POSTOPERATIVE CARE

Prompt assessment and treatment of postoperative complications is critical for the comprehensive care of surgical patients. The goal of the postoperative assessment is to ensure proper healing as well as rule out the presence of complications, which can affect the patient from head to toe, including the neurologic, cardiovascular, pulmonary, renal, gastrointestinal, hematologic, endocrine and infectious systems. Several of the most common complications after surgery are discussed below, including their risk factors, presentation, as well as a practical guide to evaluation and treatment. Of note, fluids and electrolyte shifts are normal after surgery, and their management is very important for healing and progression. Please see the module on Fluids and Electrolytes for further discussion.

Epidemiology/Pathophysiology

I. Wound Complications

Proper wound healing relies on sufficient oxygen delivery to the wound, lack of bacterial and necrotic contamination, and adequate nutritional status. Factors that can impair wound healing and lead to complications include bacterial infection (>106 CFUs/cm^2), necrotic tissue, foreign bodies, diabetes, smoking, malignancy, malnutrition, poor blood supply, global hypotension, hypothermia, immunosuppression (including steroids), emergency surgery, ascites, severe cardiopulmonary disease, and intraoperative contamination. Of note, when reapproximating tissue during surgery, whether one is closing skin or performing a bowel anastomosis, tension on the wound edges is an important factor that contributes to proper healing. If there is too much tension on the wound, there will be local ischemia within the microcirculation, which will compromise healing. Common wound complications include infection, dehiscence, and incisional hernia.

Wound infections, or surgical site infections (SSI), can occur in the surgical field from deep organ spaces to superficial skin and are due to bacterial contamination. Examples include cellulitis, superficial abscess, and deep abscess. Deep abscesses may form due to transient contamination of the peritoneal space when the GI tract is entered and contents spill out, such as when creating a bowel anastomosis or if bile spills during a cholecystectomy. Abscesses may also form if an anastomosis does not heal properly and eventually leaks GI contents in a delayed fashion. The delay occurs because the anastomosis is temporarily sealed with ischemic tissue that then breaks down in several days allowing contents to leak. Therefore, abscesses secondary to anastomotic leak usually present around postoperative day 5-7. Necrotizing soft tissues infections, which may progress rapidly, can occur in surgical wounds within hours of the operation. The more aggressive necrotizing infections are typically due to Clostridium perfringens or beta-hemolytic Group A Streptococcus, whereas, the slower onset infections are typically polymicrobial and occur over a 2-3 day period.

Most surgical wounds, despite appropriate sterile preparation of the skin, are contaminated by some bacteria, which usually consist of normal endogenous flora from the skin, respiratory, GU or GI tracts, depending on the type of surgery. Common skin flora include

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Gram positive cocci (Staphylococcus and Streptococcus), whereas GI tract flora include Gram positive microbes, Gram negative rods, and anaerobic species. However, unless there is heavy bacterial contamination, infection usually does not occur when host defenses are intact. The level of contamination of surgical wounds can be classified into four groups, shown in Table 1 below. These classifications are important predictors of the risk of postoperative surgical site infection.

Table 1

Classification	Definition	Examples	Risk of Infection
Clean	Clean wound, non-traumatic, no violation of mucosal barrier (GI, GU, respiratory tracts)	Skin, hernia repair, vascular	< 2%
Clean-contaminated	GI, GU or respiratory tract entered without spillage, non-traumatic wound, no inflammation	GI procedures (appendectomy, cholecystectomy), GU/GYN procedures, thoracic	3-5%
Contaminated	Fresh traumatic wound, gross spillage from GI tract, entrance into GU or biliary tract, non-purulent inflammation, major break in sterile technique	Traumatic wounds, gross spillage	5-10%
Dirty	Traumatic wound from a dirty source or delayed treatment, fecal contamination, abscess, bacterial inflammation/ purulence, retained foreign body or devitalized tissue	Debridement, abscess I&D, old traumatic wound	30%

Dehiscence is defined as the disruption of a wound that was primarily closed, leading to a loss of the barrier and structural functions of skin, fascia, etc. Examples of particularly dangerous wound dehiscence include abdominal fascial dehiscence, which can lead to evisceration of abdominal contents, and sternal dehiscence after median sternotomy. Dehiscence is essentially a form of poor wound healing, and risk factors include poor nutritional status, diabetes, obesity, smoking, immunosuppression, advanced age, infection, and poor surgical technique. Examples of technical errors that can lead to dehiscence include using suture material that absorbs too quickly, handling the tissue poorly, or leaving excessive tension on the wound.

Incisional hernias develop because of incomplete healing of the fascia, resulting in weakness of the surgical wound, allowing abnormal protrusion of contents through a defect. Complete wound healing occurs in the first 6-8 weeks after surgery, at which point the tensile strength across the wound is at its maximum (80% of normal tissue). Therefore, hernias can develop over this time period due to poor wound healing and/or increased stress on the abdominal

wall. Incisional hernias after abdominal surgery are the most common type of ventral hernia. Conditions resulting in increased pressure on the fascia can also contribute to development of an incisional hernia, such as chronic cough (COPD), ascites, or pregnancy. Although dehiscence and incisional hernias share similar pathogenesis, with hernias, the contents are still contained within a sack of peritoneum, which prevents exposure of the bowel to the atmosphere. Surgical site infections place patients at a higher risk of developing an incisional hernia.

II. Postoperative Fever

Postoperative fever presents a diagnostic challenge to surgeons as it can be due to a number of infectious causes, as well as a noninfectious inflammatory response to the procedure itself. Knowledge of the most common causes of fever after surgery is necessary to guide diagnostic work up, and the 5 Ws mnemonic (see Table 2) is useful for remembering a broad differential.

In general, fever is mediated by the cytokine IL-1, which is released by activated macrophages and neutrophils, among other cells. IL-1 mediates the inflammatory response, along with IL-1 and TNFα, and it causes fever by altering the temperature set point in the hypothalamus. Often surgery itself can cause such an inflammatory response with a resulting fever. However, this is self-limited and does not occur after the first 24 hours.

Table 2

Category	Timing	Description
Wind	POD1-3	• Atelectasis (POD1) – collapse of alveoli during surgery and with splinting secondary to pain is thought to activate neutrophils, which releases IL-1 • Pneumonia (POD3) – secondary to prolonged collapse of alveoli or aspiration; Pseudomonas and MRSA are the most common bacteria that cause hospital-acquired pneumonia. Pneumonia is the most common nosocomial infection in the ICU Risk factors for atelectasis and pneumonia include incisional pain, shallow breathing with poor alveolar recruitment, depressed cough from narcotics, pulmonary edema, prolonged bedrest, and smoking history (smokers should be encouraged to stop smoking 4-8 weeks before surgery)
Water	POD3-5	Urinary tract infection is secondary to Foley catheter use during surgery and/or post-operatively. Normal urogenital flora (Gram negative E. coli, Proteus) can colonize the catheter and seed the urine UTI is the most common nosocomial infection in the hospital overall
Walking (Vein)	POD3-7	Venous thromboembolic disease (DVT, PE) can cause an inflammatory reaction resulting in fever
Wound	POD3-7	Surgical site infections such as cellulitis, abscess, and anastomotic leak develop after POD3; Staphylococcus and Streptococcus are common organisms, although enteric

		bacteria may be involved, especially after colon surgery *more aggressive necrotizing soft tissue infections can develop within hours after surgery (C. perfringens and beta-hemolytic Streptococcus are common organisms)
Wonder drugs or "What did we do"	POD7+	Drug fever – any drug can cause this, but antibiotics and sulfa drugs are common culprits Central line infection – also due to colonization of the line and seeding of the bloodstream, most commonly with Staphylococcus Transfusion reactions

Of note, malignant hyperthermia, which is a serious cause of early postoperative fever, is not discussed here.

III. Respiratory Distress

Respiratory distress is a common postoperative complication encountered by surgeons. The differential for postoperative respiratory distress includes atelectasis, pneumonia, aspiration, pulmonary edema, acute respiratory distress syndrome (ARDS), pulmonary embolism, fat embolism, and narcotic overdose. Atelectasis and pneumonia frequently occur in the post-operative period. See the above section on Postoperative Fever for descriptions. Although atypical, respiratory distress may be the only presenting symptom of a heart attack, especially in elderly women and diabetics.

Aspiration can occur when gastric contents reflux beyond the esophagus and enter the respiratory tract or when oropharyngeal secretions and food enter the respiratory tract from above. Aspiration initially causes a chemical pneumonitis, but can progress to bacterial infection. It can occur during induction of anesthesia prior to surgery or postoperatively due to inability to protect the airway, such as in patients with altered mental status, stroke, or heavy vomiting. Patients that are high risk for aspiration during induction of anesthesia include those that have not fasted, all trauma patients regardless of last PO intake, pregnant patients, and patients with certain GI tract pathologies: bowel obstruction, gastroesophageal reflux disease (GERD), paraesophageal hernia, and delayed gastric emptying. In surgical patients, aspiration occurs in the supine position, and flow of aspirated contents is dependent on gravity. Therefore, aspiration usually affects the right middle and lower lobes due to the larger caliber and straighter course of the right mainstem bronchus compared to the left. Aspiration in the prone position, such as in alcoholic patients who are found down, is more likely to affect the right upper lobe.

Pulmonary edema is caused by abnormal movement of fluid across the alveolar capillary membrane due to an imbalance of Starling forces in the pulmonary capillaries. This buildup of fluid in the pulmonary interstitial and alveolar spaces leads to decreased diffusing capacity, hypoxemia and shortness of breath. It can be caused by increased hydrostatic pressure within pulmonary capillaries, as is the case with heart failure, where decreased left ventricular cardiac output causes fluid to back up in the pulmonary veins, increasing hydrostatic

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pressure in the capillaries. Fluid overload can cause pulmonary edema even in patients with a normal ejection fraction, and this is especially the case in patients with end-stage renal disease who are dependent on dialysis for removal of excess volume.

Acute respiratory distress syndrome (ARDS) is essentially non-cardiogenic pulmonary edema. This process is mediated by widespread inflammation in the lungs and can be a result of several different diseases. Increased permeability of alveolar capillary membranes, mediated by inflammatory cytokines such as IL-1, IL-6 and TNF, causes protein- and leukocyte-rich fluid to build up in the alveolar space, again causing decreased diffusing capacity of oxygen, hypoxemia and shortness of breath. The most common etiologies of ARDS include sepsis, pneumonia, aspiration, severe trauma, and massive transfusions.

Pulmonary embolism (PE) occurs when there is thrombus within the pulmonary arterial system. This can result in hemodynamic and respiratory consequences. The obstruction of pulmonary arteries and/or arterioles results in increased afterload against the right heart, which can be hemodynamically significant and result in obstructive shock. Pulmonary embolism increases the dead space, resulting in hypoxemia from a ventilation perfusion mismatch. The majority of PEs develop from migration of iliofemoral deep vein thrombosis (DVT), another form of venous thromboembolic disease (VTE). Risk factors for VTE can be categorized into three main groups using Virchow's Triad:

1. Stasis of blood flow - prolonged postoperative immobility (especially orthopedic and trauma patients) results in stasis, May-Thurner syndrome results in stasis due to mechanical obstruction of the left common iliac vein by the overlying right common iliac artery
2. Endothelial injury – all trauma patients and patients undergoing major operations
3. Hypercoagulability – malignancy, pregnancy, obesity, smokers, hyperviscosity syndromes, and genetic mutations can all cause hypercoagulability (Factor V Leiden is the most common genetic hypercoagulability disorder)

IV. Acute Kidney Injury/Oliguria

Oliguria, or low urine output, is common in the postoperative patient and is often the first presenting sign of acute kidney injury (AKI) or acute renal failure (ARF). While oliguria can occur in any patient, patients with preexisting chronic kidney disease (CKD) are at higher risk. Other risk factors include advanced age, heart failure, hypertension, peripheral vascular disease, diabetes, cardiovascular procedures, endovascular procedures requiring IV contrast, intraoperative hypotension, aortic cross-clamping, and emergency surgery. Oliguria and AKI can be broken down into prerenal, intrinsic renal, and postrenal obstructive causes (see Table 3). Of note, AKI can be non-oliguric, where the patient has normal urine output but increasing decreased clearance ability. Contrast-induced nephropathy is often non-oliguric.

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Table 3

Type of AKI	Description
Prerenal	Due to transient renal hypoperfusion from: - hypovolemia secondary to bleeding or dehydration - hypotension during surgery - decreased cardiac output (heart failure) Mediated by ADH and the renin-angiotensin axis (decreased GFR and urine output)
Intrinsic renal	Three types: - Acute tubular necrosis (ATN) – most common, >50% of intrinsic renal injury. Causes include prolonged hypoperfusion (prerenal state) which causes ischemia; or nephrotoxic substances and drugs such as myoglobin, antibiotics (vancomycin, aminoglycosides), IV contrast, etc. - Acute interstitial nephritis (AIN) – result of infection or allergic reaction to a variety of drugs (antibiotics, NSAIDs) - Acute glomerulonephritis - immune mediated inflammation of the glomerular membrane
Postrenal	Due to obstruction of the urinary tract – BPH, kidney stones, obstructed Foley catheter, GU tract malignancy

V. Hypotension

Hypotension in the postoperative patient can be due to serious causes such as bleeding, sepsis, adrenal insufficiency, or cardiac causes. It is important to diagnose and treat these conditions, since their progression can result in shock, multi-organ failure and death.

Once these life-threatening causes are ruled out, other more common causes such as hypovolemia due to insensible losses during the case or reactions to certain drugs, especially narcotics, sedatives, and epidural regional anesthesia, should be considered.

Postoperative bleeding is usually due to incomplete hemostasis during the operation or coagulopathy, which can be seen after severe trauma or in liver failure. It is imperative to rule out bleeding as the cause of hypotension in the immediate postoperative period since it may require a trip back to the operating room.

Sepsis can cause distributive shock due to a massive inflammatory response to infection. Usually the infection is bacterial, although fungi, viruses and parasites can also cause sepsis. The inflammatory cascade that occurs causes vasodilation and increased capillary permeability, resulting in hypotension due to decreased circulatory volume.

Adrenal insufficiency can result in systemic hypotension due to low levels of glucocorticoids, which are necessary for adequate systemic vascular resistance (SVR). Adrenal insufficiency can be primary (Addison's disease), or secondary as a result of suppression of the hypothalamic-pituitary-adrenal (HPA) axis. In either case, any stress on the body, such as with surgery, trauma, or major illness, will require additional glucocorticoid to maintain SVR and blood pressure. However, patients with adrenal insufficiency will be unable to endogenously produce this additional steroid, and can present with refractory hypotension postoperatively. The HPA axis will be iatrogenically suppressed in patients that are on steroids for medical conditions (COPD, rheumatoid arthritis), and it is important to recognize that refractory hypotension in these patients may be due to adrenal insufficiency. Adrenal suppression is a commonly tested side effect in patients receiving etomidate, a sedative used for induction of anesthesia. This is rarely seen as it typically only occurs with continuous etomidate infusions, which is no longer done.

Cardiac causes of postoperative hypotension include acute myocardial infarction (MI) from coronary artery plaque rupture resulting in left ventricular (LV) dysfunction, exacerbation of congestive heart failure (CHF), or arrhythmias. Atrial fibrillation with rapid ventricular response (RVR) occurs frequently after surgery due to fluid shifts and electrolyte imbalances, and can result in hypotension due to decreased filling time of the LV and decreased preload. Preoperative cardiac assessment, which may involve stress testing, should be done for all patients to evaluate their risk of perioperative cardiac complications, and coronary revascularization may be needed to decrease risk prior to non-cardiac surgery.

VI. Ileus

Ileus, or intestinal paralysis, is common in hospitalized patients, especially surgical and trauma patients, for a number of reasons. While severe ileus may lead to intestinal ischemia and perforation, it is important to rule out mechanical obstruction, which poses a much higher risk of perforation. Risk factors for development of ileus include abdominal surgery, electrolyte imbalances, narcotic pain medication, immobility, sepsis, peritoneal irritation, spinal cord injury, or metabolic acidosis; all of which can cause ileus after non-abdominal surgery. With abdominal surgery, there is a normal physiologic ileus due to handling of the bowel and irritation of the peritoneum. This especially occurs after open procedures on the intestinal tract. Physiologic ileus typically lasts up to 3 days, with the small bowel regaining function first, then the stomach (within 24 hours), then the colon (within 48-72 hours). However, after this time period, ileus may be a sign of a pathologic process, such as intra-abdominal bleeding or abscess, which causes peritoneal irritation. The most common causes of ileus in the postoperative patient include electrolyte imbalance, immobility and narcotics.

VII. Stress Ulcers

Stress ulcers, or stress gastritis, occur in severely ill patients or those under extreme physiologic stress. Patients at risk include those suffering massive burn injury, severe head injury, massive trauma, sepsis, and multisystem organ failure. The gastric lumen contains acidic fluid due to secretion of hydrogen ions by parietal cells found in the fundus and body of the stomach. This acid secretion is governed by various stimuli, such as vagal nerve

stimulation, histamine secreted by enterochromaffin-like cells, and gastrin secreted by G cells in the antrum of the stomach. The gastric mucosa itself is protected by a mucous layer, which creates a barrier between the epithelial cells and the luminal contents and buffers the acid with bicarbonate. Several factors, including prostaglandins, nitric oxide and also vagal nerve stimulation, promote the formation of this layer. However, during periods of severe illness or physiologic stress, the mucous layer is less effective due to decreased bicarbonate concentrations. During periods of stress and illness, there is also decreased blood flow to the gut, causing a lower threshold for ischemia of the mucosal lining. Of note, acid secretion is also decreased during these conditions; therefore acid hyper-secretion is not a significant factor in the pathophysiology of stress gastritis.

VIII. Neurologic Complications

Two of the most common neurologic complications, delirium and stroke, are discussed below. These can occur regardless of the type of surgical procedure.

Postoperative delirium is very prevalent in the hospital setting and is occurs in 10-50% of older surgical patients, with even higher rates in the ICU setting. It can be challenging to distinguish delirium from preexisting cognitive dysfunction, or dementia. While delirium is often a self-limited state related to the stress of undergoing surgery, it can also be the presenting sign of another serious pathologic process, similar to ileus. It is imperative that the surgical team evaluate patients presenting with delirium for underlying causes before proceeding with pharmacologic treatment. For example, before a patient is diagnosed with ICU delirium, which is due to loss of physiologic sleep patterns, other causes should be ruled out. Risk factors that can increase a patient's baseline vulnerability to developing delirium include advanced age, dementia, prior episodes of delirium, polypharmacy, and sensory impairment. Table 4 below lists some common precipitating factors that can cause delirium.

Table 4

Primary CNS Disorders	- Seizure - Infection: meningitis, encephalitis, epidural abscess - Head injury - Psychiatric disorders
Infection	- Any cause of sepsis: - UTI - Pneumonia - Abdominal abscess
Metabolic	- Electrolyte disturbances: sodium, calcium, magnesium, phosphate - Endocrine: thyroid, adrenal - Hyper/hypoglycemia - Acid/base disturbance - Acute or chronic renal failure (uremia) - Liver failure

Respiratory	• Hypoxia • Hypercapnia
Drugs/Toxins	• Any drug of abuse • Withdrawal from EtOH, benzodiazepines • Medications: opioids, benzos, sedatives, antihistamines, antimuscarinics

Stroke Although the risk of stroke is low with general surgery procedures, the risk increases with vascular and cardiac surgery procedures due to manipulation of the arteries supplying the brain and release of emboli. Most postoperative strokes are ischemic and embolic in nature, as opposed to hemorrhagic. Ischemic strokes are due to occlusion of flow in a cerebral vessel. This can be due to atheroembolic disease from within the intracranial or extracranial carotid artery, or it can be due to cardioembolic disease, as seen with atrial fibrillation. Delayed strokes may also be due to atrial fibrillation and myocardial infarction. Hemorrhagic strokes can be subarachnoid or intraparenchymal, where the bleeding compresses surrounding brain tissue. Those factors that contribute to surgery-induced hypercoagulability, such as general anesthesia, bed rest, and withholding of antiplatelet and anticoagulant therapy, can also increase the risk of stroke. Some risk factors that increase the risk of postoperative stroke are:

- Advanced age
- Comorbidities: diabetes, hypertension, chronic renal failure, COPD, peripheral vascular disease, cardiac disease (CAD, arrhythmias, heart failure)
- Smoking
- History of prior cerebrovascular accident (CVA) or transient ischemic attack (TIA)
- Carotid stenosis
- Intraoperative factors: general anesthesia, prolonged surgery, manipulation of the proximal aorta
- Hypovolemia due to bleeding or dehydration
- Postoperative heart failure

Of note, a stroke that occurs in the immediate postoperative period after carotid endarterectomy may be due to a technical complication from the operation if it affects the ipsilateral distribution. For example, an intimal flap within the lumen of the carotid artery may have been created during the surgery which then occludes flow.

Signs and Symptoms

I. Wound Complications

Wound infections can affect the superficial incisional space or the deep incisional space. Superficial infections involve only the skin and subcutaneous tissue near the incision, and these typically present with cellulitis or superficial abscess with purulent drainage. In general, cellulitis can manifest the classic signs of inflammation: erythema, induration, pain, and

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warmth. On the other hand, deep infections, such as abscesses deep to the fascial or muscular planes, may involve the peritoneal or organ spaces. These types of abscesses typically present with pain, fevers, or, in the case of GI surgery, ileus. New onset ileus after GI surgery, which presents with nausea, vomiting and failure to advance the diet, is often an indicator of peritoneal inflammation, which can be secondary to intra-abdominal abscess. Of note, necrotizing soft tissue infections, such as necrotizing fasciitis, typically progress much more rapidly and can cause the patient to deteriorate hemodynamically if they are not taken to the OR for early debridement. Necrotizing fasciitis may present a few hours after surgery, and specific signs include pain beyond external signs of wound infections, crepitus, and expression dish-water fluid. Wound infections, like all infections, will also cause signs of systemic inflammation, which are discussed below in the section on Postoperative Fever. Importantly, tachycardia may one of the first signs of an intra-abdominal anastomotic leak particularly in obese patients.

Wound dehiscence presents with spontaneous opening of the wound edges. With fascial dehiscence, the overlying skin closure may be intact. Signs of abdominal fascial dehiscence include erythema of the skin, leakage of salmon-colored peritoneal fluid through the incision, or leakage of succus or stool, which would indicate an underlying anastomotic leak and fistula as well. Often, surgeons will probe the skin incision using a sterile cotton-tipped applicator to determine if the underlying fascia is still intact, although one must be careful as this can worsen the extent of the dehiscence. Large dehiscences may present with evisceration of bowel contents, a surgical emergency. Sternal dehiscence may present with bony instability, chest pain and fevers. There is usually an underlying infection of the mediastinum.

Incisional hernia presents with bulging and pain at the site of the incision. While hernias can occur acutely in the immediate postoperative period, they can also form more gradually due to increasing pressure on the fascia during the wound healing period. Incisional hernias may be freely reducible, where the bowel contents are easily pushed back through the fascial defect, or they may have a tendency to become incarcerated. Incarceration occurs when the bowel herniates through the defect but cannot reduce back into the abdominal cavity, often due to edema that develops in the bowel while it is herniated. Smaller fascial defects, such as laparoscopic port sites, tend to have a higher risk of incarceration. Incarceration typically presents with worsening pain, obstructive symptoms (nausea, vomiting, lack of flatus or bowel movements), and it can progress to strangulation of the bowel.

II. Postoperative Fever

Post-operative fever is defined as a temperature above 38.0 Celsius (100.4 F). While fever is commonly a normal inflammatory response to surgery, it can also be a sign of infection. Often, fever will be associated other criteria indicative of the Systemic Inflammatory Response Syndrome (SIRS), shown below in Table 5.

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Table 5

Criteria	Definition
Hyper/hypothermia	Temperature > 38°C or < 36°C
Tachycardia	HR >90 beats/min
Tachypnea	RR > 20 breaths/min or $PaCO_2$ < 32 mmHg
Leukocytosis/leukopenia or left shift	WBC >12 cell/mm^3, <4 cell/mm^3, or >10% immature bands

To meet SIRS criteria, the patient must exhibit two of the four criteria. Fever and SIRS may be signs of sepsis, which is defined as meeting SIRS criteria with a known source of infection, and it is imperative to quickly recognize and treat sepsis because it can progress to shock, multi-organ failure, and death.

In addition to SIRS criteria, the other clinical signs and symptoms of the most common causes of postoperative fever are summarized in Table 6 below.

Table 6

Category	Etiology (Time)	Presentation
Infectious	UTI (POD3)	Dysuria, hematuria, cloudy urine, delirium especially in elderly patients; positive SIRS criteria may be the only signs in obtunded patients, positive UA
	Pneumonia (POD3)	Respiratory distress, hypoxemia, cough, purulent sputum, increasing ventilator support requirements, infiltrates on chest x-ray
	Wound infection (POD3-7)	Incisional erythema, purulent drainage, deep abscess, pain, intra-abdominal abscesses can cause ileus secondary to peritoneal inflammation. See section on Wound Infections above.
	Central line infection	Redness at the catheter insertion site, bacteremia
Non-infectious	DVT/PE (POD3-7)	DVTs typically cause calf swelling and pain; since PEs usually embolize from a DVT, these patients may also have calf swelling. PE presents with nonspecific signs, including respiratory distress, cough, chest pain, tachycardia, tachypnea, and hypoxemia, and one must have a high index of suspicion.
	Wonder Drug	This is a diagnosis of exclusion after other causes of fever are ruled out

Any source of infection can cause bacteremia. Central line infections directly seed the vascular space, whereas other sources of infection must be left untreated for several days to become severe enough for bacteria to translocate into the vascular space.

III. Respiratory Distress

General signs and symptoms of respiratory distress include tachypnea (respiratory rate > 20 breaths/min), use of accessory muscles to breathe (neck muscles, pectoralis, abdominal muscles, serratus anterior, latissimus dorsi), nasal flaring, chest retractions, inability to speak full sentences, grunting, difficulty breathing while lying flat, shortness of breath abnormal breath sounds, hypoxia or increasing oxygen requirements, cyanosis, altered mental status due to hypoxia, and increasing ventilator support requirements. Below are signs and symptoms specific to some common causes of respiratory distress.

Aspiration can present from mild respiratory distress to severe respiratory failure requiring immediate intubation or even cardiopulmonary bypass with ECMO. These patients will have decreased breath sounds, wheezing, tachycardia, and imaging is expected to show pathology in the right middle and lower lobes.

Pneumonia presents with decreased or abnormal breath sounds (wheezing, crackles), fever, sputum production, unilateral opacification or infiltrates on chest x-ray.

Pulmonary edema presents with bilateral wheezing or crackles, diffuse patchiness on chest x-ray. The Berlin criteria is used to clinically define ARDS using the following signs and data: acute onset of lung injury (1 week), bilateral opacities on imaging, decreased PaO_2/FiO_2 ratio (<300 = mild, <200 = moderate, <100 = severe ARDS), and no heart failure or fluid overload.

IV. Acute Kidney Injury/Oliguria

Acute kidney injury typically presents abruptly (within 48 hours) with oliguria, or low urine output, and decreased clearance ability. In adults, oliguria is defined as less than 0.5 cc/kg/hour, and 300 cc per 8 hour shift can be used as quick estimate for patient approximately 75 kg. In children, oliguria is defined as less than 1 cc/kg/hr since their nephrons have a decreased ability to concentrate urine. In patients with Foley catheters, an obstructed or clogged catheter can mimic oliguria. Flushing the catheter to ensure proper drainage and using an ultrasound bladder scanner to look for urinary retention can help rule out an obstructed Foley. Prolonged oliguria can lead to fluid retention and overload, which causes both peripheral and pulmonary edema, and this may be severe enough to cause respiratory distress. When evaluating these patients, it is important to pay attention to their volume status. Intake and output balance and increasing daily weights also suggests hypervolemia. Decreased clearance ability can result in symptomatic electrolyte and acid-base imbalances, such as hyperkalemia, uremia, and acidosis. Severe hyperkalemia can be life threatening due to destabilization of the cardiac membrane resulting in arrhythmias. The first EKG change will be peaked T waves. Uremia, or elevated BUN, can lead to mental status changes, coma, pericarditis, nausea/vomiting, pruritus, and platelet dysfunction. As mentioned above, acute renal injury can be nonoliguric, in which case the only manifestation

would be decreased clearance ability. In these patients, AKI is defined as an increase in serum creatinine greater than 1.5x the baseline or an absolute rise of 0.3 mg/dL or more in a 48 hour period according to the Acute Kidney Injury Network (AKIN). It is important to remember the indications for acute hemodialysis in these patients: refractory metabolic acidosis, electrolyte imbalances, clearance of toxins, volume overload, and uremia.

V. Hypotension

Hypotension is very common in surgical patients, and, while it can be due to relatively benign or transient causes, such as medications or mild hypovolemia from fluid losses in the OR, it can also be a sign of the clinical syndrome of shock. Shock is defined as inadequate tissue perfusion to support normal cellular functions. Because blood pressure varies among patients, there is no hard cut off defining hypotension. However, a pressure below 90/60 mmHg or mean arterial pressure (MAP) below 65 mmHg is generally considered low. In addition to hypotension, some general signs of shock and hypoperfusion include cool, pale skin, tachycardia, confusion or mental status changes, decreased urine output, and lactic acidemia.

Bleeding - when postoperative patients present with hypotension, it is critical to assess them for ongoing bleeding, which can progress to hemorrhagic shock. Early bleeding may be due to inadequate hemostasis during the case, and it must be identified quickly as the patient may need to return to the operating room. Signs of ongoing bleeding include rapid saturation of dressings with blood, surgical drains that continuously fill with blood, or peritonitis from intra-abdominal bleeding.

Sepsis - see the above section on postoperative fever for signs of sepsis, which can cause septic shock.

Adrenal insufficiency typically presents with hypotension refractory to adequate fluid resuscitation in a patient with a history of chronic steroid use or primary adrenal insufficiency (Addison's disease). This medical history is often more important than laboratory testing in making the diagnosis of postoperative adrenal insufficiency.

Cardiac hypotension, which can cause cardiogenic shock if severe, is due to pump failure, and will be accompanied by fluid overload, abnormal heart sounds, cardiomegaly on chest imaging, and LV dysfunction echocardiography.

Other types of shock include obstructive shock, neurogenic shock, although they are less commonly encountered in postoperative patients.

VI. Ileus

Ileus presents with nausea, vomiting, lack of flatus and bowel movements, decreased bowel sounds, inability to tolerate a diet, and abdominal distension and pain. The clinical signs and symptoms of ileus are identical to those of mechanical bowel obstruction, therefore, imaging and laboratory data are necessary to differentiate the two.

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VII. Stress Ulcers

Stress gastritis and ulceration is a form of upper GI bleeding and presents with coffee ground emesis, hematemesis, or melena. When patients present with melena, nasogastric tube lavage can be used to differentiate between an upper and lower GI bleed. If the NG tube returns blood, then it is an upper GI bleed and if it returns bile then it is a lower GI bleed. If it returns gastric fluid without bile, then it is considered equivocal because there could be an upper GI bleed from the duodenum which was not caught because the pylorus was closed at the time of the lavage (the presence of bile confirms that the pylorus was open when the sample was taken). Hemodynamic instability and the drop in hemoglobin are markers of the severity of the bleeding, and endoscopy is used to make the diagnosis and to potentially manage the bleeding ulcer.

VIII. Neurologic Complications

Delirium presents with altered mental status, including disturbances in cognition, consciousness, and perception. These patients are often disoriented and display odd behavior, such as agitation or hallucinations. While some of these symptoms overlap with those of dementia, one of the hallmarks of delirium is a fluctuating course which affects attention the most. Delirium has an acute onset, and this can be the most helpful feature in distinguishing it from a patient's underlying dementia. Because delirium is almost always due to a medical condition or substance, one should evaluate the patient broadly to uncover the underlying cause. For example, delirium is often the first sign of an underlying infection, such as UTI or pneumonia, especially in older patients. In addition, if a patient that underwent a bowel anastomosis presents with delirium and other SIRS criteria 3-5 days postoperatively, one should consider abscess or anastomotic leak. Blood work should be done to evaluate for an elevated WBC or electrolyte abnormalities.

Stroke may present with altered mental status and unresponsiveness, albeit rarely. In addition, large subarachnoid hemorrhage can present with headache, seizures, and obtundation. Strokes that are ischemic typically present with signs caused by dysfunction of the specific region the brain that is ischemic. For instance, a stroke affecting the motor cortex (middle cerebral artery) would present with contralateral weakness. Common signs of stroke include facial droop, aphasia, and limb weakness.

Diagnostic Strategy

1. History and Physical

The first step in approaching a patient with a postoperative complication is performing a careful history and physical geared towards discovering the important details of the operation and the above-mentioned signs and symptoms. The section on Signs and Symptoms above describes specific history and physical exam findings that are associated with each disorder.

- Interviewing the patient can provide details about the presence and timing of symptoms that are associated with the various complications. In addition, pertinent past medical history can be reviewed and may reveal risk factors for developing certain complications.

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For example, a patient with a history of chronic kidney disease is more likely to develop postoperative AKI, and a patient with preexisting coronary artery disease is more likely to develop an MI.

- Chart review can provide details about the operation that can aid diagnosis. Often, this information can be found in the operative report or the anesthesia record. For example, if the patient had a large amount of blood loss during the case and presents with continued hypotension, it is likely due to hypovolemia. Or, if there was a large amount of spillage of GI contents during abdominal surgery and the patient has a fever, abscess should be higher on the differential.

2. Diagnostic Tests

In practice, the presentation and evaluation of many post-op complications overlap, and any one sign or symptom may have multiple etiologies on the differential. For instance, respiratory distress could be due to pneumonia, heart failure, ARDS, or fluid overload from AKI. Therefore, the work up of these postoperative conditions involves common laboratory and radiographic tests. Table 7 below shows the lab and imaging test used for work up of the complications discussed above. The information gathered from these tests is discussed afterwards.

Table 7

Complication	Laboratory/Imaging Tests
Wound complication	CBC, wound culture, CT scan
Fever	CBC, BMP, UA, urine culture, blood culture, sputum culture and wound culture if applicable, CXR
Respiratory distress	CXR, ABG, EKG, echocardiogram, CT-angiography of the chest
AKI/Oliguria	BMP, UA with microscopy, urine electrolytes, EKG, renal ultrasound
Hypotension	CBC, BMP, lactate, ABG, ACTH stimulation testing and cortisol level; coagulation studies
Ileus	BMP, abdominal x-ray
Stress ulcer	CBC, upper endoscopy

Laboratory Tests

- ACTH stimulation (cosyntropin) testing and cortisol levels are necessary to diagnose adrenal insufficiency (AI). A low free cortisol levels is suggestive of AI, but the level can be affected by low protein levels even when adrenal function is normal.
- Arterial blood gas (ABG) – pO2 and pCO2 can provide details about the cause and severity of respiratory distress. Hypoxemia is expected with pneumonia, PE, and

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pulmonary edema. Hypocapnia can be seen with tachypnea due to hyperventilation. ABGs are often obtained in shock as the pH and base deficit can be helpful in defining the degree of hypoperfusion.

- Basic metabolic panel (BMP)
  - o Checking electrolytes, blood urea nitrogen (BUN), and creatinine (Cr) can determine the presence and severity of kidney injury
  - o The BUN:creatinine ratio can differentiate prerenal from renal AKI: a BUN:Cr ratio > 20:1 suggests prerenal AKI since the tubules are still able to reabsorb efficiently. A BUN:Cr <10:1 suggests renal injury and a reduced ability to reabsorb urea.
- Blood cultures should be obtained for a fever work up in order to both diagnose bacteremia and ensure appropriate antibiotics.
- Cardiac enzymes (troponins, creatine kinase total and MB isoenzyme) should be obtained in patients with postoperative hypotension to rule out MI and arrhythmias such as atrial fibrillation.
- Coagulation studies (PT/INR, PTT, fibrinogen) and a blood type and screen should be checked in any bleeding patient to ensure that there are no coagulopathies that require correction with blood products.
- Complete blood count (CBC)
  - o Hemoglobin levels are used to qualify the severity of blood loss in the setting of hypotension from bleeding or stress gastritis; however, one cannot rely on hemoglobin to diagnose bleeding since acute blood loss will not cause a change in the hemoglobin until enough time has passed for the blood to equilibrate.
  - o An elevated white blood cell count (WBC) is in an indicator of an infection
  - o Platelets should be checked if a patient is bleeding to make sure there isn't a quantitative deficit that needs transfusion to help treat the bleeding
- Lactate, a measurement of lactic acid which accumulates during anaerobic metabolism, is a marker of inadequate tissue oxygenation and is thus a sign of hypoperfusion. Lactates are measured in patients with shock.
- Urinalysis (UA) with microscopy and culture
  - o The presence of casts on microscopy can suggest renal damage. For example muddy brown casts are suggestive of acute tubular necrosis (ATN), the most common type of intrinsic AKI. White blood cell casts are associated with acute interstitial necrosis, and red blood cell casts are associated with glomerulonephritis.
  - o A UA positive for nitrites, leukocyte esterases and bacteria is highly suggestive of UTI, and it is important to obtain urine culture in the inpatient setting to ensure that appropriate antibiotics are chosen.
- Urine electrolytes
  - o Urine sodium and creatinine are also used to differentiate prerenal acute kidney injury from other types. Specifically, calculating the fractional excretion of sodium (FeNa) can be diagnostic. However, it is important to realize that FeNa is not valid in patients that have received loop diuretics which alter sodium excretion.
  - o FeNa = (Serum creatinine x Urine sodium)/(Serum sodium x urine creatinine)

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- A FeNa < 1% suggest prerenal causes – this makes sense because the kidney is still able to efficiently able to reabsorb sodium in an attempt to counteract renal hypoperfusion
- FeNa >2-3% suggest renal damage.

Imaging

- Abdominal acute series should be done for patients presenting with ileus. Dilated loops of bowel will be seen with bowel, but there should not be any air-fluid levels, which can be seen with mechanical obstruction. This includes three films:
  - An upright chest x-ray – can see free air under the diaphragm from intestinal perforation
  - An upright abdominal x-ray which can show air-fluid levels suggestive of mechanical bowel obstruction
  - A supine abdominal x-ray, also called a kidney-ureter bladder (KUB) which can estimate the level of distension of loops of bowel and show air in the rectum
- Abdominal CT scans should be done whenever a deep wound infection is suspected (intra-abdominal abscess or anastomotic leak) or there is prolonged ileus. It is important to obtain these studies with both PO and IV contrast if able. PO contrast will delineate the bowel lumen and may demonstrate a leak. IV contrast can help define the wall of an abscess.
- Chest x-rays (CXR) should be done for all patients presenting with respiratory distress and are used in the work up of fever to diagnose pneumonia
- Lower extremity venous duplex ultrasound is used to diagnose deep vein thrombosis.
- Renal ultrasounds should be done in cases of severe AKI to rule out hydronephrosis, which is associated with post-renal obstructive renal injury.
- Transthoracic echocardiography (TTE) has many applications:
  - Evaluation of LV function, which can differentiate cardiogenic pulmonary edema from non-cardiogenic causes. Therefore, a TTE is required for the diagnosis of ARDS to rule out CHF.
  - Diagnosis of structural causes of new onset arrhythmias, like atrial fibrillation.
  - Assessment of right heart strain, a measure of the severity of massive pulmonary embolism.

Prevention and Management

Most postoperative complications are best treated by prevention. Both prevention and management of complications are discussed below:

I. Wound Complications

Wound infections can be prevented with adequate skin prep prior to surgery, chlorhexidine washes at home, clipping (not shaving) hair, using preoperative antibiotics for certain cases, glycemic control, tobacco cessation at least 30 days prior to an elective case, and appropriate surgical barriers and antiseptic hand scrub by the surgical team. Preoperative

antibiotics are indicated for clean-contaminated cases and above, although they are often used in clean cases when implants will be placed. They should be given within 30-60 minutes prior to skin incision and appropriate antibiotics should cover the bacteria that will be encounter. Cefazolin can be used to cover common skin flora such as Staphylococcus and Streptococcus. Higher generations of cephalosporins offer more gram negative and anaerobe coverage and are used in cases involving entry into the GI tract. For example, cefoxitin is commonly used prior to colon surgery. Often, each hospital will provide guidelines for choosing the appropriate antibiotic for each type of surgery. In addition to prophylactic antibiotics, grossly contaminated wounds should not be closed. Instead they should be kept open with wet-to-dry dressings, allowing microdebridement of any contamination each dressing change and healing by secondary intention. Again, if a wound infection does occur, antibiotics are sufficient for simple cellulitis, but, abscesses must be drained. Superficial abscesses can often be drained by opening the skin and packing the wound with wet-to-dry dressings, which will debride the infected tissue. Deep abscesses may require surgical drainage or percutaneous drain placement. If a deep abscess is a result of anastomotic leak, the patient must be made NPO and may require reoperation and creation of a proximal stoma to reduce flow of stool across the leak.

Dehiscence and incisional hernia can be prevented by ensuring adequate preoperative nutritional status and reducing risk factors that will contribute to local tissue ischemia, such as smoking and diabetes. A preoperative albumin of less than 3.5 g/dL indicates malnutrition, and delaying elective surgery until the patient's nutrition can be optimized should be considered. Both wound dehiscence and hernia require returning to the operating room for repair. However, not all incisional hernias need to be fixed, especially large hernias that pose little risk of incarceration.

II. Postoperative Fever

Prevention and management of wound infections, atelectasis, and pneumonia are described in the sections above and below on Wound Complications and Respiratory Distress. Prevention of other infectious causes of fever involves decreasing the exposure to risk factors, such as Foley catheters and central lines. These should be aggressively removed in the postoperative period as long as they are no longer needed for treatment. Another key principle in the management of infections is that source control is usually necessary for the successful treatment of most infections, in addition to antibiotics. Antibiotics alone cannot eradicate abscesses, central line infections, and catheter-associated UTIs because these sites are not vascularized and the antibiotic will not reach meaningful concentrations to kill the bacteria. Any catheter causing an infection should be removed and abscesses must be drained, either in the operating room or with percutaneous drainage. On the other hand, pneumonia and simple cellulitis do not require source control since antibiotics in the blood stream will reach effective concentrations in the alveoli and dermis, respectively. While it is important to obtain culture data prior to starting treatment in order to ensure proper sensitivity, this should not delay treatment.

III. Respiratory Distress

Aspiration during induction of anesthesia in high risk patients can be prevented by NG tube decompression of the stomach prior to induction and rapid sequence intubation (RSI), which involves preoxygenating an unsedated patient with O2 via face mask, rapid sedation and paralysis with etomidate and succinylcholine, and rapid intubation with cricoid pressure. This foregoes bag-mask ventilation of a sedated patient, which would worsen gastric distension and risk of vomiting by insufflating the stomach. Rapid onset medications are used and the patient must be intubated quickly once they are paralyzed. Cricoid pressure during intubation (Sellick Maneuver) will compress the esophagus, preventing passive regurgitation but not active vomiting from reaching the airway if it does occur, although the effectiveness of this maneuver has been called into question. All patients with severe bowel obstruction requiring operation should receive an NG tube well before surgery.

Atelectasis, pulmonary edema, and pneumonia can often be prevented with judicious fluid use, early mobilization, and cough and deep breathing exercises with incentive spirometry. Adequate analgesia has been shown to prevent atelectasis, pulmonary edema, development of pneumonia, and even development of DVT and PE by reducing splinting from pain and promoting early mobility. Multimodal therapy including intravenous patient-controlled anesthesia, opioid-sparing regimens, epidural anesthesia, and regional blocks have been shown to be superior. Pulmonary edema can also be treated with diuretics.

Pneumonia should be treated the same as hospital-acquired pneumonia, with broad-spectrum antibiotics that cover both pseudomonas and methicillin-resistant Staphylococcus aureus (MRSA).

ARDS is managed by treating the underlying cause and using a lung-protective ventilation strategy with low tidal volumes to avoid further trauma to the alveoli.

Pulmonary embolism and DVTs are prevented by early ambulation, prophylactic doses of subcutaneous unfractionated heparin or low molecular weight heparin (LMWH), and intermittent compression hose. Treatment involves intravenous heparin, subcutaneous LMWH at therapeutic doses. With severe, hemodynamically significant PE (obstructive shock, right heart strain), more invasive treatment aimed at removing the clot from the pulmonary artery may be required. Techniques include endovascular thrombectomy and thrombolysis by Interventional Radiology and open pulmonary artery embolectomy median sternotomy.

IV. AKI/Oliguria

AKI is treated by correcting renal hypoperfusion with IV hydration and removing nephrotoxic agents. Contrast-induced nephropathy is best prevented by prehydration. Intravenous bicarbonate and N- acetylcysteine are also used prior to contrast to reduce the risk of AKI, although the data is inconclusive about their protective benefits. Bladder and urethral obstructions causing postrenal failure will require catheter drainage of the bladder, either with

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a Foley or a suprapubic catheter. Ureteral obstruction requires percutaneous nephrostomy tube drainage of the renal pelvis. If kidney injury progresses to failure, dialysis may be necessary.

V. Hypotension

Bleeding may require returning to the OR to achieve hemostasis, especially if the bleeding is from an internal site. Bleeding from skin edges can often be managed at bedside by holding direct pressure, packing the wound with hemostatic agents, or placing a suture across the bleeding vessel. Correcting any coagulopathy may also be necessary.

Sepsis – see above for management of Wound Infections and Postoperative Fever.

Adrenal insufficiency is treated with administration of an IV hydrocortisone taper, termed stress dose steroids.

Cardiac causes of hypotension:

- Post-operative MI requires immediate Cardiology consultation in order to evaluate the patient for possible percutaneous coronary intervention (PCI). These patients should also be given supplemental oxygen, adequate analgesia, and nitroglycerin to induce coronary vasodilation and beta-blockers to decrease myocardial oxygen demand only if they are not hypotensive. Aspirin and a statin are used to stabilize plaque and prevent further coronary occlusion, while anticoagulation is given to improve coronary flow. They should be transferred to the intensive care unit for hemodynamic management.

- Atrial fibrillation with rapid ventricular response (RVR) is treated with correction of electrolytes, correction of fluid overload with diuretics, and anti-arrhythmics such as metoprolol, diltiazem, or amiodarone. Patients with RVR that are hypotensive and unstable should undergo synchronized cardioversion.

Shock that is refractory to fluid resuscitation may require pressors to maintain adequate perfusion. Norepinephrine, which primarily causes peripheral vasoconstriction, is used in septic shock, whereas an inotrope, such as dobutamine, may be in cardiogenic shock. In cases of severe cardiogenic shock, mechanical circulatory support (intra-aortic balloon pump, ECMO) may be necessary. Hydrocortisone should be administered if the patient has adrenal insufficiency. These patients must be in the ICU.

VI. Ileus

After mechanical obstruction and intra-abdominal abscess or leak is ruled out, ileus is managed with bowel rest, IV fluids, NG tube decompression if there is vomiting, correction of electrolytes, and ambulation. Parenteral nutritional support may be required for prolonged ileus greater than 7 days.

VII. Stress Ulcers

Stress ulcers are prevented and treated with proton pump inhibitors or H2 blockers to suppress acid secretion. All severely ill patients at risk of stress ulcer formation should receive prophylactic acid suppression. Endoscopy with application of hemostatic agents, clipping or cautery may be required for severe bleeding.

VIII. Neurologic Complications

Delirium

After diagnosing and treating the underlying cause of delirium, there are several actions that can prevent or help improve the symptoms:

- Avoid problematic medications such as opioids, benzodiazepines, and antihistamines

- Orientation protocols

- Restoration of physiologic sleep patterns

- Regular visits with family and friends during the day time

- Early mobilization

- Ensuring patients have their visual and hearing aids

Antipsychotic agents, such as haloperidol or quetiapine, can be used to treat the hyperactive symptoms of delirium, like agitation and combativeness.

Stroke:

It is important to note that if patient presents with a stroke after a carotid endarterectomy, they may need to be taken back to the OR emergently to assess the carotid artery for technical complications.

Management of acute strokes often requires Neurology and sometimes Neurosurgery consultation. Depending on the timing of symptoms and type of stroke, IV thrombolytic therapy with alteplase (tPa) may be appropriate, and some patients require endovascular thrombectomy. Most patients will have specific blood pressure goals in the immediate period after stroke, depending on the type of stroke and whether they were treated with thrombolytics. In addition, it is important to evaluate the patient's speech and swallow function to prevent aspiration events and ensure that they have appropriate nutritional access. These patients will often require physical therapy and stroke rehabilitation. Further management, which is usually directed by the patient's Neurologist, is aimed at reducing risk factors for future stroke.

Questions

1. All of the following cytokines are mediators of the initial proinflammatory response to infection and injury EXCEPT:
   A. Interleukin 10
   B. Tumor necrosis factor α
   C. Interleukin 1
   D. Interleukin 6
   E. Interleukin 8

2. A 48 year old man is post-operative day number 5 from a right hemicolectomy for cancer. He has no nausea, no vomiting, but has subjective shortness of breath. Vital signs are T 36.5, HR 123 bpm, and BP 145/90 mmHg. Lung sounds are clear, abdomen shows a well healing midline incision. Chest x ray is unremarkable. EKG shows sinus tachycardia with no ST changes. What is the next most appropriate step in management? He has a right subclavian catheter in place.
   A. Increase pain medications
   B. Give lasix, 40 mg, IV times one now
   C. Begin a heparin drip and order CT chest
   D. Encourage incentive spirometry
   E. Insert a left sided chest tube

3. All of the following have been shown to decrease the risk of postoperative pulmonary complications, EXCEPT:
   A. Lung expansion maneuvers
   B. Preoperative smoking cessation
   C. Routine nasogastric tube decompression
   D. Postoperative epidural anesthesia
   E. Use of intraoperative short-acting neuromuscular blocking agents.

4. A 70 kg 48 year old healthy male is post-operative day number 0 from an open right hemicolectomy for cancer. His operation lasted 4 hours, and was complicated by 250 cc of blood loss. He received 1.5L of fluid during the case. His vital signs are 98.5, HR 104, BP 100/50. His physical exam is remarkable for dry mucous membranes, clear lungs, and soft abdomen with appropriate tenderness. In the 3 hours since the operating room he has made 20cc of urine. What is the next most appropriate initial step in management?
   A. Order a renal ultrasound
   B. Send a FeNa
   C. Give him a bolus of 500 cc Lactated Ringers
   D. Transfuse 2 Units PRBC
   E. Give Lasix 20mg IV times one

5. A 45 year old woman undergoes an uneventful laparoscopic cholecystectomy for which she receives one dose of a cephalosporin. One week later, she returns to the ER with fever, nausea, and copious diarrhea. Her WBC is elevated at 30,000. She undergoes a colonoscopy as part of a workup which shows colonic

pseudomembranes. After seven days of therapy with PO vancomycin, the patient still has an elevated WBC – now to 40,000. She develops hypotension requiring transfer to the ICU. What is the best course of action now?

A. Add IV Flagyl
B. Go to OR and remove affected colon (segmental)
C. Add High Dose steroids
D. Begin pressors and check serial labs
E. Perform a total abdominal colectomy

Answers

1. **A.** The immunologic response to injury involves the complement cascade, which is part of the humoral system. The complement factors, C3a and C5a, which are anaphylatoxins, induce the release of proinflammatory cytokines. IL-1 and TNFα are also key mediators of this cascade, and IL-6 and IL-8 are important for recruitment and activation of B cells and other inflammatory cells. Fever is mediated by the pyrogenic cytokines IL-1, IL-6, and TNFα. IL-10, on the other hand, is an anti-inflammatory cytokine.

2. **C.** This patient is presenting with signs and symptoms of a pulmonary embolism, which include shortness of breath, chest pain, tachycardia, and hypoxia. Patients may also have signs of lower extremity DVT, such as leg swelling and tenderness, since most pulmonary thrombi originate as emboli from the iliofemoral region. Because the symptoms of PE are nonspecific, one must have a high suspicion of it, especially in patients who exhibit the criteria outlined by Virchow's triad. For example, this patient has likely had prolonged immobility due to his recent large surgery. In addition, his malignancy has likely caused him to be in a hypercoagulable state. This patient needs CT angiography of the chest using a PE protocol to diagnose the arterial clot. However, if suspicion is high enough given the patient's history, signs and symptoms, one should not delay treatment. The treatment for a DVT or PE is usually anticoagulation with an intravenous heparin drip.

3. **C.** Postoperative pulmonary complications include atelectasis, pneumonia, inability to wean from the ventilator, bronchospasm, pulmonary edema, and worsening of preexisting lung pulmonary conditions. Pulmonary toilet, smoking cessation at least 30 days prior to elective surgery, multimodal anesthesia with epidurals, and minimization of paralysis have been shown to decrease these complications. Smoking has been shown to impair ciliary clearance in the lungs, which puts the patient at risk of developing pneumonia. Atelectasis, which is a cause of early postoperative fever, results in alveolar collapse and subsequent respiratory shunting which can cause varying degrees of hypoxemia. With persistent collapse, the lungs are prone to bacterial growth, leading to development of pneumonia. This can be prevented with lung expansion maneuvers, or pulmonary toilet, such as incentive spirometry, forced coughing, and frequent turning. Routine nasogastric tubes do not routinely reduce pulmonary complications and should only be used for patients at high risk of aspiration or for specific operative indications. Some data show that NG tubes may

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increase postoperative aspiration risk due to disruption of the lower esophageal sphincter.

4. **C.** This patient has tachycardia, mild hypotension, and oliguria (remember adequate urine output is 0.5-1.0 cc/kg/hr) with signs of dehydration on exam. This is most likely due to dehydration from his surgery given the blood loss and the long duration of the case, which can lead to a large amount of insensible fluid loss. It would be appropriate to give him a fluid challenge with a 500 cc crystalloid bolus to see if this improves his findings. If it does not, he should be further evaluated for bleeding with labs, closer monitoring, and potentially a trip back to the OR. A FeNa may eventually be helpful in differentiating prerenal AKI from renal AKI, however, given his history he almost certainly has prerenal AKI due to hypovolemia.

5. **E.** This patient has developed antibiotic-associated colitis due to Clostridium difficile infection, which typically presents with diarrhea, abdominal pain, fever, and a markedly elevated white blood cell count. C. difficile associated disease (CDAD) is classically associated with clindamycin use, but it can also occur with penicillins, cephalosporins, and ciprofloxacin. Colonic pseudomembranes seen on colonoscopy are pathognomonic for the disease. It can present with mild symptoms or it may cause severe septic shock, as with this patient, and the tenets of treatment are the same as for any case of septic shock. This patient needs timely source control, appropriate antibiotics, and she may need further resuscitation or pressor support. Source control requires a total abdominal colectomy to remove all diseased colon. Appropriate antibiotics currently include PO and rectal vancomycin and IV metronidazole (Flagyl). Of note, severe CDAD is defined as any case with a WBC >15,000 or a rise in creatinine 50% above baseline, and complicated CDAD is any case associated with hypotension, ileus, toxic megacolon, need for intensive care unit admission or colectomy, colonic perforation, lack of response to therapy. These patients require early surgical consultation. Mild CDAD is typically treated with metronidazole alone initially, while patients with severe CDAD should be started on PO vancomycin. Intravenous vancomycin is not appropriate for treatment of C. difficile colitis because it does not penetrate the GI mucosa.

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Authors/Contributors

Andre R. Campbell, MD, FACS (Section Editor)
University of California San Francisco, San Francisco, CA

Rahul J. Anand MD, FACS (Content Author)
Virginia Commonwealth University, Richmond, VA

Nina A. Wickramaratne, MD (Content Author)
Virginia Commonwealth University, Richmond, VA

Emil Petrusa, PhD (Assessment Consultant)
Massachusetts General Hospital, Boston, MA