

General Principles of Postoperative Intensive Care Unit Care

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Regionalization within a health care structure allows for more efficient control and use of limited resources. The intensive care unit (ICU) contains specially trained staff and a variety of support devices, such as mechanical ventilators, intra-aortic balloon pumps, ventricular assist devices, and dialysis machines, which in most cases cannot be used elsewhere. Optimally, the location of a patient is determined by matching the patient's needs with a location's resources and expertise.

Generally, the surgical ICU is where experience, staffing, skills, and technology converge to provide services that cannot be provided anywhere else within the hospital. Highly skilled nurses, often greater in number than the patients themselves, work intimately with intensivists and ancillary staff in an environment designed to stabilize, diagnose, and simultaneously treat the most acutely ill patients. ICU management by intensivists allows for improved staff and family satisfaction, reduced complication rates, lower costs, shorter length of stay, improved processes of care, and a morbidity and mortality risk advantage.¹⁻⁴ ICU systems focused on an environment of safety and compliance with evidence-based standards promote improvement in many outcome metrics.⁵ Safe and efficient patient throughput allows for greater institutional procedural volume, which, when paired with surgeon procedural volume, has been shown to be associated with reduced mortality risk.⁶

Classic postoperative indications for ICU admission include advanced age or prolonged duration of the operation, both criteria without specifically defined thresholds. Other factors, such as the need for mechanical ventilation,

volume resuscitation, or administration of vasoactive medications, make ICU care unavoidable. Monitoring of level of consciousness, airway, bleeding, pulses, rhythm, acidosis, urine output, and global perfusion also is facilitated by ICU admission. Identifying patients who may need postoperative ICU care can be difficult. Although there are scoring systems to assess risk and fatality (APACHE, SAPS, MPM, SOFA), it is difficult to apply these predictions to specific disease states or individual patients. Some prediction models utilize physiologic data for patients after admission to the ICU and have not been validated as preadmission screening tools.^{7,8} Physicians may predict mortality risk even better than scoring systems.⁹ In practice, most physicians do not use these tools to determine postoperative ICU admission. Admission criteria based on priority, diagnosis, and objective parameter models have been published by the Task Force of the American College of Critical Care Medicine and the Society of Critical Care Medicine.¹⁰

POSTOPERATIVE EVALUATION

Obtaining a comprehensive medical and surgical history is a fundamental step in understanding a patient in the surgical ICU. The medical record, traditionally written but now more commonly electronic, should contain all of the elements necessary to assemble the story up until the time of ICU admission, although deciphering a chart, particularly when it is long, requires time, patience, and detective skills. Data gathering usually begins by word of mouth from the

providers delivering the patient. Effective “hand-off” is essential to maintain the continuity of care and to ascertain important operative events that may have escaped documentation. It is in fact a standard expected by The Joint Commission.¹¹ Certain questions are common to virtually all admissions:

1. How old is the patient?
2. What are the highlights of the medical/surgical history?
3. Was the operation elective or emergent?
4. What operation was performed, and what are the details of the surgery?
5. Are there any drains?
6. What are the current ventilator settings if the patient is intubated?
7. What medications is the patient receiving currently?
8. Where are the vascular access points? Were they placed under sterile conditions?
9. What was the intubation and anesthetic course like?
10. What were the complications, if any?

Age, comorbid conditions, and emergency operations all affect mortality risk. The details of the operation are key, often aided by diagrams in the chart. Resections, diversions, anastomoses, transplantations, use of prosthetic materials, and other surgical findings are some of the details that should be obtained. In addition, the type and location of each drain must be accounted for. Only by knowing where a drain is placed can a care provider know how to interpret the quantity and quality of the effluent. Each drain or wire must be labeled correctly. Also, the completion of wound closure must be ascertained (skin and fascia closed?). Finally, if the operation was incomplete or intentionally staged, the health care provider needs to inquire about intentions and timing of return to the operating room.

The significance of the anesthesia record should not be minimized. The details about trends in gas exchange, blood pressure, urine output, medications, and summary fluid balance should be reviewed. Always identify if the intubation was easy or difficult. Reviewing the ventilator settings that were used in the operating room sheds some light on any possible gas exchange difficulties and provides a first opportunity to make corrections. Tidal volumes in the operating room are often much larger than those used in the ICU. Identification of current medications and the purpose of each help to formulate short-term therapeutic strategies. Assessing the adequacy of intraoperative resuscitation begins with a review of the quantifiable gains and losses. Resuscitation fluids, blood products, urine output, cavity fluid, and blood losses should all be reviewed. Evaporative and extravascular (third space) losses may be more difficult to accurately quantitate. Major surgical procedures such as bowel resection can require 7 to 8 mL/kg/hour of resuscitation fluid and severe blunt or penetrating injury 10 to 15 mL/kg/hour to match these losses. Underresuscitation may occur in patients with congestive heart failure or anuric renal failure for fear of creating a state of uncorrectable fluid overload. What amounted to adequate resuscitation in the operating room may not be the case by the time the patient arrives in the ICU. A careful reassessment of the adequacy of resuscitation is necessary in virtually all postoperative ICU admissions. Typical postoperative maintenance

intravenous fluid rates are 80 to 125 mL/hour, but can be substantially higher in the presence of ongoing intravascular volume loss. Isotonic fluids are the most appropriate maintenance fluids. It is useful to inquire about the last time the patient received narcotics, benzodiazepines, or paralytics and if reversal agents were given. Finally, any intraoperative laboratory values, particularly ones that require immediate attention, should be reviewed.

When time permits, attention should be directed back to the medical record. The clinician should scan the history and physical examination, progress notes, and consultations to develop a cohesive story line of events that led up to the operation. Did the illness have an impact on nutrition or functional state? How are other comorbid conditions or past operations related to the current presentation? The past medical history and the medication list should be scrutinized; the two are complementary. Inclusion of a disease in the past medical history and absence of an expected medication warrants further investigation (and vice versa). The medication list should be scanned in particular for anti-seizure medications, bronchodilators, antihypertensives, antiarrhythmics, anticoagulants, diuretics, steroids, thyroid replacement, and insulin. It must be decided which medications must be continued in the immediate postoperative period and which can be temporarily delayed. If antibiotics were administered preoperatively, the clinician should identify what they were and how long had they been given and for what indication. In general, if administered preoperatively, bronchodilators, steroids, and insulin are resumed postoperatively. Long-acting antihypertensives should be avoided in the early postoperative period, and short-acting intravenous agents should be used to control hypertension. Diuretics should be avoided in the immediate postoperative period unless directed by invasive monitoring or required because of some other medical necessity. The use of early postoperative beta blockade in patients with coronary artery disease is encouraged if the overall hemodynamic performance allows. Most other medications can be safely delayed until the postoperative patient has shown satisfactory cardiopulmonary performance and stability.

Postoperative laboratory, imaging, and electrocardiogram studies should be selected on a case-by-case basis. Patients who have been moved from operating room table to bed and then transported for any distance are at risk for displacement of tubes and catheters. The admission chest radiograph allows for the evaluation of intravascular catheter and endotracheal, nasogastric, and thoracostomy tube positions in addition to visualization of the pleural, mediastinal, and parenchymal structures. Measurements of blood counts and chemistries are usually routine, but may be deemed unnecessary if preoperative or intraoperative values were unremarkable and the operation was uneventful. Laboratory abnormalities should be followed closely until a favorable trend is established. Patients at risk for perioperative myocardial injury or with new intraoperative arrhythmias should have an electrocardiogram and possibly cardiac enzyme determination.

The physical examination of the patient completes the initial postoperative evaluation. It starts as a cursory survey and concludes as a detailed examination. The examination should expose all parts of the patient that can be accessed, and the examiner should inspect and palpate the patient.

Box 35.1 Support for Adequate Clinical Perfusion

Mean arterial blood pressure >70 mm Hg
 Heart rate <100 beats/minute
 Warm, pink skin without cyanosis or mottling over the digits, thighs, or knees
 Palpable pulses
 Good capillary refill
 Clear yellow urine >0.5-1 mL/kg/hour

Areas that are not under examination should be kept covered to preserve body temperature. If the bed sheets are being changed, it presents an opportunity to examine the back of the patient. An initial assessment of the vital signs, skin, pulses, and urine output provides preliminary insight into clinical perfusion (Box 35.1).

The endotracheal tube, if present, needs to be secured adequately. The health care provider should listen for obvious air leaks around the cuff. The presence of nasal or oral gastric tubes should be noted. All drainage tubes should be identified, and the quality and quantity of output should be scrutinized: Is it serous? Sanguineous? Bilious? Drainage from raw, inflamed surfaces is often serosanguineous. Frankly bloody drainage in quantities of more than 100 mL/hour suggests either surgical bleeding or coagulopathy. All intravascular catheters should be identified with the goal of determining which should be retained for use and which should be removed. Diagnostic catheters often remain unnoticed, and unused, particularly when in femoral vessels. Intravenous catheters not placed under sterile conditions should be removed immediately.

The neurologic examination may be suboptimal if the patient is still under the effects of anesthesia. Reducing or temporarily withholding narcotics and sedation can provide a window to complete a neurologic assessment. If further analgesia or sedation is still required, it may be resumed after the neurologic assessment. However, withholding sedation should not be done in the early postoperative course if it results in a state of competition with care (severe agitation, inability to oxygenate/ventilate, hemodynamic instability).

Intubation, general anesthesia, and mechanical ventilation can result in a variety of airway or parenchymal injuries. Breath sounds should be equal bilaterally. Asymmetry can be caused by atelectasis (possibly endotracheal tube malposition), pleural effusions, or pneumothorax and can be excluded by careful review of the chest radiograph. Examination of the respiratory system should include evaluation of thoracostomy tubes and the mechanical ventilator if present. Except in the case of pneumonectomy, thoracostomy tubes should be placed to suction pending demonstration of sustained lung inflation or resolution of significant drainage. The mechanical ventilator settings and airway pressures should be noted. Adjustments to mechanical ventilation may need to be made to accommodate shivering, metabolic abnormalities, and hypoxia in the early postoperative period. The clinician should ensure satisfactory initial oxygen saturation and avoid excessive tidal volumes. End-tidal carbon dioxide monitoring facilitates adjustment in ventilation and progress in weaning. Routine blood gas

analysis is unnecessary but will be required to manage the more challenging derangements in gas exchange and acid/base disorders.

The cardiovascular examination is primarily directed at assessment of adequate clinical perfusion. Impressions from the initial survey of clinical perfusion plus any available data from invasive monitoring can be used to assess appropriate hourly maintenance fluid rate and the need for further volume resuscitation. Cardiac surgery patients may have mediastinal drains and pacing wires. The former should be connected to suction, and the quantity and quality of drainage should be scrutinized. Pacing wires should be tested for function on admission and can be capped if pacing is not needed. If a postoperative patient comes to the ICU with a permanent pacemaker or an implantable cardiac defibrillator, the device should be interrogated for mode and function at the earliest convenience.

In contrast to the lungs and heart, which can be imaged easily and whose function can be monitored objectively, the abdomen and its contents cannot be evaluated handily. The persistence of anesthesia or administration of narcotics can remove many of the signs and symptoms typically relied on to signal problems. Examination should focus on baseline location and quantity of pain, presence of abdominal distention, firmness to palpation, and quality and quantity of effluent from drains. Bleeding and progressive visceral edema can cause a rapid distention and loss of compliance of the abdomen, often before other findings occur, such as reduction in hemoglobin concentration, urine output, and blood pressure. Frequent follow-up examinations compared with baseline data may be the earliest way of recognizing an intra-abdominal catastrophe. The practitioner should be alert to abdominal distention with associated changes in clinical perfusion (such as low urine output) as a marker of abdominal compartment syndrome. Measurement and trending of bladder pressures can supplement other clinical findings in guiding decision making.

Knowing where the tip of each abdominal drain lies is necessary to evaluate the effluent. A drain lying outside the bowel or biliary system should not drain succus or bile. A drain that suddenly shows these fluids may herald loss of integrity of a surgical repair or de novo perforation. Unexplained or unexpected changes in the quantity of effluent from a drain also are notable.

Abdominal wounds are not always closed at the end of an operation. The clinician needs to determine if the skin or fascia has been left open and, if so, what kind of temporary closure is employed. If a temporary abdominal closure device is used, the quality and quantity of effluent from that device should be examined and documented. It is important to remember that temporary abdominal closure devices are not proof against abdominal compartment syndromes. The provider should be prepared to loosen the outer layers of an abdominal closure or dressing to provide temporary relief. Surgical or traumatic wounds, regardless of location on the body, should be examined for closure integrity, erythema, and induration.

Examination of pulses is important after vascular surgical procedures. Scheduled reassessments should document the presence and strength of pulses. Sudden reduction or loss of pulse signal can represent proximal vascular occlusion, a distal outflow obstruction, or increase in compartment

pressures. Baseline cyanosis and mottling of extremities should be noted for subsequent comparison. In addition, a clinical examination (palpation) of the compartments should be performed to provide the practitioner a baseline for further comparison. Should the mechanism of injury increase the risk of muscle swelling and compartment syndromes, the practitioner can utilize invasive monitoring to measure compartment pressures, and should be prepared to pursue extremity fasciotomies.

Evaluation of a postoperative trauma patient in the ICU can be restricted by the presence of dressings and immobilizing casts and neck collars. Sometimes only toes or fingers are visible for examination. Postoperative admission to the ICU is a good opportunity to look for injuries missed during the initial evaluation and management period. In addition, the practitioner should be alert to potential iatrogenic injuries from intraoperative events; this would include electrical burns from ungrounded cautery circuits, infiltrated intravenous lines, and compression injuries from positioning in the operating room.

RECOVERY FROM ANESTHESIA

POSTOPERATIVE RESUSCITATION

ASSESSMENT

“Adequate resuscitation” is a state, often temporary, that allows for good clinical perfusion and physiologic stability. Patients with good clinical perfusion (expected heart rates, blood pressures, and urine outputs; absence of acidosis) may require no further resuscitation other than maintenance intravenous fluids. The correct maintenance fluid rate will be just enough to match intravascular losses out of proportion to that which is mobilizable from the interstitium but not so much as to needlessly expand the third space or interstitium with edema. Subtle abnormalities in any of these parameters of perfusion may suggest a more serious physiologic derangement warranting further investigation and intervention. Resuscitation is the process of optimizing macroscopic and microscopic metabolic substrate delivery with the goal of avoiding an imbalance between supply and demand. The most fundamental concept is to ensure adequate oxygen delivery (DO_2) and meet the oxygen consumption ($\dot{V}O_2$) needs of tissues and organelles. Because the moment when $\dot{V}O_2$ exceeds DO_2 is difficult to determine, resuscitation “targets” serve as proxy markers of adequate DO_2 . Resuscitation targets are reproducible, quantifiable values, such as pressures, outputs, metabolites, inflammatory mediators, or oxygen saturations, which represent therapeutic goals. Resuscitation targets provide an important opportunity for study and outcome validation. Despite the seemingly simple logic of employing resuscitation targets, few of these therapeutic goals have been shown to improve clinical outcome. Even routine data derived from a pulmonary artery catheter have not been shown to improve outcome in patients undergoing surgery with decompensated cardiogenic shock or acute lung injury.^{12,13}

MANAGEMENT THEORY

Evaluation and optimization of blood pressure, filling pressures, DO_2 , heart rate, and rhythm often occur

simultaneously, particularly in unstable patients (Fig. 35.1). This may require ongoing volume resuscitation and support with vasopressors and inotropes. Restoration of “normal” blood pressure, heart rate, and urine output, however, do not ensure adequate DO_2 at the level of the microvasculature.¹⁴ Overzealous resuscitation and supranormal DO_2 not only do not improve outcome but also may be detrimental.¹⁵ Not all patients require the same type of resuscitation. Although the fundamental principles are the same, the particular resuscitation technique end points may differ among the different types of shock.^{16,17} Crystalloid resuscitation may be appropriate in septic shock but detrimental in the early resuscitation of penetrating traumatic injury.^{18,19} Even low-volume resuscitation plays a role in the management of patients with penetrating traumatic injury or severe intraoperative hemorrhage.²⁰ Early goal-directed therapy with parameter-specific targets has not completely survived prospective validation. However, the principle of timely intervention remains a cornerstone for virtually all types of resuscitation. End points specific to particular mechanisms of injury can vary significantly.²¹⁻²³

Targeted resuscitation strategies provide an orderly approach to resuscitation, monitoring, and outcome validation. In general, such strategies optimize cardiovascular performance and concurrently measure markers of adequate global DO_2 and $\dot{V}O_2$. Increased serum lactate concentration, decreased mixed venous oxygen saturation, and decreased central venous oxygen saturation are the proxy markers for inadequate global DO_2 . However, normal values of mixed venous oxygen saturation and central venous oxygen saturation do not guarantee normal use of oxygen in the tissues, particularly at the regional level. Appropriate targets for microcirculatory resuscitation remain elusive. Noninvasive techniques have reduced the need to obtain physiologic data by the use of a pulmonary artery catheter.²⁴ Pulse and pressure wave analysis along with their derivatives (cardiac output and stroke volume variation) offer a less invasive way of measuring hemodynamic performance and predict volume responsiveness in the appropriate patient population.²⁵ Gastric tonometry, sublingual capnography, near-infrared spectroscopy, and orthogonal polarization spectral imaging are less mainstream technologies available to assess the effectiveness of resuscitation at the regional level.²⁶

Resuscitation products should target the intravascular components that are inadequate, including red blood cell concentrates, platelets, coagulation factors, and acellular resuscitation fluids. Fluid type, bolus volume, and maintenance rate must be individualized. The optimal resuscitation fluid effectively should expand the intravascular space and minimize the inflammatory response (particularly in hemorrhagic shock^{27,28}). All resuscitation fluids leak to some degree out of the intravascular space into the interstitium of the extracellular space. Hypotonic resuscitation fluids are inappropriate for volume resuscitation because of their inability to remain exclusively in the extracellular space. Volume per volume, hypertonic fluids cause more intravascular expansion than isotonic fluids. Hypertonic fluids yield no better outcomes than isotonic crystalloids, however, in the resuscitation of trauma patients.²⁹ Similarly, isotonic crystalloids are at least as efficacious or may be better than colloids to reach the same end points.¹⁴ In trauma, burn,

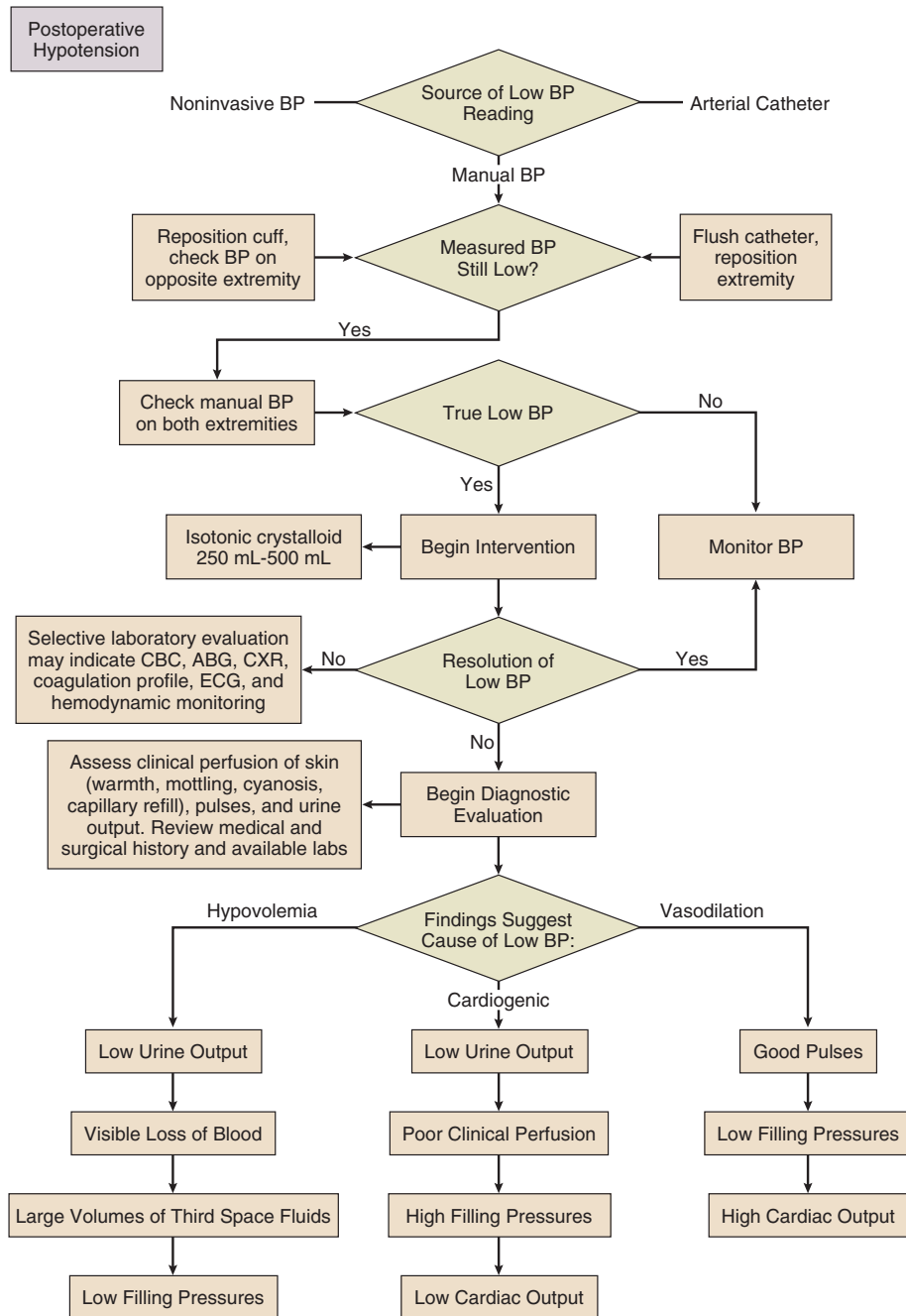


Figure 35.1 Approach to managing postoperative hypotension. ABG, arterial blood gases; BP, blood pressure; CBC, complete blood count; CXR, chest x-ray study; ECG, electrocardiogram.

and general surgery patients, resuscitation with colloids, as compared to crystalloids, has not been shown to reduce the risk of death.³⁰

Metabolic consequences are associated with virtually all resuscitation fluids. Ringer’s lactate can activate neutrophils and cause a potent inflammatory response.³¹ Hypertonic saline and dextran combinations cause less of an inflammatory response but any mortality benefit is unproved.^{32,33} Greater than 1 L of hypertonic saline typically results in the development of hypernatremia. Resuscitation exclusively with isotonic NaCl results in a hyperchloremic acidosis. Recent literature has suggested that hetastarch is associated

with greater adverse events when compared to saline resuscitation.³⁴ Hetastarch can cause coagulopathy if greater than 1.5 L is given. All acellular resuscitation fluids, if given in sufficient quantities, cause dilutional anemia. As one can infer from this confusing and sometimes contradictory collection of recommendations, no single resuscitation fluid is satisfactory on its own.

TEMPERATURE CONTROL

Postoperative patients can come to the ICU with moderate to severe hypothermia. Heat is lost in the operating room as a result of vasodilation from volatile anesthetics, cool

intravenous fluids and air temperature, large open surfaces, and evaporation. Excluding patients with potentially anoxic central nervous system injuries,³⁵ hypothermia complicates initial postoperative care by creating an *in vivo* coagulopathy, even when *in vitro* coagulation studies (normalized to 37°C) are normal. In trauma patients, reduction in enzyme activity and platelet function, leading to abnormal fibrin polymerization, occurs at temperatures less than 34°C.³⁶ Care must be taken when administering large volumes of cold blood products or even room temperature crystalloids. Fluid warming devices are available not only to prevent but also to treat hypothermia. All patients with postoperative hypothermia less than 36°C should be actively warmed with forced air blankets, and when normothermia has been achieved, patients should be kept covered to prevent heat loss. Active warming does not cause peripheral vasodilation and subsequent hypotension, and it does not paradoxically cause core cooling owing to heat exchange in cold extremities.

AWAKENING FROM ANESTHESIA

Before completing a successful resuscitation, sedation, analgesia, and anxiolysis should be maintained to facilitate patient comfort and to prevent interference with medical care (e.g., mechanical ventilation or motor activity jeopardizing airway, drains, and intravenous catheters). Selected agents should have minimal hemodynamic sequelae and relatively short duration of action so that frequent neurologic assessment can be performed. Daily interruption of continuous sedation has been shown to reduce ICU length of stay, duration of mechanical ventilation, and incidence of posttraumatic stress disorder.^{37,38}

Narcotics such as fentanyl, morphine, and hydromorphone make ideal first-line analgesics. Delivered by continuous infusion and supplemented as needed, successful analgesia reduces pain-driven tachycardia and hypertension and facilitates cough and deep breathing. The sensation of anxiety is a potent dysphoric stimulus that can result in restlessness and interfere with care. Anxiety can be treated with short-acting intravenous benzodiazepines, such as lorazepam. Very short-acting benzodiazepines, such as midazolam, are less useful because of the dosing frequency necessary to prevent symptoms from returning. It is important not to use scheduled benzodiazepines to treat restlessness due to delirium. This practice can exacerbate delirium and worsen outcomes. Delirium can be identified using simple evaluation tools such as the Confusion Assessment Method for the ICU (CAM-ICU). Competitive restlessness due to delirium is best managed with atypical antipsychotics such as haloperidol, ziprasidone, and quetiapine.³⁹ Persistent restlessness, agitation, or delirium can compete with mechanical ventilation, confound hemodynamic stability, and impede the provision of care. If further reduction of level of consciousness is necessary, propofol or dexmedetomidine can be added and titrated to desired effect. Dexmedetomidine, a weak analgesic, can reduce narcotic requirements.⁴⁰ Propofol, however, has no intrinsic analgesic properties. In a patient who has serious pain, neither propofol nor dexmedetomidine should be used without the concurrent administration of a narcotic. The use of most agents mentioned can be limited by their

tendency to reduce blood pressure and, in the case of dexmedetomidine, decrease heart rate.

When patients are resuscitated adequately, consideration can be given to awakening from residual sedation. On arrival to the surgical ICU or recovery room, unconsciousness, if present, is due to the residual effects of volatile anesthetics, narcotics, benzodiazepines, and paralytics. The effects of volatile anesthetics can persist for 20 to 60 minutes after their discontinuation, particularly if the agent is fat-soluble, the patient is obese, and the surgery was long. Paralytics can have longer than expected duration of action, and this should be suspected when a postoperative patient remains very weak (cannot perform a 10-second head lift) or does not move. A train-of-four twitch monitor can address this issue. Persistent chemical paralysis can be reversed with neostigmine and glycopyrrolate.

Reentry into consciousness may be accompanied by disorientation, anxiety, pain, and varying degrees of restlessness. In the absence of underlying encephalopathy, it is usually possible to get patients to follow commands, answer questions, and participate in the extubation process. The discomfort of an endotracheal tube can lead to unplanned self-extubation. It is important for the bedside care provider to maintain control of the recovery process by ensuring analgesia and anxiolysis. Small doses of narcotic or benzodiazepine or both can usually correct these problems without inducing further sedation and delay of extubation.⁴¹ Patients with encephalopathy resulting from sepsis or shock may not recover a level of consciousness that allows participation in the weaning process. It is controversial whether such a patient should be extubated (avoiding the complications of prolonged extubation) or remain intubated until the ability to protect the airway is more certain. Dexmedetomidine can reduce restlessness without respiratory suppression and may be useful to facilitate extubation of a restless patient. Patients who require sedation for an extended time should receive doses of medication no higher than necessary to achieve the therapeutic target. Sedation scales, such as the Ramsay and Richmond Agitation Sedation Scale,⁴² are useful to avoid oversedation and ultimately promote earlier liberation from mechanical ventilation.

POSTOPERATIVE EXTUBATION

Liberation from mechanical ventilation requires clinical readiness to begin weaning and demonstration of adequate physiologic reserve before extubation. Clinical readiness assesses completion of perioperative tasks at hand and questions any need for early return to the operating room. Resuscitation should be complete, hemostasis should be achieved, metabolic acidosis should be resolving, vasoactive support and gas exchange abnormalities should be minimized, anesthetic agents should be cleared, the ability to protect the airway should be present, and the patient should be awake and reasonably cooperative. These criteria have not been validated clinically, but similar consensus guidelines have been published.⁴³ Daily, if not more frequent, reassessment of clinical readiness is necessary to determine if it is reasonable to consider weaning.⁴⁴

Patients who are ready clinically to progress to extubation should have an assessment of physiologic reserve. Having the patient breathe without mechanical assistance

allows observation of respiratory rate, mechanical coordination of chest and abdomen, vital signs, end-tidal carbon dioxide concentration, and subjective comfort. If the patient was not mechanically ventilated preoperatively, the perioperative course has been uneventful, the patient is comfortable with stable vital signs, no tachypnea or respiratory muscle dyssynchrony is present, and there is no short-term plan to return to the operating room, the patient should begin spontaneous breathing trials and be evaluated for extubation.

Patients who do not achieve these basic criteria may require continued mechanical ventilation that maximizes patient comfort and unloads the respiratory muscles. These patients require a structured, evidence-based approach to ventilator weaning and assessment of adequate physiologic reserve. For more detailed information on weaning, refer to Chapter 43.

BEST PRACTICES

Achieving optimal outcomes should be pursued by providing optimal care. This is especially true for patients with longer length of stay. Effort should be expended pursuing interventions that have been shown to reduce complications, cost, morbidity, and mortality risk. Because a postoperative ICU patient is different in many ways from other ICU patients, some of these fundamental practices are applied with slight nuance and warrant additional mention.

PREVENTION OF VENOUS THROMBOEMBOLISM AND DEEP VENOUS THROMBOSIS

All postoperative ICU patients should be considered for venous thromboembolism (VTE) or deep venous thrombosis (DVT) prophylaxis. The risk of postoperative VTE depends upon both the type of procedure and modifying attributes such as age, prior VTE, history of cancer, obesity, or hypercoagulable state. Risk has been quantified and grouped based on the Modified Caprini Risk Assessment Model.⁴⁵ Low-risk general and abdominal-pelvic surgery patients should receive intermittent pneumatic compression (IPC) over no prophylaxis or anticoagulant-based prophylaxis. Moderate-risk general and abdominal-pelvic surgery patients should receive anticoagulant-based prophylaxis. Low-dose unfractionated heparin, low-molecular-weight heparin, or fondaparinux should be started in the absence of postoperative bleeding. High-risk general and abdominal-pelvic surgery patients should receive low-dose unfractionated heparin three times a day, low-molecular-weight heparin, or fondaparinux. The highest risk patients should receive mechanical prophylaxis via IPC devices, in addition to low-dose unfractionated heparin, low-molecular-weight heparin, or fondaparinux. In general surgery patients with a high risk of postoperative bleeding, mechanical prophylaxis should be the initial preventive modality until the risk of bleeding has decreased enough to allow for anticoagulant prophylaxis.⁴⁶

Neurosurgical procedures or the use of neuraxial analgesia also require special consideration. Anticoagulant prophylaxis should not be in effect while epidural catheters are placed or removed and should be used with caution while an epidural catheter is in place. Patients

undergoing intracranial surgery should receive mechanical prophylaxis with sequential compression devices. Anticoagulant prophylaxis should be added in neurosurgical patients at high risk for VTE/DVT beginning 24 hours postoperatively.

Trauma patients constitute an extremely heterogeneous group, making it difficult to study the strategies of VTE/DVT prophylaxis. There is disagreement in the literature about valid independent risk factors for VTE/DVT in trauma patients. Older age, spinal fractures, spinal cord injuries, traumatic brain injuries, prolonged mechanical ventilation, pelvic fractures, venous injuries, and multiple major operative procedures are often cited. In trauma patients, there are few large, prospective, randomized studies validating the efficacy of any method of VTE/DVT prevention.⁴⁷ Low-dose unfractionated heparin, which has proven efficacy in the general surgery population, is no better than absence of prophylaxis in a trauma patient.⁴⁸ Low-molecular-weight heparin given twice daily does offer a statistical benefit, however, in the prevention VTE/DVT in trauma patients.⁴⁹ Trauma patients without significant risks for bleeding should begin anticoagulant prophylaxis or postoperatively. Data are insufficient to make recommendations as to when anticoagulant prophylaxis in trauma patients with brain injury or liver or spleen fracture is safe. Waiting 24 hours after bleeding has ceased is a conservative time to delay.⁵⁰ In trauma patients at high risk for bleeding, mechanical prophylaxis can be used, although benefit is unproved. Note that IPCs cannot be applied to lower extremities with fractures, fasciotomies, or external fixators. Compression devices applied to the feet may be used as a substitute for IPC but have not been shown to be as efficacious as leg devices. In selected trauma patients expected to have prolonged immobilization or with significant risks for bleeding, inferior vena cava filters may be placed as VTE prophylaxis.³⁰ Inferior vena cava filters should not be used as a primary prophylactic strategy in trauma patients.²⁹ If available, removable filters should be considered, despite the low removal rates. In a trauma patient at high risk for VTE/DVT, the addition of mechanical prophylaxis to anticoagulant prophylaxis may be useful, but synergistic benefit is unproved.

STRESS ULCER PROPHYLAXIS

Stress-related mucosal disease (SRMD) is manifest as diffuse gastric mucosal petechiae, erosions (loss of epithelium, necrosis, and hemorrhage), and discrete ulcers. SRMD can progress to clinically significant bleeding resulting in hemodynamic instability and need for transfusion. It can develop as early as 24 hours after ICU admission. Patients at risk for SRMD include critically ill patients who require mechanical ventilation for greater than 48 hours; patients with coagulopathy, traumatic brain or spinal cord injury, or severe burns; and patients with a history of gastrointestinal bleeding or ulceration within the past year. Minor risks include sepsis, corticosteroids, and prolonged ICU admission.⁵¹ The risk of clinically significant bleeding increases with the severity of illness, duration of mechanical ventilation, increased length of stay, and low intragastric pH. Hemodynamic compromise secondary to acute blood loss occurs in only a small percentage of patients with SRMD, but it is associated with a significantly increased mortality rate.⁵²

Because of the morbidity and mortality rates associated with the complications of SRMD, it is important to identify patients at risk for SRMD and employ effective prophylaxis before bleeding occurs. Although early enteral nutrition has many benefits, the effects of enteral nutrition on SRMD are controversial and should not be used as a sole prophylactic strategy.⁵³ Pharmacologic prophylaxis targets mucosal protection or the suppression of acid secretion. Proton-pump inhibitors may be a good first choice for SRMD prophylaxis owing to degree of acid suppression, duration of action, lack of tolerance, and cost. Parenteral H₂ receptor antagonists may offer a cost advantage over proton-pump inhibitors. Prophylaxis with sucralfate is not preferred because of the efficacy profile of acid-suppression therapies and a higher rate of bleeding with sucralfate prophylaxis.

PREVENTING NOSOCOMIAL PNEUMONIA

The most significant risk for hospital-acquired pneumonia (HAP) in the postoperative patient is mechanical ventilation. Other significant risks include age more than 70 years, chronic lung disease, and depressed levels of consciousness. Though gastric acid suppression is also associated with an increased incidence of HAP, withholding ulcer prophylaxis can hardly be avoided in the patient mechanically ventilated for more than 48 hours.⁵⁴ Postoperative patients should be encouraged to take deep breaths, cough, ambulate, and use incentive spirometry. Semirecumbent body positioning, keeping the head of bed elevated more than 30 degrees, has been shown to reduce ventilator-associated pneumonia in mechanically ventilated patients.⁵⁵ Placing the bed in reverse Trendelenburg position can simulate this elevation without flexing the back, as could be difficult in trauma patients or patients with large open abdomens. Iatrogenic spread of bacteria that can cause pneumonia can be reduced by the enforcement of handwashing and by the use of appropriate barrier protection when performing procedures.⁵⁶ Before deflating the cuff of an endotracheal tube for tube removal or position change, ensure that secretions are suctioned clear from above the cuff.³⁶ Endotracheal tubes designed to provide drainage to the subglottic area above the tube's cuff have been shown to reduce the risk of ventilator-associated pneumonia.^{57,58} The use of 0.12% chlorhexidine oral rinse has been associated with reductions in the rate of ventilator-associated pneumonia in surgical ICU patients and should be part of good oral hygiene.⁵⁹ Although there is evidence that selective digestive decontamination beyond the oropharynx also can reduce the risk of ventilator-associated pneumonia, it is unclear how the routine use of this technique would affect antimicrobial resistance.⁶⁰ The use of noninvasive ventilation in patients with exacerbations of chronic obstructive pulmonary disorder and congestive heart failure is associated with reductions in rates of nosocomial pneumonia, but there are few studies evaluating application of this technique in the management of postoperative respiratory failure.⁶¹

MANAGEMENT OF AGITATION AND DELIRIUM

Delirium is a major problem in postoperative ICU patients.⁶² Previously believed to be an expected and unavoidable result of critical illness that resolves with clinical improvement, it is now known to be a significant marker of increased

morbidity,⁶³ resource use, and long-term cognitive deficit. Delirium is an acute, variable change in mental status with inattention and either altered level of consciousness or disorganized thinking. Delirium can be hypoactive or hyperactive, the majority of patients being in the former group. Occurring in about 70% to 80% of ICU patients, delirium had been underdiagnosed until validated assessment tools such as the CAM-ICU became available.⁶⁴ Delirium is believed to be due to imbalances between the stimulatory and inhibitory neurotransmitters, particularly an increase in dopaminergic and decrease in γ -aminobutyric acid and cholinergic activity. Risk factors include age, preexisting dementia, sepsis, metabolic abnormalities, and medications. The use of benzodiazepines, narcotics, anticholinergics, and antipsychotics is associated with a substantial increase in risk. It is currently unclear whether prevention or treatment of delirium changes clinical outcomes such as fatality and long-term cognitive deficits.

Preventive strategies include avoidance of hypoxemia (Fig. 35.2), correction of metabolic disturbances, and adequate pain control. In addition, environmental normalization with minimization of unnecessary physical and auditory stimulation, restoration of sleep/wake cycles, frequent reorientation (particularly with family involvement), and early mobilization can help decrease rates of ICU delirium.⁴² Pharmacologic treatment of delirium is suboptimal because the same medications intended to reduce disorganized thought may simultaneously increase sedation, prolonging the undesired state. Benzodiazepines may aggravate disorganized thought and should not be used to treat delirium. Haloperidol is the most commonly prescribed neuroleptic to treat delirium,⁶⁵ although its efficacy is yet to be validated. Other atypical antipsychotics such as olanzapine, quetiapine, ziprasidone, and risperidone have also recently gained popularity.^{66,67} Until efficacy of any pharmacologic intervention is shown, medications should be used in the lowest doses possible for as brief a time as possible.

MANAGEMENT OF BLOOD GLUCOSE LEVEL

Hyperglycemia in a critically ill patient can be due to diabetes mellitus (established or new) or stress-induced release of counterregulatory mediators. It is associated with increased mortality risk after acute myocardial infarction, stroke, and severe traumatic brain injury. Hyperglycemia also is associated with reduced functional outcome after neurologic injury, the development of polyneuropathy in critically ill patients, increased rates of infectious complications in the postoperative period, and defective collagen formation in wound healing. Earlier studies⁶⁸⁻⁷⁰ about the benefits of intensive insulin therapy had been published touting improved outcomes, but more contemporary evidence has shown results to the contrary.

Blood glucose less than 60 mg/dL occurs up to 32% of the time when intensive insulin strategies are utilized. Hypoglycemia can have a negative impact on mortality risk and neurologic outcome.⁷¹ Identification of appropriate blood glucose target ranges and management techniques has required the prospective study of thousands (NICE-SUGAR)⁷² of medical and surgical patients. From this data, and other meta-analyses, we can make some observations and logical management recommendations. Intensive insulin treatment, targeting a blood glucose of 80 to

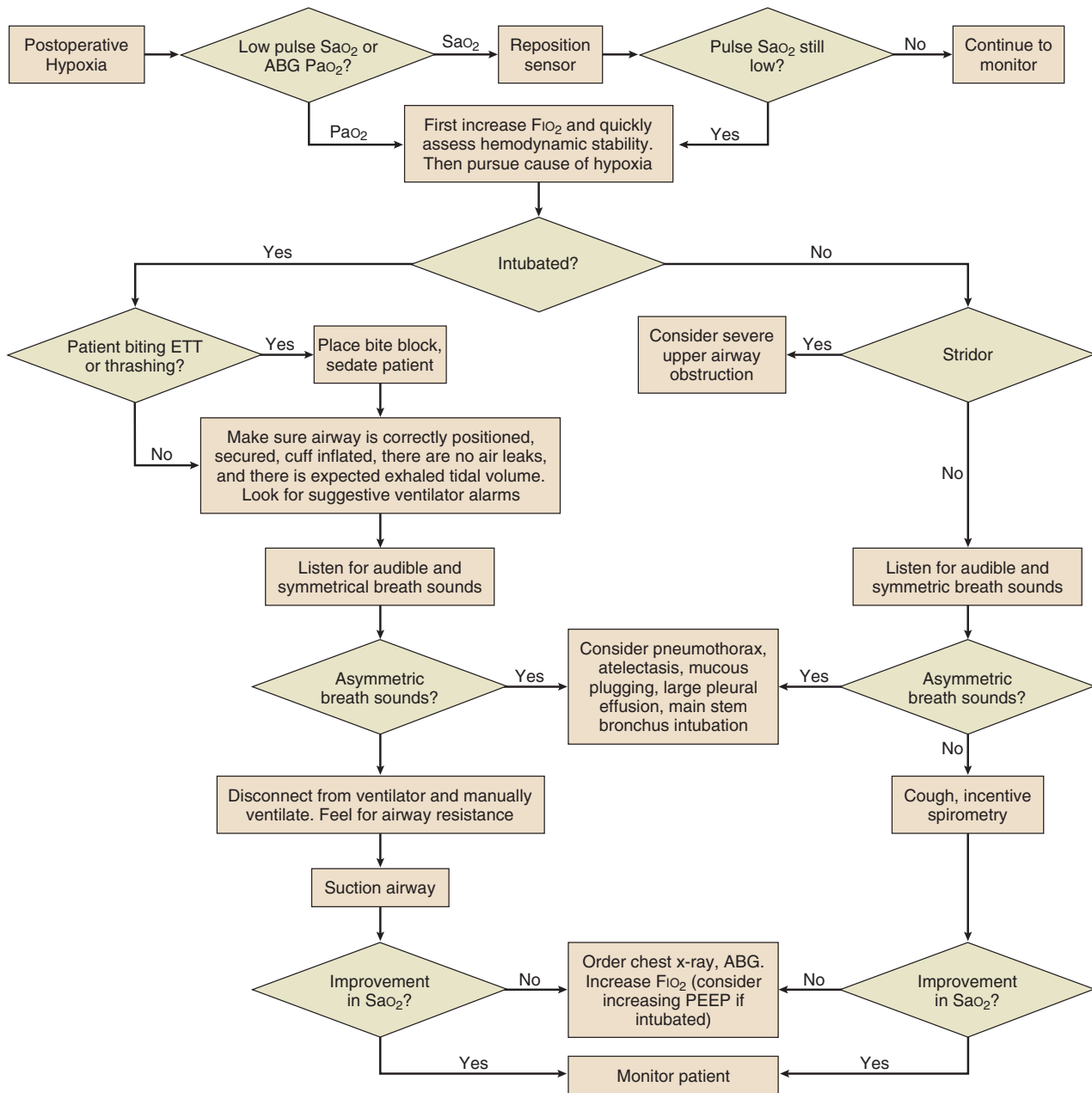


Figure 35.2 Approach to managing postoperative hypoxemia. ABG, arterial blood gas; ETT, endotracheal tube; FiO_2 , fraction of inspired oxygen; PaO_2 , arterial oxygen tension; PEEP, positive end-expiratory pressure; SaO_2 , arterial oxygen saturation.

110 mg/dL, increases the incidence of episodes of severe hypoglycemia and either has no effect on mortality risk or increases mortality risk when compared to more liberal blood glucose target ranges of 140 to 180 mg/dL and 180 to 200 mg/dL.

A rational approach to the management of blood glucose begins with minimizing causes of hyperglycemia such as unnecessary dextrose in intravenous fluids, choosing appropriate balances of carbohydrates and fats in the diet, and avoidance of overfeeding. Blood glucose targets in the 140 to 180 mg/dL range and an insulin regimen that minimizes hypoglycemia appear to be the most beneficial strategy.

Unreliable subcutaneous absorption, extreme or labile hyperglycemia, and inconsistent caloric intake are reasons to use short-acting, continuous intravenous insulin rather

than slower-onset, longer-acting subcutaneous insulin. The treatment of hypoglycemia also needs to be considered an urgent therapy.

POSTOPERATIVE NUTRITION

Postoperative surgical patients are exposed to unique nutritional challenges as a result of the enhanced metabolic demands of wound healing and the abnormalities of bowel motility, anastomotic function, and swallowing. Nutritional support provides calories for metabolic processes, reduces catabolism of protein stores as an energy source, supplies substrate for anabolic processes, and provides an opportunity to reduce net protein losses in the face of ongoing

protein catabolism. In an otherwise well-nourished postoperative patient, beginning nutritional support may be unnecessary, unless it is anticipated that oral intake at nutritional goal would be delayed for 7 days.⁷³ There are considerably fewer studies showing nutritional support strategies that work in the postoperative patient than ones that do not work.⁷⁴

TIMING AND ROUTE

There are three routes of nutritional support—enteral nutrition (including nasogastric tube or postpyloric tube), parenteral nutrition, and oral feedings. With respect to outcomes, it is important to consider not only the route of administration but also the timing. Neither enteral nutrition nor parenteral nutrition seems to have an effect on mortality rates whether given preoperatively or postoperatively.⁷⁵ Preoperative nutritional support seems to benefit only severely malnourished patients by reducing complication rates.^{76,77} Parenteral nutrition, which requires vascular access, is associated with complications related to non-catheter-related infection and catheter-related bloodstream infection. In addition to avoiding the complications associated with parenteral nutrition, enteral nutrition possibly reduces gut mucosal atrophy and up-regulates gut-associated immunity. In theory this protects against infections elsewhere by the common mucosal immune hypothesis.⁷⁸ In perioperative patients, sufficient evidence is lacking, however, to suggest that the effect of enteral nutrition on the gut barrier has any outcome advantage over parenteral nutrition.^{79,80} Enteral nutrition has been shown to be associated with a lower risk of infection compared with parenteral nutrition.⁸¹ Early enteral nutrition also has been shown to be associated with a shorter length of stay and lower incidence of infections compared with delayed enteral nutrition.⁸² Enteral nutrition is the preferred route over parenteral nutrition because of the reduction in complications and cost. Early postoperative parenteral nutrition does not improve clinical outcomes and should be reserved only for patients who are unable to receive timely enteral nutrition.⁸³

The combination of parenteral nutrition and early enteral nutrition has no advantage over early enteral nutrition alone in patients who are not malnourished.⁸⁴ Patients who are malnourished or are not expected to be tolerating enteral feedings at nutritional goal by about postoperative day 7 should begin parenteral nutrition. If otherwise adequately nourished postoperative ICU patients are expected to be tolerating enteral feedings at nutritional goal by postoperative day 7, early parenteral nutrition may not provide substantial benefit. Finally, patients who are able to tolerate enteral feedings but are unable to tolerate an amount equal to the nutritional goal require supplemental nutrition, typically parenteral nutrition.

When the decision is made to deliver enteral nutrition, tube feedings should be increased quickly in volume to reach nutritional goal. The initial destination for enteral nutrition is the stomach. Nothing about laparotomy itself precludes enteral nutrition with the return of bowel function (e.g., bowel sounds, flatus). Although bowel motility continues through surgery or returns shortly thereafter, gastroparesis is common postoperatively and may result in

delayed gastric emptying. It may be recognized by abdominal distention, high daily nasogastric output (>500 mL/day), or high residual volume in the stomach (>300 mL). Gastroparesis has the potential to delay achieving delivery of adequate enteral nutrition and has resulted in a trend toward delivering enteral nutrition via a postpyloric route. There are recent data to suggest that postpyloric feedings in patients with severe traumatic brain injury reduces the incidence of overall and late pneumonia and in addition improves nutritional efficacy.⁸⁵ Other data suggest that there is no clinical benefit to postpyloric feeding with respect to incidence of pneumonia, ICU length of stay, mortality rate, or time to reach nutritional goal compared with the prepyloric route.⁸⁶ Evidence to demonstrate the clinical benefit of postpyloric to prepyloric feedings is possibly still equivocal. Gastroparesis often can be improved with prokinetic agents, such as metoclopramide or erythromycin.⁸⁷ It is reasonable to continue gastric enteral nutrition in the presence of gastric residual volumes of 150 to 300 mL as long as the patient is not experiencing nausea, vomiting, or progressive abdominal distention or has any evidence of functional gastric outlet obstruction or ileus. The nasogastric route of feeding is preferred, but if establishing stomach function is anticipated to be problematic, implantation placement of a jejunostomy feeding tube should be considered during laparotomy.

FEEDING CONSIDERATIONS IN GENERAL SURGERY PATIENTS

ESOPHAGUS

Patients requiring esophageal resection may present with some degree of malnutrition. It is important to resume nutritional support as soon as technically possible after the operation. These patients have fragile anastomoses in their chests, however, which usually have a suction catheter placed across the repair to decompress the postanastomotic structures. An oral diet is delayed to ensure mechanical integrity of the anastomosis. Some patients have a distal feeding tube placed at the time of surgery so that enteral nutrition does not need to be delayed. Patients who cannot receive oral or enteral nutrition by postoperative day 7 should be considered for institution of parenteral nutrition.

STOMACH

Gastric surgery may result in delayed gastric emptying. Vagal denervation can cause some degree of gastroparesis, and functional outlet obstruction may occur owing to edema at the site of anastomosis. Gastric enteral nutrition cannot be started until gastric emptying improves. If it seems that gastric enteral nutrition would be unacceptably delayed, a more distal enteral route should be secured, or parenteral nutrition should be started. Patients with new gastrostomies, whether placed percutaneously or via an open procedure, rarely have postoperative motility disturbances. It is common, however, to wait for 24 hours before the use of gastronomy feeding tubes.

SMALL AND LARGE INTESTINE

Postoperative ICU patients with manipulation, resection, or diversion of the bowel may have a transient ileus. Small bowel hypomotility, if present, resolves 6 to 8 hours after

surgery, and some absorptive capacity is present even without normal peristalsis.^{88,89} Large bowel hypomotility, if present, begins to resolve 24 hours postoperatively, heralded by the passage of flatus. Recognized postoperatively as abdominal distention on physical examination or a non-obstructed gas pattern on abdominal x-ray study, ileus usually resolves over 24 to 72 hours with conservative therapy including nasogastric suctioning. Refractory ileus in the absence of mechanical obstruction should suggest some unresolved inflammatory process. In the absence of such unresolved problems, ileus also can be improved with prokinetic agents. Neostigmine has been successful in decompressing acute colonic pseudo-obstruction.⁹⁰ The presence of enterotomy repairs, bowel anastomoses, or new ostomies should not be barriers to enteral nutrition with the return of bowel function.⁹¹

FISTULAS

Nutritional support in the presence of an enterocutaneous fistula is problematic because enteral nutrition can exacerbate fistula output. This output, particularly when high, can perpetuate or worsen malnutrition owing to the loss of nitrogen and also lead to significant losses of intravascular volume and total body water. With the exception of some colcutaneous fistulas, conservative therapy consists of bowel rest (nothing per mouth), parenteral nutrition, control of infection, correction of electrolyte disturbances, and local wound care.

PANCREATITIS

Acute pancreatitis is treated commonly in the surgical ICU. In mild acute pancreatitis, enteral nutrition has no effect on outcome and is recommended only in patients who cannot tolerate oral nutrition after 5 to 7 days.⁹² In severe acute pancreatitis, the therapeutic pendulum has swung from bowel rest and parenteral nutrition back toward early enteral nutrition. Although no differences in mortality rate have been shown in severe acute pancreatitis between groups treated with enteral nutrition and parenteral nutrition, the early enteral nutrition group has significant reductions in stress response, infections, surgical interventions, and length of stay.^{93,94} The theoretical benefit of feeding beyond the ligament of Treitz versus gastric feeding in patients with severe acute pancreatitis remains controversial in the available literature.

NUTRITION IN WOUND HEALING

Nutritional deficiencies can impede wound healing. Large open wounds are metabolically demanding and may be a source of substantial protein loss. Daily dietary goals of calorie and protein need to be increased accordingly. Deficiencies of vitamins and minerals (micronutrients) are infrequent, but should be suspected in malnourished (including unusual dietary habits) patients, elderly patients, and patients who have been receiving parenteral nutrition. Vitamin and mineral supplementation should accompany dietary calorie and protein in patients with deficiencies, but the benefit of pharmacologic doses of these micronutrients in the absence of deficiency is unproved. Vitamin A has been shown to antagonize the detrimental effects of corticosteroids on inflammation, epithelialization, and collagen synthesis. However, it does not lower infection rates

or ameliorate impaired wound contraction associated with corticosteroid therapy.^{95,96} Currently, vitamin A is not routinely used to treat patients with corticosteroid-induced immunosuppression because evidence for benefit in clinical practice is lacking. Vitamin C is needed for hydroxylation of lysine and proline in collagen formation (see earlier discussion). The benefit of vitamin C supplementation in patients receiving a normal diet is not validated. Zinc is an essential trace mineral for protein synthesis, cell division, and protein synthesis; however, its supplementation has not been shown to be beneficial in patients who are not zinc deficient.⁷⁵ Glucosamine is required for the synthesis of hyaluronic acid, an abundant component of the extracellular matrix, but also lacks clinical validation of benefit.

WOUND HEALING AND CARE

PHYSIOLOGY AND BIOLOGY OF WOUND HEALING

Many tissues in the body respond to injury by undergoing a reparative process, which can be described histologically, biochemically, chronologically, or functionally. There are many ways to label these processes, but a simple and useful paradigm includes inflammatory, proliferative, and remodeling phases.^{97,98} The process begins with hemostasis, inflammation, and generation of an extracellular matrix on which proliferating cells can attach. Wound healing is locally coordinated by cytokines and facilitated by systemically mobilized cellular elements and noncellular substrate. Ultimately, the normal healing process ends with collagen maturation. Collagen develops its tensile strength through intermolecular cross-linking of fibrils into larger and longer bundles. The collagen mass undergoes continual synthesis and degradation as weaker, randomly oriented collagen fibers are reorganized into stronger, linear, highly cross-linked bundles aligned toward mechanical stress placed on the wound. This remodeling process may last 6 to 12 months, with the tissue never fully recovering its original strength. In normal circumstances, these phases tend to be sequential with generous overlap between the end of one phase and the beginning of the next.

Surgical site infection (SSI), the presence of necrotic tissue, the presence of a foreign body, an immunocompromised state, ischemia, and poor surgical closure technique all can contribute to failed wound healing and possibly wound dehiscence. Wounds are classified by their native propensity for infection as clean, clean contaminated, contaminated, and dirty. Clean wounds are uninfected with little or no inflammation, and dirty wounds are those with gross contamination such as fecal matter. Clean-contaminated and contaminated wounds lie somewhere in the middle of this spectrum.⁹⁹ Although a clean or clean-contaminated surgical wound may be purposely closed by primary intention, a contaminated or dirty wound is left open to close slowly by granulation and wound contraction (secondary intention). Alternatively, a contaminated wound may be left open for several days prior to being closed (delayed primary closure) to prevent infection. The healing processes are similar in these various approaches to wound

management. Successful healing of a closed surgical wound yields mechanical integrity by virtue of high tensile strength. Successful healing in an open wound may be measured by epithelialization with the promise of satisfactory mechanical integrity (scarring) over time. Understanding these interrelated processes facilitates logical wound care and helps to avoid diversions from normal wound healing.

EPITHELIALIZATION AND WOUND CARE

Development of an epithelial barrier begins within hours of injury. In partial-thickness wounds, the source of epithelial repopulation is remaining dermal structures, sweat glands, and hair follicles. Epithelial cells from the basal layers of the wound migrate across the underlying extracellular matrix, re-forming the characteristic basal to apical differentiation, until migration halts in the center of the wound because of contact inhibition. Wound coverage can be complete 24 to 48 hours after a clean surgical incision is closed by primary intention. At this time, no further wound protection is necessary, and skin cleansing with water is permitted. Bacteria, necrotic tissue, wound exudates, inflammatory cells, inflammatory mediators, and desiccation all retard re-epithelialization. Deeper or open wounds also show delayed epithelialization. Open wounds first must fill in with proliferating fibroblasts, capillaries, and a loose extracellular matrix made of collagen and proteoglycans (granulation tissue) before epithelialization can occur. Such tissue is of poor mechanical integrity.

The ability of epithelialization to occur from the margin of the wounds over the granulation tissue depends on the presence of adequate angiogenesis, absence of bacterial burden, the provision of a moist environment, and the removal of excess necrosis and proteinaceous exudates (which contain proteases and inflammatory mediators and support bacterial growth). With optimal circumstances, the maximal rate of epithelialization from the margins occurs at 1 to 2 mm/day. As the epithelial cells mature and stratification progresses, keratinization occurs.

Without moist, occlusive dressings over superficial wounds, eschar forms, delaying epithelialization. Only with clot proteolysis can the wound be resurfaced successfully. If the wound is kept moist with an occlusive dressing,¹⁰⁰ however, and accumulated exudates and necrotic tissue are removed frequently, epithelialization can occur. Small amounts of wound exudates and necrotic tissue can be removed with frequent, moist dressing changes and water irrigation; larger amounts may require surgical débridement. The optimal wound dressing provides a moist environment, has absorptive reserve to trap wound exudates, possesses bacteriostatic properties, and does not adhere to the wound. Large, open wounds may be dressed with moist gauze at the surface and reinforced with dry gauze packing (wet-to-dry dressing). Absorptive capacity is limited, however, and frequent dressing changes are required. Dressings made of hydrocolloids, materials that incorporate high-capacity absorptive materials into a self-adhering occlusive backing, are useful for open wounds of moderate size and allow for less frequent dressing changes. More recently, the vacuum-assisted closure has gained popularity for the management of large open wounds. Vacuum-assisted closure therapy is the combination of moderate suction applied

above an absorptive surface, such as a towel or sponge, which is covered by an occlusive plastic drape. The application provides for increased blood flow, the promotion of angiogenesis, a reduction of wound surface area in certain types of wounds, and induction of cell proliferation. However, at this time, vacuum-assisted closure therapy has not been shown to reduce edema, improve bacterial clearance, or increase the speed of healing in chronic wounds.^{101,102}

OPTIMIZING WOUND HEALING

The first rule of wound evaluation is “take off the dressing and look at the wound.” Wounds should be evaluated at least daily or with each dressing change in the case of vacuum-assisted closure therapy for progression of healing and development of infection. Normally healing surgical incisions should be dry with a minimal dry eschar at the point of closure. The edges should have at most a 3- to 4-mm border of erythema and induration when fresh, which should resolve over about 1 week.

ANTIBIOTICS

The routine use of systemic antibiotics to aid wound healing, in the absence of actual SSI, should be avoided. Wound surfaces are typically colonized by bacteria, and this colonization is not detrimental to wound healing. An increased bacterial load, more than the typical colonization, may impede wound healing, however. Distinguishing between common colonization and an increased bacterial burden requires microbiologic confirmation. Simple swab cultures lack specificity, and quantitative tissue cultures revealing greater than 10^5 organisms per gram are necessary to identify true bacterial infection. Topical antibiotics are commonly applied to wound surfaces, but the benefits of topical antibiotics are not well documented.^{72,103} The incorporation of silver into dressing materials adds bacteriostatic properties and may be useful to limit bacterial overgrowth in the wound.

SURGICAL SITE INFECTIONS

Infections of surgical incisions are referred to as *surgical site infections* (SSIs).^{104,105} SSIs are *superficial* incisional SSIs when limited to skin and subcutaneous tissues above the fascia or *deep* incisional SSIs if extending below. Intracavitary SSIs are referred to as organ-space SSIs. The surgical site becomes inoculated either inward from the skin or outward from the structures beneath the incision. Most SSIs are caused by the gram-positive cocci found on the skin, such as *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Enterococcus* species. The type of operation also can influence the causative organisms of the SSI such that enteric aerobic gram-negative rods (*Escherichia*, *Enterobacter*) and anaerobic organisms (*Bacteroides*) are more likely after intestinal or head and neck surgery.⁷⁶

Although it was once believed that mechanical bowel preparation would decrease postoperative infectious complication rates, this practice has not survived prospective validation.¹⁰⁶ The use of skin preparations, in addition to the use of narrow-spectrum “prophylactic” systemic antibiotics, has reduced the incidence of SSIs by decreasing bacterial numbers. However, the administration of prophylactic antibiotics beyond 24 hours, even in the presence

of colonic perforation or shock, does not contribute further to reducing the rate of SSIs.¹⁰⁷ In addition, prolonged use of prophylactic antibiotics may result in the emergence of multiple drug-resistant strains of organisms, *Clostridium difficile* colitis, nosocomial pneumonia, and catheter-related infections.¹⁰⁸ It is important to discontinue prophylactic antibiotics before the benefits of such therapy are overshadowed by the risks that their continuation brings with them.

A daily wound evaluation is necessary to identify early signs of wound infections. Nonpurulent drainage is not likely to be infected. Clear drainage from the wound may simply be escaping subcutaneous edema fluid or may signify seroma formation. However, wounds with an enlarging border of erythema and induration, without fluctuance or drainage, particularly when painful to palpation, suggest cellulitis or infection of deeper structures. Fluctuance and drainage may be from an abscess beneath the wound. Drainage that is turbid or frankly purulent should suggest true SSI. SSIs require opening of the incision for irrigation and drainage. Antibiotics may not be needed for uncomplicated SSIs, which respond to this intervention and local care.⁷⁶

More complicated SSIs require systemic antibiotics directed at the likely pathogens. Culture of pus collected aseptically is useful to guide therapy, but simple swab cultures of the wound surface are of low specificity because of the presence of wound colonizers. Necrotizing SSIs can spread rapidly through soft tissues and involve the fascia (necrotizing fasciitis). Necrotizing soft tissue infections can have subtle findings at the skin surface (e.g., an advancing border of erythema), while forging a destructive path just below. Wounds that dehisce superficially or at the fascia should suggest aberration of normal wound healing. Dehiscence almost always requires surgical evaluation. When an abdominal wound has open skin, evaluation for status of the fascial closure is needed. The mechanical integrity can be evaluated by gently probing the closure with sterile cotton-tipped swabs. The edges of these wounds should show yellow fat or pink granulation. Dark gray, nonviable tissue should be obvious on inspection and should be debrided.

DRAINS

Few things in the postoperative patient are more puzzling and sometimes intimidating to the uninitiated than drains. Seemingly simple in construction and intuitive in purpose, the efficacy of these devices and their application is quite limited. A study of the history of drainage is a study in the evolution of medicine and surgery itself. The earliest description of drains shows their application for the removal of fluid from large cavities, such as the pleural space, abdomen, and bladder, and for the treatment of wounds.

Drains can be classified on many levels.¹⁰⁹ Drains with one end open to the atmosphere are known as “open” systems and constitute most early devices. Before the recognition of germ theory, it was not appreciated that open systems provided a free route for entrance of infectious agents into the body. Some open systems employed a filter at the open end to limit the ability of microorganisms to enter the system. “Closed” systems of drainage have no opening to the atmosphere directly; fluid collection terminates in a bag or canister.

Structurally, drains can be classified as “hollow” or “capillary.” Hollow drains take on many shapes, but all have one or multiple internal lumens and have fenestrations throughout a portion of their length, sometimes including their ends. Fenestrations must be large enough to allow fluid and debris to enter, but not so large as to allow significant portions of tissue, such as omentum or intestine, to enter. Such migration into the drain has been the cause of drain failure, tissue adhesion, and organ injury. Capillary-type drains leverage the physical interaction that occurs between liquids and the walls of thin tubes and fibers. Structurally, capillary-type devices are made from tufts of thin fibers, fabrics (e.g., gauze), or thin tubes. Drains should be soft and flexible, but not so much that the lumen collapses with suction. Irritating materials, such as latex rubber, should be avoided (except in cases in which development of a fibrous tract is desired, such as in T-tube biliary drainage). Siliconized materials (Silastic) and polyvinyl chloride are commonly used in contemporary drainage systems.

Drains can be classified as “passive” or “active.” Passive drains provide a route of low resistance to the body’s exterior and are driven by capillary action and pressure gradients. Capillary-type drains are classified as passive drains. Active drains use an external source of negative pressure to establish a pressure gradient. Active drainage of deep recesses is classified as sump drainage. Sump drains were ultimately modified so that an additional lumen running alongside the primary lumen supplied atmospheric gas into the drainage site to prevent the intestine and omentum from occluding the fenestra.¹¹⁰ Sump drains are used to drain the gastrointestinal tract and abscess cavities. Active drainage employing a closed system is used to obliterate potential spaces, particularly under skin/muscle flaps or other wounds.

Drains also are classified as therapeutic or prophylactic.¹¹¹ Therapeutic drainage is intended to remove necrotic debris, pus, or fistula drainage or to prevent premature closure of wounds. Prophylactic drainage is intended to prevent the accumulation of blood, pus, bile, pancreatic secretions, intestinal contents, and fluids. In the historical literature of medicine and surgery, it was noted that patients with ovariectomy developed accumulations of blood and fluid in the pelvis. It was believed that this fluid, in stagnation, would decompose and release toxins whose absorption resulted in fatal outcomes. In 1882, drains were used to “remove from cavities fluids liable to undergo putrefactive changes if retained and to cleanse such cavities by injection of disinfectants.”⁸⁰

The popularity of drainage in certain applications waxed and waned owing to its controversial effect on outcome, particularly mortality risk. When surgeons abandoned the use of abdominal drains during World War II, mortality rates decreased by 50% compared with those of World War I.¹¹² The use of prophylactic drains, particularly in abdominal surgery, was equally controversial. Capillary-based systems, which did more to prevent drainage of necrotic or purulent material than facilitate its removal, ultimately fell out of favor. Complications increased from the use of multiple or unnecessary drains and included ventral hernias, pain on removal, omental penetration of the drain’s fenestrations, intestinal obstruction, adhesions (occasionally pulling omentum or bowel into the abdominal wall), fecal fistulas,

and persistent sinus tracts. The pioneering surgeon Halsted believed that good surgical technique and obliteration of dead space obviated the need for drainage in nonseptic instances. He believed that drains “invariably produce some necrosis of tissue with which it comes in contact and enflees the power of resistance of tissues toward organisms. But given necrotic tissue plus infections, drains become almost indispensable.”⁸⁰ Prophylactic drainage ultimately gave way to therapeutic drainage. In the 1920s, indications for drains included the “presence of free purulent material in considerable quantity . . . and the presence of an abscess sac.”⁸⁰

Currently, the indications for drainage include the following:

- Removal of cerebrospinal fluid (CSF) from the brain’s ventricles or spinal cord for the purpose of reducing pressure in a closed space and improving perfusion pressure
- Removal of blood or fluid from the subdural space to prevent compression or shift of intracranial contents
- Closure of certain soft tissue wounds to minimize dead space and remove excess fluid and debris; often seen in neck surgery, breast surgery, and certain reconstructive procedures
- Drainage of the pleural space in the event of pneumothorax, hemothorax, or large pleural effusions
- Drainage of the pericardium to treat large pericardial effusions
- Drainage of abscess cavities; drains can be placed directly in the operating room or percutaneously with the guidance of imaging technologies
- Drainage of existing fistulas to create a controlled route of elimination; includes drainage of bile or pancreatic secretions, succus, or stool
- Surveillance drainage over the sites of complicated procedures involving the stomach, duodenum, pancreas, and rerouting anastomoses

Placement of surveillance drains is controversial because of the risk of creating a fistula by the drains themselves. However, in the event of a catastrophic breach in enteral integrity, such as the highly morbid duodenal stump “blow-out,” early identification and controlled drainage may be facilitated by placement of such a drain.

In general, the following questions must be answered for all drains:

1. What is the intended anatomic location of the drain?
2. How can location be confirmed?
3. What is the expected quantity and quality of the drain’s output?
4. Is the drain functioning normally?
5. When should a drain be removed and according to what criteria?

Only by knowing the intended anatomic location of a given drain can a clinician determine the best way to confirm location and assess function. The visual location of a drain on physical examination does not ensure proper placement; a thoracostomy tube seen to penetrate the chest wall may not be in the pleural space, and a gastrostomy tube seen to penetrate the abdominal wall does not guarantee that the

tip lies in the stomach. Sometimes the location of a drain cannot be confirmed, such as drains left in the peritoneum. This, short of advanced imaging techniques, leaves only assessment of quantity and quality of drain output as a guide to the drain’s proper location and function. For these reasons, it is useful to know certain characteristics of specific drains.

The most common drains seen after neurosurgical procedures are the subdural drain and the ventriculostomy. The former drain is usually a Silastic drain left in the subdural space to drain blood or fluid after craniotomy. There is no way to confirm its location. These drains typically drain about 20 to 30 mL of serosanguineous fluid per hour until tapering off to minimal drainage after about 6 hours. Frankly bloody drainage, particularly when in higher volumes or persisting longer than a few hours, suggests active bleeding that requires correction of coagulopathy or neurosurgical intervention. The ventriculostomy tube, also made of Silastic, has its tip located in a lateral ventricle. The proper tip location can be confirmed by seeing a pulsatile waveform when the catheter has continuous pressure monitoring and by seeing CSF output. About 450 mL of CSF is produced a day; the volume of CSF drained depends on the height of the drainage system’s external port relative to the height of the catheter’s tip in the ventricle and the ability of the arachnoid granulations to reabsorb CSF. The fluid may be clear or sanguineous depending on the intracranial surgery performed. CSF that changes from clear or serosanguineous to frankly bloody suggests a serious problem, particularly in subarachnoid hemorrhage. Declining or absent CSF drainage or loss of a pulsatile waveform suggests tube occlusion by clot or malposition and requires neurosurgical attention.

Thoracostomy tubes are placed to drain pleural effusions and treat pneumothorax. Thoracostomy tubes can be inadvertently placed subcutaneously. Proper location is confirmed by chest radiograph. The tube may be intentionally positioned in many orientations; however, the most proximal “sentinel” hole should always lie within the pleural space, and the tube should not be kinked. A properly functioning, correctly located thoracostomy tube should show a cycling of intrapleural pressure with respiration when the drainage system is on “water seal.” Absence of cycling may suggest tube occlusion or inappropriate location. Bubbling across the water seal suggests an air leak, but does not indicate the source of the leak. Persistence of the bubbles across the water seal when the thoracostomy tube is clamped close to the chest wall indicates a leak in the drainage system, not in the lung. Variable amounts of suction can be applied to the thoracostomy tube, particularly when draining an effusion or reinflating a lung after pneumothorax. Initial suction of -20 cm H_2O is appropriate in this clinical situation. Persistence of sanguineous drainage greater than 200 mL/hour for 2 to 3 hours after the correction of hypothermia and coagulopathy suggests surgical bleeding and requires attention. When fluid drainage has diminished to about 100 to 200 mL/day or air leaks have ceased, external suction can be removed, and the water seal alone can be used to prevent lung collapse. If effusions or pneumothorax do not return, as assessed on chest radiograph, the thoracostomy tube can be removed.

Nasogastric or orogastric tubes are used to decompress the stomach or provide a route for nutrition. Double-lumen sump tubes should never have the secondary port clamped; this secondary port prevents mucosal injury in the presence of suction. Inadvertent placement in the airways can be disastrous if enteral feedings are administered. Confirmation of gastric placement cannot be guaranteed by listening over the epigastrium during insufflation. Correct placement on radiograph is recognized by identifying the distal tip well below the diaphragm. Salivary and gastric output can be 0.75 to 1.5 L/day each. Continuous gastric suction can result in significant volume and chloride loss, leading to metabolic alkalosis. Gastric suction should be maintained until resolution of enteral obstructions or ileus. When the daily volume of gastric aspirate is less than 200 to 300 mL, gastric suction can be discontinued as long as nausea, vomiting, or abdominal distention does not result.

The color of gastric aspirate should be clear or yellow-green. Large volumes of bilious aspirate suggest the distal port of the drain is positioned beyond the pylorus. “Coffee grounds” or frank blood in the aspirate suggests bleeding in the stomach or duodenum. The stomach also can be accessed by placement of a surgical or percutaneous endoscopically assisted gastrostomy. These tubes infrequently migrate out of the stomach to lie in the peritoneum. Should acute abdominal pain or absence of typical gastric drainage occur in a patient with a recently placed gastrostomy, a radiographic contrast study of the gastrostomy should be done to exclude tube migration.

The liver produces 500 to 1500 mL of bile daily. Drainage of the common bile duct via a T-tube is used after complicated biliary surgery, often for obstruction. T-tubes are used less than in the past now that transhepatic catheter drainage and common bile duct stents/sphincterotomy are more commonplace. The drainage tube itself causes a modest inflammatory reaction resulting in the formation of a fibrous tract. The drainage system is closed, without suction, and terminates in a collection bag. Significant reduction or cessation of biliary output may suggest either obstruction or malposition of the T-tube or resolution of the obstruction.

With the exception of drains placed in abscess cavities and to control the direction of pancreatic and enteral fistula output, drains left in the abdominal cavity are seen less frequently than in the past. Drains left in the peritoneum should have relatively little output. Confirmation of their location is usually unnecessary. A change in the quality or quantity of drainage is important to note. New bile, succus entericus, or stool in a drain suggests a breach in the integrity of some part of the viscera and requires investigation or surgical attention.

Drains placed in subcutaneous spaces or areas of reconstruction are placed to gentle suction to obliterate potential spaces and remove excessive fluid and blood collection. Confirmation of absolute location is generally unnecessary. The quality of the fluid should be serous to serosanguineous in volumes less than 100 mL hourly for the first 3 to 6 hours postoperatively before tapering off. Frankly bloody drainage in higher volumes or of longer durations suggests surgical bleeding in the absence of coagulopathy.

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KEY POINTS

- Optimally, the location of a patient in the ICU is determined by matching the patient’s needs with a location’s resources and expertise.
- The postoperative evaluation should include a thorough evaluation of the patient’s medical and surgical history and a physical examination, which should encompass all parts of the patient that can be accessed by sight and touch.
- “Adequate resuscitation” is a state, often temporary, that allows for good clinical perfusion and physiologic stability. The most fundamental concept is to ensure adequate DO_2 and meet the $\dot{V}\text{O}_2$ needs of tissues and organelles.
- Resuscitation targets are reproducible, quantifiable values, such as pressures, outputs, metabolites, inflammatory mediators, or oxygen saturations, that represent therapeutic goals. Targeted resuscitation strategies optimize cardiovascular performance and concurrently measure markers of adequate global DO_2 and tissue use.
- Analgesics should be administered as the patient is resuscitated from anesthesia to facilitate comfort and avoid interference with medical care.
- Resuscitation should be complete, with hemostasis achieved, metabolic acidosis resolving, vasoactive support and gas exchange abnormalities minimized, anesthetic agents cleared, the ability to protect the airway present, and the patient awake and reasonably cooperative prior to initiating weaning from mechanical ventilation.
- Nutritional support is an important consideration in ICU patients due to the ongoing caloric demands of these complex patients. Early assessment of the available routes should be performed and the appropriate supplementation should be incorporated into the patient’s daily care.
- Successful healing of a closed surgical wound yields mechanical integrity by virtue of high tensile strength. Successful healing in an open wound may be measured by epithelialization with the promise of satisfactory mechanical integrity (scarring) over time.
- The ability of epithelialization to occur from the margin of the wounds over the granulation tissue depends on the presence of adequate angiogenesis, the absence of bacterial burden, the provision of a moist environment, and the removal of excess necrosis and proteinaceous exudates (which contain proteases and inflammatory mediators and harbor bacterial growth).
- The first rule of wound evaluation is “take off the dressing and look at the wound.” Wounds should be

KEY POINTS (Continued)

evaluated at least daily for progression of healing and for development of infection.

- Only by knowing the intended anatomic location of a given drain can a clinician determine the best way to confirm location and assess its function.

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