KAPLAN MEDICAL

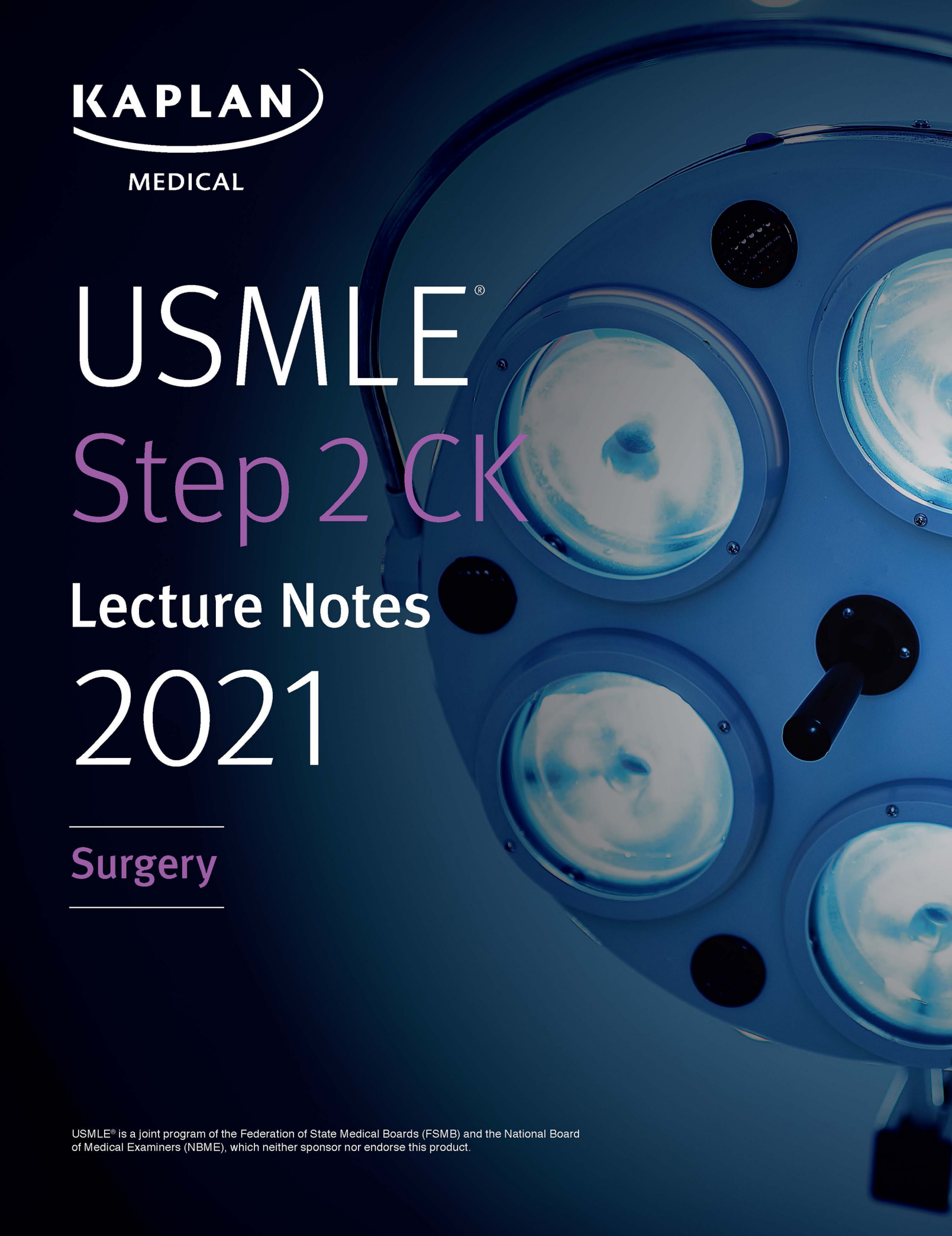
USMLE®

Step 2 CK

Lecture Notes

2021

Surgery



KAPLAN MEDICAL

USMLE®
Step 2 CK
Lecture Notes
2021

Surgery

Published by Kaplan Medical, a division of Kaplan, Inc.
750 Third Avenue
New York, NY 10017

10 9 8 7 6 5 4 3 2 1

Course ISBN: 978-1-5062-6153-9
Course Kit ISBN: 978-1-5062-6135-5

Retail ISBN: 978-1-5062-6151-5
Retail Kit ISBN: 978-1-5062-6137-9
Kit items come as a set and should not be broken out and sold separately.

Kaplan Publishing print books are available at special quantity discounts to use for sales promotions, employee premiums, or educational purposes. For more information or to purchase books, please call the Simon & Schuster special sales department at 866-506-1949.

Editors

Mark Nolan Hill, MD, FACS
Professor of Surgery
Chicago Medical School
Chicago, IL

Gary Schwartz, MD, FACS
Department of Thoracic Surgery & Lung Transplantation
Baylor University Medical Center
Dallas, TX

Contributors

Ted A. James, MD, MSc, FACS
Medical Director and Vice Chair,
Academic Affairs at Beth Israel
Deaconess Medical Center

Associate Professor of Surgery at
Harvard Medical School

Teaching Faculty for Patient Safety at
Harvard Medical School, AAMC, and the
Institute for Healthcare Improvement
Boston, MA

Carlos Pestana, MD, PhD
Emeritus Professor of Surgery
University of Texas Medical School at San Antonio
San Antonio, TX

We want to hear what you think. What do you like or not like about the Notes?
Please email us at **medfeedback@kaplan.com**.

Table of Contents

Additional resources available at kaptest.com/usmlebookresources

Preoperative and Postoperative Care 1

Learning Objectives

- ❑ Recognize the factors essential to a preoperative assessment
- ❑ Describe the approach to diagnosis and management of postoperative complications

PREOPERATIVE ASSESSMENT

Prior to **elective** surgery, a patient should be evaluated for potential risks associated with surgery and general anesthesia. These include cardiac, pulmonary, hepatic, nutritional, and metabolic factors that can contribute to intra- and postoperative complications.

Cardiac Risk

The revised cardiac risk index (RCRI) can be used to estimate the risk of cardiac complications for patients undergoing noncardiac operative procedures under general anesthesia. This index is composed of the following variables:

- History of ischemic cardiac disease
- History of congestive heart failure (CHF)
- History of stroke or cerebrovascular accident (CVA)
- Diabetes mellitus
- Chronic kidney disease (CKD, or creatinine >2 mg/dL)
- Planned surgery for thoracic, intra-abdominal, or infrainguinal vascular disease

The risk of cardiac death, cardiac arrest, or nonfatal perioperative myocardial infarction is based on total score.

Score	Risk
0 factors	<0.4% risk
1 factor	0.9% risk
2 factors	6.6% risk
3 or more factors	>11% risk

Note

All general anesthetics **decrease inotropy** and **increase ectopy**.

Pulmonary Risk

Smoking is by far the most common cause of increased pulmonary risk; the problem is compromised ventilation more than compromised oxygenation. Increased pCO_2 and decreased FEV_1 are the most significant predictors of advanced disease.

Smoking history or the presence of chronic obstructive pulmonary disease (COPD) should lead to evaluation with pulmonary function testing.

Smoking cessation and intensive respiratory therapy (physical therapy, expectorants, incentive spirometry, humidified air) should precede elective surgery when possible.

Hepatic Risk

Perioperative risk due to hepatic disease is stratified by several systems, most notably the Child-Pugh classification system and the Model for End-Stage Liver Disease (MELD). The most common disease affecting the liver is alcoholism.

The Child-Pugh system incorporates **A**scites, **B**ilirubin, **C**lotting (prothrombin time), **D**iet (serum albumin), and **E**ncephalopathy (presence/absence). Predicted surgical mortality is as follows:

- Mortality of ~40% is predictable with bilirubin >2 mg/dL, albumin <3 g/dL, prothrombin time >16 sec, or encephalopathy.
- Mortality of ~80–85% is predictable if 3 of the above are present (close to 100% if all 4 exist) or if bilirubin alone is >4 mg/dL, albumin alone is <2 g/dL, or blood ammonia concentration alone is >150 mg/dL.

The MELD score uses the patient's serum bilirubin, creatinine, and INR (normalized prothrombin time) to predict survival and estimate hepatic reserve. Online and app-based calculators are available to calculate the score.

The table shows MELD scores and their associated mortality rates.

MELD score	Mortality rate
<9	1.9%
10–19	6%
20–29	19.6%
30–39	52.6%
≥40	71.3%

Nutritional Risk

Malnutrition results in immunodeficiency and impairs healing, significantly increasing the risk of major surgery. Severe nutritional depletion is identified by one or more of the following:

- Loss of 20% of body weight over 6 months
- Serum albumin <3 g/dL; prealbumin <16 mg/dL
- Serum transferrin level <200 mg/dL

Operative risk is increased significantly in the presence of malnutrition. As few as 4–5 days of preoperative nutritional support (preferably enteral) can make a big difference; 7–10 days is optimal if the surgery can be deferred for that long.

Metabolic Risk

Diabetic ketoacidosis is an absolute contraindication to surgery. Rehydration, return of urinary output, and at least partial correction of the acidosis and hyperglycemia must be achieved before surgery can be undertaken.

Cardiac Risk

> A 72-year-old man with a history of multiple myocardial infarctions is scheduled to have an elective sigmoid resection for diverticular disease. A preoperative echocardiogram shows ejection fraction 35%.

With this ejection fraction, the incidence of perioperative myocardial infarction is ~75%, and the associated mortality rate is 50–90%. In this case, elective surgery is most likely not an option. Continue with medical therapy for the diverticular disease and to optimize cardiac function. If the patient develops an abscess, consider percutaneous drainage to avoid surgical intervention.

> A 72-year-old chronically bedridden man is being considered for emergency cholecystectomy for acute cholecystitis that is not responding to medical management. Four months ago he had a myocardial infarction. Currently he has paroxysmal atrial fibrillation.

This patient has multiple risk factors correlating to a ~20% predicted mortality. Nonsurgical treatment (in this case, percutaneous cholecystostomy tube under local anesthesia) should be pursued.

A 72-year-old man is scheduled to have an elective sigmoid resection for diverticular disease. In the preoperative evaluation it is noted that he has venous jugular distention.

Not a lot of information is provided, but what is given raises suspicion for CHF, which is the worst cardiac risk predictor. Further evaluation starting with echocardiography should be pursued, and the patient should be medically optimized prior to surgery with ACE inhibitors, beta-blockers, and diuretics.

A 61-year-old man with a 20-pack-year smoking history needs elective surgical repair of an abdominal aortic aneurysm. He has cut back on smoking to half a pack per day.

Smoking is by far the most common cause of increased pulmonary risk; smoking cessation and respiratory therapy should precede surgery. Do a complete pulmonary evaluation with pulmonary function testing and optimization with bronchodilators and secretion management. A rapidly growing aneurysm at risk for rupture will need more urgent intervention prior to optimization.

A 49-year-old alcoholic presents with upper gastrointestinal bleeding from a duodenal ulcer. On examination she has bilirubin 3.5 mg/dL, prothrombin time 22 seconds, and serum albumin 2.5 g/dL. Ascites is present.

This patient likely has advanced cirrhosis. Surgical intervention is contraindicated.

- If only one of these conditions is present (bilirubin >2 mg/dL, prothrombin time >16, albumin <3), mortality is predicted at >40%.
- If 3 of these conditions are present, mortality is as high as 85%.

Attempt nonsurgical treatment with blood product resuscitation and consider nonsurgical options such as endoscopic clipping or endovascular embolization.

A 78-year-old man needs palliative surgery for an obstructing cancer of the colon. He has lost 20% of his body weight over the past 2 months. Serum albumin is 2.7.

Any one of these findings indicates severe nutritional depletion. Delaying surgical intervention for several days of preoperative nutrition would decrease some of the risk. This must be taken into consideration when contemplating a palliative procedure.

An older diabetic man presents with a clinical picture of acute cholecystitis that has been present for 3 days. He is profoundly dehydrated and confused, and has blood sugar 550 mg/dL with severe metabolic acidosis.

Diabetic ketoacidosis is a contraindication to surgical intervention. This vignette presents a challenging situation because the patient's hyperglycemia will continue to worsen as long as sepsis is present. Therefore, when the acidosis has resolved, nonsurgical management of the

infection should be pursued—in this case, a percutaneous cholecystostomy tube and definitive source control with cholecystectomy.

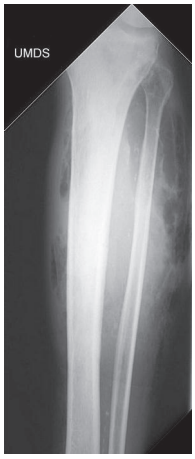
PERIOPERATIVE COMPLICATIONS

Fever

Malignant hyperthermia develops shortly after the onset of the anesthetic (most commonly halothane or succinylcholine). Symptoms include temperature >40 C (104 F), metabolic acidosis, hypercalcemia, and hyperkalemia. A family history may exist; the patient should always be questioned preoperatively. Treatment is **IV dantrolene**, 100% oxygen, correction of the acidosis, and cooling blankets. Monitor postoperatively for the development of myoglobinuria (very uncommon).

Bacteremia is seen within 30–45 minutes of invasive procedures (instrumentation of the urinary tract is a classic example) and presents as chills and a temperature spike as high as 40 C (104 F). Draw multiple sets of blood cultures and start empiric broad-spectrum antibiotics.

Although the condition is rare, severe wound pain and very high fever within hours of surgery should alert you to the possibility of a **necrotizing soft tissue infection**. Immediately remove surgical dressings to examine the wound and promptly return to the OR for wound reopening, debridement, and washout.



Courtesy of SRS-X, Scottish Radiological Society

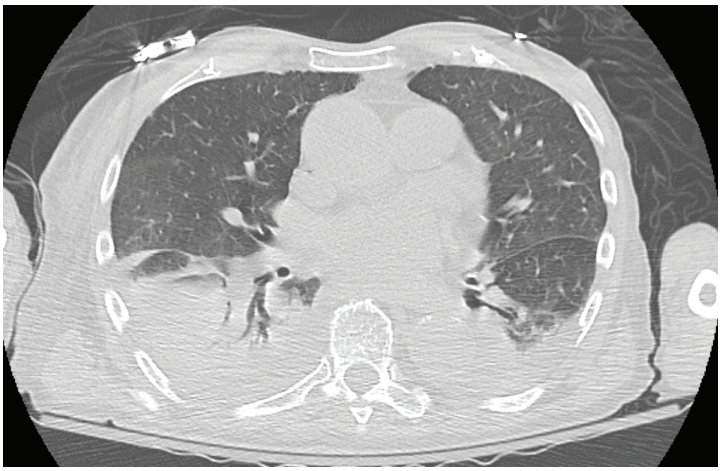
Figure 1-1. Necrotizing Soft Tissue Infection due to *Clostridium Perfringens*

Postoperative fever typically is not as high as in the previous examples, usually 38.3–39.4 C (101–103 F). Fever in the postoperative period is caused (in order of time sequence) by atelectasis, pneumonia, urinary tract infection (UTI), deep vein thrombosis (DVT), wound infection, or deep abscesses.

Atelectasis is the most common source of fever on the first postoperative day. Assess the risk of the other causes already noted, listen to the lungs, do a chest x-ray, and improve ventilation (deep breathing and coughing, postural drainage, incentive spirometry). No need to order a CT or blood cultures in this early postoperative period, as this is generally an empiric diagnosis. Bronchoscopy with clearing of secretions is occasionally necessary.

Clinical Pearl

In post-op patients, fever commonly arises from **W**ind (atelectasis), **W**ater (UTI), and **W**ound (wound infection). (Note that **W**alking [DVT]—historically part of the 4 Ws—is now much less common in the post-op period due to the absolute necessity of DVT prophylaxis.)



Courtesy of Gary Schwartz, MD

Figure 1-2. Atelectasis

Pneumonia will happen in about 3 days if atelectasis is not resolved (atelectasis is a prelude to pneumonia). Fever will persist, leukocytosis will be present, and chest x-ray will demonstrate infiltrate(s). There may be purulent sputum. Obtain sputum cultures and treat with appropriate antibiotics.

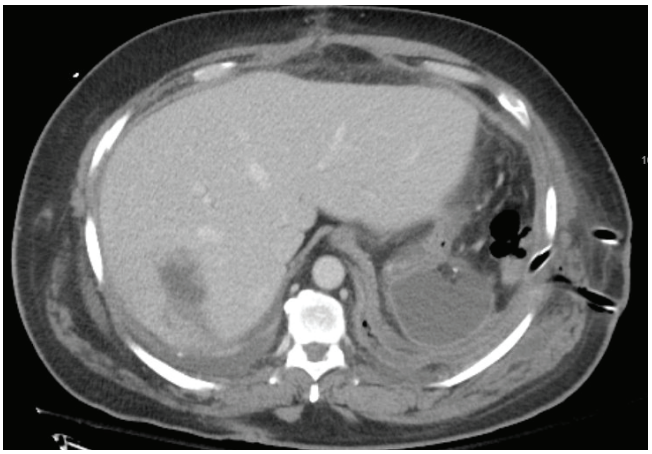
UTI typically produces fever starting on postoperative day 3. Work up with a urinalysis and urinary cultures and treat with appropriate antibiotics. The most common cause is instrumentation (catheterization).

Deep vein thrombosis can result in fever starting around postoperative day 5. Diagnosis requires a high index of suspicion. Physical exam is not very sensitive; U/S is diagnostic. Treatment is systemic anticoagulation, initially with heparin or unfractionated low molecular weight heparin and then transitioned to a long-term anticoagulant. Prophylaxis is mandatory in all surgical patients with early ambulation, compression devices, and/or chemical prophylaxis with low-dose heparin.

Wound infection typically begins to produce fever around postoperative day 7. Physical exam will reveal erythema, warmth, tenderness, and fluctuance. The 7-day delay is because it takes that long from **colonization to infection** (a numbers game).

- If only cellulitis is present, treat with antibiotics.
- If an abscess is present or suspected (most important physical finding is **fluctuance**), the wound must be opened and drained.
- If the case is unclear, use U/S or CT to diagnose.

A **deep abscess** (e.g., intraperitoneal: subphrenic, pelvic, or subhepatic) will start to produce fever around postoperative days 10–15. CT of the appropriate body cavity is diagnostic. Percutaneous image-guided drainage is therapeutic.



Courtesy of Gary Schwartz, MD

Figure 1-3. CT Splenic Bed Abscess

Shortly after the onset of a general anesthetic with inhaled halothane and muscle relaxation with succinylcholine, a patient develops a rapid rise in body temperature, exceeding 40 C (104 F). Metabolic acidosis and hypercalcemia are also noted. A family member died under general anesthesia several years earlier.

This is a classic case of malignant hyperthermia. The history should have been a warning, but once the problem develops, discontinue the anesthetic gas, treat with IV dantrolene, and take the essential support measures:

- 100% oxygen
- Correction of the acidosis
- Cooling blankets

Monitor for myoglobinuria and an acute kidney injury.

Forty-five minutes after completion of a cystoscopy, a patient develops chills and a fever spike to 40 C (104 F).

This is early on after an invasive procedure, and a fever this high means bacteremia. Take blood cultures and start broad-spectrum, empiric IV antibiotic therapy.

On postoperative day 1 after a right hemicolectomy, a patient develops a fever of 38.9 C (102 F).

Fever on day 1 is most commonly due to atelectasis, but all the other potential sources have to be ruled out. Examine the wound and IV sites and take a chest x-ray. Inquire about urinary tract symptoms. Improve the patient's ventilation: deep breathing and coughing, postural drainage, and incentive spirometry. This is all referred to as "pulmonary toilet."

On postoperative day 1 after an abdominal procedure, a patient develops a fever of 38.9 C (102 F). The patient is not compliant with treatment for atelectasis and by postoperative day 3 still has daily fever in the same range.

Bacterial pneumonia has mostly likely developed in the atelectatic lung. Chest x-ray, sputum cultures, and appropriate antibiotics are needed.

A patient who had a right colectomy for colon cancer is afebrile during the first 2 postoperative days, but on day 3 she has a fever spike to 39.4 C (103 F).

A patient who had a right colectomy for colon cancer is afebrile during the first 4 postoperative days, but on day 5 he has a fever spike to 39.4 C (103 F).

A patient who had a right colectomy for colon cancer is afebrile during the first 6 postoperative days, but on day 7 she has a fever spike to 39.4 C (103 F).

Every potential source of post-op fever always has to be investigated, but the timing of the first febrile episode gives a clue as to the most likely source. Remember the "4 Ws": UTI, thromboembolism (now less common because of mandatory prophylaxis), and wound infection are the likely culprits in these vignettes. Urinalysis and urinary culture, lower extremity venous U/S, and physical examination are the respective tests.

A patient who had major abdominal surgery has a normal postoperative course, with no significant episodes of fever until day 10, when his temperature begins to spike up to 39.4 C (103 F) daily.

At this postoperative stage, a deep abscess is the most likely source. CT is diagnostic, and treatment typically is percutaneous drainage.

Chest Pain

Perioperative myocardial infarction (MI) may occur during the operation (most commonly triggered by hypotension), in which case it is detected by the EKG monitor (ST elevation or depression, T-wave flattening). When it happens postoperatively, MI typically presents with chest pain in the first 2–3 days. The most reliable diagnostic test is serum troponin I level. Mortality is 50–90%, greatly exceeding that of MI not associated with surgery. Treatment is directed at the complications. Emergency coronary angiography with percutaneous intervention (angioplasty, stenting) may be lifesaving.

On postoperative day 2 after an abdominoperineal resection for rectal cancer, a 72-year-old man complains of severe retrosternal pain radiating to the left arm. He is short of breath and tachycardic.

During an abdominoperineal resection for rectal cancer, the patient unexpectedly has severe bleeding and is hypotensive on and off for almost 1 hour. The anesthesiologist notes ST depression and T-wave flattening in the EKG monitor.

Perioperative MI happens intraoperatively or within the first 3 days, and the biggest triggering cause is hypovolemic shock and hypotension. The 2 vignettes presented here are typical scenarios, although in practice the classic chest pain picture is often obscured by other ongoing events. Check a 12-lead EKG and serum troponin levels, and contact cardiology.

Pulmonary Embolism

Pulmonary embolism (PE) typically occurs around postoperative day 7 but can occur at any time postoperatively. Elderly patients and those with cancer are at increased risk; postoperative immobilization alone increases the risk. Typical presentation is sudden-onset pleuritic pain accompanied by shortness of breath.

Look for a patient who is anxious, diaphoretic, and tachycardic, with prominent distended veins in the neck and forehead. (Note that a low central venous pressure [CVP] virtually excludes the diagnosis.) Arterial blood gases demonstrate hypoxemia and often hypocapnia due to tachypnea.

CT angiogram (the gold standard) is used for diagnosis. This diagnostic test is a spiral CT with a large IV contrast bolus timed to pulmonary artery filling. **This diagnostic test is not to be delayed.**

Treatment is systemic anticoagulation, initially with heparin, and should be started immediately following diagnosis.

- In decompensating patients with a high index of suspicion, consider starting treatment even before confirming the diagnosis.
- If a PE recurs while the patient is anticoagulated or if anticoagulation is contraindicated, place an inferior vena cava filter to prevent further embolization from lower extremity deep venous thromboses.

Prevention of thromboembolism will also prevent PE.

- Use a sequential compression device on anyone who does not have a lower extremity fracture or significant lower extremity arterial insufficiency.
- In moderate- or high-risk patients, prophylactic anticoagulation is indicated with low-dose heparin (typically 5,000 units subcutaneously/8–12 hours until mobile) or enoxaparin (30–40 mg/24 hours, based on renal function). This is referred to as **chemoprophylaxis**.
- Risk factors for DVT include age >40, pelvic/leg fracture, venous injury, femoral venous catheter, presence of cancer, and anticipated prolonged immobilization.

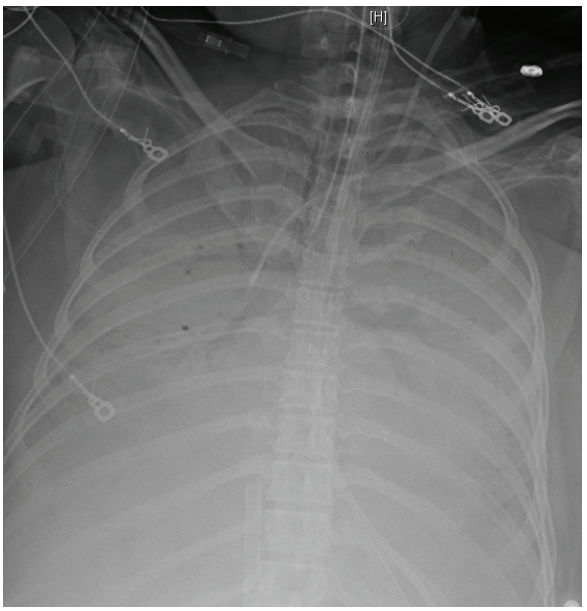
On postoperative day 7 after a broken hip is pinned, a 76-year-old man suddenly develops severe pleuritic chest pain and shortness of breath. When examined, he is found to be anxious, diaphoretic, and tachycardic. He has prominent distended veins in his neck and forehead.

Chest pain this late post-op is most likely due to a pulmonary embolus (PE). This patient is obviously at high risk, and the findings are classic. Arterial blood gas or pulse oximetry is the first test, and hypoxemia and hypocapnia are the expected findings; in their absence, it is not a PE. CT angiogram is the immediate gold standard diagnostic test of choice. Therapy starts with systemic heparinization. Fibrinolysis with tissue plasminogen activator (tPA), either systemic or catheter-directed, is indicated for extreme cases with hemodynamic compromise, as well as consideration of surgical embolectomy. If a PE recurs despite anticoagulation, an inferior vena cava filter is indicated.

Other Pulmonary Complications

Aspiration is a distinct hazard when intubating patients with a full stomach. It can be lethal right away, but more commonly causes a chemical injury of the tracheobronchial tree—pneumonitis—that can progress to pneumonia and respiratory failure. Prevention includes strict restriction of oral intake prior to surgery and antacids before induction. Therapy starts with bronchoscopic lavage and removal of acid and particulate matter, followed by bronchodilators and respiratory support. Steroids have not been demonstrated to improve outcomes and therefore are not usually indicated. Antibiotics are indicated only where there is evidence of the resultant pneumonia—e.g., leukocytosis, sputum production and culture, and focal consolidation on chest x-ray. This typically does not present for several days following the insult.

Adult respiratory distress syndrome (ARDS) is seen in patients with a complicated post-op course, often with sepsis as the precipitating event. These patients demonstrate bilateral pulmonary infiltrates and hypoxia with no evidence of CHF. The centerpiece of therapy is positive end-expiratory pressure (PEEP) with low volume ventilation. (Excessive ventilatory volumes have been demonstrated to result in barotrauma.) A source of sepsis must be sought and corrected.



Courtesy of Gary Schwartz, MD

Figure 1-4. ARDS

Intraoperative tension pneumothorax can develop in patients with traumatized or emphysematous lungs who are subjected to positive-pressure breathing. They become progressively more difficult to ventilate, with rising airway pressure, steadily declining BP, and steadily rising CVP.

- If the abdomen is open, quick decompression can be achieved through the diaphragm (but the risk is contamination of the pleural cavity).
- Alternatively (and better), needle decompression in the midclavicular line followed by formal chest tube is indicated.

> An awake intubation is being attempted in a drunk and combative man who has sustained a gunshot wound to the abdomen. In the struggle, the patient vomits and aspirates a large amount of gastric contents with particulate matter.

This is every anesthesiologist's nightmare. Aspiration results in a chemical injury to the tracheobronchial tree ("aspiration pneumonitis"). This is an inflammatory problem, not an infectious process, so antibiotics are not immediately indicated. However, the irritation results in pulmonary failure and increases the risk of secondary pneumonia. Prevention is best: empty stomach, antacids before induction, rapid sequence induction with manual cricoid pressure. Once aspiration happens, however, bronchoscopic lavage and removal of particulate matter are the first steps, followed by bronchodilators and respiratory support. Steroids are usually not helpful.

> In week 2 of a complicated postoperative period, a young patient with multiple gunshot wounds to the abdomen becomes progressively disoriented and unresponsive. The patient has bilateral pulmonary infiltrates and PaO_2 of 65 mm Hg while breathing 40% oxygen.

The reason for the mental changes is obvious: the patient is not getting enough oxygen in the blood. The rest of the findings, however, specifically identify ARDS. The centerpiece of therapy for ARDS is mechanical ventilation with high PEEP and low tidal volumes. Also consider why this has developed now: in an older patient with preexisting lung disease, an acute illness can exacerbate the problem; in a patient with normal lungs, chest trauma and sepsis are the most common etiologies.

> A trauma patient is undergoing a laparotomy for a seatbelt injury. She also sustained several broken ribs. Halfway through the case it becomes progressively difficult to ventilate the patient, and oxygen saturation and blood pressure steadily decline. There is no evidence of intra-abdominal bleeding.

This patient has an intraoperative tension pneumothorax. Likely, while the patient was receiving positive-pressure ventilation, one of the broken ribs punctured the lung. The best approach is immediate transdiaphragmatic decompression, or better, transthoracic needle decompression followed by formal chest tube placement.

Disorientation/Coma

When a postoperative patient becomes confused and disoriented, **hypoxia** is the first concern and **sepsis** is a close second. If airway protection is threatened, check an arterial blood gas and provide respiratory support.

Delirium tremens (DTs) is very common in the alcoholic whose drinking is suddenly interrupted by hospital admission. During postoperative day 2 or 3, the patient gets confused, has hallucinations, and becomes combative. IV benzodiazepines are the standard therapy, but oral alcohol is sometimes available at hospitals for this indication (not frequently given in today's environment). DTs must be recognized and treated: it can be fatal!

Electrolyte imbalances, particularly alterations in sodium concentration, can have a profound effect on a patient's mental status. Both hypernatremia and hyponatremia can produce confusion, seizures, lethargy, and coma.

Ammonium intoxication is a common source of coma in the cirrhotic patient. In patients with cirrhosis, inability to detoxify absorbed protein from GI bleeding can produce "hepatic coma"; this effect may also be seen after implementation of a portosystemic shunt (e.g., TIPS procedure).

> Eighteen hours after abdominal aortic aneurysm repair, a patient becomes disoriented.

This is a very brief vignette, but of the long list of things that can produce post-op disorientation, the most lethal one if not promptly recognized and treated is hypoxia. Physical examination and vital signs will likely indicate hypoxemia; obtain an arterial blood gas if unsure or to quantify. Alternative etiologies are mostly metabolic: uremia, hyponatremia, hypernatremia, ammonium, hyperglycemia, DTs, or medications.

> A recovered alcoholic undergoes an elective colon resection for recurrent diverticular bleeding. The patient reports that he has not touched a drop of alcohol for the past 6 months. On postoperative day 3 he becomes disoriented and combative, and claims to see elephants crawling up the walls. The spouse then reveals that the patient actually drank heavily up until the day of hospital admission.

This case clearly describes DTs. The standard management relies on benzodiazepines. Some hospitals allow oral intake of alcohol, but that is less common these days.

> Twelve hours after completion of an abdominal hysterectomy, a 42-year-old woman becomes confused and lethargic, complains of severe headache, has a grand mal seizure, and finally goes into a coma. Review of the chart reveals that an order for D5W, to run in at 125 mL/h, was mistakenly implemented as 525 mL/h.

This is a classic example of water intoxication. A very low serum sodium concentration will confirm it. Mortality for this iatrogenic condition is very high, and therapy is quite controversial. Very careful use of hypertonic saline (3%) is a reasonable answer in this extreme scenario. Indications are generally coma or seizures.

> Eight hours after completion of a transsphenoidal hypophysectomy for prolactinoma, a young woman becomes lethargic, confused, and eventually comatose. Review of the record shows that her urinary output since surgery has averaged 600 mL/h, although her IV fluids are going in at 100 mL/h.

This case illustrates the reverse of the previous vignette: large, rapid, unreplaced water loss from surgically induced diabetes insipidus. The labs will show significant hypernatremia. The safest therapy is an infusion of 1/3 or 1/4 normal saline to replace the lost fluid; in this acute setting, D5W would be acceptable.

> A cirrhotic patient goes into coma after receiving an emergency portocaval shunt for bleeding esophageal varices.

This clinical case is brief but unmistakable: the culprit here will be ammonia. If the case also involves hypokalemic alkalosis and high cardiac output combined with low peripheral resistance, overt liver failure has occurred.

Urinary Complications

Postoperative urinary retention is extremely common, particularly after surgery in the lower abdomen, pelvis, perineum, or groin. The patient feels the need to void but cannot. Bladder scanning and catheterization should be performed 6–8 hours postoperatively if no spontaneous voiding has occurred. Indwelling Foley catheter placement is indicated at the second consecutive catheterization.

Zero urinary output typically is caused by a mechanical problem (not a biologic one), as even patients with renal failure will have some output. Look for a plugged or kinked catheter, and flush the tubing to dislodge any clot that may have formed. You need to know this, but likely the nurse will irrigate and replace the catheter if blocked without your definitive order.

Low urinary output (<0.5 mL/kg/hr) in the presence of **normal BP** (i.e., not because of shock) represents either fluid deficit or an acute kidney injury. Always check the BP, as hypotension will cause this (renal blood flow follows cardiac output). The treatment is fluids, not diuretics.

- A low-tech diagnostic test is a fluid challenge: a bolus of 500 mL of IV fluids infused over 10–20 minutes. Patients who are dehydrated will respond with a temporary increase in urinary output; those in renal failure will not.
- A more scientific test is to measure urinary sodium: it will be <10 or 20 mEq/L in the dehydrated patient with normally functioning kidneys; it will exceed 40 mEq/L in cases of renal failure.
- An even more scientific test is to calculate the fractional excretion of sodium, or FeNa. This involves measuring plasma and urinary sodium and creatinine. In acute kidney injury, the ratio >2; in hypovolemia it is <1.

> Six hours after undergoing a hemorrhoidectomy under spinal anesthesia, a 62-year-old man complains of suprapubic discomfort and fullness. He feels the need to void but has been unable to do so since the operation. There is a palpable suprapubic mass that is dull to percussion. Bladder scanning reveals a significant volume of urine.

By far the most common urinary problem post-op is the inability to void, and men are more commonly affected. Treatment is in-and-out bladder catheterization. If bladder catheterization has to be repeated again after another 6–8 hours, a Foley catheter should be left in place for 24–48 hours before removal is attempted.

> A man has had an abdominoperineal resection for cancer of the rectum, and an indwelling Foley catheter was left in place after surgery. The nurses are concerned because even though the patient's vital signs have been stable, urinary output in the last 2 hours has been zero.

In the presence of renal perfusing pressure, urinary output of zero invariably means a mechanical problem: the catheter is plugged or kinked. More ominous possibilities include injury of both ureters or thrombosis of the renal vessels, but these causes are much more rare.

> Several hours after completion of multiple surgery for blunt trauma in an average-sized adult, the patient's urinary output in 3 consecutive hours is reported as 12 mL/h, 17 mL/h, and 9 mL/h. Blood pressure has hovered around 95–130 mm Hg systolic during that time.

The patient's kidneys are perfusing, but she is either behind in fluid replacement or has gone into renal failure. A fluid challenge would suggest which situation exists: a bolus of 500 mL given over 10–20 minutes should produce diuresis in the dehydrated patient but will not do so in renal failure.

The more precise technique—and the preferred exam answer—is to measure urinary sodium (<10–20 mEq/L in dehydration, >40 mEq/L in renal failure). An even more elegant calculation is measurement of FeNa (<2 in renal failure).

Abdominal Distention

Paralytic ileus is to be expected in the first few days after abdominal surgery. Presentation includes:

- Bowel sounds: absent or hypoactive
- No passage of gas
- Mild distention (some cases)
- **No pain**

The condition is prolonged by electrolyte abnormalities, especially hypokalemia and hypomagnesemia. Be patient, as it will usually resolve with time.

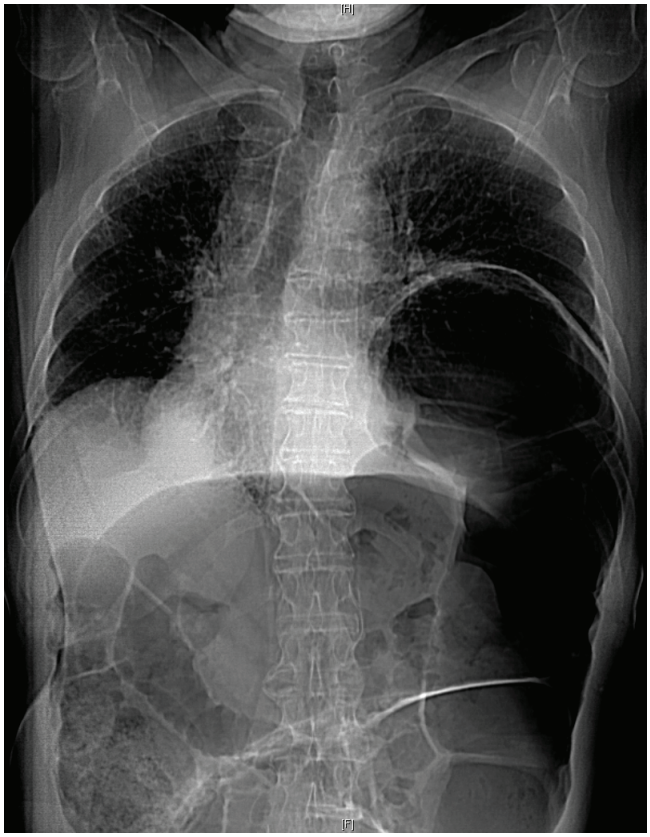
Early mechanical bowel obstruction can happen during the postoperative period because of adhesions. Apparent paralytic ileus that fails to resolve after 5–7 days is most likely an early mechanical bowel obstruction. X-ray will show dilated loops of small bowel and air-fluid levels.

Diagnosis is confirmed with an abdominal CT that demonstrates a transition point between proximal dilated bowel and distal collapsed bowel at the site of the obstruction. Surgical intervention is needed to correct the problem. Remember that with bowel obstruction, **there is pain**.

Ogilvie syndrome (or pseudo-obstruction) is a poorly understood but very common condition that could be described as a paralytic ileus of the colon.

- A functional (physiologic) obstruction, not mechanical obstruction
- Classically seen in elderly, sedentary patients (Alzheimer's, nursing home residents) who have become further immobilized owing to either surgery (broken hip, prostatic surgery) or anticholinergic or psychotropic medications
- Symptoms include abdominal distention without tenderness
- X-ray shows a massively dilated colon
- Treatment
  - Fluid and electrolyte correction first
  - Then, mechanical obstruction must be ruled out radiologically
  - Endoscopy (diagnostic and therapeutic) may include leaving a rectal tube in place
  - IV neostigmine to restore colonic motility, but given the risk of significant bradycardia, can be done only during continuous telemetric monitoring

If this syndrome is not treated, the cecum is the usual point of rupture (law of Laplace).



Courtesy of Gary Schwartz, MD

Figure 1-5. X-Ray Dilated Colon

> Four days after exploratory laparotomy for blunt abdominal trauma with resection and anastomosis of damaged small bowel, a patient has abdominal distention without abdominal pain. She has no bowel sounds and has not passed flatus, and abdominal x-ray shows dilated loops of small bowel without air-fluid levels.

This case is likely a postoperative paralytic ileus, which can be expected under the circumstances. NPO and NG suction should be continued until peristaltic activity resumes. If it does not, CT of the abdomen should be taken to rule out a mechanical obstruction, visible as a transition point between the proximal dilated bowel and the distal collapsed bowel at the site of obstruction. Hypokalemia should also be ruled out. A technical error at the anastomosis site should always be considered. Be patient!

> An 80-year-old man with Alzheimer's disease living in a nursing home undergoes surgery to repair a fractured femoral neck. On postoperative day 5 the patient's abdomen is noted to be grossly distended and tense, but nontender. He has occasional bowel sounds. X-ray shows a very distended colon and a few distended loops of small bowel.

In the elderly, who are not very active to begin with and are now further immobilized, massive colonic dilatation (Ogilvie syndrome) is commonly seen. Correct the fluids and electrolytes first. Neostigmine can dramatically improve colon motility at the cost of **very significant side effects**. Colonoscopy is the most successful treatment should intervention become indicated.

Wound Infections

Wound infections are typically seen around postoperative day 7. Manage with IV antibiotics and, potentially, by reopening the wound partially or completely to allow for drainage.

Wound dehiscence is typically seen around postoperative day 5 after open laparotomy. The wound may look intact, but a **large volume** of pink ("salmon-colored") fluid is noted to be soaking the dressing; this is peritoneal fluid draining through a dehiscence in the fascial closure. Reoperation is indicated to avoid evisceration and peritonitis. In high-risk patients, nonsurgical management with negative pressure wound therapy may suffice.

Evisceration is a catastrophic complication of wound dehiscence where the fascia closure opens and the abdominal contents herniate. It typically happens when the patient (who may not have been recognized as having a dehiscence) coughs, strains, or gets out of bed. The patient must be kept in bed and the bowel covered with large sterile dressings soaked with warm saline. Emergency abdominal closure is mandatory.

Enterocutaneous fistula is a devastating complication that develops between the gastrointestinal tract and the skin, typically through a surgical wound or drain site.

- If the enterocutaneous fistula does not empty completely to the outside ("uncontrolled") but instead leaks into a body cavity, it may precipitate an abscess and lead to sepsis; treatment is complete drainage.
- If it drains freely ("controlled"), sepsis does not usually develop, but fluid and electrolyte loss, nutritional depletion, and erosion of the abdominal wall are potential problems.

Complications associated with GI fistulas depend on the location and volume of the fistula:

- Less problematic in the distal colon
- Present but manageable in low-volume fistula (up to 200–300 mL/day)
- Daunting in high-volume fistulas (several liters per day) arising from upper GI tract

Fluid and electrolyte replacement, nutritional support, and protection of the skin of the abdominal wall are done to keep the patient alive until nature heals the fistula.

Fistulas are a nightmare to both the patients and surgeons, as the healing of even a controlled fistula can take weeks or months.

> On postoperative day 5 after a laparotomy, it is noted that large amounts of salmon-colored clear fluid are soaking the patient's dressings.

This is the classic presentation of a wound dehiscence. Surgical exploration is indicated, with reclosure of the fascia. In a very high-risk patient, consider nonsurgical management with negative pressure wound therapy.

> Nurses report that on postoperative day 5 after a laparotomy, a patient is draining clear pink fluid from his abdominal wound. A medical student removes the dressing and asks the patient to sit up so he can be helped out of bed to the treatment room. When he complies, the wound opens wide and small bowel rushes out.

This variant describes evisceration, a serious problem. Put the patient back in bed, cover the bowel with large, moist dressings soaked in warm saline (moist and warm are the key), and then get him to the OR for reclosure.

> A patient presents to the surgeon's office postoperative day 7 after an open appendectomy. The incision is noted to be red, hot, tender, and fluctuant. She reports fever for the past 2 days.

If there were just a bit of redness, or symptoms occurred earlier in the postoperative course, this could be a case of superficial cellulitis and managed with antibiotics alone. However, this far post-op and with the physical examination findings, this scenario describes a postoperative wound infection. There is likely to be pus; the wound must be opened to allow for drainage, and antibiotics must be administered. If there is doubt as to the presence or absence of a drainable collection, U/S is diagnostic.

> Nine days after a patient undergoes sigmoid resection for cancer, the wound drains a brown fluid that is clearly feculent. The patient is afebrile and otherwise doing quite well.

This scenario describes a fecal fistula. If draining to the outside, it is unruly and inconvenient but not life-threatening. The fistula will close eventually with little or no therapy if there are no limiting factors (FRIENDS mnemonic). If feces were accumulating on the inside ("uncon-

Note

Natural wound healing will take place unless the following **FRIENDS** (mnemonic) are present:

- **F**oreign body
- **R**adiation injury
- **I**nfection or **I**BD
- **E**pithelialization
- **N**eoplasm
- **D**istal obstruction
- **S**teroid use

trolled"), the patient would be febrile and sick and would need drainage and probably a diverting colostomy.

> Eight days after a difficult hemigastrectomy and gastroduodenostomy for gastric ulcer, a patient begins to leak 2–3 L of green fluid per day through the right corner of her bilateral subcostal abdominal wound.

A patient who is febrile and sick, with an acute abdomen, needs to be explored for what is likely an uncontrolled fistula. However, if all the gastric and duodenal contents are leaking to the outside ("controlled"), further immediate surgery can often be avoided:

- Provide fluid/electrolyte replacement
- Deliver elemental nutritional support into the upper jejunum
- Provide local wound care to prevent skin breakdown
- Consider somatostatin or octreotide to diminish the volume of GI fluid loss

Total parenteral nutrition (TPN) is second choice, but it is less effective and carries greater potential risk of bloodstream infections.

Fluid and Electrolyte Imbalance

Hypernatremia means that the patient has lost water (or other hypotonic fluids) and become dehydrated. The condition typically presents as alterations in neurologic function; the extent of brain dysfunction depends on the magnitude and time frame over which the hypernatremia developed. Every 3 mEq/L that serum sodium concentration exceeds 140 represents approximately 1 L of water lost.

- If the problem develops slowly (i.e., over several days), the brain will adapt, and the only clinical manifestations will be those of volume depletion.
- If the problem happens rapidly (e.g., osmotic diuresis, diabetes insipidus), the brain will not be able to adapt, and thus more profound CNS symptoms will develop.

Treatment is volume repletion to correct overall volume rapidly (hours), while tonicity is corrected slowly (days). This is achieved by using 5% dextrose in half-normal saline rather than D5W.

Hyponatremia means the patient has retained a net excess of water and hypotonicity has developed. There are 2 potential scenarios:

- A patient who is losing large amounts of isotonic fluids (typically from the GI tract) is forced to retain water if she has not received appropriate replacement with isotonic fluids. Volume restoration with isotonic fluids (NS or lactated Ringer's [LR]) will correct the hypovolemia and allow the body to unload the retained water slowly and safely and return the tonicity to normal.
- A patient who starts with normal fluid volume adds to it by retaining water in the presence of inappropriate amounts of antidiuretic hormone (ADH) (e.g., post-op water intoxication or inappropriate ADH secreted by tumors). Rapidly developing hyponatremia (water intoxication) produces CNS symptoms because the brain has not had time to adapt; it requires careful use of hypertonic saline (3 or 5%). In hyponatremia that develops slowly in response to inappropriate ADH, the brain has time to adapt; therapy should be water restriction.

Hypokalemia develops slowly, over days, when potassium is lost from the GI tract (all GI fluids have lots of potassium) or in the urine (because of loop diuretics or excess aldosterone) and is not replaced. Hypokalemia develops rapidly, over hours, when potassium moves into the cells—for example, when diabetic ketoacidosis is corrected. Treatment is IV potassium replacement at a rate not faster than 10 mEq/hr.

Hyperkalemia will develop slowly if the kidney cannot excrete potassium (renal failure, aldosterone antagonists), or rapidly if potassium is being released from cells into the blood (crushing injuries, dead tissue, acidosis).

Treatment must take into account whether the kidneys are functioning. Emergent management includes stabilizing cellular membranes with IV calcium and "pushing potassium into the cells" through the use of IV glucose and insulin. Loop diuretics excrete potassium in the urine (if the kidneys are working), and sodium polystyrene sulfonate (oral or rectal) may absorb potassium via the GI tract. Dialysis may be needed in the event of renal failure.

Metabolic acidosis can result from any of the following:

- Excessive production of fixed acids (diabetic ketoacidosis, lactic acidosis, low-flow states)
- Inability of the kidney to eliminate fixed acids (renal failure)
- Loss of buffers (loss of bicarbonate-rich fluids from GI tract)

In all cases, blood pH is low (<7.4), serum bicarbonate is low (<22), and there is a base deficit. When abnormal acids are piling up in the blood, there is also an "anion gap" in which serum sodium exceeds the sum of chloride and bicarbonate by >10 or 15. The anion gap does not exist when the problem is loss of buffers. This can be fatal, as metabolic acidosis increases ectopy and decreases inotropy.

Treatment of metabolic acidosis is aimed at treating the underlying cause. Bicarbonate therapy will correct the pH temporarily but can risk producing a "rebound alkalosis." For chronic acidosis, renal loss of K^+ will cause a deficit that does not become obvious until the acidosis is corrected. Be prepared to replace K^+ as part of the treatment.

Metabolic alkalosis classically occurs in scenarios involving loss of acidic gastric fluid, e.g., prolonged emesis or NG suction. It can also develop if excess bicarbonate is administered. Symptoms include low K+, low Cl−, and high bicarbonate (hypokalemic, hypochloremic metabolic alkalosis).

Treatment of metabolic alkalosis is chloride and potassium replacement, thereby allowing the kidneys to correct the problem.

Respiratory acidosis and alkalosis result from impaired ventilation (acidosis) or abnormal hyperventilation (alkalosis). Symptoms include abnormal PCO_2 (low in alkalosis, high in acidosis) and abnormal blood pH. Treatment is correction of the underlying respiratory problem.

Note that metabolic acid-base derangements may be accompanied by respiratory compensatory changes. For example, acute metabolic acidosis will result in tachypnea with lowering of pCO_2 to mitigate the decrease in pH arising from the primary derangement (in this case, metabolic acid).

Remember that metabolic acidosis has the same effects on the heart that general anesthetics have: **decreased inotropy** and **increased ectopy**.

> Eight hours after completion of a transsphenoidal hypophysectomy for a prolactinoma, a young woman becomes lethargic, confused, and eventually comatose. Review of the record shows that her urinary output since surgery has averaged 600 mL/h, while IV fluids are infusing at 100 mL/h. Serum sodium determination shows concentration 152 mEq/L.

Elevated concentration of serum sodium invariably means that the patient has lost pure water (or hypotonic fluids). Every 3 mEq/L above the normal of 140 represents 1 L lost. This woman is 4 L shy, which fits her history of a diuresis of 500 mL/h more than her intake. She should be given 4L of D5W or possibly D5-1/3NS.

> Several friends go on a weekend camping trip in the desert. On day 2 they get lost and aren't rescued until one week later. One patient is brought to your hospital—awake and alert—with obvious clinical signs of dehydration. Serum sodium concentration is 155 mEq/L.

The patient has also lost water, about 5 L, but has done so slowly by pulmonary and cutaneous evaporation over 5 days. He is hypernatremic, but his brain has adapted to the slowly changing situation. Were he to be given 5 L of D5W, the rapid correction of his hypertonicity would be dangerous. Five liters of D5-1/2NS is a much safer plan.

> Twelve hours after undergoing an abdominal hysterectomy, a 42-year-old woman becomes confused and lethargic, complains of severe headache, has a grand mal seizure, and finally goes into coma. Review of the chart reveals that an order for D5W to run in at 125 mL/h was mistakenly implemented as 525 mL/h. Her serum sodium concentration is 122 mEq/L.

In the surgical patient with normal kidneys, hyponatremia invariably means that water (without sodium) has been retained, so the body fluids have been diluted. In this case a lot of IV water was given, and the ADH produced as part of the metabolic response to trauma has held onto it. Rapidly developing hyponatremia (water intoxication) is a big problem, as the brain has no time to adapt; once it has occurred, therapy is controversial. For the sake of the exam, replete with hypertonic saline (3 or 5%) given 100 mL at a time and reassess (clinically and with bloodwork) before each subsequent dose.

> A 62-year-old woman comes in for her scheduled chemotherapy administration for metastatic cancer of the breast. Although she is quite asymptomatic, the lab reports serum sodium concentration of 122 mEq/L.

In this setting, water has also been retained (by ADH produced by the tumor), but so slowly that the brain has kept up with the developing hypotonicity. Rapid correction would be ill-advised at best and lethal at worst. Water restriction, on the other hand, will slowly allow the abnormality to reverse itself.

A 68-year-old woman comes in with an obvious incarcerated umbilical hernia. She has gross abdominal distention, is clinically dehydrated, and reports persistent fecaloid vomiting for the past 5 days. She is awake and alert. Serum sodium concentration is 118 mEq/L.

Hyponatremia means water retention, but in this case the problem began with loss of isotonic (sodium-containing) fluid from her gut. As the patient's extracellular fluid became depleted, her body retained whatever water it could: exogenous from oral intake and endogenous from catabolism. Consequently, she is now simultaneously volume-depleted and hyponatremic (hypotonic).

This patient desperately needs volume replacement, but it must not be corrected too quickly. Administer isotonic fluids in quantity: Start with 1 or 2 L/h of normal saline or Ringer's lactate, depending on the acid-base status (use clinical variables to fine tune the rate). Once fluid volume is replenished, the patient's body will unload the retained water and correct its own tonicity.

A patient with severe diabetic ketoacidosis comes in with profound dehydration and a serum potassium concentration of 5.2 mEq/L. After several hours of vigorous therapy with insulin and IV fluids (saline, without potassium), the patient's serum potassium concentration is 2.9 mEq/L.

Severe acidosis precipitates a loss of potassium in the urine. While the acidosis is present, however, the serum concentration is high because potassium ions have come out of the cells in exchange for hydrogen ions. Once the acidosis is corrected, that potassium rushes back into the cells, and the true magnitude of the potassium loss becomes evident.

The patient obviously needs potassium. Under most circumstances, 10 mEq/h is a safe limit for a peripheral IV line. In extreme settings, 20 mEq/h can be justified, but central venous catheter placement is indicated.

An 18-year-old woman slips and falls under a bus, and her right leg is crushed. On arrival at the ED she is hypotensive, and she receives several units of blood. Over the next several hours the patient is in and out of hypovolemic shock and develops acidosis. Serum potassium concentration, which was 4.8 mEq/L at the time of admission, is reported at 6.1 a few hours later.

The elevated serum potassium could have multiple etiologies: rhabdomyolysis from the crushed leg, hemolysis from multiple blood transfusions, and/or transcellular migration from acidosis. With low perfusing pressure (in and out of shock), the kidneys have failed to eliminate it.

This patient needs multiple treatment strategies: BP improvement to allow for urinary clearance, intracellular transport using D50 and insulin, GI elimination with exchange resins, and an NG tube. If those are not successful, urgent hemodialysis is indicated. During the stabilization period, IV calcium should be administered to stabilize the cellular membrane.

Clinical Pearl

The definitive compensatory mechanism in acid-base balance is the kidney: "**urine follows serum**," i.e., if acidotic, the kidney will excrete acid (retain bicarb), and vice versa.

An elderly man with alcoholism, diabetes, and marginal renal function sustains multiple traumas while driving under the influence of alcohol. In the course of his resuscitation and multiple surgeries, he is in and out of shock for prolonged periods of time. Blood gases show pH 7.1 and PCO_2 32 mm Hg. Serum electrolytes are sodium 138 mEq/L, chloride 98 mEq/L, and bicarbonate 15 mEq/L.

This man has every risk factor for developing metabolic acidosis through retention of fixed acids (rather than by loss of bicarbonate). The driving force in this case is the state of shock, with lactic acid production; the diabetes, alcohol, and bad kidney are also contributing.

Labs confirm metabolic acidosis here (low pH and low bicarbonate). His body is trying to compensate by hyperventilating (low PCO_2), and he exhibits the classic anion gap: the sum of chloride and bicarbonate is 25 mEq/L less than the serum sodium concentration, instead of the normal 10–15 mEq/L.

The classic treatment for metabolic acidosis is bicarbonate or a bicarbonate precursor such as lactate or acetate. However, in a case like this, this tends to result in alkalosis once the low-flow state is corrected. Thus the emphasis here should be on fluid resuscitation with Ringer's lactate. Avoid large volumes of saline, which would deliver too much chloride.

A patient who has undergone a subtotal gastrectomy for cancer with a Billroth II reconstruction develops a blowout of the duodenal stump and, subsequently, a duodenal fistula. For the past 10 days, 750–1,500 mL/day of green fluid has been draining from the incision. Serum electrolytes are sodium 132 mEq/L, chloride 104 mEq/L, and bicarbonate 15 mEq/L. Blood pH is 7.2 and PCO_2 is 35 mm Hg.

This is another case of metabolic acidosis, but with a normal anion gap. The patient has been losing lots of bicarbonate out of the fistula. The problem would not have developed if his IV fluid replacement had contained lots of bicarbonate (or lactate, or acetate). The use of those agents is now indicated.

Clinical Pearl

Pyloric stenosis in children presents with the same electrolyte abnormality as in adults. Treatment for both populations is surgical (pyloromyotomy). In preparation for surgery, children should be resuscitated with only 1/2 NS to avoid hypernatremia, which can be a consequence.

A patient with severe peptic ulcer disease develops pyloric obstruction and has protracted vomiting of clear gastric contents (i.e., without bile) for several days. Serum electrolytes show sodium 134 mEq/L, chloride 82 mEq/L, potassium 2.9 mEq/L, and bicarbonate 34 mEq/L.

This is a classic case of hypochloremic, hypokalemic, metabolic alkalosis secondary to loss of acidic gastric juice. The patient needs to be rehydrated (choose saline rather than Ringer's lactate) and infused with a lot of potassium chloride ($\geq$10 mEq/hr).

Trauma 2

Learning Objectives

- ❑ Describe the algorithm for evaluating a trauma patient
- ❑ Recognize management of burns, bites, and stings

The initial evaluation of a trauma patient requires a systematic approach to identify life-threatening injuries. This involves 2 parts:

- **Primary survey** to evaluate for all potential injuries; necessary interventions are performed during this time
- After the primary survey is complete and the patient is deemed to be stable, **secondary survey** to do a head-to-toe examination and evaluate all organ systems

PRIMARY SURVEY

The primary survey uses a systematic ABCDE approach to assess a rapidly deteriorating patient: **A**irway, **B**reathing, **C**irculation, **D**isability (neurological exam), and **E**xposure. This is also the **order of priority**.

Airway (A)

An airway should be secured before the situation becomes critical. The first step in the evaluation of trauma is airway assessment and protection.

- If the patient is conscious and speaking in a normal tone of voice, the airway is considered intact.
- If the patient has facial or neck trauma, an expanding hematoma or subcutaneous emphysema in the neck, noisy or "gurgly" breathing, or neurologic deficit with Glasgow coma score ≤8, the airway is considered unprotected.

In the field, an airway can be secured via laryngeal mask airway or orotracheal intubation.

In the ED, an airway can be established by orotracheal intubation or cricothyroidotomy. If the use of intubation is precluded or unsuccessful, surgical cricothyroidotomy may be needed.

Clinical Pearl

If an airway is secured via cricothyroidotomy, formal tracheostomy must be performed later to prevent airway stenosis.

Clinical Pearl

In patients age <8, tracheostomy is preferred over cricothyroidotomy, which can cause airway stenosis (the cricoid is much smaller in children than it is in adults).