuscitation
  - globin <10 g/dL (100 g/L) in high-risk patients (eg, elderly, coronary artery disease)
  - globin <7 g/dL (70 g/L) in low-risk patients
  - over-transfusion with possible variceal bleeding
- fresh frozen plasma for coagulopathy; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin
- immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleed
- acotherapy for all patients with suspected or known severe bleeding:
  - a proton pump inhibitor (eg, Esomeprazole 80 mg IV bolus, followed by 8 mg/hour OR Pantoprazole 80 mg IV bolus, followed by 8 mg/hour
- acotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:
  - somatostatin or an analogue (eg, Octreotide 50 mcg bolus, followed by 50 mcg/hour infusion)
  - an antibiotic (eg, Ceftriaxone, Amoxicillin-clavulanate, or Quinolone)
- n tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using
  nore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior

Initial management

abilize the patient

- *Protect the airway*. Position patient on left side if actively vomiting.
- *Rapidly assess circulatory status*. Feel temperature of hands and feet. Does the patient look unwell? Is there pallor or sweating? Measure BP (including postural drop: significant if >20mmHg drop in systolic pressure on standing) and HR.
- *IV access*. Insert two large IV cannulae (e.g. 14–16G). Jugular, subclavian, or femoral vein cannulation may be necessary to assess CVP or if peripheral access limited. If the patient is shocked (systol BP <100mmHg, HR >100/min) or has other signs of hypovolaemia (such as pallor, sweating, cold peripheries, weak pulse, or postural hypotension), infuse 1L of 0.9% saline or 500mL colloid (e.g. Gelofusine®) 'stat'.

توجه

- Hb and PCV do not fall till the plasma volume has been restored, but if low at presentation suggest massive blood loss or acute-on-chronic bleeding.
- WCC may be elevated but usually is <15 x 10^9/L. If elevated, look for evidence of sepsis, which can predispose to haemorrhage.
- Low platelet count may suggest hypersplenism and chronic liver disease. Other causes of thrombocytopaenia may predispose to GI bleeding.
- An elevated plasma urea out of proportion to plasma creatinine indicates renal hypoperfusion or the absorption of blood proteins from the gut. It signifies a significant GI bleed or dehydration. A ratio of (urea (mmol/L) x 100) divided by creatinine (μmol/L) of >7.0 indicates that the urea is disproportionately high.

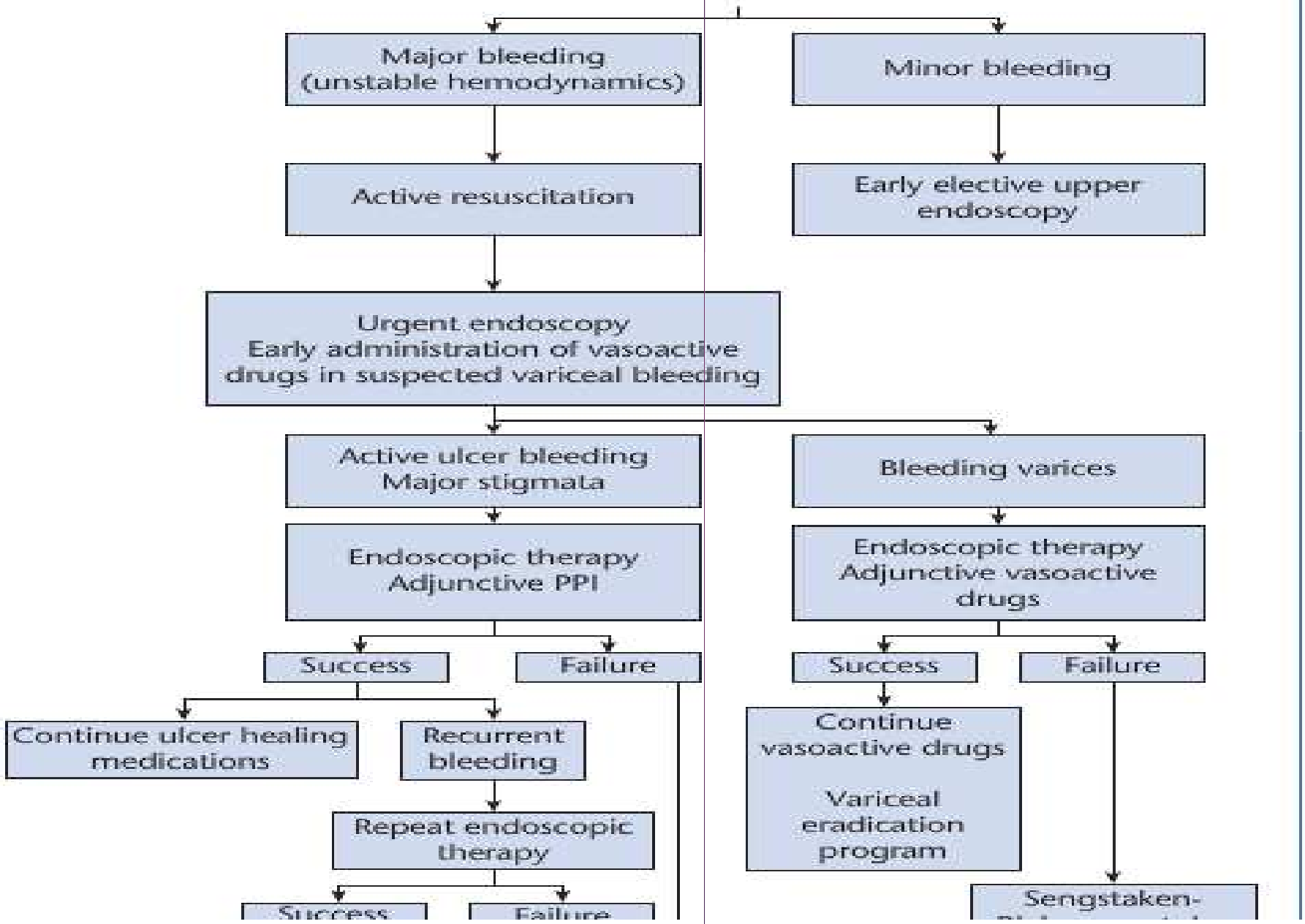
store circulating volume

chycardia, hypotension, or postural hypotension suggest low intrav
ar volume. Initially, give 1–2L of crystalloid (0.9% saline or Hartman
ution) or 500mL–1L of colloid (e.g. Haemaccel® or Gelofusine®) 'stat
re are no signs of hypovolaemia, use a slower rate of infusion. Contin

ood transfusion

ood transfusion is indicated in moderate or massive, but not minor, haem
rhage (e.g. Hb >10g/dL after fluid resuscitation). Give blood at 1 unit
til the circulating volume is restored or the CVP is between 5–10cm
easured from the mid-axilla with the patient supine. O-negative bloo
n be transfused immediately in massive bleeds. Serum calcium may f
er several units of citrate-containing blood. Give 10mL (4.5mEq) of 10
lcium gluconate for every 3–4U transfused. Supplement magnesium a
osphate as necessary (often low in alcoholics).

Algorithm for management of acute GI bleeding



Glasgow Blatchford bleeding score – admission risk markers and

n risk markers	Score value
ea (mmol/L)	
	2
	3
9	4
	6
bin for men (g/L)	
	1
	3
	6

Hemoglobin for women (g/L)	
100–119	1
<100	6
Systolic blood pressure (mmHg)	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥ 100/min	1
Presentation with melaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

.2 Hypovolemic shock: symptoms, signs and fluid replacement

loss (mL)	<750	750–1500	1500–2000	>2000
loss (%)	<15	15–30	30–40	>40
ate	<100	>100	>120	>140
pressure	Normal	Normal	Decreased	Decreased
ressure	Normal or increased	Decreased	Decreased	Decreased
tory rate	14–20	20–30	30–40	>35
utput (mL)	>30	20–30	5–15	Negligible
status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and let
eplacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and b

20.3 Bleeding ulcers: prevalence, risk, and need for surgery

scopic characteristics	Prevalence (%)	Further bleeding (%)	Surgery (%)	Mortalit
base	42	5	0.5	2
pot	20	10	6	3
rent clot	17	22	10	7
leeding visible vessel	17	43	34	11
e bleeding	18	55	35	11

Endoscopic view of a clean based antral gastric ulcer ir
g a nonsteroidal anti-inflammatory drug. Tests for infecti
acter pylori were negative.

Bahavar Medicine Lit

Figure 53-2. Endoscopic appearance of a gastric ulcer at the angularis with a flat spot (Forrest type IIc).

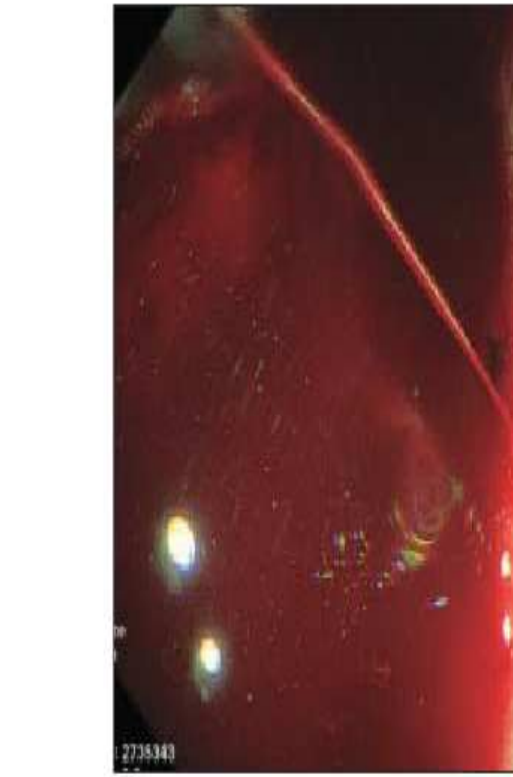
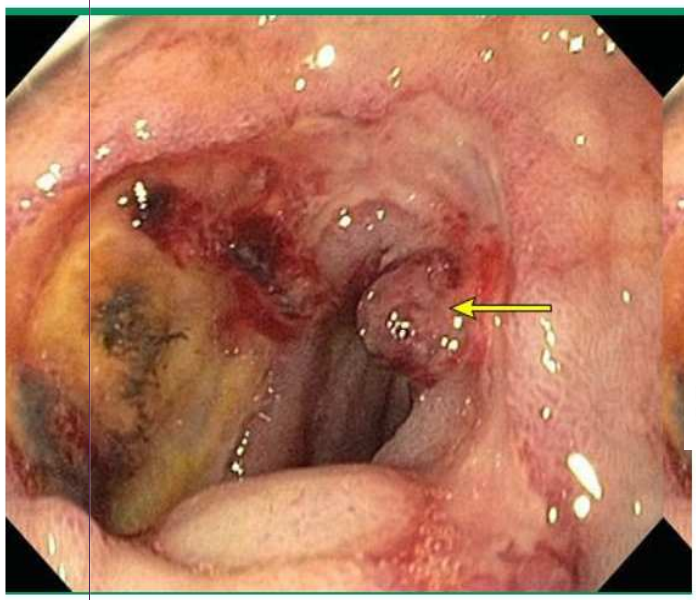


Figure 20.1 Ulcer with spurting hemorrhage.

ulcer with adherent clot

doscopy showing a gastric ulcer with an adherent clot (Forrest
tion IIb).

Duodenal ulcer with visible vessel



Upper endoscopy showing a duodenal ulcer with a nonbleeding visible vessel in a large circumferential ulcer (Forrest classification IIa)

Acute lower gastrointestinal bleeding

ESSENTIALS OF TREATMENT OF SEVERE HEMATOCHEZIA

- Initial resuscitation in a monitored care setting
- Colonoscopy to provide both diagnosis and therapeutic hemostasis of focal lesions (with epinephrine-saline injection, hemoclips, multipolar electrocoagulation), angiomas (with MPEC) and internal hemorrhoids (with band ligation)
- Angiography with transcatheter embolization
- Emergency surgery when bleeding not controlled by endoscopic hemostasis and angiography

CAUSES OF SEVERE HEMATOCHEZIA

Colonic source

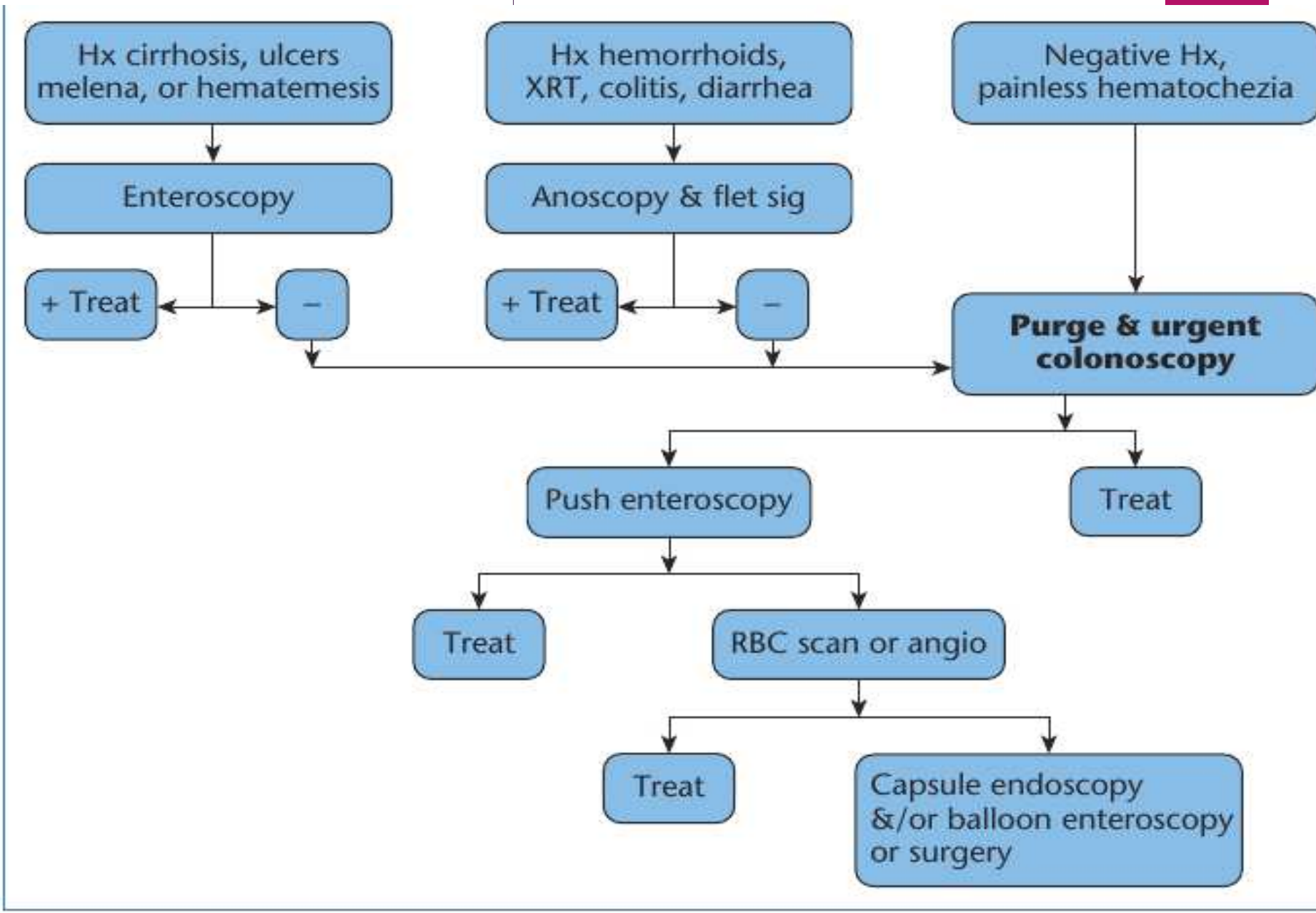
Diverticulosis
Internal hemorrhoids
Ischemic colitis
Rectal ulcers
Other colitis
Post-polypectomy ulcer
Polyp/cancer
Angiomas

UGI source

Ulcer
Varices
Angiomas

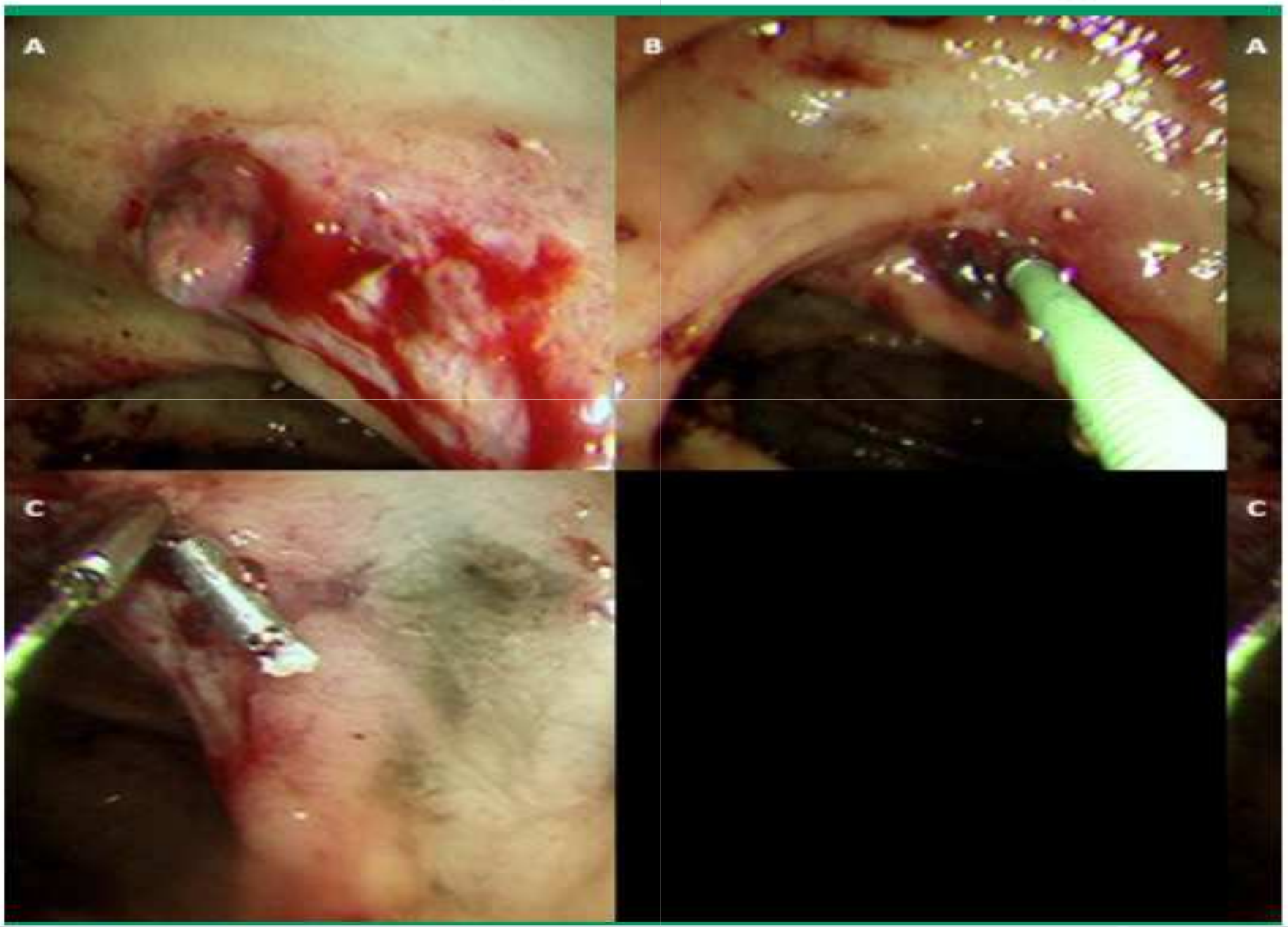
Small bowel source

Angiomas



gure 21.1 Algorithm for severe hematochezia.

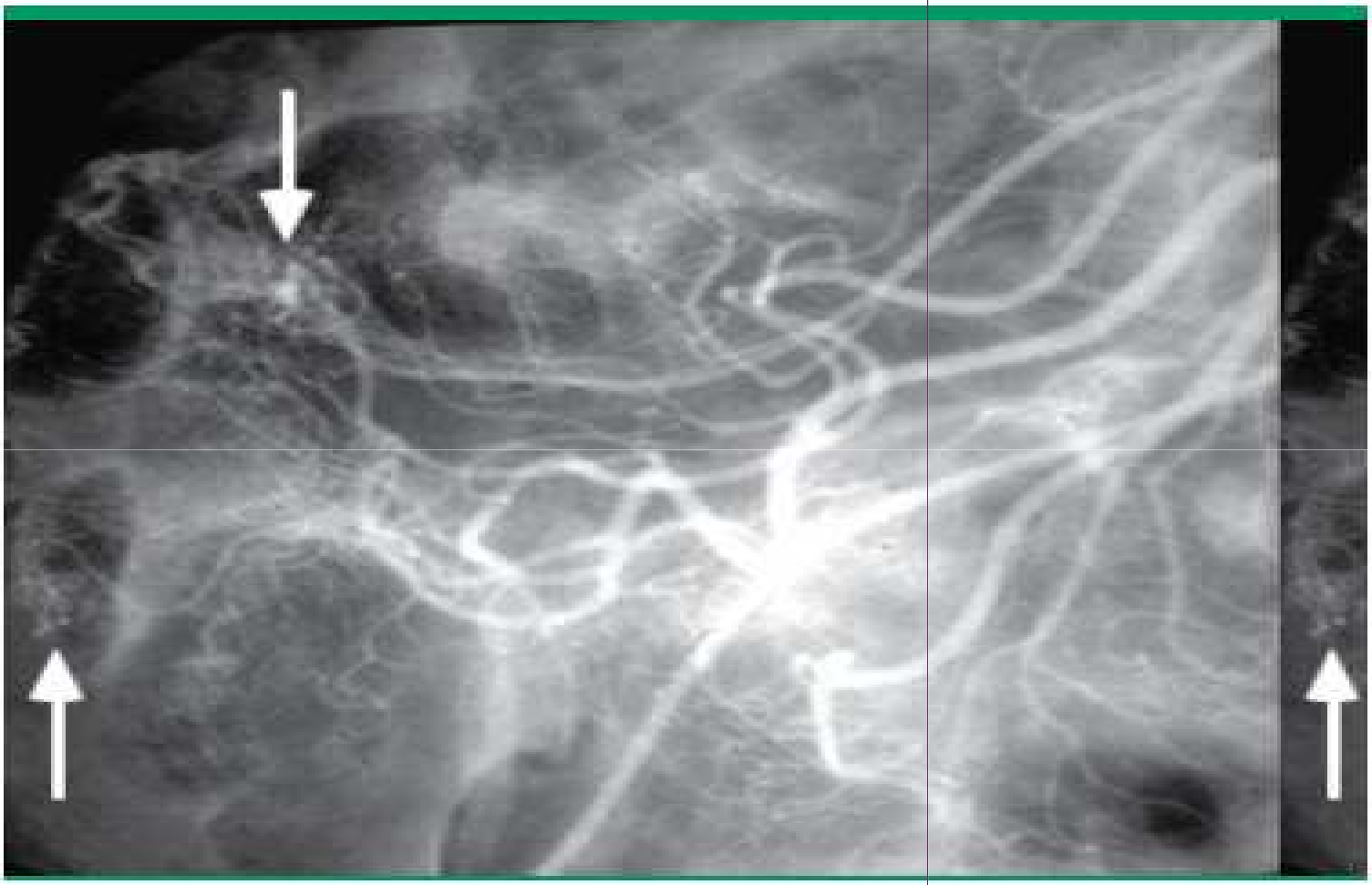
Treatment of lower gastrointestinal bleeding



dures used for evaluation of lower gastrointestinal bleeding

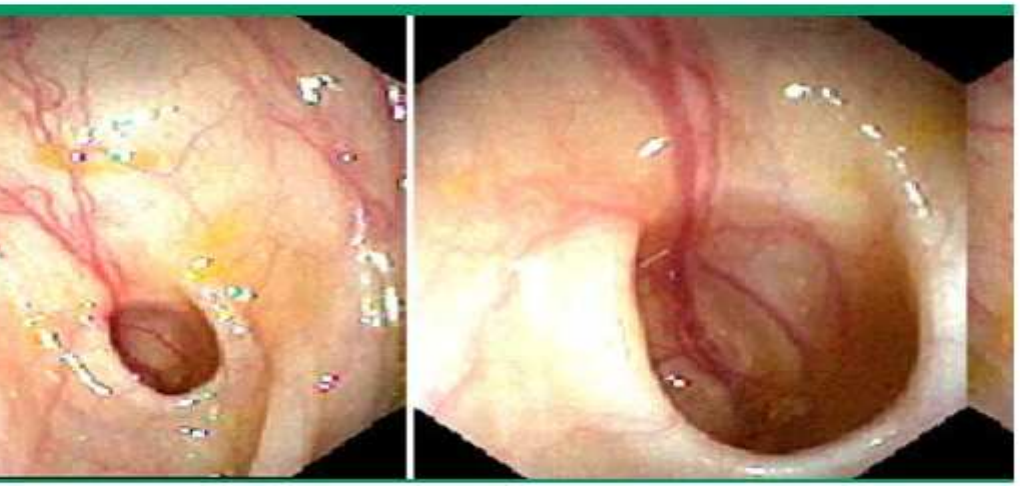
Technique	Advantages	Disadvantages
nuclide imaging	Noninvasive Sensitive to low rates of bleeding Can be repeated for intermittent bleeding	Has to be performed during active bleeding Poor localization of bleeding site Not therapeutic Not widely available
giography	Noninvasive Accurately localizes bleeding source Provides anatomic detail Widely available	Has to be performed during active bleeding Not therapeutic Radiation and IV contrast exposure
graphy	Accurately localizes bleeding source Therapy possible with super-selective embolization Does not require bowel preparation	Has to be performed during active bleeding Potential for serious complications
oscopy	Precise diagnosis and localization regardless of active bleeding or type of lesion Endoscopic therapy is possible	Need colon preparation for optimal visualization Risk of sedation in acutely bleeding patient Definite bleeding source (stigmata) infrequently ide

Angiodysplasia of the colon



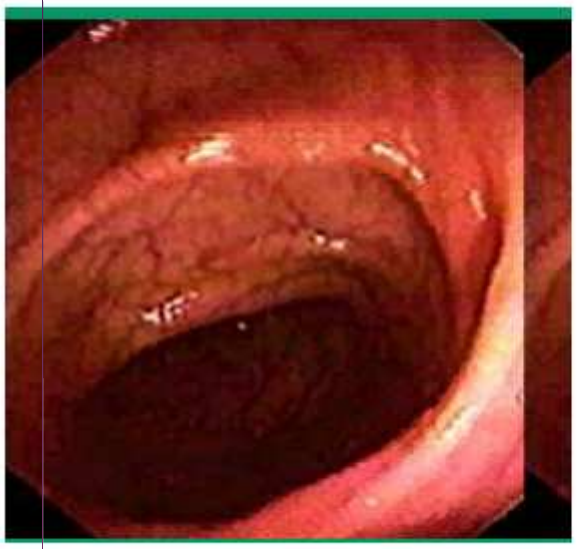
A superior mesenteric arteriogram demonstrates puddling of contrast material in tortuous distended vessels in the cecal wall (arrows).

essel within a colonic diverticulum



by showing a blood vessel within a diverticulum. The blood separated from the bowel lumen only by mucosa. Over time, el wall is exposed to injury along its luminal aspect, possibly o segmental weakness which predisposes to rupture into the

Normal sigmoid colon

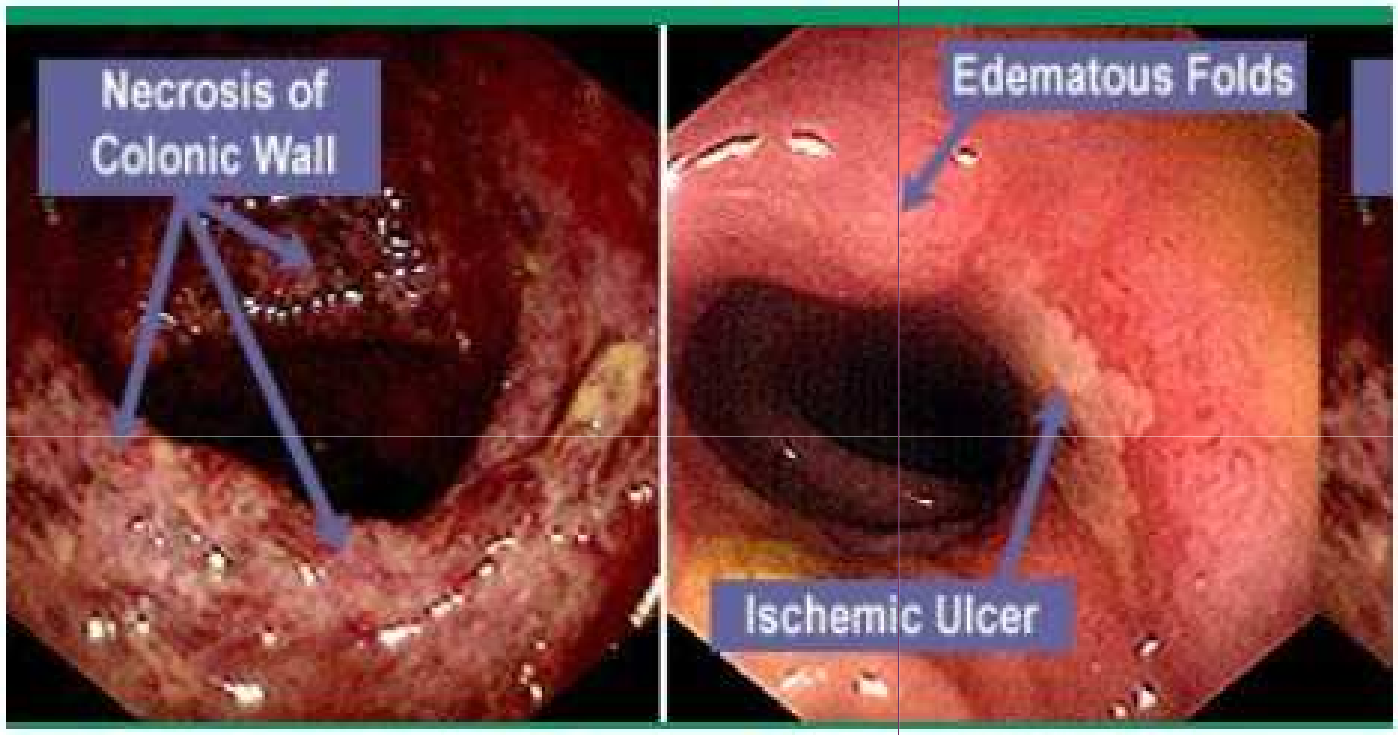


Endoscopic appearance of the normal sigmoid colonic mucosa. The fine vasculature is easily visible, and the surface is shiny and smooth. The folds are of normal thickness.

Ulcerative colitis

Endoscopic appearance of ulcerative colitis. Extensive ulceration of the mucosa is the most common endoscopic finding (panel A). The surface is irregular, friable, and erythematous, with loss of the normal vascular markings. Pseudopolyps may form as a reaction to inflammation (panel B); these can become quite extensive (panel C).

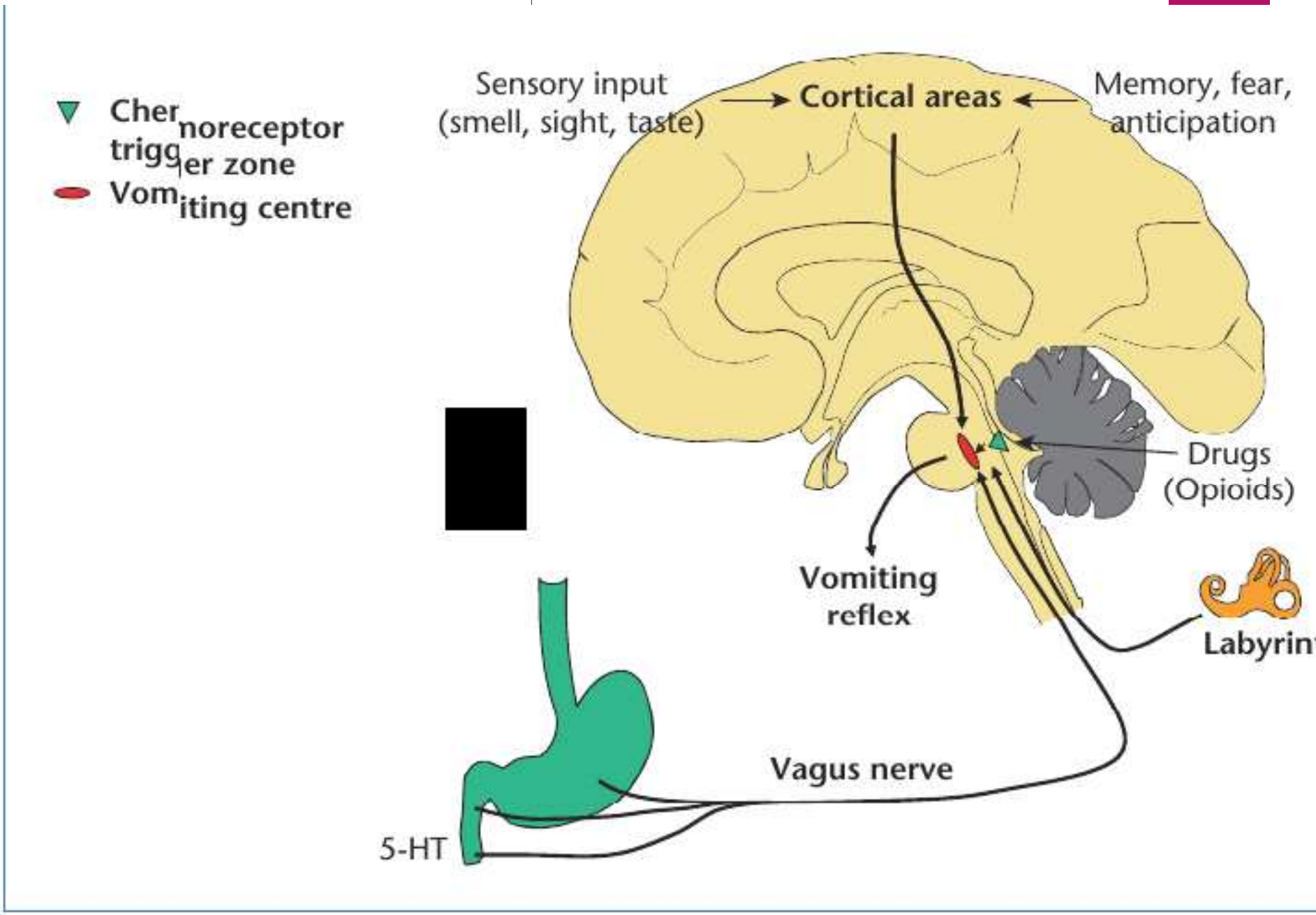
Ischemic colitis on colonoscopy



Endoscopy of ischemic colitis may reveal continuous necrosis and mucosal friability that resembles ulcerative colitis (left panel); discrete ulcers with surrounding edema may also be seen (right panel).

Diagnosed lesion	Frequency (%)[2]
1. Diverticulosis	31.9
2. Internal hemorrhoids	12.8
3. Ischemic colitis	11.9
4. Rectal ulcers	7.6
5. Colon angiomas or radiation telangiectasia	7.0
6. Ulcerative colitis, Crohn's disease, other colitis	6.2
7. Other LGI diagnoses	5.6
8. Post-polypectomy ulcer	4.7

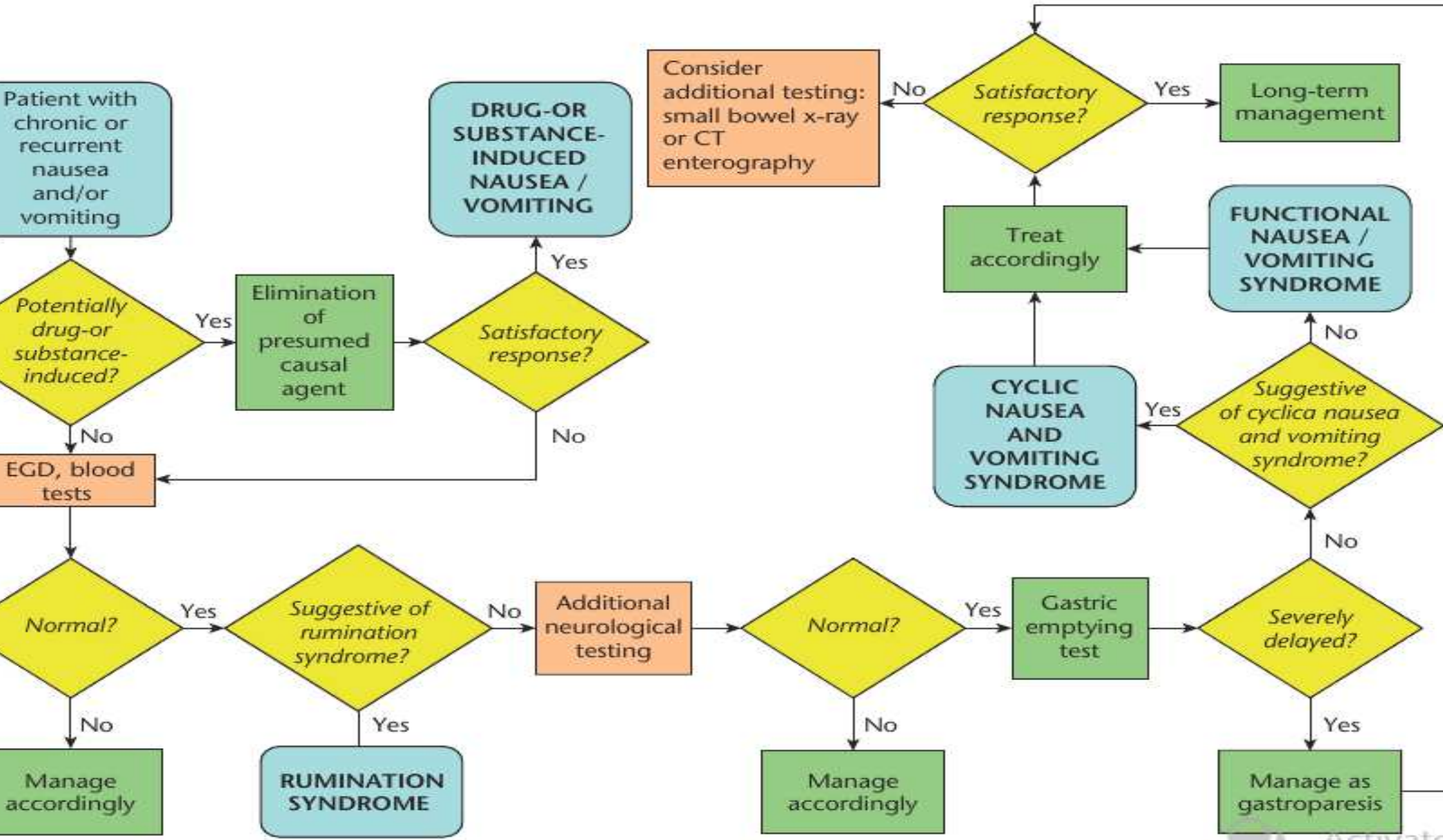
SEVERE VOMITING



ure 5.1 Pathways involved in triggering nausea and vomiting. (This figure was published in *Clinical Gastroenterology and Hepa*

AUSES OF NAUSEA AND VOMITING

TRAPERITONEAL	EXTRAPERITONEAL	MEDICATIONS/METABOLIC DISORDERS
bstructing disorders	Cardiopulmonary disease	Drugs
Pyloric obstruction	Cardiomyopathy	Cancer chemotherapy
Small bowel obstruction	Myocardial infarction	Antibiotics
Colonic obstruction	Labyrinthine disease	Cardiac antiarrhythmics
Superior mesenteric artery syndrome	Motion sickness	Digoxin
nteric infections	Labyrinthitis	Oral hypoglycemics
Viral	Malignancy	Oral contraceptives
Bacterial	Intracerebral disorders	Endocrine/metabolic disease
flammatory diseases	Malignancy	Pregnancy
Cholecystitis	Hemorrhage	Uremia
Pancreatitis	Abscess	Ketoacidosis
Appendicitis	Hydrocephalus	Thyroid and parathyroid disease
Hepatitis	Psychiatric illness	Adrenal insufficiency
tered sensorimotor function	Anorexia and bulimia nervosa	Toxins
Gastroparesis	Depression	Liver failure
Intestinal pseudoobstruction	Postoperative vomiting	Ethanol
Gastroesophageal reflux		
Chronic idiopathic nausea		
Functional vomiting		
Cyclic vomiting syndrome		
liary colic		
odominal irradiation		



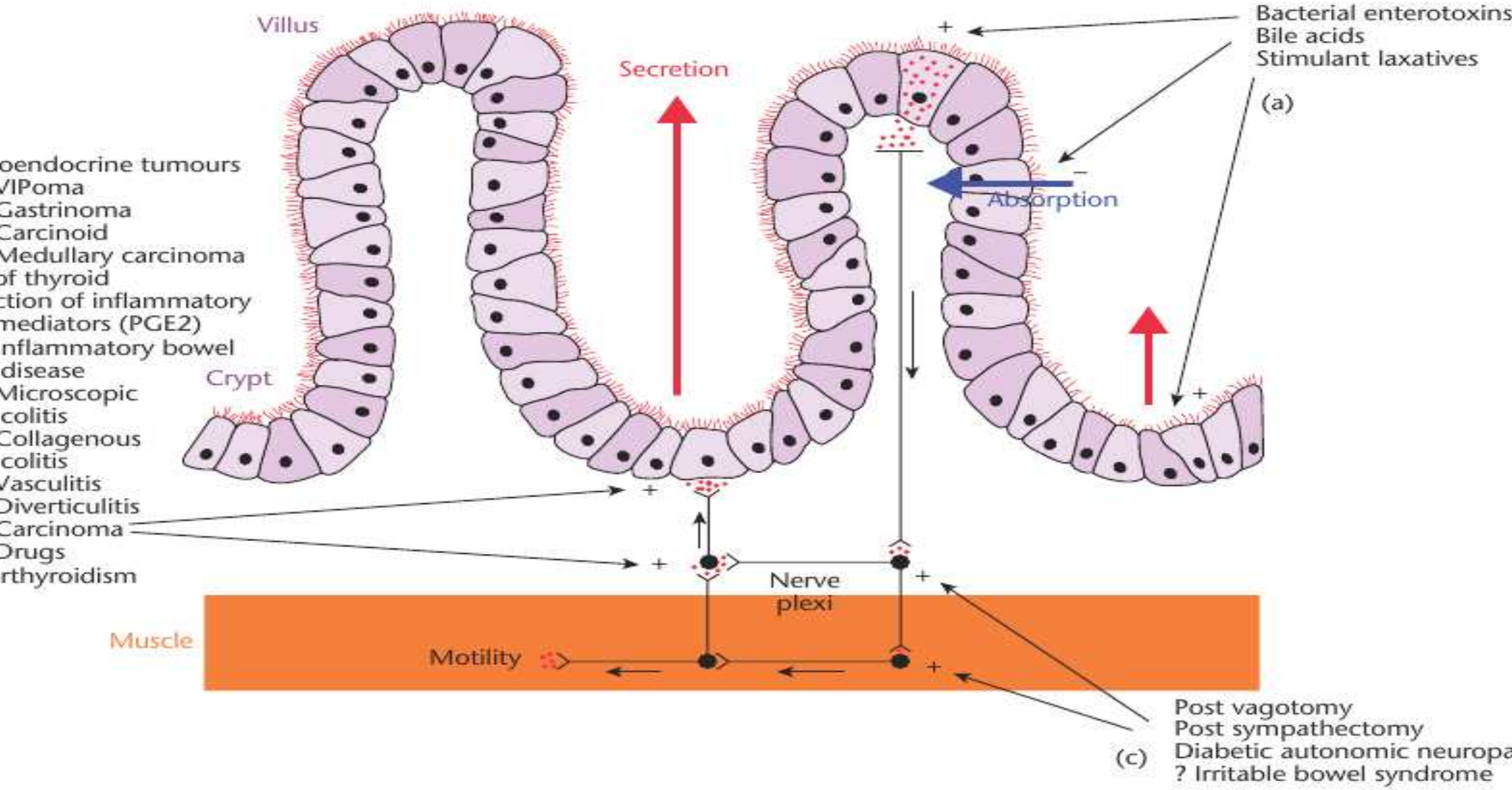
.2 Algorithm for a clinical approach to p

omography; EGD, esophagogastroduodenoscopy.

REATMENT OF NAUSEA AND VOMITING

REATMENT	MECHANISM	EXAMPLES	CLINICAL INDICATIONS
ntiemetic agents	Antihistaminergic	Dimenhydrinate, meclizine	Motion sickness, inner ear disease
	Anticholinergic	Scopolamine	Motion sickness, inner ear disease
	Antidopaminergic	Prochlorperazine, thiethylperazine	Medication-, toxin-, or metabolic-induced emesis
	5-HT_3 antagonist	Ondansetron, granisetron	Chemotherapy- and radiation-induced emesis, postoperative emesis
	NK_1 antagonist	Aprepitant	Chemotherapy-induced nausea and vomiting
	Tricyclic antidepressant	Amitriptyline, nortriptyline	Chronic idiopathic nausea, functional vomiting, cyclic vomiting syndrome, ?gastroparesis
	Other antidepressant	Mirtazapine	?Functional vomiting, ?gastroparesis
rokinetic agents	5-HT_4 agonist and antidopaminergic	Metoclopramide	Gastroparesis
	Motilin agonist	Erythromycin	Gastroparesis, ?intestinal pseudoobstructio
	Peripheral antidopaminergic	Domperidone	Gastroparesis
	Somatostatin analogue	Octreotide	Intestinal pseudoobstruction
	Acetylcholinesterase inhibitor	Pyridostigmine	?Small intestinal dysmotility /pseudoobstruction
pecial settings	Benzodiazepines	Lorazepam	Anticipatory nausea and vomiting with chemotherapy
	Glucocorticoids	Methylprednisolone,	Chemotherapy-induced emesis

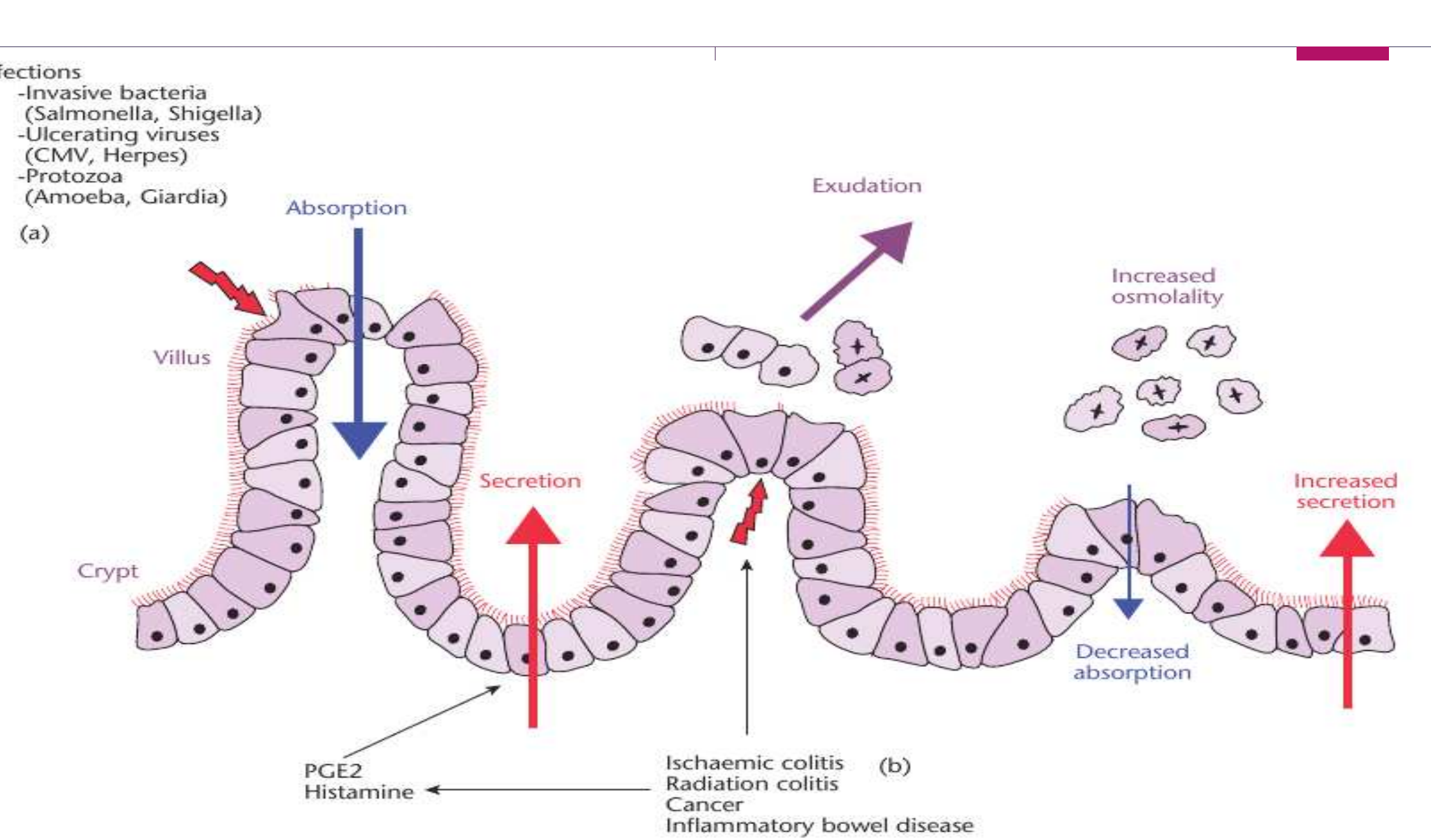
Severe and acute diarrhea



.1 Causes and pathophysiology of secretory diarrhea. The figure rates the intestinal mucosa with the villi projecting into the lumen. The muscle layers represent the lamina propria and the present the afferent, efferent and interneurons of the enteric

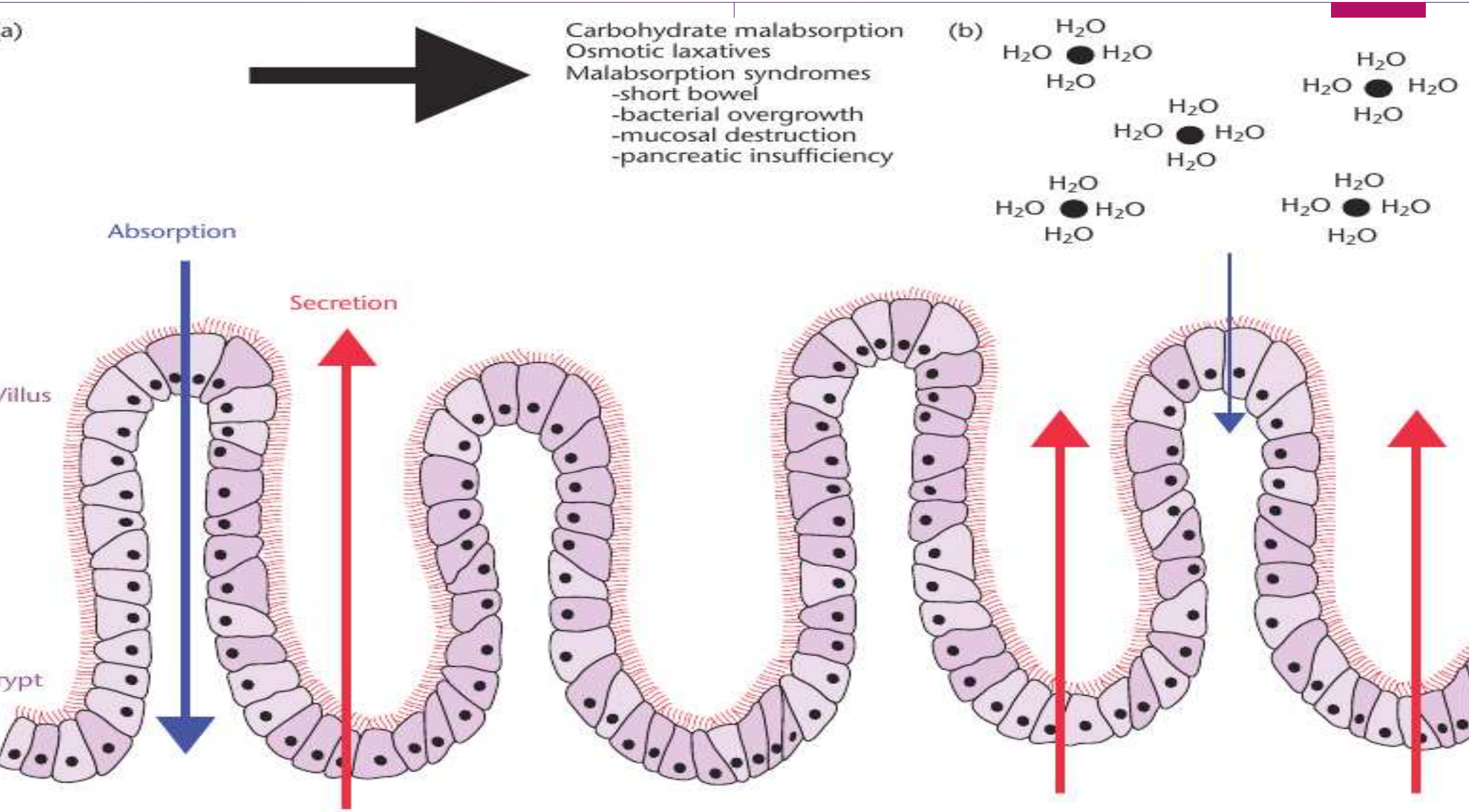
induce secretion by binding to the serosal surface of the epithelium c activating the secretomotor nerves. These include intestinal hormones as vasoactive intestinal peptide (VIP) and inflammatory mediators suc prostaglandin E_2 and histamine. (c) The function of the enteric nervo

fections
-Invasive bacteria
(Salmonella, Shigella)
-Ulcerating viruses
(CMV, Herpes)
-Protozoa
(Amoeba, Giardia)



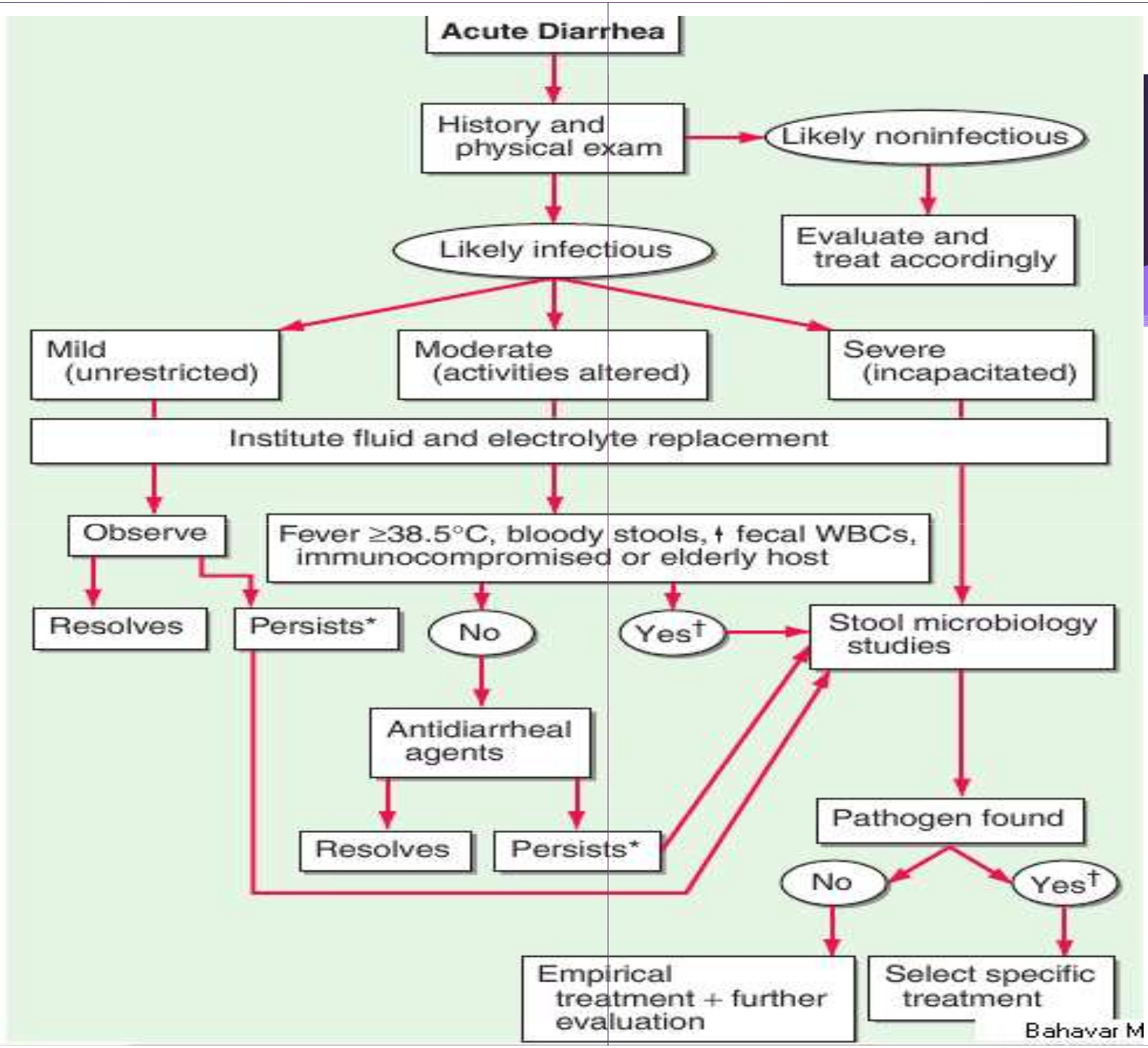
2 Causes and pathophysiology of inflammatory diarrhea. The luminal osmolality; 2. cell exudation further increases the luminal osmola
monstrates the destruction of the intestinal mucosa, with a loss of 3. immune recruitment of inflammatory mediators such as prostaglandin
inal invasive infections (a) and intrinsic causes of i

e secretion directly and through intermediate



.3 Causes and pathophysiology of osmotic diarrhea. The figure
rates the pro-secretory effects of osmotic
carbohydrates. (This figure was published in *Clinical Gastroenterolog*
d M. Weinstein, Christopher J. Hawkey, Jaime Bos

ATHOBIOLOGY/AGENTS	INCUBATION PERIOD	VOMITING	ABDOMINAL PAIN	FEVER	DIARRHEA
oxin producers					
Preformed toxin					
Bacillus cereus, Staphylococcus aureus, Clostridium perfringens	1–8 h 8–24 h	3–4+	1–2+	0–1+	3–4+, watery
Enterotoxin					
Vibrio cholerae, enterotoxigenic *Escherichia coli, Klebsiella pneumoniae, Aeromonas* species	8–72 h	2–4+	1–2+	0–1+	3–4+, watery
nteroadherent					
Enteropathogenic and enteroadherent *E. coli, Giardia* organisms, cryptosporidiosis, helminths	1–8 d	0–1+	1–3+	0–2+	1–2+, watery, mushy
ytotoxin producers					
C. difficile	1–3 d	0–1+	3–4+	1–2+	1–3+, usually watery, occasionally bloody
Hemorrhagic *E. coli*	12–72 h	0–1+	3–4+	1–2+	1–3+, initially watery, quickly bloody
nvasive organisms					
Minimal inflammation					
Rotavirus and norovirus	1–3 d	1–3+	2–3+	3–4+	1–3+, watery
Variable inflammation					
Salmonella, Campylobacter, and	12 h			3–4+	1–4+, watery or


Acute Diarrhea
History and physical exam
Likely noninfectious
Evaluate and treat accordingly
Likely infectious
Mild (unrestricted)
Moderate (activities altered)
Severe (incapacitated)
Institute fluid and electrolyte replacement
Observe
Fever ≥38.5°C, bloody stools, ↑ fecal WBCs, immunocompromised or elderly host
Resolves
Persists*
No
Yes†
Stool microbiology studies
Antidiarrheal agents
Resolves
Persists*
Pathogen found
No
Yes†
Empirical treatment + further evaluation
Select specific treatment
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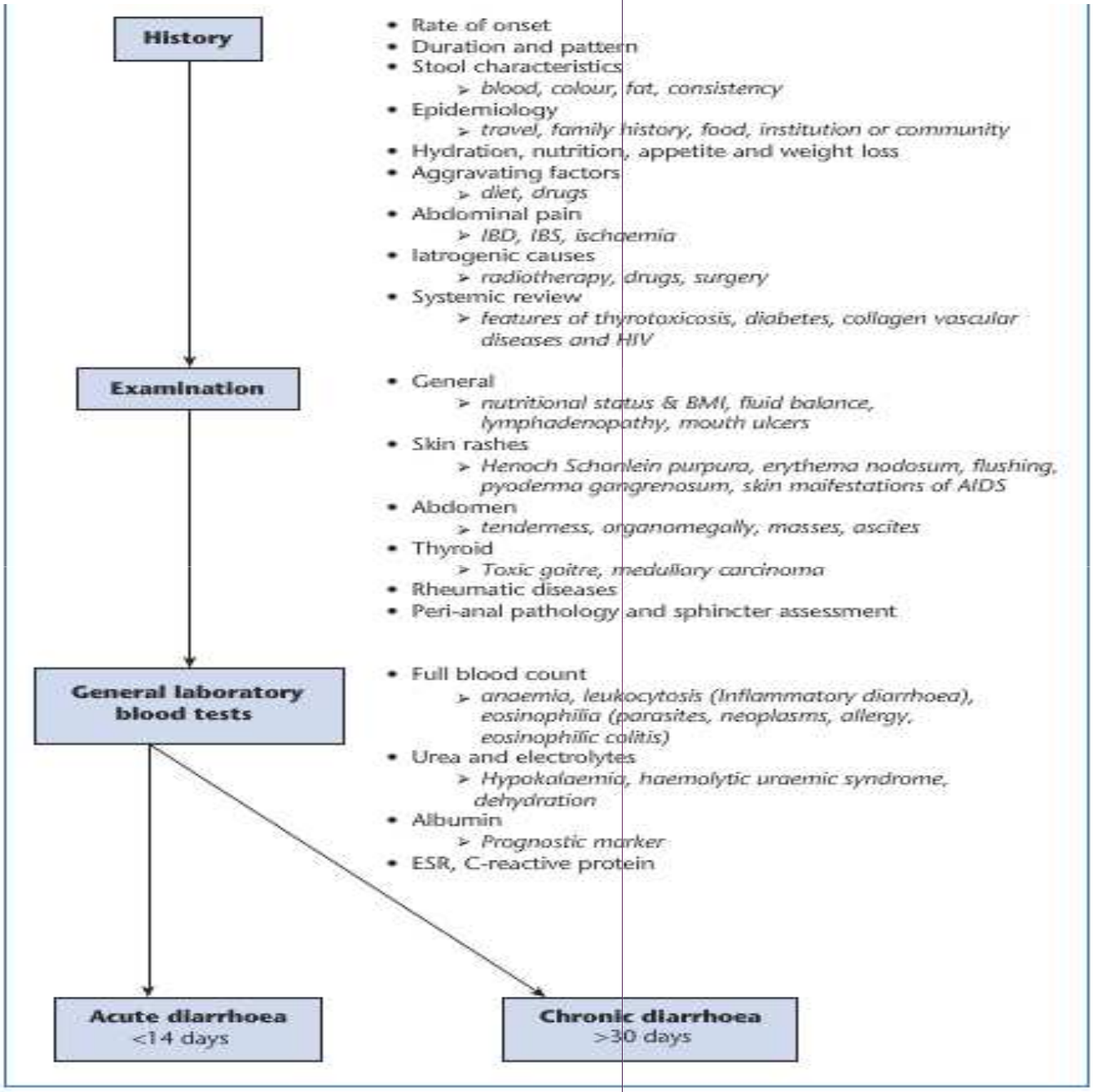
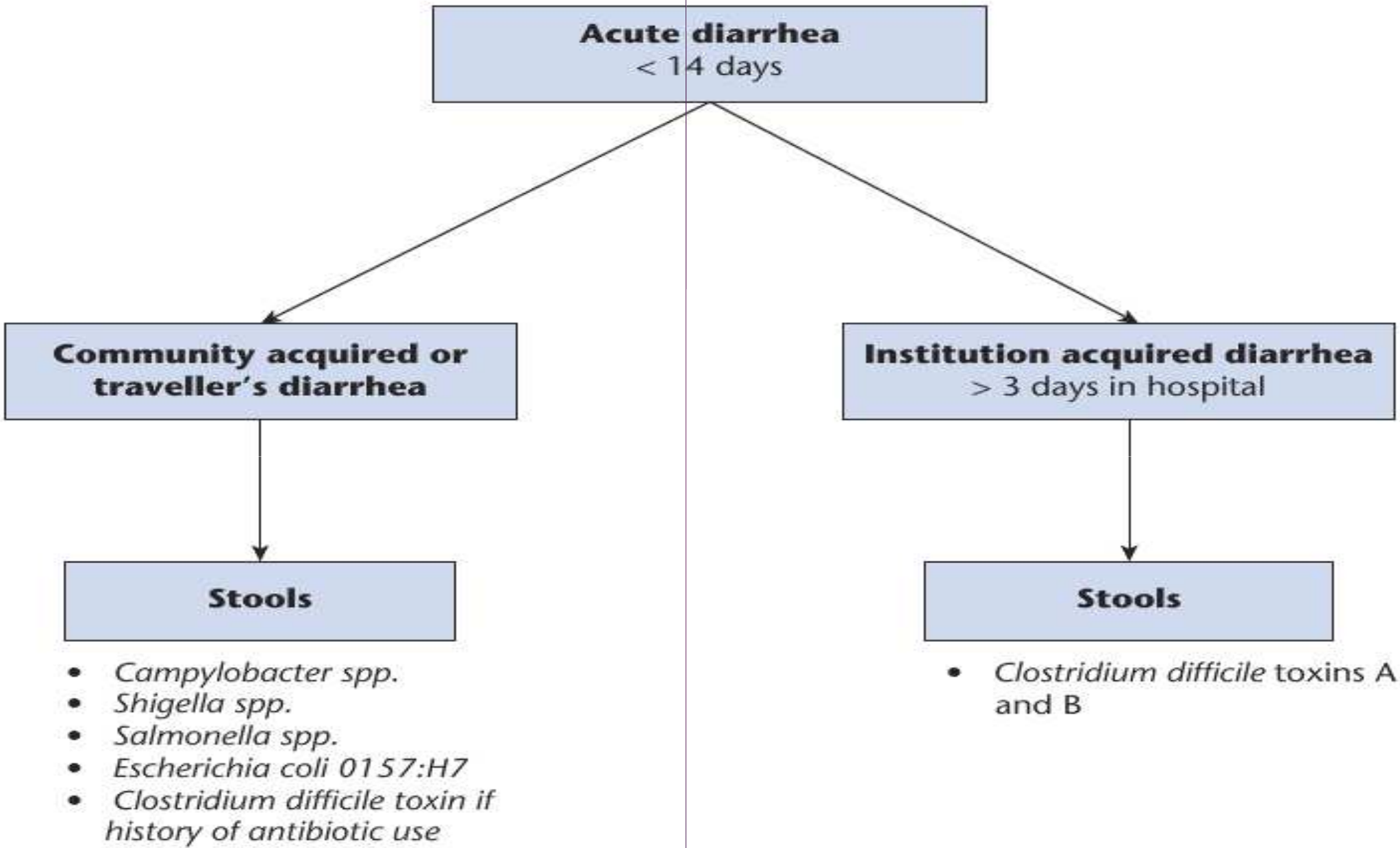


Figure 6.4 General investigations for diarrhea. (This figure was published in *Clinical Gastroenterology and Hepatology*, Wilfred M. Weinstein, Christopher J. Hawkey, Jaime Bosch, Diarrhea, Pages 1–8, Copyright Elsevier, 2005.)



estigations for acute diarrhea. (This figure was published in *Clinical Gastroenterology and Hepatology*, Wilfred M.

(a)

Chronic diarrhea
> 30 days

Stool examination

Basic investigations
- Observation
  - *consistency, pale, blood, fat*
- Microscopy and culture
  - *leukocytes (Inflammatory diarrhea), bacteria and protozoa (Giardia, Cryptosporidia, Cyclospora, Isospora belli)*

Comprehensive investigations
- Quantitative 48 hr stool weight, >500 g/day
- Osmotic gap and osmolality
  - *osmotic or secretory diarrhea, dilutional diarrhea*
- pH
  - *<5 indicates carbohydrate malabsorption*
- Fat content

Additional blood tests
- TSH
- Celiac disease serology

Endoscopy
- Sigmoidoscopy or colonoscopy and biopsy
- Small bowel biopsy and aspirate

Radiology
- Small intestinal barium
- CT abdomen

Investigation for specific categories of chronic diarrhea
- Watery: secretory or osmotic
- Fatty

(b)

Watery diarrhea

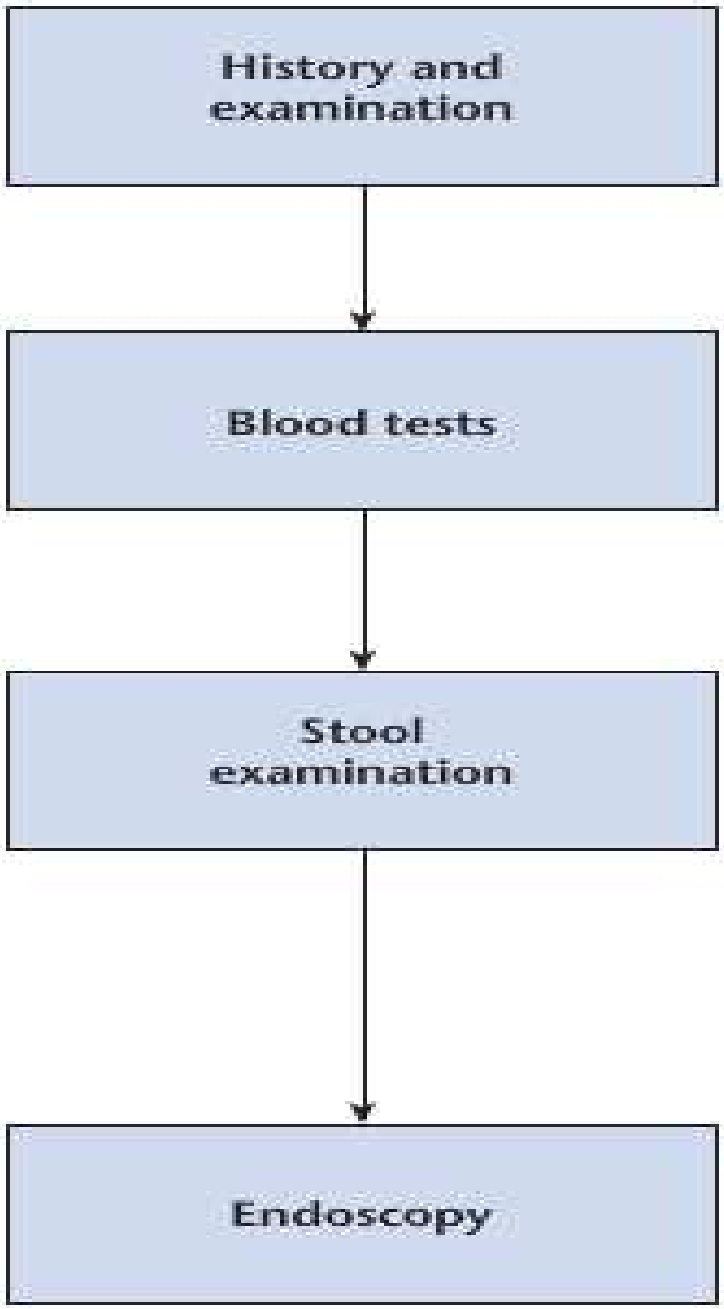
Osmotic gap <50 mOsm/kg Unresponsive to fasting → **Secretory diarrhea**

Osmotic gap >50 mOsm/kg Responsive to fasting → **Osmotic diarrhea**

- **Blood tests:** VIP, gastrin, calcitonin, enteroglucagon
- **Urine:** 5-hydroxyindole acetic acid, Vanillymandelic acid
- **SeHCAT test.** If not available a trial of bile salt sequestrants

- **Stools:** Clinitest for reducing sugars, pH < 5 suggests carbohydrate malabsorption, Laxative screen, including Mg
- **Lactose H_2 breath** test

(c)
- **Small intestinal biopsy**
- **Breath tests:** H_2 glucose or lactulose breath test for bacterial overgrowth
- **Pancreatic radiology:** pancreatic protocol CT scan or MRCP
- **Pancreatic function testing:** fecal elastase, secretin test, pancreolauryl test, trial of pancreatic enzymes.
- **Small intestinal barium study**



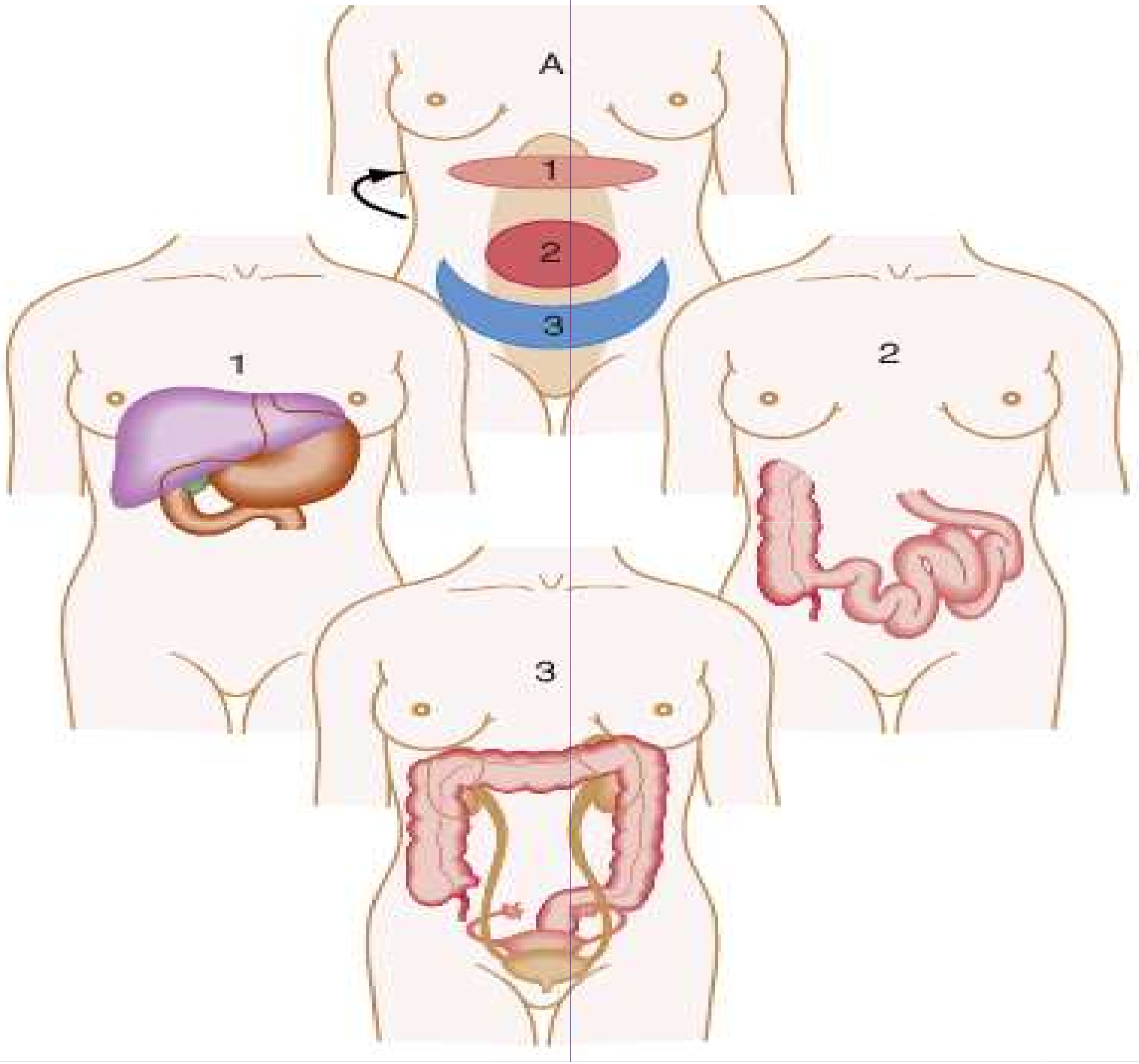
- Association of the onset of diarrhea and use of protease inhibitor
- Features of systemic infection

- HIV load
- CD4 count
- If febrile, blood culture

- Culture for *Shigella* spp., *Salmonella* spp., *Campylobacter* spp.
- Ova, cysts and paracytes
- *Clostridium difficile* toxins A and B
- Smears for acid-fast bacilli (AFB) and culture for *Mycobacterium avium* complex (MAC)
- Weber's modified trichrome for microsporidia
- Cryptosporidia ELISA

- Select patients with CD4 < 200 cells/mm3, fever and weight loss
- Colonoscopy and ileoscopy (39% greater yield for CMV than sigmoidoscopy)
- Small intestinal biopsy

Acute abdominal pain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1
2
3
1
2
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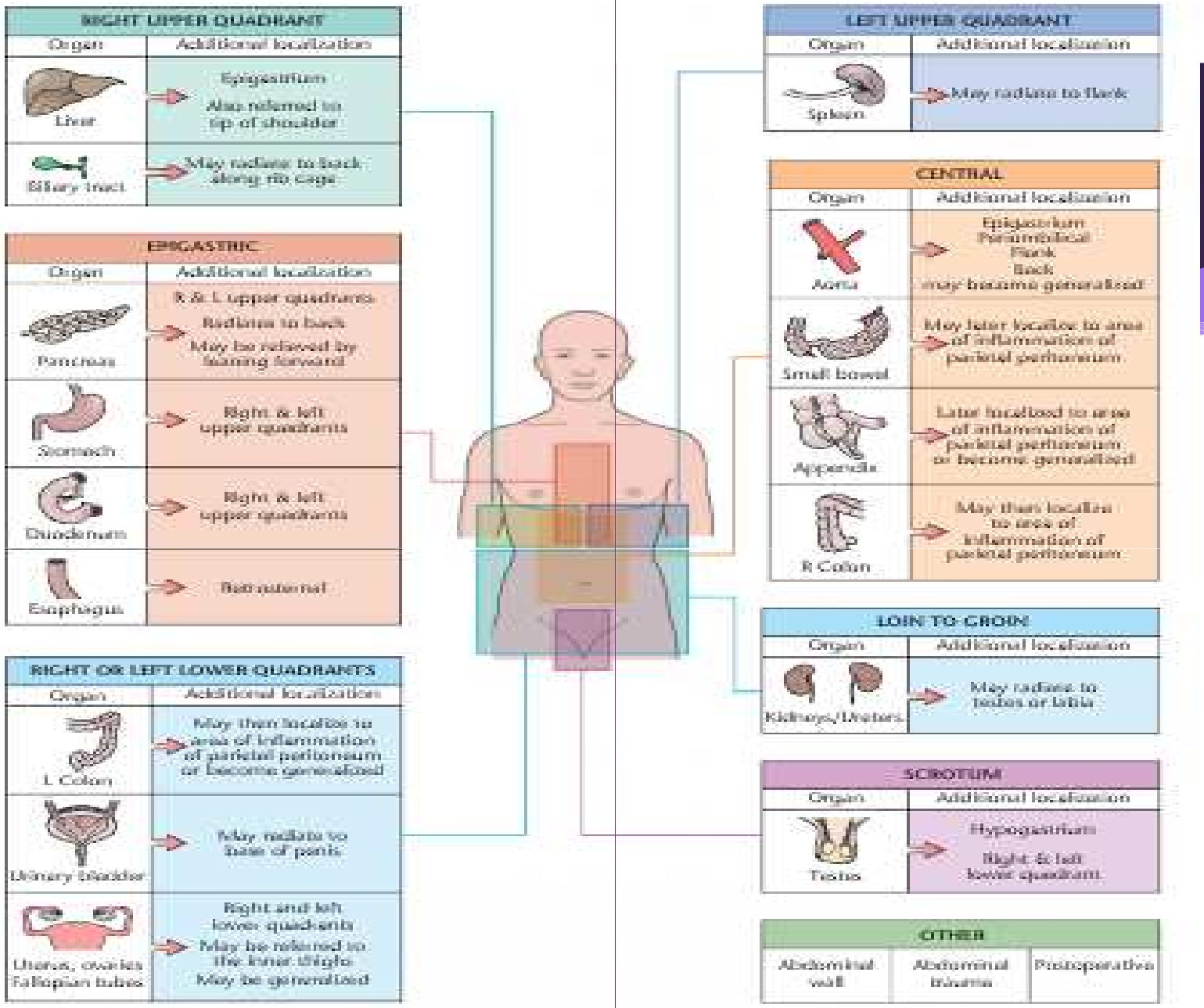


Figure 19.1 Sites of acute abdominal pain. (This figure was published in Clinical Gastroenterology and Hepatology, Wilfred M. Weinstein, Christopher J. Hawkey, Jaime Bosch, Acute abdominal pain, Pages 1–16, Copyright Elsevier, 2005.)

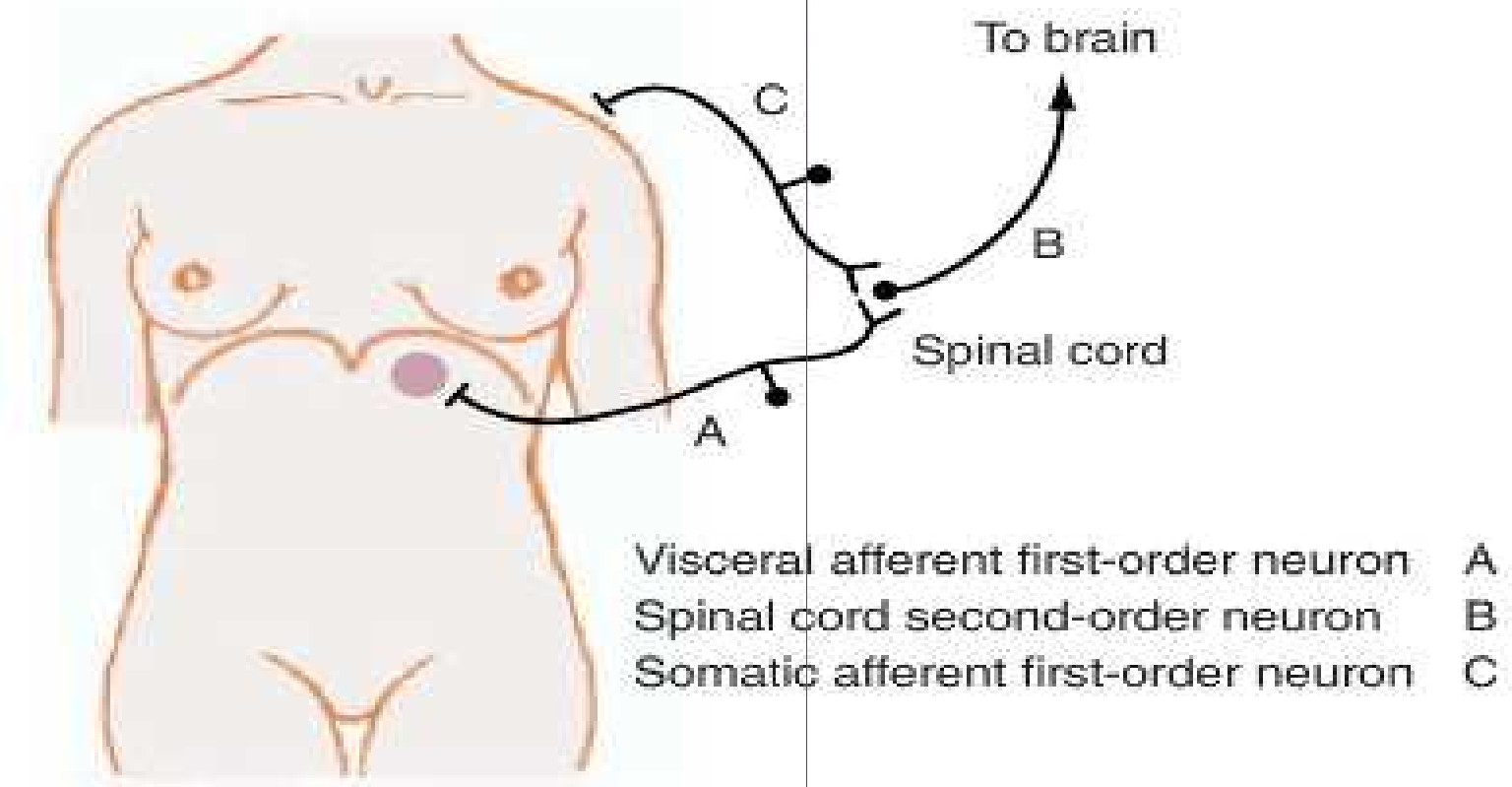


Figure 10-3. Demonstration of the neuroanatomic basis of referred pain. Visceral afferent fibers that innervate the diaphragm can be stimulated by local irritation (e.g., subdiaphragmatic abscess [*circle*]). These visceral afferent fibers (*A*) synapse with second-order neurons in the spinal cord (*B*) as well as somatic afferent fibers (*C*) arising from the left shoulder area (cervical roots 3 to 5). The brain interprets the pain to be somatic in origin and localizes it to the shoulder.

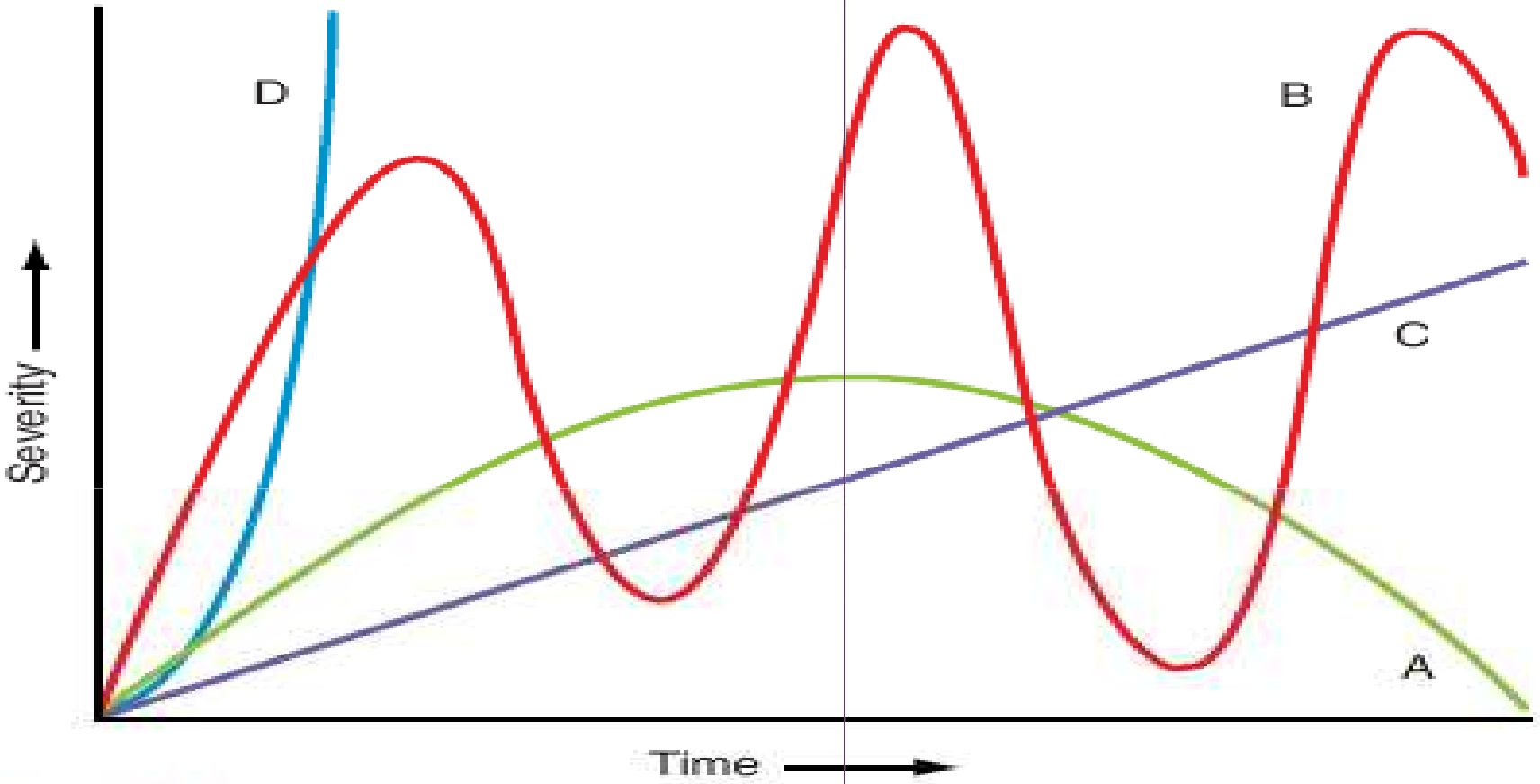


Figure 10-4. Patterns of acute abdominal pain. *A*, Many causes of abdominal pain subside spontaneously with time (e.g., gastroenteritis). *B*, Some pain is colicky (i.e., the pain progresses and remits over time); examples include intestinal, renal, and biliary pain (colic). The time course may vary widely from minutes in intestinal and renal pain to days, weeks, or even months in biliary pain. *C*, Commonly, acute abdominal pain is progressive, as in acute appendicitis or diverticulitis. *D*, Certain conditions have a catastrophic onset, such as ruptured abdominal aortic aneurysm.

10-1 Comparison of Common Causes of Acute Abdominal Pain

E	ONSET	LOCATION	CHARACTER	DESCRIPTOR	RADIATION	INTEN
dicitis	Gradual	Periumbilical area early; RLQ late	Diffuse early; localized late	Ache	None	++
cystitis	Acute	RUQ	Localized	Constricting	Scapula	++
eatitis	Acute	Epigastrium, back	Localized	Boring	Midback	++ to
culitis	Gradual	LLQ	Localized	Ache	None	
ated peptic ulcer	Sudden	Epigastrium	Localized early, diffuse late	Burning	None	+++
bowel obstruction	Gradual	Periumbilical area	Diffuse	Cramping	None	++
teric ischemia, rction	Sudden	Periumbilical area	Diffuse	Agonizing	None	+++
red abdominal aortic urysm	Sudden	Abdomen, back, flank	Diffuse	Tearing	None	+++
enteritis	Gradual	Periumbilical area	Diffuse	Spasmodic	None	+ to +
inflammatory disease	Gradual	Either LQ, pelvis	Localized	Ache	Upper thigh	++
red ectopic pregnancy	Sudden	Either LQ, pelvis	Localized	Sharp	None	++

++ = moderate; +++ = severe; LLQ = left lower quadrant; LQ = lower quadrant; RLQ = right lower quadrant; RUQ = right upper quadrant.

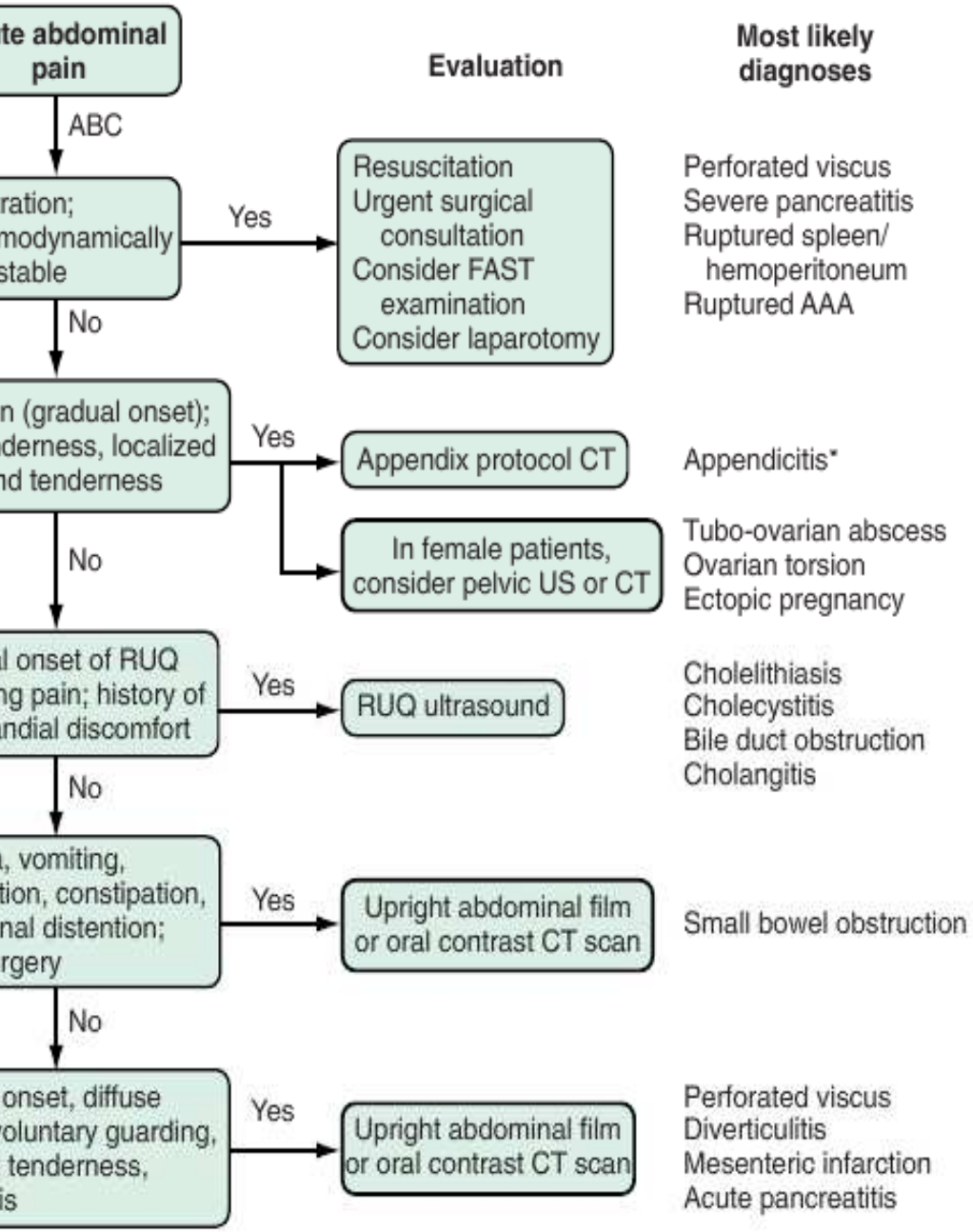


Figure 10-5. An approach to the urgent evaluation of abdominal pain. Specific complaints and physical examination findings are coupled with appropriate radiologic imaging. AAA, abdominal aortic aneurysm; ABC, airway, breathing, circulation; CT, computed tomography; FAST, focused abdominal sonogram for trauma; RLQ, right lower quadrant; RUQ, right upper quadrant; US, ultrasound. *For left lower quadrant pain, the most likely diagnosis is diverticulitis.

SOME IMPORTANT CAUSES OF ABDOMINAL PAIN	
Pain Originating in the Abdomen	
Parietal peritoneal inflammation Bacterial contamination Perforated appendix or other perforated viscus Pelvic inflammatory disease Chemical irritation Perforated ulcer Pancreatitis Mittelschmerz Mechanical obstruction of hollow viscera Obstruction of the small or large intestine Obstruction of the biliary tree Obstruction of the ureter	Vascular disturbances Embolism or thrombosis Vascular rupture Pressure or torsional occlusion Sickle cell anemia Abdominal wall Distortion or traction of mesentery Trauma or infection of muscles Distention of visceral surfaces, e.g., by hemorrhage Hepatic or renal capsules Inflammation of a viscus Appendicitis Typhoid fever Typhlitis
Pain Referred from Extraabdominal Source	
Cardiothoracic Acute myocardial infarction Myocarditis, endocarditis, pericarditis Congestive heart failure Pneumonia Pulmonary embolus	Pleurodynia Pneumothorax Empyema Esophageal disease, spasm, rupture, inflammation Genitalia Torsion of the testis
Metabolic Causes	
Diabetes Uremia Hyperlipidemia Hyperparathyroidism	Acute adrenal insufficiency Familial Mediterranean fever Porphyria C'1 esterase inhibitor deficiency (angioneurotic edema)
Neurologic/Psychiatric Causes	
Herpes zoster Tabes dorsalis Causalgia Radiculitis from infection or arthritis	Spinal cord or nerve root compression Functional disorders Psychiatric disorders
Toxic Causes	
Lead poisoning Insect or animal envenomations Black widow spiders Snake bites	
Uncertain Mechanisms	
Narcotic withdrawal Heat stroke	

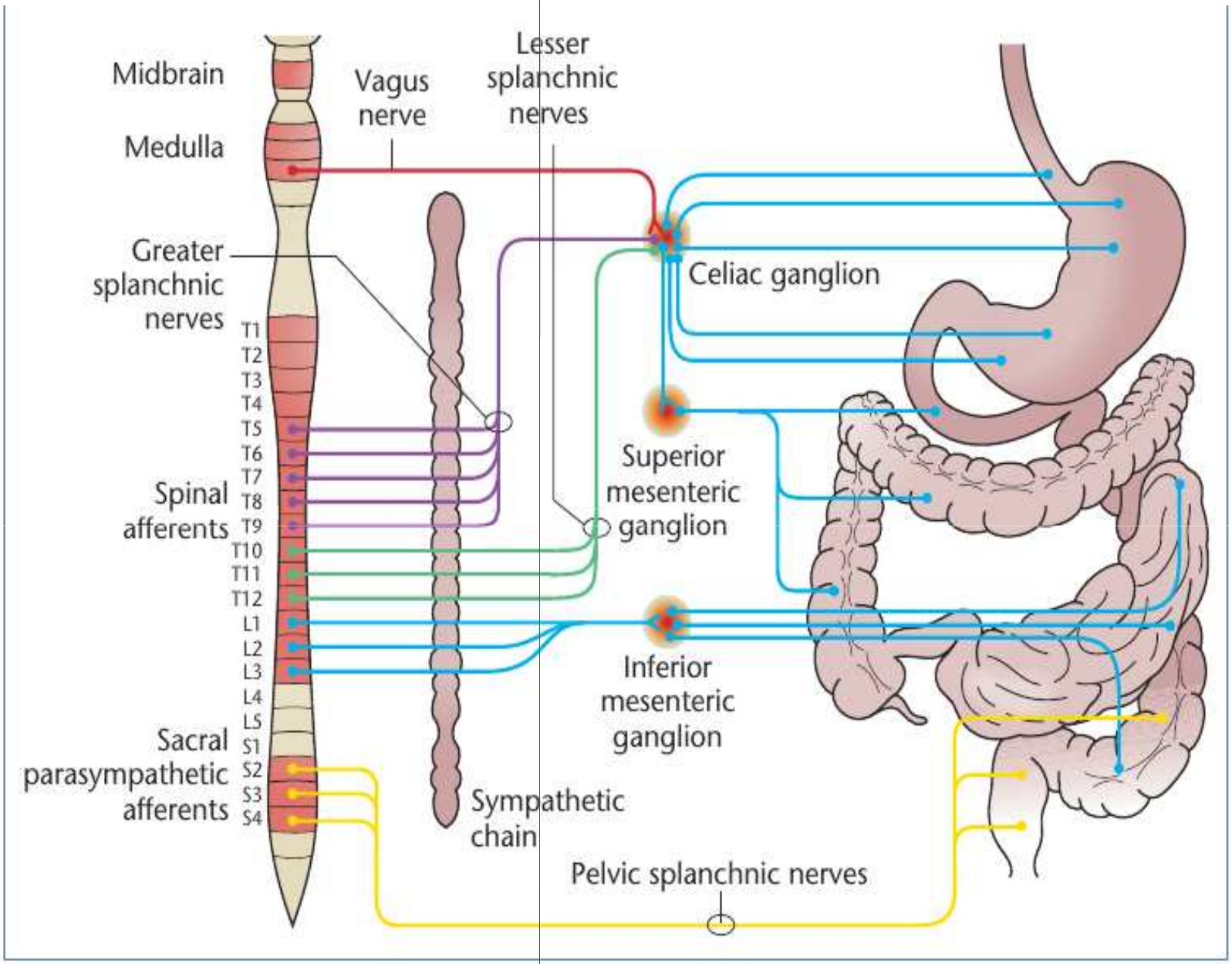
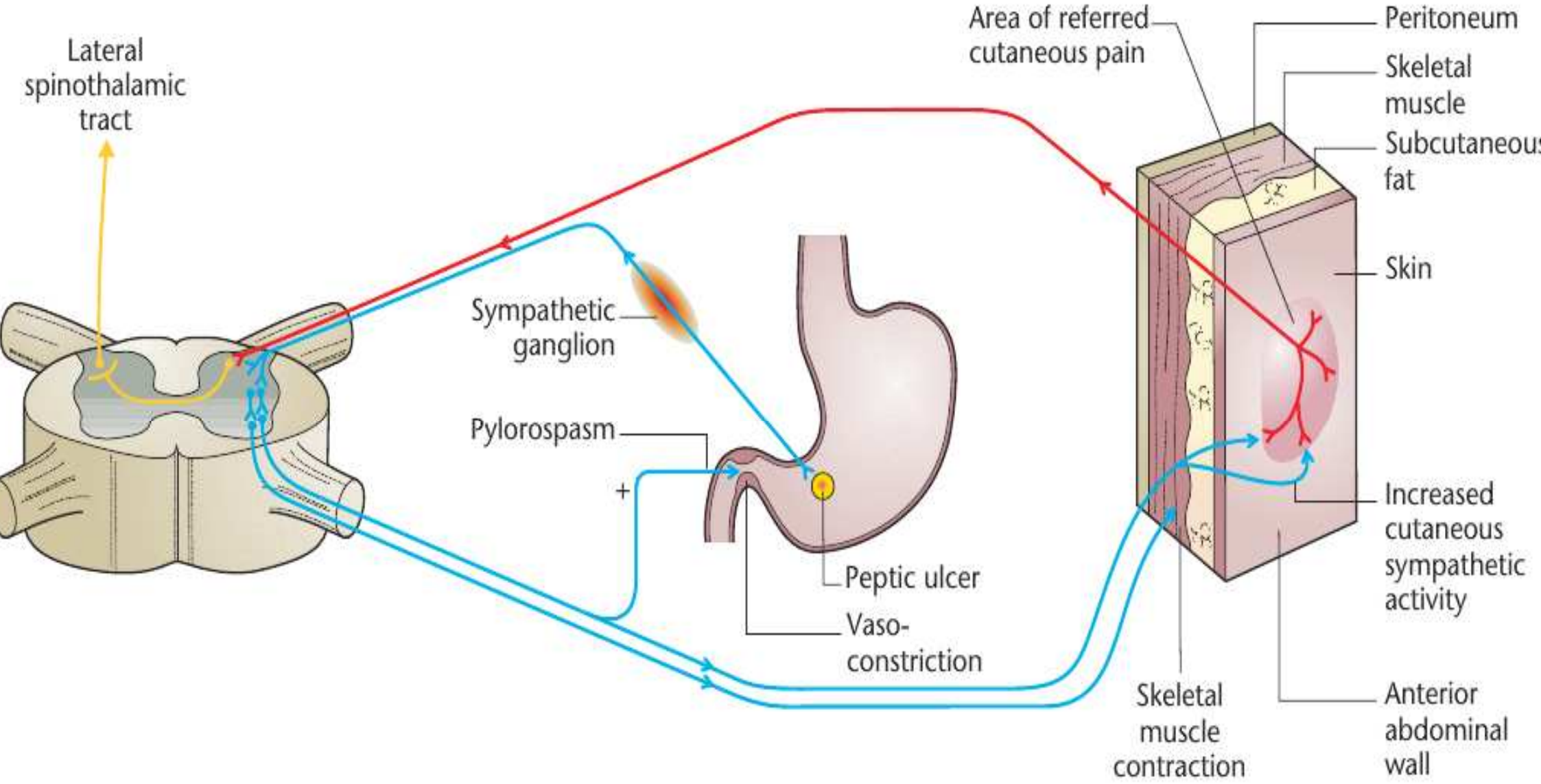


Figure 19.1 Efferent autonomic pathways of the gastrointestinal tract. The parasympathetic pathways are shown in purple and the sym red. (Reproduced from Mertz HR, Mayer EA. Functional gastrointestinal syndromes. In: Zinner MJ, Schwartz SI, Ellis H, eds. *Maingot's ab*



e 19.2 Visceral pain pathway from the gastrointestinal tract. (This figure was published in *Clinical Gastroenterology and Hepatology*, Wilfred M. Weinste

Table 19.2 Extra-abdominal/systemic causes of acute abdominal pain

Lungs	Lobar pneumonia Pleurisy Pulmonary embolism
Heart	Acute myocardial infarction Congestive cardiac failure Myocarditis
Metabolic/endocrine	Porphyrias Diabetic ketoacidosis Lead poisoning Hypercalcemia Adrenal insufficiency
Vasculitis	Henoch-Schonlein purpura Systemic lupus Polyarteritis nodosa Familial Mediterranean fever

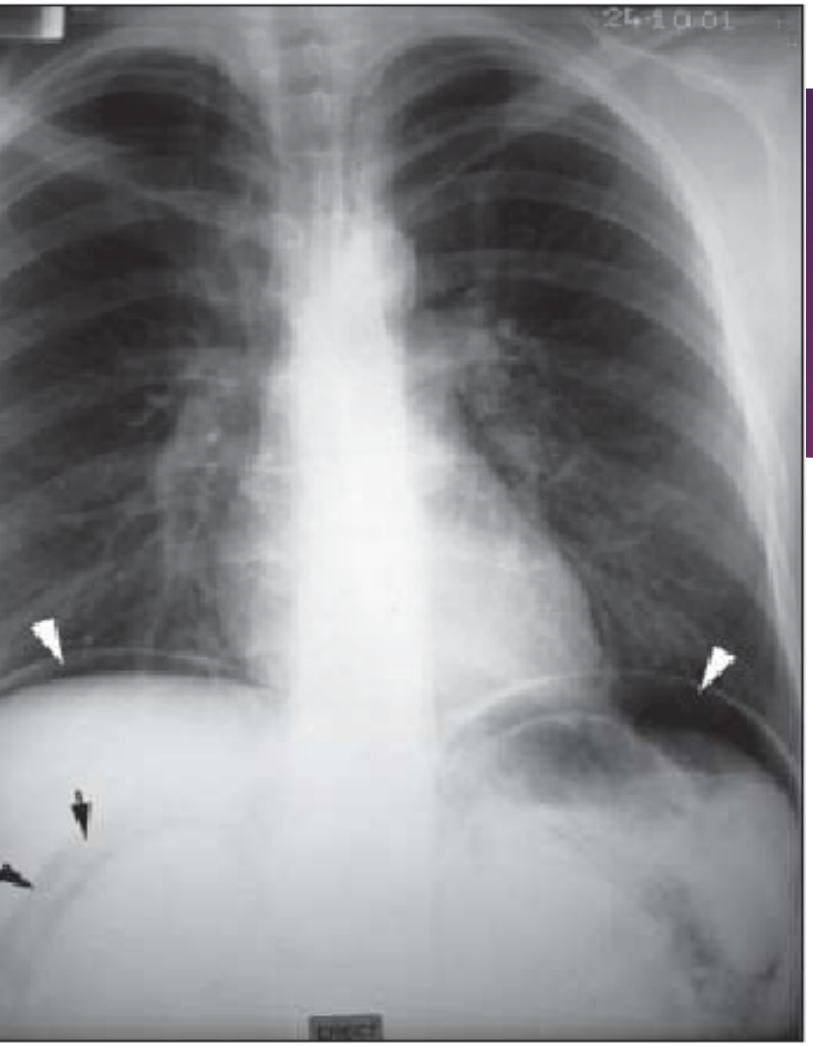
Table 19.4 Questions to ask when taking the patient history

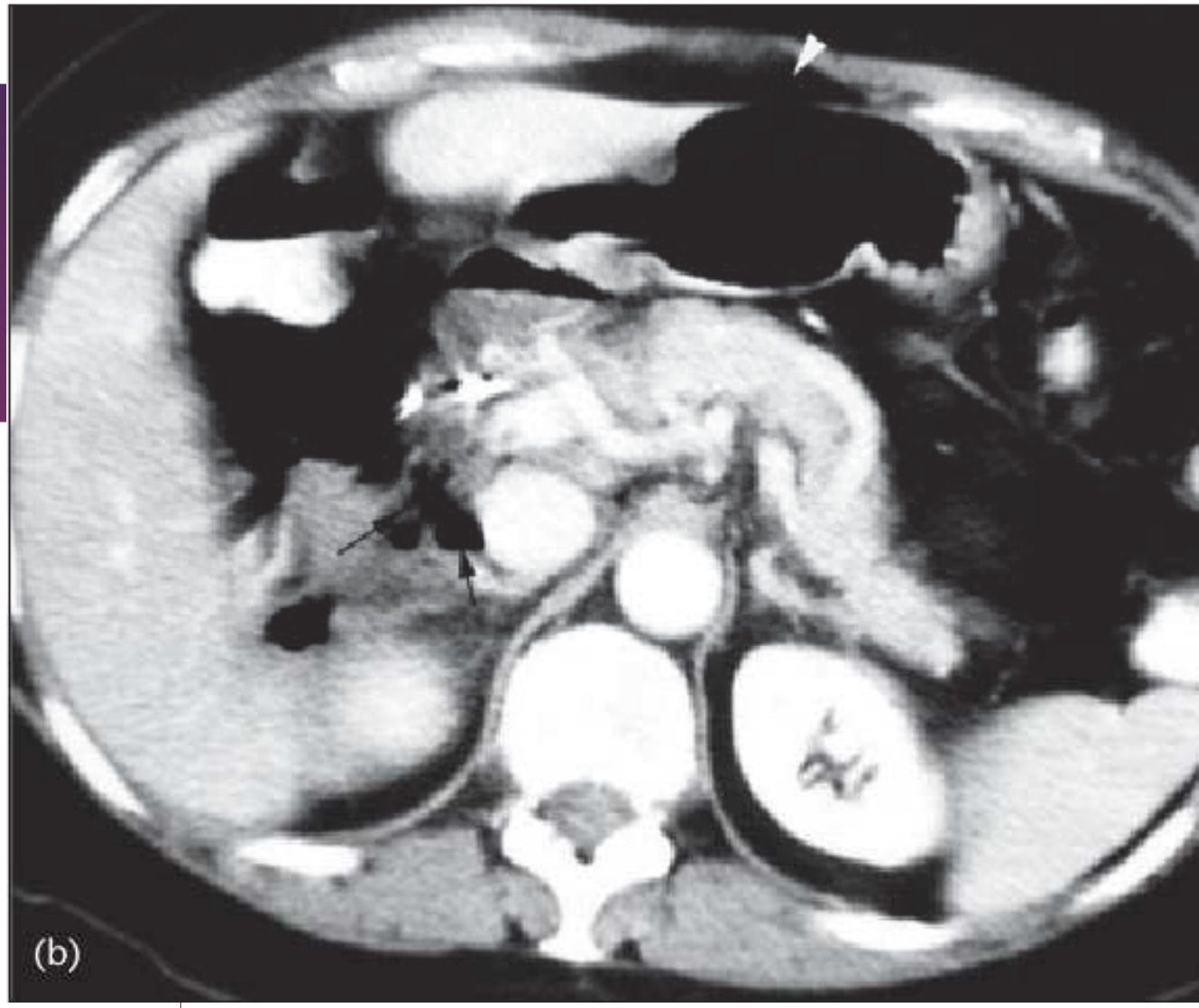
- When and where did the pain start?
- Was the onset sudden and what brought the pain on?
- Where is it now?
- What is the character of the pain?
- How severe is it?
- Does the pain radiate elsewhere?
- Are there any aggravating or relieving factors?
- Has this happened before?
- Are there any associated symptoms? (e.g., distention, nausea, vomiting, fever, diarrhea, absolute constipation, anorexia, jaundice, prutitus, gastrointestinal bleeding, dysuria, oliguria, chest pain)
- When was your last period and is there any chance of you being pregnant?
- History of alcohol intake
- Drug (medicinal and recreational) history
- History of ingestion of toxins, poisons or foreign bodies
- History of previous surgery
- History of pre-existing disease
- History of travel, especially foreign travel
- Family history

e **19.5** Investigations for acute abdominal pain

estigations	Subcategory/comment	Interpretation/usefulness
blood count	Hemoglobin ↓	Blood loss
	Hemoglobin ↑	Dehydration/hemoconcentration/polycythemia
	Leukocytes ↑	Inflammation or infection
	Leukocytes ↓	Overwhelming inflammation
	Platelets ↑	Active inflammatory bowel disease
	Platelets ↓	Overwhelming sepsis
nical chemistry	Creatinine ↑	Renal failure
	Urea ↑	Dehydration/hemoconcentration
	Electrolytes	
um amylase	Fourfold increase	Acute pancreatitis (but may be normal)
	Lesser increases	Almost any acute abdominal condition
er function tests	Biliary enzymes	Obstructive jaundice(but alkaline phosphatase slow to rise with acute obstruction)
	Transaminases/mixed	Acute liver injury sepsis, cholangitis
cium	Calcium ↑	Medical cause of abdominal pain
od glucose	Glucose ↑	Ketoacidosis can cause abdominal pain
ture	Urine/blood	Prelude to antibiotics
oup/cross mach		Prelude to surgery, transfusion

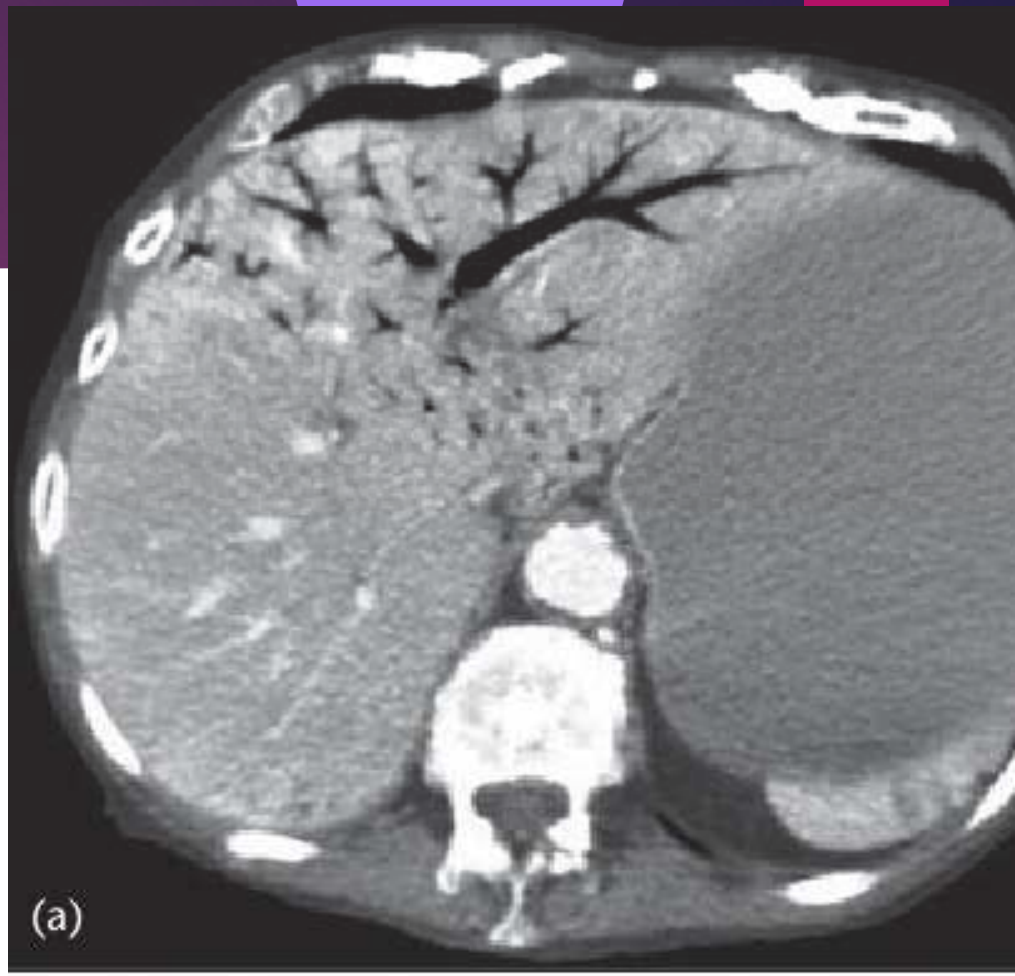
Investigations	Subcategory/comment	Interpretation/usefulness
Pregnancy test		Ruptured ectopic
Electrocardiogram		Myocardial infarction as cause of abdominal pain. Preoperative investigation for patients over the age of 50 years
Supine abdominal	Intestinal lumen and pattern	Obstruction, ileus, IBD
X-ray	Calcification Pneumobilia Foreign bodies Skeletal abnormalities Soft tissue masses	Gallstones(10%), renal stones, pancreatic calcification, aortic rim Ascending cholangitis
Erect chest x-ray	Detection of free subdiaphragmatic air	Perforation. Also look at lung fields and cardiac contour
Lateral decubitus film	≥1 mL of free peritoneal gas can be visualized	Perforation
Abdominal ultrasound	Percutaneous ultrasound Transvaginal ultrasound	Bile duct dilatation, gallstones, fluid collections, aortic aneurysms Gynecological causes
Abdominal CT scan	Value high especially with intravenous and intraluminal contrast enhancement	Multiple diagnoses Provides both anatomical and etiological diagnosis
Intravenous urography/CT kidney, ureters and bladder (KUB)	Important if blood in urine	Urological causes Useful in urological trauma
Magnetic resonance imaging	Not as popular as CT for the acute abdomen. MRCP	Excellent images of biliary tract pathology
Gastrointestinal contrast studies	Lower GI	Large bowel obstruction vs. pseudo-obstruction (may be therapeutic)
	Upper GI	Cryptic perforation or obstruction
Visceral angiography	Diagnostic	Intestinal ischemia, obscure bleeding
	Therapeutic	Embolization in gastrointestinal bleeding
Endoscopy	Upper and lower GI endoscopy	Helpful in selected cases
	Sigmoidoscopy	May be therapeutic for sigmoid volvulus
	ERCP	Therapeutic in biliary obstruction, especially ascending cholangitis

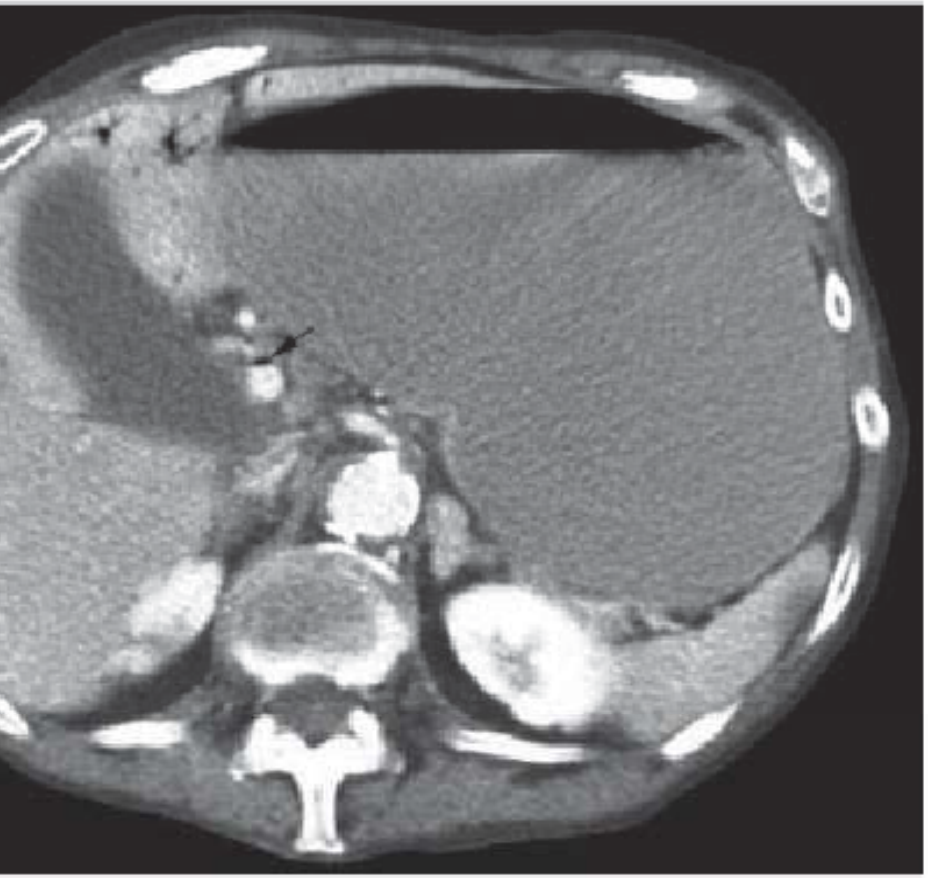




9.4 Intraperitoneal and retroperitoneal gas. Chest X

9.5 Gallstone ileus. Supine abdominal X-ray demonstrating
ilia (black arrowhead) and a radiopaque gallstone obstructing the
vel (white arrow).





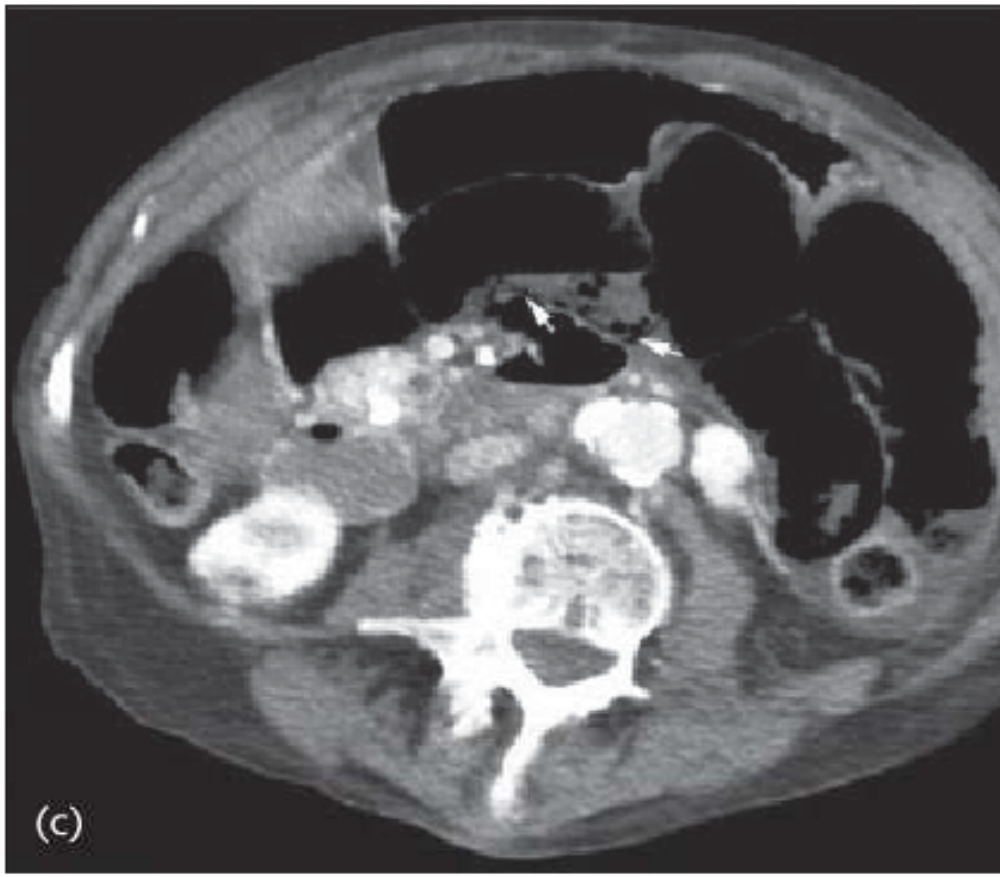
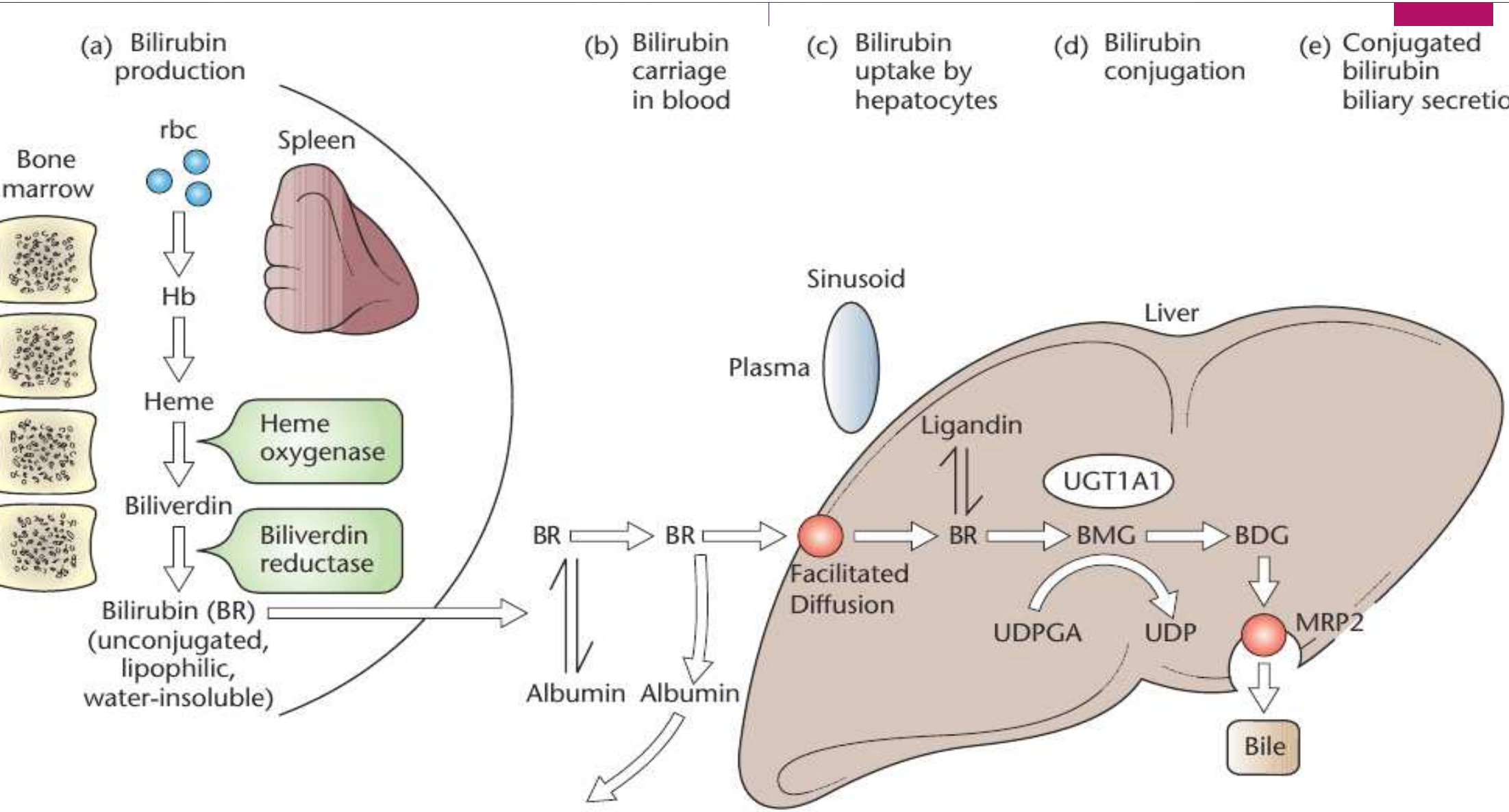
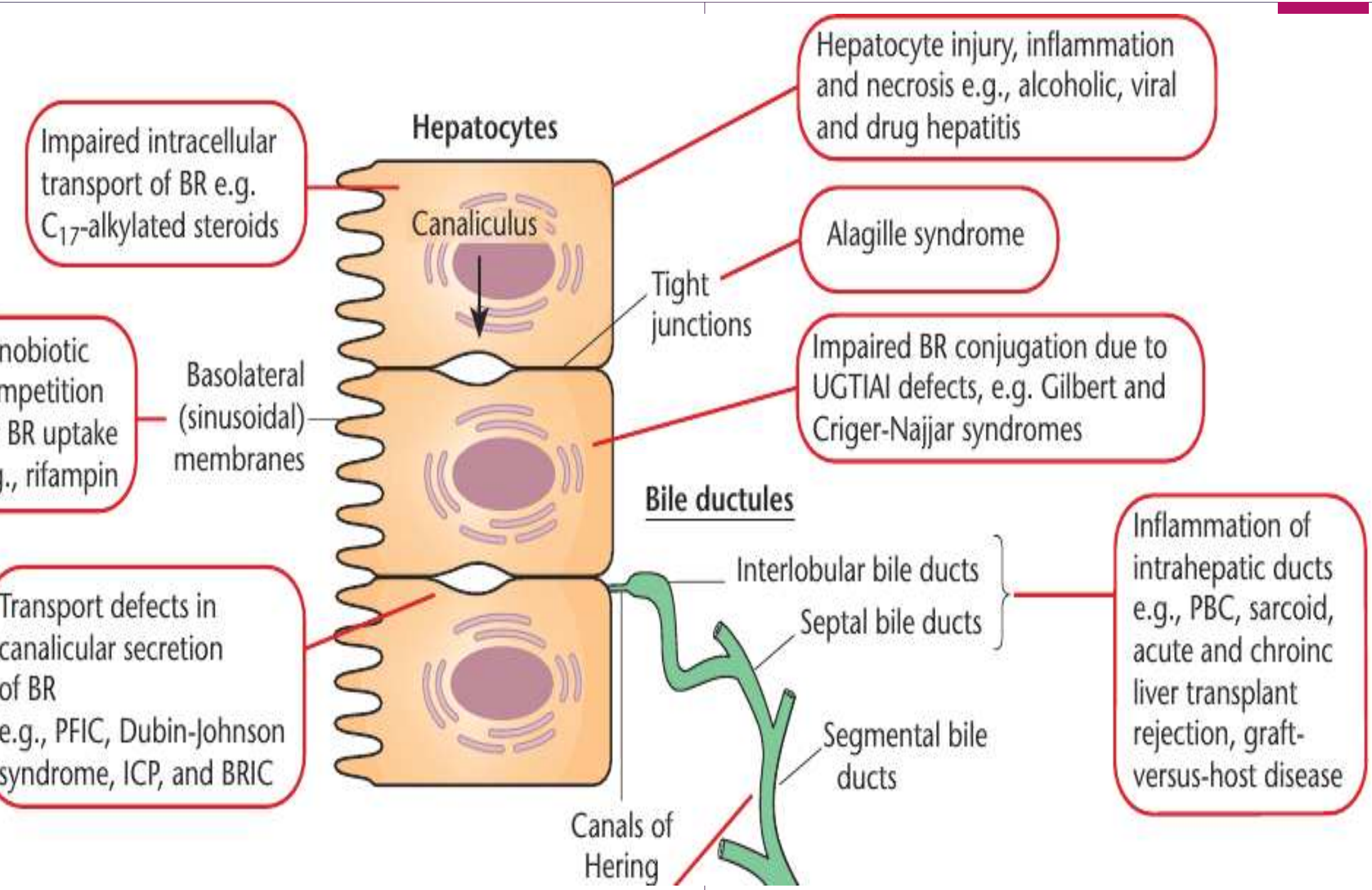


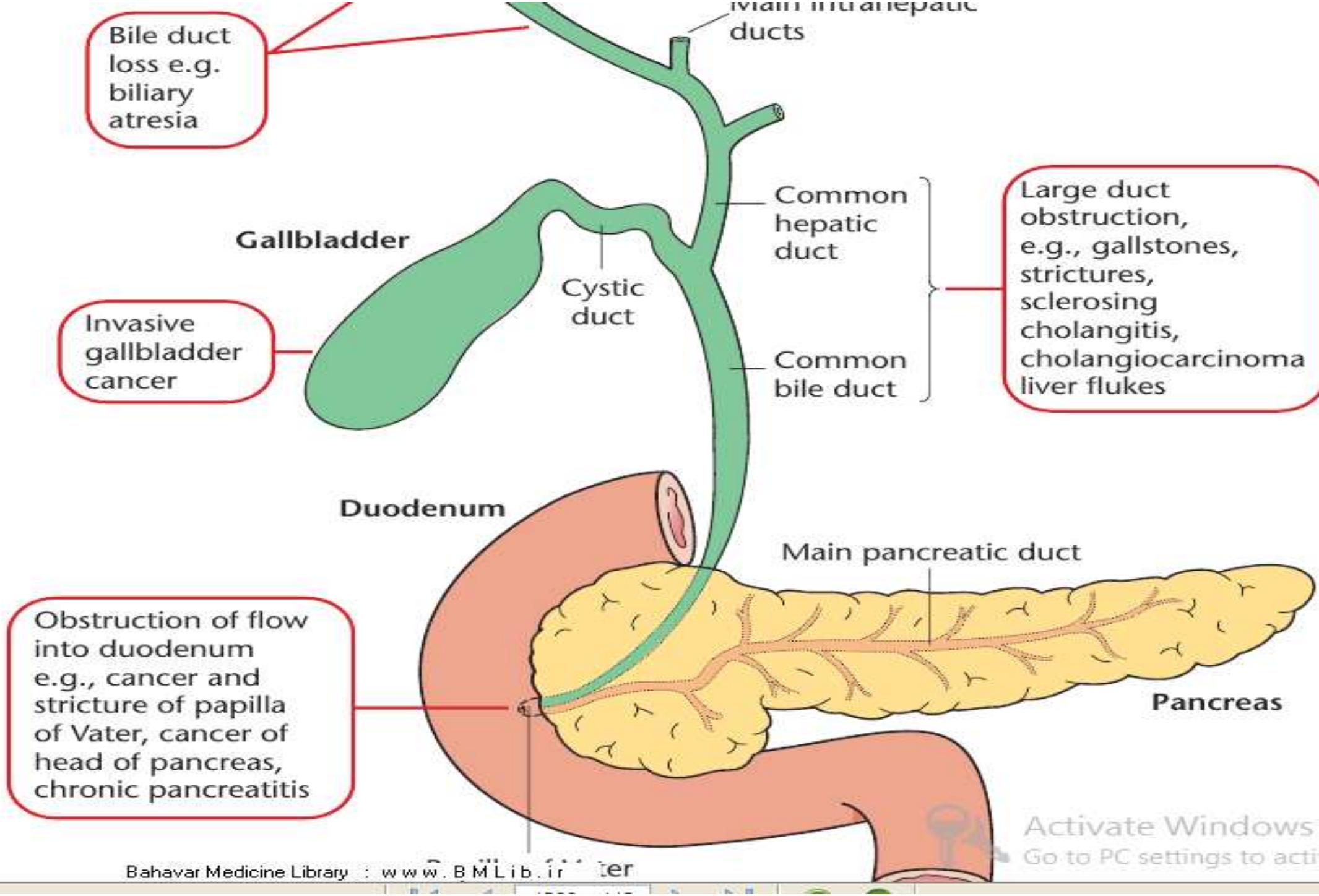
Figure 19.8 Acute mesenteric ischemia. Abdominal CT scans done at admission showing **(a)** gas in the intrahepatic branches of the left portal vein, **(b)** intravenous contrast and gas (arrow) in the main portal vein and **(c)** intramural bowel gas (arrows).

Jaundice



e 15.1 Overview of bilirubin production, transport in blood, ocyte uptake, conjugation, and biliary secretion. BDG, bilirubin uronide; BMG, bilirubin monoglucuronide; BR, bilirubin; Hb UGT1A1, 1A1 (bilirubin-specific) uridine diphosphoglucuronosyltransfe enzyme. (This figure was published in *Clinical Gastroenterology and Hepatology*. Wilfred M. Weinstein, Christopher J. Hawkey, Jaime Bosch,


Hepatocyte injury, inflammation and necrosis e.g., alcoholic, viral and drug hepatitis
Impaired intracellular transport of BR e.g. C_{17}-alkylated steroids
Hepatocytes
Canaliculus
Alagille syndrome
Tight junctions
nobiotic
mpetition
BR uptake
., rifampin
Basolateral (sinusoidal) membranes
Impaired BR conjugation due to UGTIAI defects, e.g. Gilbert and Criger-Najjar syndromes
Bile ductules
Interlobular bile ducts
Septal bile ducts
Inflammation of intrahepatic ducts e.g., PBC, sarcoid, acute and chroinc liver transplant rejection, graft-versus-host disease
Transport defects in canalicular secretion of BR
e.g., PFIC, Dubin-Johnson syndrome, ICP, and BRIC
Segmental bile ducts
Canals of Hering



CAUSES AND DIFFERENTIAL DIAGNOSIS

Causes of jaundice

Bilirubin overproduction

- Excessive red cell breakdown (hemolysis, hematomas, etc.)
- Ineffective erythropoiesis
- Nonhemoglobin hemoprotein degradation (myoglobin)

Disordered plasma transport of bilirubin

- Intravenous albumin infusion
- Conjugated bilirubin–albumin, irreversibly bound

Impaired uptake of bilirubin by hepatocytes

- Disruption of sinusoid–hepatocyte interface (cirrhosis)
- Reduced or bypassed hepatic blood flow
- Xenobiotic competition for bilirubin uptake (e.g., by rifampin)

Reduced intrahepatic bilirubin conjugation

- Immature UGT1A1 enzyme (physiologic jaundice of the newborn)
- Defective UGT1A1 enzyme biosynthesis (Gilbert and Crigler-Najjar syndromes)
- Xenobiotic competition for UGT1A1 activity (indinavir)

Nonobstructive cholestatic syndromes

- Hereditary and acquired transport defects

Obstructive cholestasis

- Inflammation, fibrosis, injury, malignant and non-malignant obstruction of the microscopic and macroscopic biliary tree

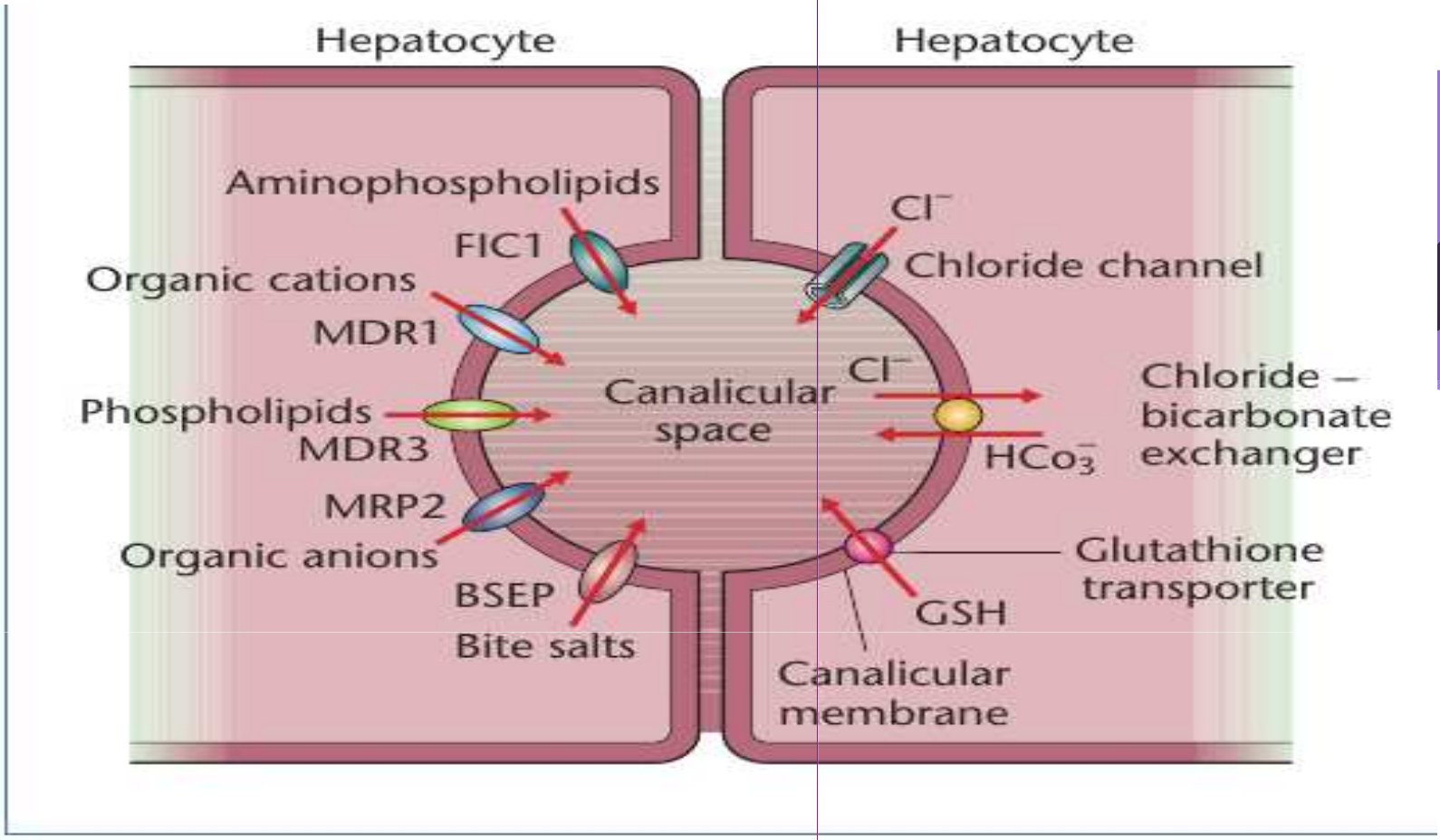
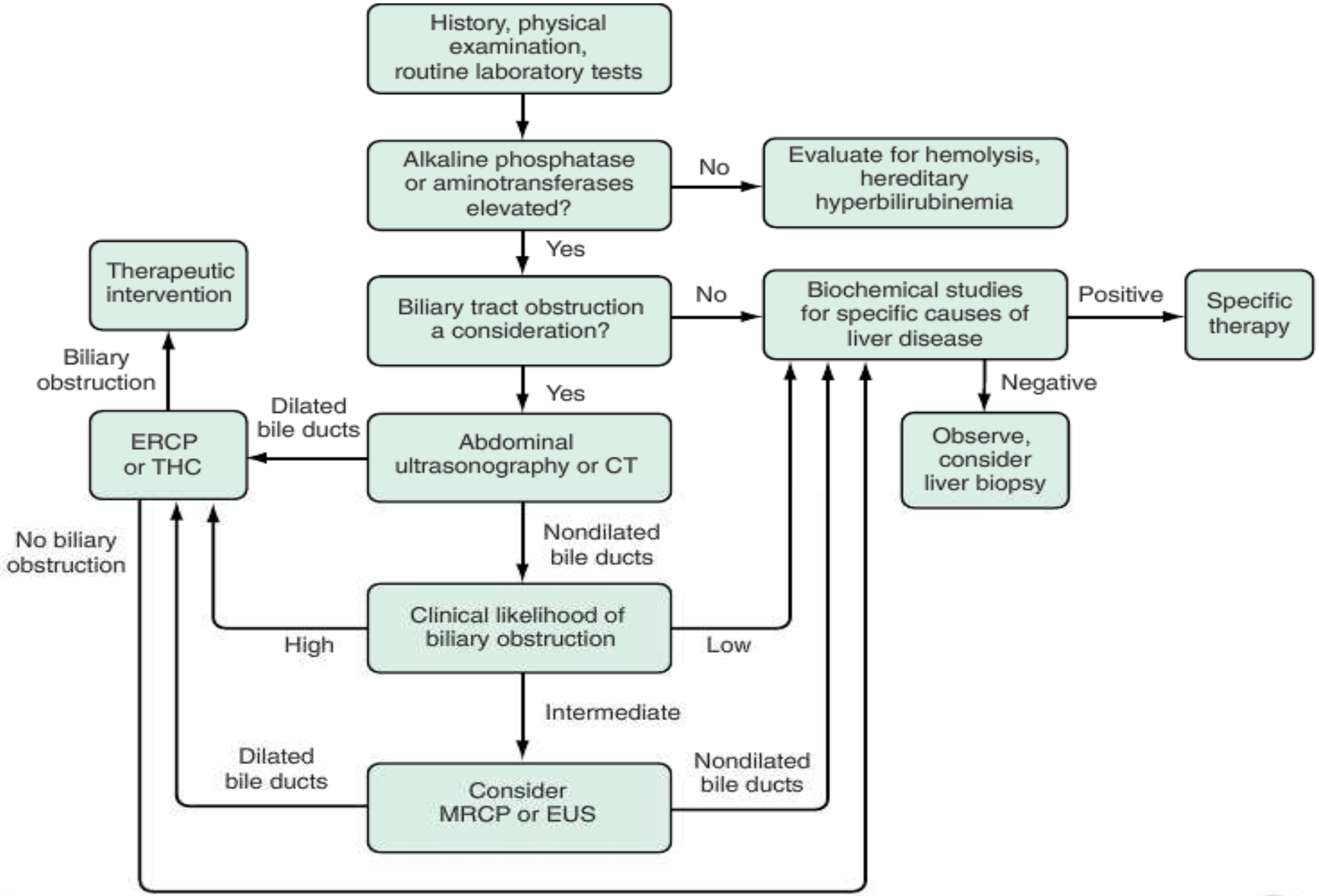
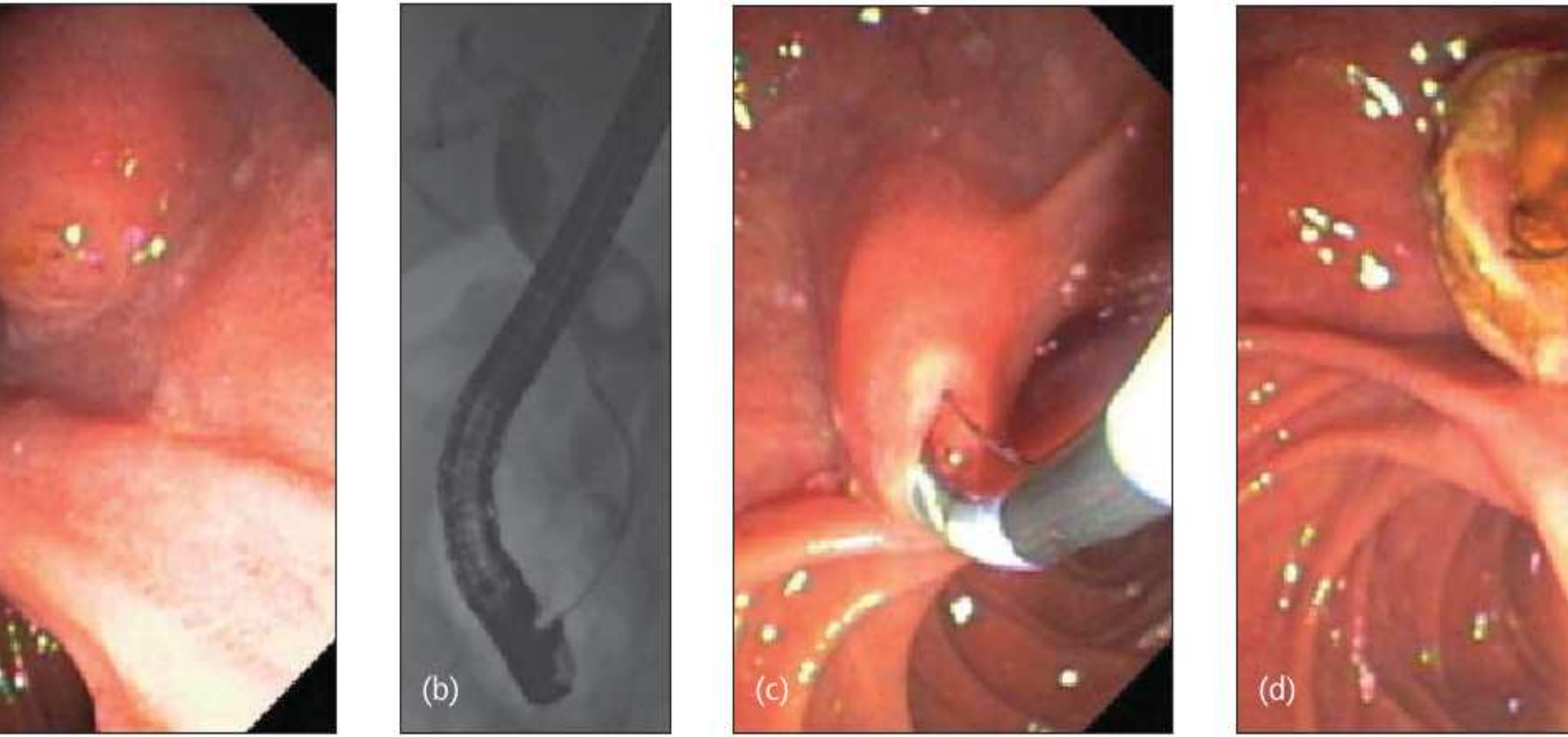


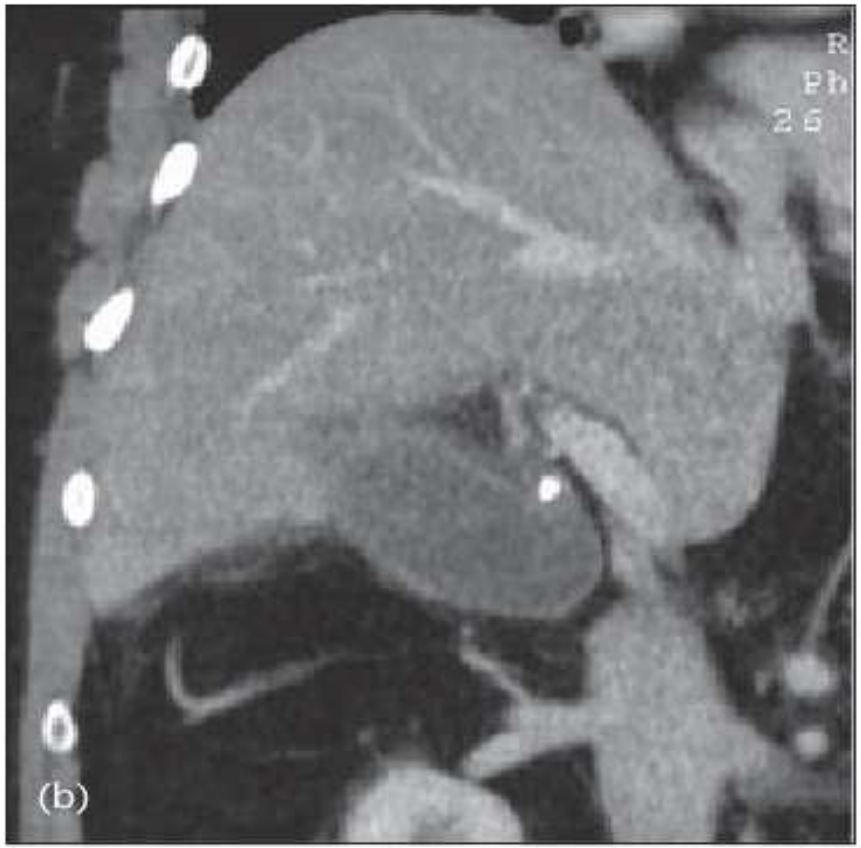
Figure 15.4 Canalicular membrane transporters – a selection. A canaliculus between two neighboring hepatocytes is represented. The canalicular membranes contain several ATP-dependent export pumps: the multidrug resistance-1 P-glycoprotein (MDR1) that transports organic cations into bile; the phospholipid transporter multidrug resistance-3 P-glycoprotein (MDR3). The multidrug resistance-associated protein 2 (MRP2, previously termed the canalicular multispecific organic anion transporter, cMOAT); and the canalicular bile salt export pump (BSEP). The canalicular membrane also contains ATP-dependent transport systems for chloride

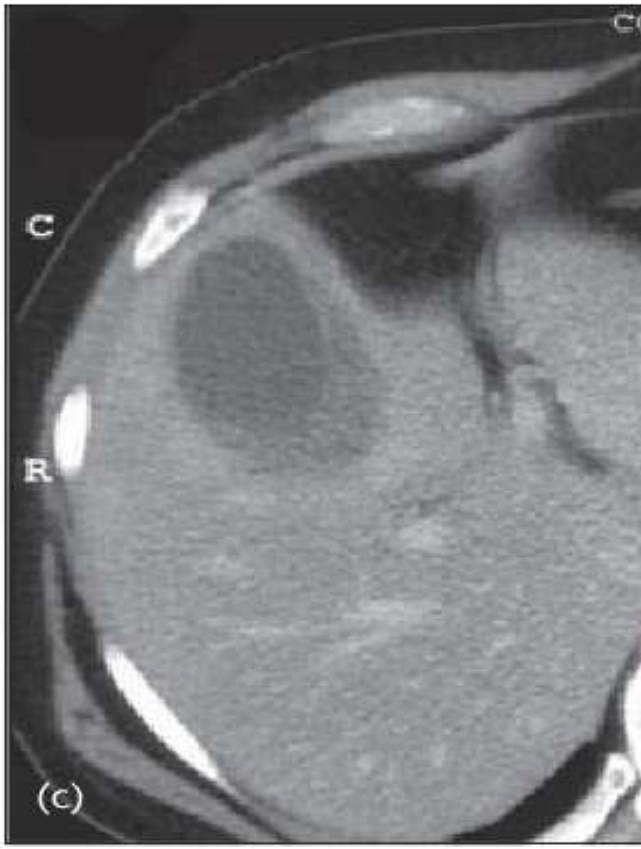


20-2. Algorithm for the evaluation and management of jaundice and hyperbilirubinemia. CT, computed tomography; ERCP, endoscopic retrograde giopancreatography; EUS, endoscopic ultrasound; MRCP, magnetic resonance cholangiopancreatography; THC, transhepatic cholangiography.

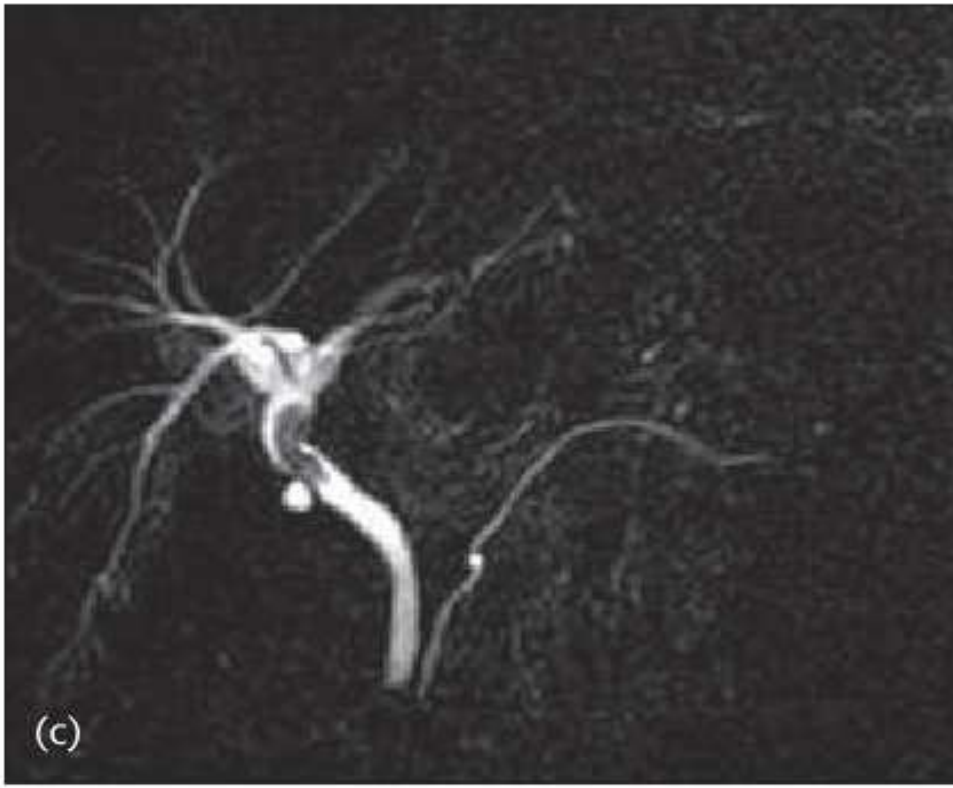

(b)


(b)
(c)
(d)





74.5 Diagnosis of cholecystitis. (a) Abdominal ultrasound showing a ericholecystic fluid collection suggestive of perforated cholecystitis llbladder sludge. The diagnosis was confirmed at laparoscopic stectomy. (b) Detection of a small stone obstructing the infundibulum of the gallbladder. (c) Computed tomography (CT) appearance of pericholecystitis as hypointense fluid collection in th patient. (CT courtesy of Prof. Dr. H.H. Schild, Department of Radiol University of Bonn, Germany).



74.4 Detection of common bile duct stones. (a) Endoscopic
de cholangiopancreatography (ERCP) showing multiple intrahepatic
The 80-year-old patient had been cholecystectomized 20 years
Note the dilated left-sided bile duct system with multiple filling
In such a patient the possibility for a genetic predisposition (e.g.,
olymorphism) should be kept in mind. (b) Endoscopic ultrasound
gh sensitivity and specificity even for the detection of stones <5 mm.
(c) Magnetic resonance cholangiopancreatography (MRCP) has a simila
sensitivity and specificity for the detection of common bile duct stones
although small stones in the distal part of the common bile duct are b
visualized by endoscopic ultrasound (MRCP courtesy of PD Dr. W. Willi
and Prof. Dr. H.H. Schild, Department of Radiology, University of Bonn
Germany).

Acute liver failure

ble 15.1 Characteristics of hyperacute, acute, and subacute liver fail

	Hyperacute	Acute	Subacute
dice to ephalopathy (wk)	0–1	1–4	4–12
ease in INR	Marked	Moderate	Mild to moderate
erity of jaundice	Moderate	Moderate	Severe
acranial ertension	Severe	Moderate	May occur
vival without liver splantation	Good	Moderate	Fair
ical causes	Paracetamol, HAV, HEV	HBV	Non-paracetamol DILI

etiology

auses of acute liver failure in the UK include:

Drug-induced hepatitis (68%):

- Paracetamol overdose.
- See Chapters 14 and 18 for other causes of DILI.

Viral hepatitis (9%) (see Chapter 14).

Toxins (2%):

- *Amanita phalloides*.
- Herbal remedies, khat (see Chapter 19).

Malignancy (1%):

- Lymphoma.
- Malignant infiltration. Often associated with high Al

Vascular (1%):

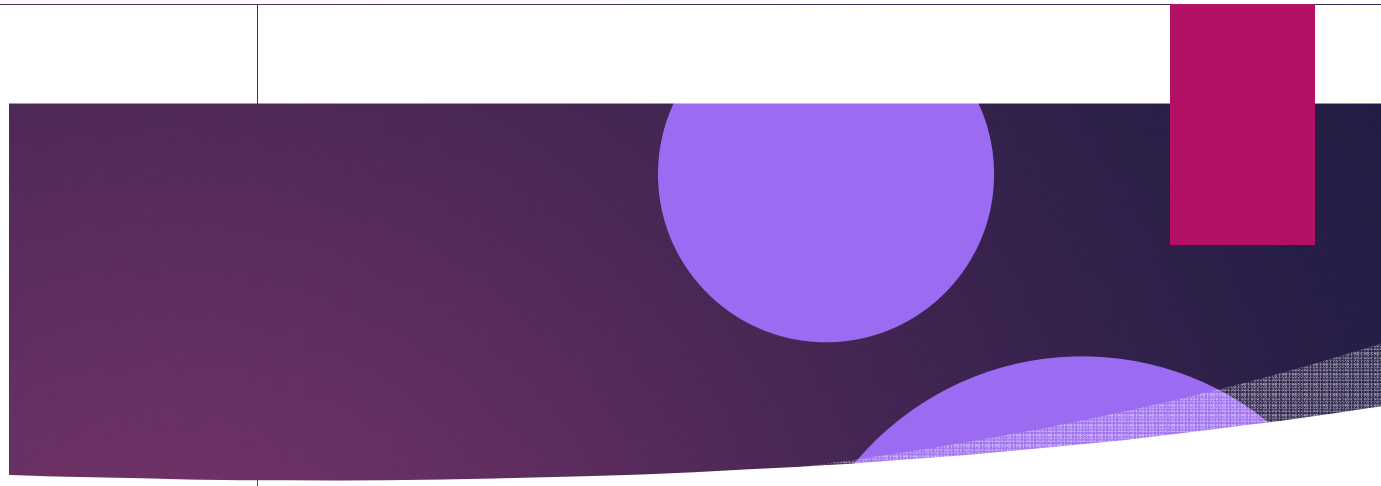
- Budd–Chiari syndrome.
- Veno-occlusive disease.
- Ischaemic hepatitis.

Miscellaneous (2%):

- Wilson's disease. Not strictly acute as many patients are cirrhoti
  but similar in other clinical respects.
- Autoimmune hepatitis.
- Malignant hyperthermia (including secondary to the drug 'ecstasy
- Pregnancy-related liver disease (see Chapter 19).
- Reye's syndrome.

Unknown (17%).

resentation



story

t specifically about:
ecent viral illnesses.
aracetamol.
lcohol and drug history.
ravel history and vaccinations.
bdominal pain, vomiting.

mination

n presents with a complication of liver failure:
gns of chronic liver disease suggest 'acute-on-chronic' liver failure
ee 📖 p. 244).
lenomegaly rare but occurs with Wilson's disease, autoimmune
patitis, and lymphoma.
racetamol overdose causes severe abdominal pain and retching.

atients are usually dehydrated on admission. Rehydrate, using
rystalloid (e.g. Hartmann's, which provides less renal Cl^- load tha
aline, thus reducing risk of hyperchloraemic metabolic acidosis),
olloid (e.g. Gelofusine®), or 4.5% human albumin solution (HAS).
void 5% glucose, as risk of causing hyponatraemia and cerebral
edema.
lace urinary, central venous, and arterial catheters, unless patient
esponds rapidly to fluid challenge. Reassess fluid balance, keeping
VP 6–10cm above mid-axillary point with colloid, HAS, or blood
se of vascular monitoring technologies (e.g. PiCCO/LiDCO) or
esophageal Doppler in ITU setting allows more accurate assessm
f haemodynamic status and cardiac output, and optimization of
ling pressures. Avoid over-hydration, which increases ICP. Pay s
ttention to aseptic insertion of any intravascular device and ensu

Start prophylactic antibiotics and antifungals (e.g. cefotaxime 2g IV od and fluconazole 200mg IV od) if at least grade 2 encephalopathy, systemic inflammatory response syndrome (SIRS; see Box 15.1), or refractory hypotension. Reduce doses if GFR <30mL/min. Avoid nephrotoxic antibiotics (e.g. aminoglycosides).

If encephalopathy is grade 1, 2, or 3, and there is no ileus, provide lactulose 10–15mL PO qds (avoid NGT in conscious patients to avoi gagging, which can raise ICP).

Stop all hepatotoxic medications (e.g. NSAIDs) and those that worse complications (e.g. ACE inhibitors, opioids).

Correct electrolyte abnormalities (if required, monitor serum levels 8–12 hourly):

- If serum Na^+ <120mmol/L, replace with crystalloid or colloid rath than fluid restrict (otherwise, hepatorenal syndrome may develop
- Correct hypokalaemia (add 40mmol KCl to each litre of fluid).
- Correct hypophosphataemia. If PO_4^{3-} <0.4mmol/L, give 25–50 mmol/L IV phosphate [illegible] 24h via a dedicated

Box 15.1 Systemic inflammatory response syndrome (SIRS)

At least two of:

- Temperature >38.5°C or <35°C.
- HR >90/min.
- RR >20/min or $PaCO_2$ <32mmHg.
- WCC >12 x 10^9/L or <4 x 10^9/L, or >10% immature (band) forms.

If ICP >20mmHg or worsening encephalopathy, provide 20% mannitol (100mL as slow IV injection qds PRN for a 70kg patient This may cause fluid overload and is less effective if renal failure (in which case remove 3–5 times the volume of mannitol infused by haemofiltration). Hypertonic 3% saline 500mL IV, or 10–20mL boluses of 30% saline, to maintain serum Na^+ at 145–150mmol/L are alternatives. Pentobarbital induced coma may be required. There is no role for hyperventilation (compromises cerebral blo flow). Indometacin 25mg IV over 1min can acutely depress ICP. N-acetylcysteine (NAC) is second-line treatment.
Moderate hypothermia (to 32–34°C) can reduce ICP by modulat cerebral blood flow.

NAC provided for 72h with a loading dose regime has been
hown in one report to be effective in mild encephalopathy from
non-paracetamol acute liver injury, with 40% vs 27% transplant-fre
urvival.
Specific management strategies have been used in certain settings
lso Chapter 14):

- Anti-viral therapy in acute HBV infection.
- Aciclovir IV in acute herpes virus infection.

Activated charcoal and forced diuresis following *Amanita phalloides* mushroom poisoning. There is some evidence for high-dose benzylpenicillin ± silibinin 20–50mg/kg/d IV (prevents uptake of toxin by undamaged hepatocytes).

TIPS, surgical decompression, or thrombolysis in acute Budd–Chiari syndrome.

Steroids can be used in autoimmune hepatitis, although often the presentation is too late for them to be effective.

Wilson's disease (see 📖 p. 243).

Expedite delivery for ALF due to acute fatty liver of pregnancy or HELLP syndrome.

ALF due to malignant infiltration has a dismal prognosis, although occasional patients with lymphomatous/leukaemic infiltration survive and then undergo successful chemotherapy. The majority

Markers of disease severity

The main prognostic factors for survival are degree of encephalopat
(see 📖 p. 245), patient age (prognosis worse if <10y or >40y), cause
ALF (better prognosis with paracetamol, HAV, and HBV), and INR.

Progressive renal dysfunction can also result from type 1 hepatorenal syndrome (see Box 15.2). If present, terlipressin 2g IV qds and 4.5% HAS IV enhance renal perfusion. Preliminary reports suggest benefit providing 7.5–12.5mg tds midodrine and 100–200µg octreotide SC tds, or noradrenaline and albumin IV; both titrated to raise MAP >15mmHg. Seek early renal support with haemofiltration.

Box 15.2 Hepatorenal syndrome

Diagnose if:

- Acute or chronic liver disease with hepatic failure.
- Creatinine >200μmol/L, or creatinine clearance <40mL/min.
- No sustained improvement with 1.5L volume expansion.
- Proteinuria <0.5g/d.
- Normal renal tract US.
- Other causes of renal impairment excluded.

- Correct hypophosphataemia. If PO_4^{3-} <0.4mmol/L, give 25–50 mmol/L IV phosphate Polyfusor® over 12–24h via a dedicated large bore peripheral cannula, then recheck levels. Provide 40–8 mmol in divided doses over 24h if PO_4^{3-} between 0.4–0.7mmol/L Hypophosphataemia is more common with paracetamol poisoni
- Hyperphosphataemia in paracetamol overdose also carries prognostic significance, with worse outcomes if PO_4^{3-} >1.0mmol
- Respiratory alkalosis is common early in illness. Lactic acidosis is often present early, when it may rapidly correct with volume replacement. Where it persists or develops later, it carries a poo prognosis.

Keep blood glucose >3.5mmol/L with continuous 10–20% glucose nfusion.

Give vitamin K 10mg IV as a single dose (usually has no effect on IN

Maintain platelets >20 x 10^9/L with transfusions.

Drug-induced liver injury

g-induced liver injury (DILI) accounts for ~1% of general medica
issions, <5% of all cases of jaundice, but up to 30% of acute live
re. It is associated with >1,000 medications and herbal products. Th
owing principles apply:

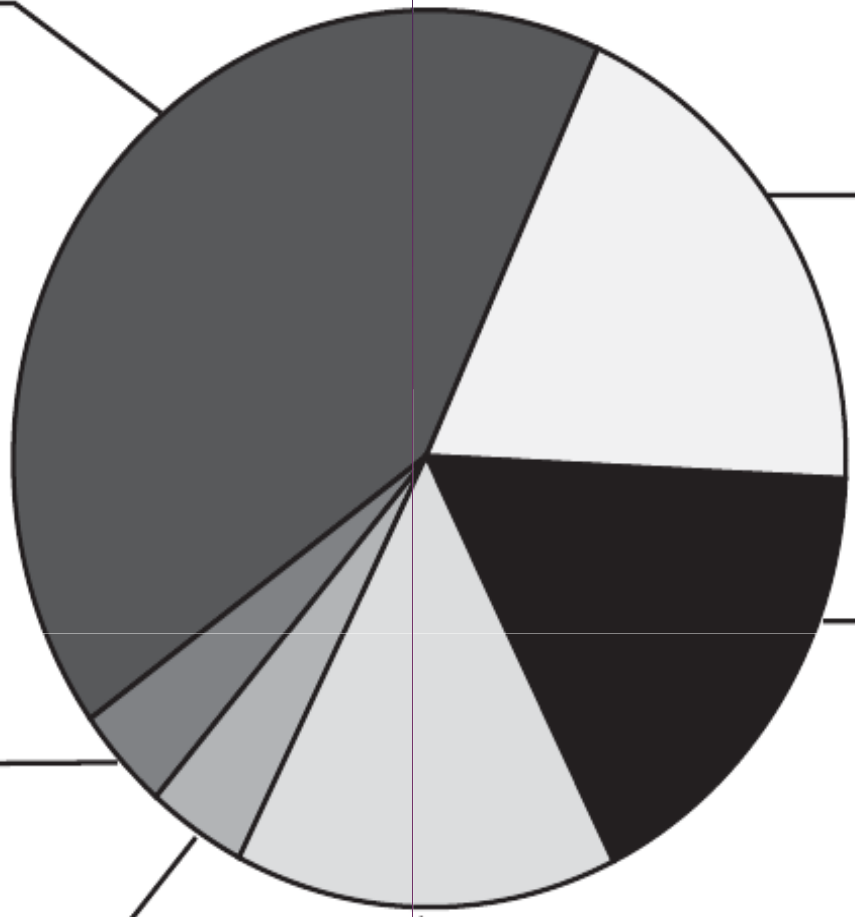
- ll drugs may cause acute liver injury.
- lost forms of DILI lead to an acute hepatitis with raised transaminases
- ome drugs cause predominantly cholestatic liver injury.
- ome drugs can cause fatty change, or fibrosis and cirrhosis.
- One drug may cause multiple patterns of injury.
- lost drug reactions are idiosyncratic.
- he diagnosis is one of exclusion.
- rug withdrawal does not always lead to improvement.
- rug challenge is rarely justified.

Table 18.1 Drug-injury liver disease

Pattern	Common drugs
Fulminant hepatitis	Paracetamol, halothane
Acute or chronic hepatitis	Co-amoxiclav (younger patients), anabolic steroids, anti-tuberculosis drugs, anti-retrovirals, aspirin, bupropion, disulfiram, ketoconazole, lisinopril, losartan, methyldopa, nitrofurantoin, sodium valproate, SSRIs, statins
Subclinical liver disease	Antibiotics, antidepressants, isoniazid, lipid-lowering drugs, sulfonamides, salicylates, sulfonylureas, quinidine
Granulomatous hepatitis	Allopurinol, carbamazepine, diltiazem, hydralazine, phenytoin, quinine, sulphur-containing drugs
Hepatic fibrosis	Vitamin A, methotrexate
Steatosis (macrovesicular)	Amiodarone, tamoxifen, valproate
Steatosis (microvesicular)	Nucleoside reverse transcriptase inhibitors, sodium valproate, tetracycline
Hepatic neoplasia	Anabolic steroids, oestrogens
Budd–Chiari syndrome	Oestrogens
Hepatic sinusoidal obstruction	Azathioprine, busulfan, mercaptopurine, tetracycline, vitamin A
Cholestasis	Anabolic steroids, azathioprine, carbamazepine, chlorpromazine, clopidogrel, co-trimoxazole, diclofenac, efavirenz, erythromycin, ezetimibe, ketoconazole, nevirapine, oestrogens, penicillins (e.g co-amoxiclav in elderly patients), phenytoin, rifampicin, rosiglitazone, co-trimoxazole, tricyclics
Mixed patterns	Phenytoin, quinolones

Table 18.2 Drugs that are more toxic in chronic liver disease

Drug	Risk factor
Anti-retrovirals	Hepatitis B, hepatitis C
Anti-tuberculosis drugs	Hepatitis B, hepatitis C
Ibuprofen	Hepatitis C
Methimazole	Hepatitis B
Methotrexate	Alcoholic liver disease, steatohepatitis
OCP	Liver tumours
Rifampicin	PBC
Vitamin A	Alcoholic liver disease



Other drugs:
Propylthiouracil (19)
Disulfiram (9)
Halothane (8)
Herbal (6)
Amitriptyline (2)
Nefazodone (2)
Methotrexate (5)
Troglitazone (4)
Methyldopa (5)
Mercaptopurine or azathioprine (3)
Fialuridine (3)

Anti-tuberculosis:
Isoniazid (48)
Isoniazid plus another anti-tuberculosis drug (2)

Anti-epileptics:
Phenytoin (20)
Valproate (20)
Carbamazepine (3)
Single case:
Felbamate

Non-steroidal anti-inflammatories:
Diclofenac (3)
Bromfenac (2)
Ibuprofen (2)
Single cases:
Etodolac
Naproxen
Indometacin

Statins:
Atorvastatin (3)
Cerivastatin (2)
Simvastatin (2)
Single cases:
Pravastatin
Ezetimibe

Antibiotics:
Nitrofurantoin (12)
Ketoconazole (8)
Amoxicillin and clavulanate (5)
Trimethoprim-sulfamethoxazole (2)
Minocycline (2)
Single cases:

Treatment is largely supportive, as there are no specific treatments fo
LI other than:
NAC for paracetamol toxicity (see 📖 p. 296).
L-carnitine for valproate toxicity. Loading dose of 100mg/kg IV (to
maximum of 6g) over 30min, followed by 15mg/kg every 4h over
10–30min until clinical improvement occurs.

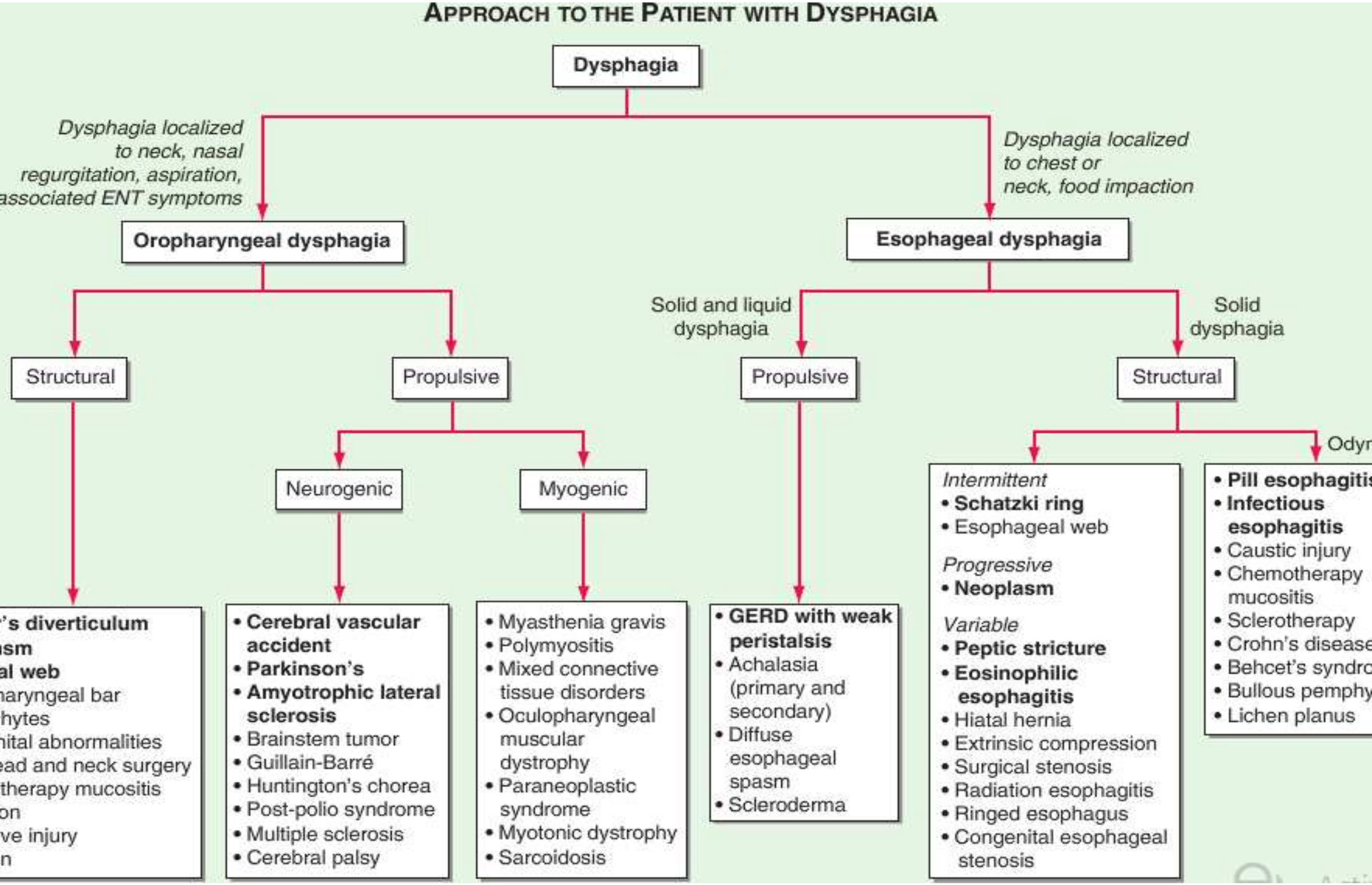
Paracetamol overdose

- Arterial pH <7.3 following adequate volume resuscitation, or
- Combination of:
  - Encephalopathy > grade 3.
  - Creatinine >300µmol/L.
  - INR >6.5 (PTT >100s).

Non-paracetamol overdose

- Any grade encephalopathy and INR >6·5 (PTT >100s), or.
- Three of:
  - INR >3·5 (PTT >50s).
  - Bilirubin >300µmol/L.
  - Age <10y or >40y.
  - Unfavourable cause (DILI, non-A non-B hepatitis).
  - Jaundice >7d pre-encephalopathy.

Acute thoracic pain and dysphagia


APPROACH TO THE PATIENT WITH DYSPHAGIA
Dysphagia
Dysphagia localized to neck, nasal regurgitation, aspiration, associated ENT symptoms
Dysphagia localized to chest or neck, food impaction
Oropharyngeal dysphagia
Esophageal dysphagia
Structural
Propulsive
Solid and liquid dysphagia
Solid dysphagia
Propulsive
Structural
Neurogenic
Myogenic
's diverticulum
sm
al web
haryngeal bar
hytes
nital abnormalities
ead and neck surgery
therapy mucositis
on
ve injury
n
• Cerebral vascular accident
• Parkinson's
• Amyotrophic lateral sclerosis
• Brainstem tumor
• Guillain-Barré
• Huntington's chorea
• Post-polio syndrome
• Multiple sclerosis
• Cerebral palsy
• Myasthenia gravis
• Polymyositis
• Mixed connective tissue disorders
• Oculopharyngeal muscular dystrophy
• Paraneoplastic syndrome
• Myotonic dystrophy
• Sarcoidosis
• GERD with weak peristalsis
• Achalasia (primary and secondary)
• Diffuse esophageal spasm
• Scleroderma
Intermittent
• Schatzki ring
• Esophageal web
Progressive
• Neoplasm
Variable
• Peptic stricture
• Eosinophilic esophagitis
• Hiatal hernia
• Extrinsic compression
• Surgical stenosis
• Radiation esophagitis
• Ringed esophagus
• Congenital esophageal stenosis
Odyn
• Pill esophagitis
• Infectious esophagitis
• Caustic injury
• Chemotherapy mucositis
• Sclerotherapy
• Crohn's disease
• Behcet's syndro
• Bullous pemphy
• Lichen planus

1 Causes of dysphagia and odynophagia

aryngeal dysphagia	Esophageal dysphagia	Odynophagia
ural	**Structural**	Gastroesophageal reflux disease (unusual)
entition	*Intraluminal*	Medication induced esophagitis
omia	Stricture	Infectious esophagitis
minal	Schatzki's ring	(Candida, herpes, CMV)
's diverticulum	Cancer	Radiation injury
al web	Hiatal hernia	Caustic ingestion
aryngeal tumor	Eosinophilic esophagitis	
minal	*Extraluminal*	
al osteophytes	Mediastinal tumors (lymphoma, lung cancer)	
negaly	Vascular structures	
adenopathy	(dysphagia lusoria, dysphagia aortica)	
	Duplication cyst	
	Postsurgical changes (fundoplication)	
enic	**Motility abnormalities**	
henia gravis	*Primary*	
tomyositis	Achalasia	
yositis	Distal esophageal spasm	
lic myopathy	Hypercontractile motility	
idosis	Hypertensive LES	
hyperthyroidism	Nutcracker esophagus	
g's syndrome	Hypocontractile motility	
	Hypotensive LES	
	Ineffective esophageal motility	
	Secondary	
	Secondary achalasia	

Table 2.2 Symptom complexes

Oropharyngeal dysphagia	Esophageal dysphagia
Dysphagia within 1 sec of swallowing	Dysphagia delayed until mid chest
Choking, cough with initiation of eating	Heartburn
Nasal regurgitation	Regurgitation
Dysarthria and diplopia	Chest pain
Facial muscle weakness (ptosis, facial droop)	Cough
Dysphonia/nasal speech	
Halitosis/gurgling noise	

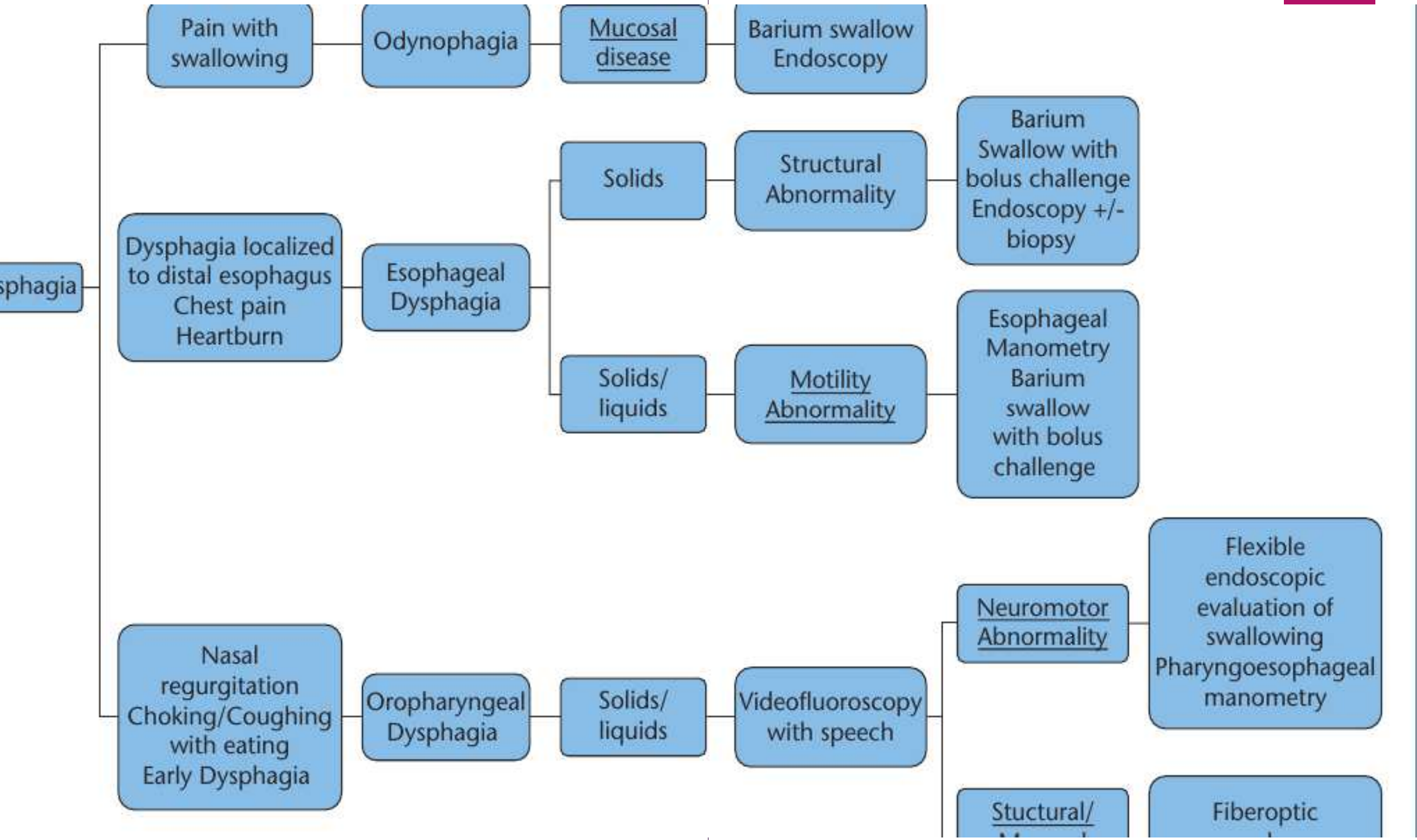

sphagia
Pain with swallowing
Odynophagia
Mucosal disease
Barium swallow Endoscopy
Dysphagia localized to distal esophagus Chest pain Heartburn
Esophageal Dysphagia
Solids
Structural Abnormality
Barium Swallow with bolus challenge Endoscopy +/- biopsy
Solids/ liquids
Motility Abnormality
Esophageal Manometry Barium swallow with bolus challenge
Nasal regurgitation Choking/Coughing with eating Early Dysphagia
Oropharyngeal Dysphagia
Solids/ liquids
Videofluoroscopy with speech
Neuromotor Abnormality
Flexible endoscopic evaluation of swallowing Pharyngoesophageal manometry
Stuctural/
Fiberoptic

Table 2.3 Medications implicated in esophageal injury and dysphagia

Drugs implicated in esophageal injury	Drugs and dysphagia
Antibacterials	**Effects on striated muscle function**
Doxycycline	Sedatives
Tetracycline	Narcotics
Amoxicillin	Antipsychotics
Penicillin	Neuroleptic therapy (extrapyramidal motor disturbances)
Clindamycin	
Rifampin	**Effects on smooth muscle function**
Nonsteroidal anti-inflammatory drugs	*Inhibitory*
Aspirin	Alcohol
Ibuprofen	Tricyclic antidepressants
Naproxen	Theophylline
Diclofenac	Calcium channel blockers
Indomethacin	Alcohol
Bisphosphonates	*Excitatory*
Alendronate	Cholinergic agonists
Pamidronate	Prokinetics
Etidronate	**Decrease lower esophageal pressure**
Risedronate	Progesterone
Others	Calcium channel blockers
Ascorbic acid	Nitrates
Ferrous sulfate	Alcohol
Prednisone	**Xerostomia**
Potassium chloride	Anticholinergics
	Antiemetics

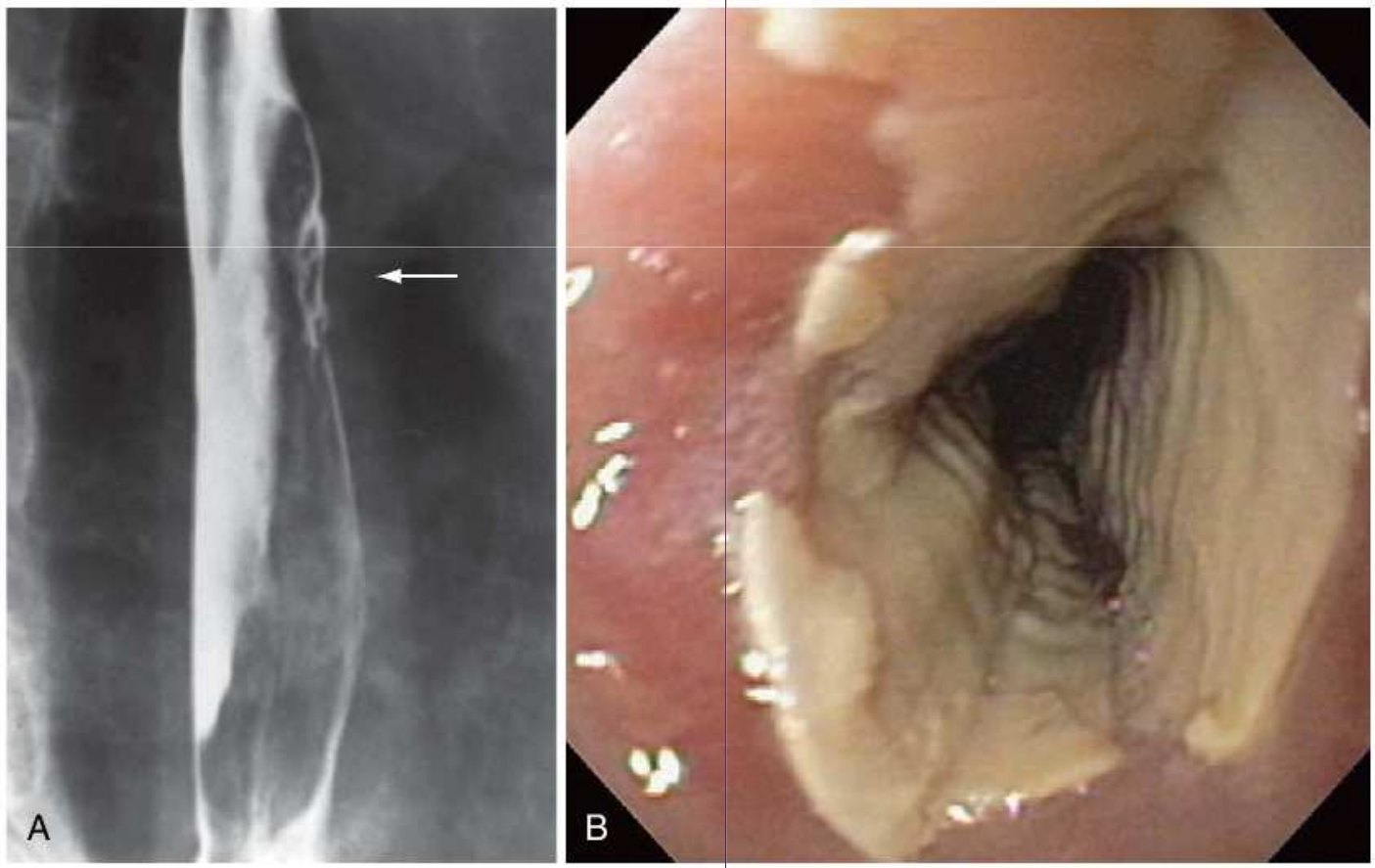
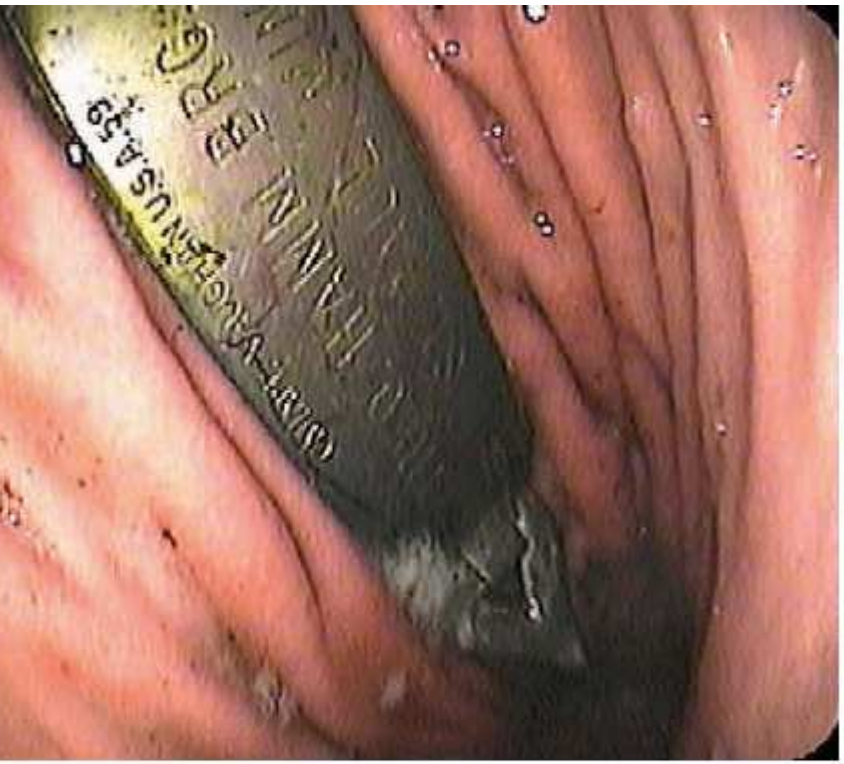


Figure 45-1. *A*, Esophageal ulceration secondary to tetracycline, with arrow pointing to area of ulcerations demonstrated by barium esophagography. *B*, Endoscopic image of tetracycline-induced esophageal burn. (*A*, Courtesy Dr. Marc Levine, Philadelphia, Penn.)



1. Endoscopic image of a bottle opener (in the stomach) y an intoxicated patient.

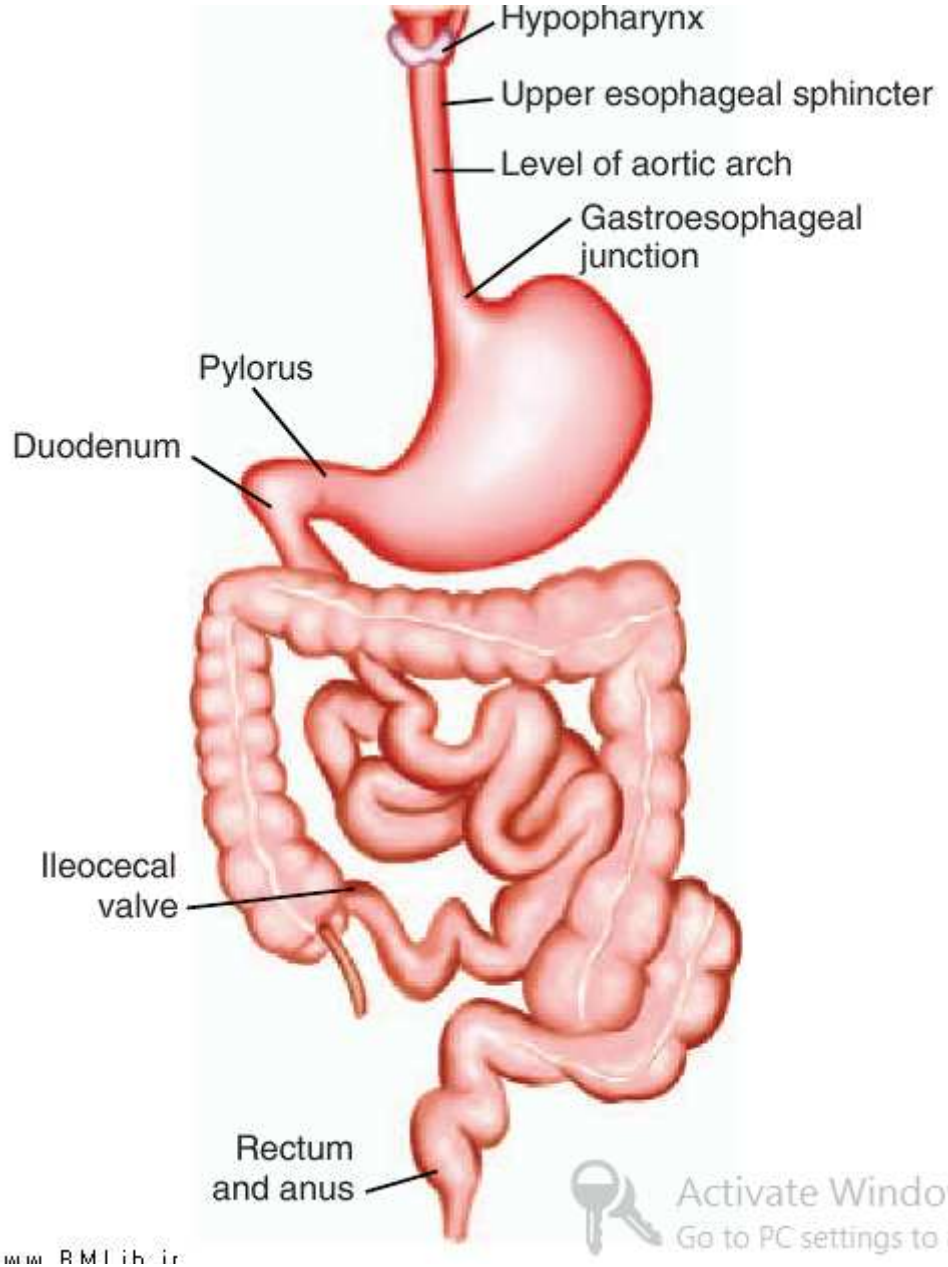


Figure 25-4. Radiograph of the chest demonstrating pneumomediastinum and bilateral pneumothoraces in a patient who developed esophageal perforation secondary to food impaction left untreated for longer than 24 hours.