Chapter

39 POSTANESTHESIA RECOVERY

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The postanesthesia care unit (PACU), sometimes referred to as the recovery room, is designed and staffed to monitor and care for patients who are recovering from the immediate physiologic effects of anesthesia and surgery. PACU care spans the transition from delivery of anesthesia in the operating room to the less acute monitoring on the hospital ward and, in some cases, independent function of the patient at home. Also, PACUs provide critical care to patients for whom there is no intensive care unit bed in busy medical centers. To serve this unique transition period, the PACU must be equipped to monitor and resuscitate unstable patients while simultaneously providing a tranquil environment for the "recovery" and comfort of stable patients. The proximity of the unit to the operating room facilitates rapid access to postoperative patients by anesthesia providers and surgical caregivers.

ADMISSION TO THE POSTANESTHESIA CARE UNIT

Upon arrival in the unit, the anesthesia provider should inform the PACU nurse of pertinent details on the patient's history, medical condition, anesthesia, and surgery. Particular attention is directed to monitoring oxygenation (pulse oximetry), ventilation (breathing frequency, airway patency, capnography), and circulation (systemic arterial blood pressure, heart rate, electrocardiogram [ECG]).

Vital signs are recorded as often as necessary but at least every 15 minutes while the patient is in the unit. The American Society of Anesthesiologists (ASA) has adopted Standards for Postanesthesia Care that delineate the minimal requirements for PACU monitoring and care.[1] More specific recommendations addressing clinical evaluation and therapeutic intervention can be found in the ASA Practice Guidelines for Postanesthesia Care.[2]

EARLY POSTOPERATIVE PHYSIOLOGIC DISORDERS

A variety of physiologic disorders affecting multiple organ systems must be diagnosed and treated in the PACU during emergence from anesthesia and surgery (Box 39.1). Nausea and vomiting, the need for upper airway support, and systemic hypotension are among the most frequently encountered complications.[3] Not surprisingly, serious outcomes may result from airway, respiratory, or cardiovascular compromise.[4] Airway problems and cardiovascular events accounted for the majority (67%) of 419 recovery room incidents reported to the 2002 Australian Incident Monitoring Study (AIMS).[5] In addition, transport of the patient from the operating room to the PACU is also a time when patients are especially vulnerable to airway obstruction, as discussed next.

Box 39.1 Physiologic Disorders Manifested in the Postanesthesia Care Unit

- Upper airway obstruction
- Arterial hypoxemia
- Hypoventilation
- Hypotension
- Hypertension
- Cardiac dysrhythmias
- Oliguria
- Bleeding
- Decreased body temperature
- Delirium (emergence agitation)
- Delayed awakening
- Nausea and vomiting
- Pain

UPPER AIRWAY OBSTRUCTION

Loss of Pharyngeal Muscle Tone

Airway obstruction is a common and potentially devastating complication in the postoperative period (also see Chapter 16). The most frequent cause of airway obstruction in the PACU is the loss of pharyngeal tone in a sedated or obtunded patient. The residual depressant effects of inhaled and intravenous anesthetics and the persistent effects of neuromuscular blocking drugs (also see Chapter 11) contribute to the loss of pharyngeal tone in the immediate postoperative period.

In an awake, unanesthetized patient, the pharyngeal muscles contract synchronously with the diaphragm to pull the tongue forward and tent the airway open against the negative inspiratory pressure generated by the diaphragm. This pharyngeal muscle activity is depressed during sleep, and the resulting decrease in tone promotes airway obstruction. With the collapse of compliant pharyngeal tissue during inspiration, a vicious circle may ensue in which a reflex compensatory increase in respiratory effort and negative inspiratory pressure promotes further airway obstruction. This effort to breathe against an obstructed airway is characterized by a paradoxic breathing pattern consisting of retraction of the sternal notch and exaggerated abdominal muscle activity. Collapse of the chest wall plus protrusion of the abdomen with inspiratory effort produces a rocking motion that becomes more prominent with increasing airway obstruction.

Obstruction secondary to loss of pharyngeal tone can be relieved by simply opening the airway with the "jaw thrust maneuver" or continuous positive airway pressure (CPAP) applied via a face mask (or use of both). Support of the airway is needed until the patient has adequately recovered from the effects of drugs administered during anesthesia. In selected patients, placement of an oral or nasal airway, laryngeal mask airway, or endotracheal tube may be required (also see Chapter 16).

Residual Neuromuscular Blockade

When evaluating upper airway obstruction in the PACU, the possibility of residual neuromuscular blockade must be considered in any patient who received neuromuscular blocking drugs during anesthesia (also see Chapter 11). Residual neuromuscular blockade may not be evident on arrival in the PACU because the diaphragm recovers from neuromuscular blockade before the pharyngeal muscles do. With an endotracheal tube in place, end-tidal carbon dioxide concentrations and tidal volumes may indicate adequate ventilation while the ability to maintain a patent upper airway and clear upper airway secretions remain compromised. The stimulation associated with tracheal extubation, followed by the

activity of patient transfer to the gurney and subsequent mask airway support, may keep the airway open during transport. Only after the patient is calmly resting in the PACU does upper airway obstruction become evident. Even patients treated with intermediate- and short-acting neuromuscular blocking drugs may manifest residual paralysis in the PACU despite what was deemed clinically adequate pharmacologic reversal in the operating room (OR).

The association between intermediate-acting neuromuscular blocking drugs and postoperative respiratory complications is dose dependent.[6] Also, inappropriate dosing of the reversal drug neostigmine can cause postoperative respiratory complications. A large prospective study of over 3000 PACU patients showed the unwarranted use or inappropriate dosing of neostigmine to be an independent risk factor for reintubation of the trachea.[7,8] Therefore, determining the appropriate dose of neostigmine, and specifically avoiding inappropriate dosing or overdosage, is essential to assure full recovery of neuromuscular function in the PACU. Over the years qualitative measurement of the train-of-four (TOF) ratio by tactile response or visualization was the most commonly used method to assess the degree of reversal of neuromuscular blockade at the end of surgery. However, more recent evidence suggests that the qualitative measurement of the TOF ratio may not accurately reflect recovery of neuromuscular function. Instead, the use of quantitative TOF measurement using acceleromyography provides a more objective and accurate method of monitoring neuromuscular function.[9] It is hoped that use of a newly approved reversal drug, sugammadex, will decrease the frequency of inadequate reversal of neuromuscular blockade.

When patients with residual neuromuscular blockade are awake in the PACU, their struggle to breathe may manifest as agitation. In an awake patient, clinical assessment of reversal of neuromuscular blockade is preferred to the application of painful TOF or tetanic stimulation. Clinical evaluation includes grip strength, tongue protrusion, the ability to lift the legs off the bed, and the ability to lift the head off the bed for a full 5 seconds. Of these maneuvers, the 5-second sustained head lift is considered the gold standard because it reflects not only generalized motor strength but, more important, the patient's ability to maintain and protect the airway. In patients whose tracheas have been extubated, the ability to strongly oppose the incisor teeth against a tongue depressor is another reliable indicator of pharyngeal muscle tone in the awake patient. This maneuver correlates with an average TOF ratio of 0.85. Inadequate ventilation or airway obstruction is less likely if the neuromuscular blockade has been reversed with neostigmine or sugammadex (also see Chapter 11).

If persistence or return of neuromuscular weakness in the PACU is suspected, prompt review of possible etiologic factors is indicated (Box 39.2). Common factors include respiratory acidosis and hypothermia, alone or in combination. Residual depressant effects of volatile anesthetics or opioids (or both) may result in progressive respiratory acidosis only after the patient is admitted to the PACU and external stimulation is minimized. Similarly, a patient who becomes hypothermic during anesthesia and surgery may show signs of weakness in the PACU that were not noted following extubation in the operating room. Simple measures such as warming the patient, airway support, and correction of electrolyte abnormalities can facilitate recovery from neuromuscular blockade.

Laryngospasm

Laryngospasm refers to a sudden spasm of the vocal cords that completely occludes the laryngeal opening. It typically occurs in the transitional period when the patient whose trachea has been extubated is emerging

Box 39.2 Causes of Prolonged Neuromuscular Blockade

Factors Contributing to Prolonged Nondepolarizing Neuromuscular Blockade

- Drugs
  - Inhaled anesthetic drugs
  - Local anesthetics (lidocaine)
  - Cardiac antidysrhythmics (procainamide)
  - Antibiotics (polymyxins, aminoglycosides, lincosamines [clindamycin], metronidazole [Flagyl], tetracyclines)
  - Corticosteroids
  - Calcium channel blockers
  - Dantrolene
  - Furosemide
- Metabolic and physiologic states
  - Hypermagnesemia
  - Hypocalcemia
  - Hypothermia
  - Respiratory acidosis
  - Hepatic/renal failure
  - Myasthenia syndromes

Factors Contributing to Prolonged Depolarizing Neuromuscular Blockade

- Excessive dose of succinylcholine
- Reduced plasma cholinesterase activity
  - Decreased levels
  - Extremes of age (newborn, old age)
  - Disease states (hepatic disease, uremia, malnutrition, plasmapheresis)
  - Hormonal changes
  - Pregnancy
  - Contraceptives
  - Glucocorticoids
- Inhibited activity
  - Irreversible (echothiophate)
  - Reversible (edrophonium, neostigmine, pyridostigmine)
- Genetic variant (atypical plasma cholinesterase)

from general anesthesia. Although it is most likely to occur in the operating room at the time of tracheal extubation, patients who arrive in the PACU asleep after general anesthesia are also at risk for laryngospasm when awakening.

Jaw thrust with CPAP (up to 40 cm H_2O) is often sufficient stimulation to "break" the laryngospasm. If jaw thrust and CPAP maneuvers fail, immediate skeletal muscle relaxation can be achieved with intravenously (IV) or intramuscularly (IM) administered succinylcholine (0.1 to 1.0 mg/kg IV or 4 mg/kg IM). A tracheal tube should not be passed forcibly through a glottis that is closed because of laryngospasm.

Airway Edema

Airway edema is a possible postoperative complication in patients undergoing prolonged procedures in the prone or Trendelenburg position and in procedures with large amounts of blood loss requiring aggressive intravascular fluid resuscitation. Surgical procedures on the tongue, pharynx, and neck, including thyroidectomy, carotid endarterectomy, and cervical spinal procedures, can result in upper airway obstruction because of tissue edema or hematoma, or both. Although facial and scleral edema are important physical signs that can alert the clinician to the presence of airway edema, significant edema of pharyngeal tissue is often not accompanied by visible external signs. If tracheal extubation is attempted in these patients in the PACU, evaluation of airway patency must precede removal of the endotracheal tube (ETT). The patient's ability to breathe around the ETT can be evaluated by suctioning the oral pharynx and deflating the ETT cuff. With occlusion of the proximal end of the ETT, the patient is then asked to breathe around the tube. This qualitative assessment of adequate air movement suggests that the patient's airway will remain patent after tracheal extubation. More quantitative methods include (1) measuring the intrathoracic pressure required to produce an audible leak around the ETT when the cuff is deflated and (2) measuring the exhaled tidal volume before and after ETT cuff deflation in a patient receiving volume control ventilation. Though helpful, none of these cuff leak "tests" takes the place of sound clinical judgment when deciding when to safely extubate the patient.[10] If concern for airway compromise is significant, a tracheal tube exchange catheter can be used.

Obstructive Sleep Apnea

Special consideration must be given to patients with obstructive sleep apnea (OSA) in the PACU (also see Chapters 27 and 50).[11] Patients with OSA have a more frequent risk for postoperative desaturation, respiratory failure, postoperative cardiac events, and the need for intensive care unit transfer.[12] As such, it is important to recognize and diagnose OSA in the preoperative setting and be mindful of its implications in the intraoperative and postoperative settings. Many screening tools such as the STOP-BANG questionnaire are effective in predicting OSA.[13] Because patients with OSA are particularly prone to airway obstruction, their tracheas should not be extubated until they are fully awake and following commands. Any redundant compliant pharyngeal tissue in these patients not only increases the incidence of airway obstruction but also makes mask ventilation and intubation by direct laryngoscopy difficult or at times impossible. Once in the PACU, a patient with OSA whose trachea has been extubated is exquisitely sensitive to opioids, and, when possible, regional anesthesia and multimodal analgesia techniques should be used to provide postoperative analgesia and minimize opioid consumption. The combination of benzodiazepines and opioids can cause significant episodes of hypoxemia and apnea in patients with OSA.[14]

For patients with OSA, plans should be made preoperatively to provide CPAP in the immediate postoperative period. Patients are often asked to bring their CPAP machines with them on the day of surgery so that the equipment can be set up before the patient's arrival in the PACU. Patients who do not routinely use CPAP at home or who do not have their machines with them may require additional attention from the respiratory therapist to ensure proper fit of the CPAP delivery device (mask or nasal airways) and to determine the amount of positive pressure needed to prevent upper airway obstruction. For patients with known or suspected OSA, consideration should also be given to postoperative continuous pulse oximetry monitoring.

Management of Airway Obstruction

A patient who has an obstructed upper airway requires immediate attention. Efforts to open the airway by noninvasive measures should be attempted before reintubation of the trachea. Jaw thrust with CPAP (5 to 15 cm H_2O) is often enough to tent the upper airway open in patients with decreased pharyngeal muscle tone. If CPAP is not effective, an oral, nasal, or laryngeal mask airway can be inserted rapidly. After successfully opening the upper airway and ensuring adequate ventilation, the cause of the upper airway obstruction should be identified and treated. The sedating effects of opioids and benzodiazepines can be reversed with persistent stimulation or small, titrated doses of naloxone or flumazenil, respectively (see Chapter 8). Residual effects of neuromuscular blocking drugs can be reversed pharmacologically or by correcting contributing factors such as hypothermia (see Chapter 11).

Ventilating the lungs of a patient with severe upper airway obstruction as a result of edema or hematoma may not be possible via a mask. In the case of hematoma after thyroid or carotid surgery, an attempt can be made to decompress the airway by releasing the clips or sutures on

the wound and evacuating the hematoma. This maneuver is recommended as a temporizing measure, but it will not effectively decompress the airway if a significant amount of fluid or blood (or both) has infiltrated the tissue planes of the pharyngeal wall. If emergency tracheal intubation is required, ready access to difficult airway equipment should be arranged and, if possible, surgical backup for performance of an emergency tracheostomy. If the patient is able to move air by spontaneous ventilation, an awake endotracheal intubation technique is preferred because visualization of the cords by direct laryngoscopy may not be possible.

Monitoring Airway Patency During Transport

Upper airway patency and the effectiveness of the patient's respiratory efforts must be monitored during transportation from the operating room to the PACU. Hypoventilation in a patient receiving supplemental oxygen will not be reliably detected by monitoring with pulse oximetry during transport.[15] Adequate ventilation must be confirmed by watching for the appropriate rise and fall of the chest wall with inspiration, listening for breath sounds, or simply feeling for exhaled breath with the palm of one's hand over the patient's nose and mouth. As indicated previously, this can be a critically dangerous time in the immediate postoperative period.

HYPOXEMIA IN THE POSTANESTHESIA CARE UNIT

Atelectasis and alveolar hypoventilation are the most common causes of transient postoperative arterial hypoxemia in the immediate postoperative period. Filling the patient's lungs with oxygen at the conclusion of anesthesia, as well as the administration of supplemental oxygen, should blunt any effect of diffusion hypoxia as a contributor to arterial hypoxemia. Review of the patient's history, operative course, and clinical signs and symptoms will direct the workup to determine possible causes of persistent hypoxia (Box 39.3) (also see Chapter 5).

Alveolar Hypoventilation

Postoperative ventilatory failure can result from a depressed drive to breathe or generalized weakness from either residual neuromuscular blockade or underlying neuromuscular disease. Restrictive pulmonary conditions such as preexisting chest wall deformity, postoperative abdominal binding, or abdominal distention can also contribute to inadequate ventilation (Box 39.4).

Review of the alveolar gas equation demonstrates that hypoventilation alone is sufficient to cause arterial hypoxemia in a patient breathing room air (Fig. 39.1). At sea level, a normocapnic patient breathing room air will have an alveolar oxygen partial pressure of 100 mm Hg.

Box 39.3 Causes of Postoperative Hypoxemia

- Right-to-left shunt
  - Pulmonary: atelectasis
  - Intracardiac: congenital heart disease
- Mismatching of ventilation to perfusion
- Congestive heart failure
- Pulmonary edema—fluid overload, postobstructive
- Alveolar hypoventilation—residual effects of anesthetics and neuromuscular blocking drugs
- Diffusion hypoxia—unlikely if patient is receiving supplemental oxygen
- Aspiration of gastric contents
- Pulmonary embolus
- Pneumothorax
- Posthyperventilation hypoxia
- Increased oxygen consumption (e.g., shivering)
- Acute respiratory distress syndrome (ARDS)
- Sepsis
- Transfusion-related acute lung injury
- Advanced age
- Obesity

Box 39.4 Factors Leading to Postoperative Hypoventilation

- Drug-induced central nervous system depression (volatile anesthetics, opioids)
- Residual effects of neuromuscular blocking drugs
- Suboptimal ventilatory muscle mechanics
- Increased production of carbon dioxide
- Coexisting chronic obstructive pulmonary disease

$$PAO_2 = FIO_2(PB - PH_2O) - \frac{PaCO_2}{RQ}$$

$PaCO_2$ = 40 mm Hg

$$PAO_2 = 0.21(760 - 47) - \frac{40}{0.8} = 150 - 50 = 100 \text{ mm Hg}$$

$PaCO_2$ = 80 mm Hg

$$PAO_2 = 0.21(760 - 47) - \frac{80}{0.8} = 150 - 100 = 50 \text{ mm Hg}$$

PAO_2 = alveolar oxygen pressure
FIO_2 = fraction of inspired oxygen concentration
PB = barometric pressure
PH_2O = vapor pressure of water
RQ = respiratory quotient

Fig. 39.1 Hypoventilation as a cause of arterial hypoxemia.

Thus, a healthy patient without a significant alveolar-arterial (A-a) gradient will have a Pa_{O_2} near 100 mm Hg. In the same patient, an increase in Pa_{CO_2} from 40 to 80 mm Hg (alveolar hypoventilation) results in an alveolar oxygen partial pressure (P_{AO_2}) of 50 mm Hg. This exercise

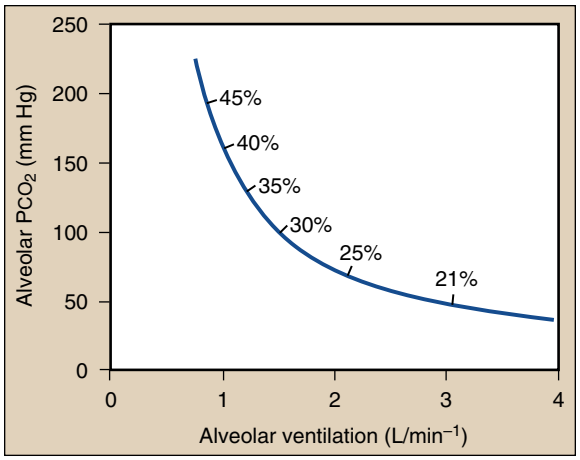

Fig. 39.2 Alveolar P_{CO_2} as a function of alveolar ventilation at rest. The percentages indicate the inspired oxygen concentration required to restore alveolar P_{O_2} to normal. (Adapted from Lumb AB, ed. *Nunn's Applied Respiratory Physiology*. 6th ed. Philadelphia: Elsevier/Butterworth-Heinemann; 2005, used with permission.)

demonstrates that even a patient with normal lungs will become hypoxic if allowed to significantly hypoventilate while breathing room air.

Normally, minute ventilation increases by approximately 2 L/min for every 1 mm Hg increase in arterial P_{CO_2}. This linear ventilatory response to carbon dioxide can be significantly depressed in the immediate postoperative period by the residual effects of drugs (e.g., inhaled anesthetics, opioids, sedative-hypnotics) administered during anesthesia.

Arterial hypoxemia secondary to hypercapnia alone can be reversed by the administration of supplemental oxygen or by restoring the P_{aCO_2} to normal, or both (Fig. 39.2).[16] In the PACU, P_{aCO_2} can be returned to normal by external stimulation of the patient to wakefulness, pharmacologic reversal of opioid or benzodiazepine effect, or controlled mechanical ventilation. Fig. 39.2 demonstrates why pulse oximetry is an unreliable marker of hypoventilation in a patient receiving supplemental oxygen.

Decreased Alveolar Partial Pressure of Oxygen

Diffusion hypoxia refers to the rapid diffusion of nitrous oxide into alveoli at the end of a nitrous oxide anesthetic. Nitrous oxide dilutes the alveolar gas and produces a transient decrease in P_{AO_2} and P_{ACO_2}. In a patient breathing room air, the resulting decrease in P_{AO_2} can produce arterial hypoxemia. In the absence of supplemental oxygen administration, diffusion hypoxia can persist for 5 to 10 minutes after a nitrous oxide anesthetic and thus contribute to arterial hypoxemia in the initial moments as the patient is admitted to the PACU.

When providing supplemental oxygen to a patient during transport to the PACU, care should be taken to avoid the relative decrease in the fraction of inspired oxygen (F_{IO_2}) that can result from an unrecognized disconnection of the oxygen source or from an empty oxygen tank.

Ventilation-to-Perfusion Mismatch and Shunt

Hypoxic pulmonary vasoconstriction (HPV) is an attempt of normal lungs to optimally match ventilation and perfusion. This response constricts vessels in poorly ventilated regions of the lung and directs pulmonary blood flow to well-ventilated alveoli. The HPV response is inhibited by many conditions and medications, including pneumonia, sepsis, and vasodilators. In the PACU, the residual effects of inhaled anesthetics and vasodilators such as nitroprusside and dobutamine will blunt HPV and contribute to arterial hypoxemia.

Unlike a ventilation-to-perfusion mismatch, a true shunt will not respond to supplemental oxygen. Causes of postoperative pulmonary shunt include atelectasis, pulmonary edema, gastric aspiration, pulmonary emboli, and pneumonia. Of these, atelectasis is probably the most common cause of pulmonary shunting in the immediate postoperative period. Mobilization of the patient to the sitting position, incentive spirometry, and positive airway pressure via a face mask can be effective in treating atelectasis.

Increased Venous Admixture

Increased venous admixture typically refers to low cardiac output states. It is due to mixing of desaturated venous blood with oxygenated arterial blood. Normally, only 2% to 5% of cardiac output is shunted through the lungs, and this small amount of shunted blood with a normal mixed venous saturation has a minimal effect on P_{aO_2}. In low cardiac output states, blood returns to the heart severely desaturated. Additionally, the shunt fraction increases significantly in conditions that impede alveolar oxygenation, such as pulmonary edema and atelectasis. Under these conditions, mixing of desaturated shunted blood with saturated arterialized blood decreases P_{aO_2}.

Decreased Diffusion Capacity

A decreased diffusion capacity is caused by underlying lung disease such as emphysema, interstitial lung disease, pulmonary fibrosis, or primary pulmonary hypertension. The differential diagnosis of arterial hypoxemia in the PACU must include the contribution of any preexisting pulmonary condition.

PULMONARY EDEMA IN THE POSTANESTHESIA CARE UNIT

Pulmonary edema in the immediate postoperative period is often cardiogenic in nature, the result of increased

intravascular volume or cardiac dysfunction. Noncardiogenic edema may occur in the PACU as a result of pulmonary aspiration of gastric contents or sepsis. Rarely, postoperative pulmonary edema is the result of airway obstruction (postobstructive pulmonary edema) or transfusion of blood products (transfusion-related acute lung injury) (also see Chapter 24).

Postobstructive Pulmonary Edema

Postobstructive pulmonary edema (POPE), also referred to as negative-pressure pulmonary edema, and the resulting arterial hypoxemia are rare but significant consequences of upper airway obstruction and may follow tracheal extubation at the conclusion of anesthesia and surgery. POPE is characterized by a transudative edema produced by one of two mechanisms: the exaggerated negative pressure generated by inspiration against acute airway obstruction (type I) or following relief of a chronic partial airway obstruction (type II).[17] The pathophysiology of type I POPE involves exaggerated negative intrathoracic pressure, which increases venous return, afterload, and pulmonary venous pressures, and promotes the transudation of fluid. Muscular healthy patients are at increased risk because of their ability to generate significant inspiratory force.

Laryngospasm is the most common cause of upper airway obstruction leading to type I POPE, but it may result from any condition that occludes the upper airway including epiglottitis, bilateral vocal cord paralysis, goiter, and occlusion of the ETT. Arterial hypoxemia with respiratory distress is usually manifested within 90 minutes after relief of airway obstruction and is frequently accompanied by tachypnea, tachycardia, rales, rhonchi, and evidence of bilateral pulmonary edema on the chest radiograph. The diagnosis depends on clinical suspicion once other causes of pulmonary edema are ruled out. Treatment is supportive and includes supplemental oxygen, diuresis, and, in severe cases, positive-pressure ventilation utilizing CPAP or mechanical ventilation.

Transfusion-Related Acute Lung Injury

The differential diagnosis of pulmonary edema in the PACU should include transfusion-related acute lung injury (TRALI) in any patient who received blood, coagulation factor, or platelet transfusions intraoperatively and is described in Chapter 24. Treatment is generally supportive and includes supplemental oxygen and diuresis. Rarely, TRALI results in a prolonged course of acute respiratory distress syndrome (ARDS). Historically, the lack of specific diagnostic criteria has led to the underdiagnosis and underreporting of TRALI. In a 2007-2008 study, implementation of TRALI risk mitigation policies that utilized a predominantly male plasma supply indicated a significant reduction in the incidence of TRALI[18] (see Chapter 24 for more details).

OXYGEN SUPPLEMENTATION

The delivery of supplemental oxygen in the immediate postoperative period is usually routine for the prevention of possible hypoxemia. Still, the "optimal" perioperative oxygenation procedure remains controversial. Whether increased oxygenation delivery results in a reduction in the incidence of postoperative nausea and vomiting (PONV) and promotion of surgical wound healing is not clear.[19]

Oxygen Delivery

The choice of oxygen delivery systems in the PACU is determined by the degree of hypoxemia, the surgical procedure, and patient compliance. Patients who have undergone head and neck surgery may not be candidates for administration of oxygen via a face mask owing to the risk of pressure necrosis of incision sites and microvascular flaps, whereas nasal packing prohibits the use of nasal cannulas in others.

Delivery of oxygen by traditional nasal cannula should be limited to 6 L/min flow to minimize discomfort and complications that result from inadequate humidification. As a general rule each 1 L/min of oxygen flow through nasal cannula increases F_{IO_2} by 0.04, with 6 L/min resulting in approximately 0.44 F_{IO_2}.

Until recently maximum oxygen delivery to patients whose tracheas have been extubated required a nonrebreather mask or high-flow nebulizer. Delivery of oxygen via mask can be inefficient when mask fit is inadequate or large minute ventilation is required, which results in significant entrainment of room air. Alternatively, oxygen can be delivered up to 40 L/min by high-flow nasal cannulas. These high-flow nasal cannula delivery systems humidify and warm the gas to 99.9% relative humidity and 37° C. Unlike nonrebreather masks, these devices deliver oxygen directly to the nasopharynx throughout the respiratory cycle. The efficacy of these systems may be enhanced by a CPAP effect produced by the high gas flow.

Continuous Positive Airway Pressure and Noninvasive Positive-Pressure Ventilation

Approximately 8% to 10% of patients who undergo abdominal surgery require endotracheal intubation and mechanical ventilation for hypoxemia postoperatively. Application of CPAP in the PACU reduces the incidence of reintubation of the trachea, pneumonia, infection, and sepsis.[18-21] Even with the application of CPAP in the PACU, many patients will require additional ventilatory support. Ventilatory failure in the immediate postoperative period may result from many conditions including excessive intravascular volume, splinting due to pain,

diaphragmatic dysfunction, muscular weakness, and pharmacologically depressed respiratory drive.

Although the use of noninvasive positive-pressure ventilation (NPPV) in both chronic and acute respiratory failure is well established, there is limited experience with its application in the PACU. NPPV can be used in the PACU for patients with increased risk for pulmonary complications and as a rescue technique for patients in postoperative respiratory distress. NPPV is often avoided in the immediate postoperative period because of the potential for gastric distention, aspiration of gastric contents, and wound dehiscence, especially in patients who have undergone gastric or esophageal surgery. Thus, the decision to use noninvasive modes of ventilation in the PACU must be guided by careful consideration of both patient and surgical factors. Contraindications include hemodynamic instability or life-threatening arrhythmias, altered mental status, increased risk of aspiration of gastric contents, inability to use a nasal or face mask (head and neck procedures), and refractory hypoxemia. In the appropriate patient population, particularly for prophylactic use in patients following bariatric surgery and for patients in postoperative respiratory distress, NPPV is effective in avoiding endotracheal intubation in the PACU.[22]

HEMODYNAMIC INSTABILITY

Hemodynamic instability in the immediate postoperative period can have a negative impact on outcome. Surprisingly, postoperative systemic hypertension and tachycardia are more predictive of unplanned admission to the critical care unit and mortality rate than are hypotension and bradycardia.[23]

Systemic Hypertension

Patients with a history of essential hypertension are at greatest risk for significant systemic hypertension in the PACU. Additional factors include pain, hypoventilation and associated hypercapnia and hypoxia, emergence excitement, advanced age, a history of cigarette smoking, and preexisting renal disease (Box 39.5). Complications that may arise as a result of postoperative hypertension include myocardial ischemia, cardiac arrhythmia, congestive heart failure with pulmonary edema, stroke, and encephalopathy.[24] Acute postoperative hypertension increases the risk for intracranial hemorrhage following craniotomy and postoperative bleeding at the surgical site, and may compromise vascular anastomoses.[25] Surgical procedures that predispose the patient to postoperative hypertension include craniotomy, carotid endarterectomy, cardiothoracic procedures, and head and neck procedures.[24]

Box 39.5 Factors Leading to Postoperative Hypertension

- Arterial hypoxemia
- Preoperative essential hypertension
- Enhanced sympathetic nervous system activity—hypercapnia from hypoventilation, pain, gastric distention, bladder distention
- Hypervolemia
- Emergence agitation
- Shivering
- Drug or alcohol withdrawal—clonidine, β-blockers, narcotics
- Increased intracranial pressure

Box 39.6 Causes of Hypotension in the Postanesthesia Care Unit

- Intravascular fluid volume depletion
  - Ongoing fluid losses—bowel preparation, gastrointestinal losses, surgical bleeding
  - Increased capillary permeability—sepsis, burns, transfusion-related lung injury
- Decreased cardiac output
  - Myocardial ischemia/infarction
  - Cardiomyopathy
  - Valvular disease
  - Pericardial disease
  - Cardiac tamponade
  - Cardiac dysrhythmias
  - Pulmonary embolus
  - Tension pneumothorax
  - Drug-induced—β-blockers, calcium channel blockers
- Decreased vascular tone
  - Sepsis
  - Allergic reactions—anaphylactic, anaphylactoid
  - Spinal shock—cord injury, iatrogenic: spinal or epidural anesthesia
- Adrenal insufficiency

Systemic Hypotension

Postoperative hypotension may be characterized as (1) hypovolemic, (2) cardiogenic, or (3) distributive (Box 39.6). Regardless of the cause, postoperative hypotension can lead to decreased tissue perfusion and impaired end organ function and requires immediate attention (also see Chapter 5).

Hypovolemia (Decreased Preload)

Systemic hypotension in the PACU is usually due to decreased intravascular fluid volume and preload and, as such, responds favorably to intravenous fluid administration. The most common causes of decreased intravascular volume in the immediate postoperative period include ongoing third-space translocation of fluid, inadequate intraoperative fluid replacement (especially in patients who undergo major intra-abdominal procedures or preoperative bowel preparation), and loss of sympathetic

nervous system tone as a result of neuraxial (spinal or epidural) blockade (also see Chapter 23).

Persistent bleeding should be ruled out in hypotensive patients who have undergone a surgical procedure in which significant blood loss is possible. This is true regardless of the estimated intraoperative blood loss. If the patient is unstable, hemoglobin can be measured at the bedside to eliminate laboratory turnover time. It is also important to remember that tachycardia may not be a reliable indicator of hypovolemia or anemia (or both) if the patient is taking β-adrenergic or calcium channel blockers.

Cardiogenic Hypotension (Intrinsic Pump Failure)

Significant cardiogenic causes of postoperative systemic hypotension include myocardial ischemia and infarction, cardiomyopathy, and cardiac dysrhythmias. The differential diagnosis depends on the surgical procedure, intraoperative course, and the patient's preoperative medical condition.

Distributive Hypotension (Decreased Afterload)

Iatrogenic Sympathectomy

Iatrogenic sympathectomy secondary to regional anesthetic techniques is an important cause of hypotension in the PACU. An extensive sympathetic block (to T4) will decrease vascular tone and block the cardioaccelerator fibers. If not treated promptly, the resulting bradycardia in the presence of severe hypotension can lead to cardiac arrest, even in young healthy patients. Vasopressors, including phenylephrine and ephedrine, are pharmacologic treatments of hypotension caused by residual sympathetic nervous system blockade.

Critically Ill Patients

Critically ill patients may rely on exaggerated sympathetic nervous system tone to maintain systemic blood pressure and heart rate. In these patients even minimal doses of inhaled anesthetics, opioids, or sedative-hypnotics can decrease sympathetic nervous system tone and produce marked systemic hypotension.

Allergic Reactions

Allergic (anaphylactic or anaphylactoid) reactions may be the cause of hypotension in the PACU. These reactions are likely underreported and have an estimated incidence of 100 per 1 million procedures.[26] Anaphylaxis should be considered in all cases of sudden refractory extreme hypotension, even when not accompanied by the classic sequelae of bronchospasm and rash. Increased serum tryptase concentrations confirm the occurrence of an allergic reaction, but this change does not differentiate anaphylactic from anaphylactoid reactions. The blood specimen for tryptase determination must be obtained within 30 to 120 minutes after the allergic reaction, but the results may not be available for several days. Neuromuscular blocking drugs (also see Chapter 11) are the most common cause of anaphylactic reactions in the operative setting, followed by latex and antibiotics. Treatment begins with withdrawal of the triggering agent, and epinephrine is the drug of choice for severe reactions. Patients should receive counseling after a suspected anaphylactic reaction, and allergy testing is recommended 4 to 6 weeks after the initial reaction.[27]

Sepsis

If sepsis is suspected as the cause of hypotension in the PACU, blood should be obtained for culture, after which empiric antibiotic therapy should be initiated before transfer of the patient to the ward (also see Chapter 41). Urinary tract manipulations and biliary tract procedures are examples of interventions that can result in a sudden onset of severe systemic hypotension in the PACU. In these cases hypotension is often accompanied by fever and rigor.

Myocardial Ischemia

Detection of myocardial ischemia in the PACU can be challenging because of the patient's inability to identify or communicate symptoms related to cardiac ischemia. In one study, only approximately 35% of postoperative patients with myocardial infarction complained of typical chest pain.[28] The ASA Practice Guidelines for Postanesthesia Care recommend routine pulse, blood pressure, and ECG monitoring to detect cardiovascular complications such as myocardial ischemia.[2]

Low-Risk Patients

Interpretation of ST-segment changes on the ECG in the PACU should be interpreted in light of the patient's cardiac history and risk index. In low-risk patients (<45 years of age, no known cardiac disease, only one risk factor), postoperative ST-segment changes on the ECG do not usually indicate myocardial ischemia. Relatively benign causes of ST-segment changes in these low-risk patients include anxiety, esophageal reflux, hyperventilation, and hypokalemia. In general, low-risk patients require only routine PACU observation unless associated signs and symptoms warrant further clinical evaluation. A more aggressive evaluation is indicated if the changes are accompanied by cardiac rhythm disturbances, hemodynamic instability, angina, or associated symptoms.

High-Risk Patients

In contrast to low-risk patients, ST-segment and T-wave changes on the ECG in high-risk patients can be significant even in the absence of typical signs or symptoms of myocardial ischemia. In this patient population, any ST-segment, T-wave, or rhythm changes that are compatible with myocardial ischemia should prompt further

Box 39.7 Factors Leading to Postoperative Cardiac Dysrhythmias

- Hypoxemia
- Hypercarbia
- Intravascular volume shifts
- Pain, agitation
- Hypothermia
- Hyperthermia
- Anticholinesterases
- Anticholinergics
- Myocardial ischemia
- Electrolyte abnormalities
- Respiratory acidosis
- Hypertension
- Digitalis intoxication
- Preoperative cardiac dysrhythmias

evaluation to rule out myocardial ischemia. Determination of serum troponin levels is indicated when myocardial ischemia or infarction is suspected in the PACU. Once blood samples for measurement of troponin and a 12-lead ECG are completed, arrangements must be made for the appropriate cardiology follow-up.

Routine Postoperative 12-Lead Electrocardiogram and Troponin Measurement

Even small increases of troponin in the postoperative period are associated with an increased 30-day mortality rate, and there is currently no defined management strategy for these patients.[27-30] As a result, current American Heart Association/American College of Cardiology (AHA/ACC) guidelines cite insufficient evidence regarding routine postoperative 12-lead ECG or troponin measurements in patients at high risk for perioperative myocardial ischemia but without ongoing signs or symptoms of myocardial ischemia. These guidelines recommend against routine screening of an unselected patient population using troponin measurements.[31]

Cardiac Dysrhythmias

Perioperative cardiac dysrhythmias are frequently transient and multifactorial in cause (Box 39.7). Reversible causes of cardiac dysrhythmias in the perioperative period include hypoxemia, hypoventilation and associated hypercapnia, endogenous or exogenous catecholamines, electrolyte abnormalities, acidemia, excessive intravascular fluid, anemia, and substance withdrawal.

Tachydysrhythmias

Common causes of sinus tachycardia in the PACU include postoperative pain, agitation (rule out arterial hypoxemia), hypoventilation with associated hypercapnia, hypovolemia (continued postoperative bleeding), shivering, and the presence of a tracheal tube. Additional causes include cardiogenic or septic shock, pulmonary embolism, thyroid storm, and malignant hyperthermia.

Atrial Dysrhythmias

The incidence of new postoperative atrial dysrhythmias may be as high as 10% after major noncardiothoracic surgery. The incidence is even higher after cardiac and thoracic procedures when the cardiac dysrhythmia is often attributed to atrial irritation. These new-onset atrial dysrhythmias are not benign because they are associated with a longer hospital stay and increased mortality rate.[32]

Atrial Fibrillation

Control of the ventricular response rate is the immediate goal in the treatment of new-onset atrial fibrillation. Hemodynamically unstable patients may require prompt electrical cardioversion, but most patients can be treated pharmacologically with intravenous β-blocker or calcium channel blocker. Diltiazem is the calcium channel blocker of choice for patients in whom β-blockers are contraindicated. Ventricular rate control with these drugs is often enough to chemically cardiovert the postoperative patient whose arrhythmia may be catecholamine driven. If the goal of therapy is chemical cardioversion, an amiodarone infusion can be initiated in the PACU.

Ventricular Dysrhythmias

Ventricular tachycardia is uncommon, whereas premature ventricular contractions (PVCs) and ventricular bigeminy are common. PVCs often reflect increased sympathetic nervous system stimulation, as may accompany tracheal intubation and transient hypercapnia. True ventricular tachycardia is indicative of underlying cardiac disease, and in the case of torsades de pointes, QT-interval prolongation on the ECG may be intrinsic or drug related (amiodarone, procainamide, haloperidol [Haldol], or droperidol).

Bradydysrhythmias

Bradycardia in the PACU is often iatrogenic. Drug-related causes include β-adrenergic blocker therapy, neostigmine reversal of neuromuscular blockade, opioid administration, and treatment with dexmedetomidine. Procedure- and patient-related causes include bowel distention, increased intracranial or intraocular pressure, and spinal anesthesia. A high spinal block of cardioaccelerator fibers originating from T1 through T4 can produce severe bradycardia. The resulting sympathectomy, and possible intravascular volume depletion and associated decreased venous return can result in sudden cardiac arrest, even in young healthy patients.

Treatment

The urgency of treatment of a cardiac dysrhythmia depends on the physiologic consequences (principally systemic hypotension and myocardial ischemia) of the dysrhythmia. Tachydysrhythmia decreases diastolic and

coronary perfusion time and increases myocardial oxygen consumption. Its impact depends on the patient's underlying cardiac function, and it is most harmful in patients with coronary artery disease. Bradycardia has a more deleterious effect in patients with a fixed stroke volume, such as infants and patients with restrictive pericardial disease or cardiac tamponade.

DELIRIUM

Delirium is a transient disturbance in attention, awareness, and cognition that is not otherwise explained by another disorder or process (also see Chapter 35). The estimated incidence of postoperative delirium ranges from 4% to 75% of patients, depending on patient characteristics and type of surgery. The incidence is much higher for certain procedures, such as repair of a hip fracture, cardiac surgery, abdominal aneurysm repair, and bilateral knee replacement, as opposed to outpatient cataract surgery. Early recognition and treatment of postoperative delirium are important because it is associated with increased morbidity and mortality rates, length of stay, and cost.[33,34] Additionally, delirium following cardiac surgery was associated with prolonged cognitive impairment at 1 year follow-up, indicating that postoperative delirium is not just a short-term disorder.[35]

Risk Factors

Persistent postoperative delirium is generally a condition of elderly patients. In adults, patients should be screened in the preoperative setting to identify those at risk for postoperative delirium. Risk factors for postoperative delirium can be divided between predisposing factors, such as advanced age, and precipitating factors, such as medication administration or withdrawal (Box 39.8). Other intraoperative and postoperative factors that increase the likelihood of postoperative delirium include larger surgical blood loss and intraoperative blood transfusions, anemia, and use of a urinary bladder catheter.[34] Intraoperative hemodynamic derangements and the anesthetic technique do not seem to be predictors of postoperative delirium. Patients with an increased risk for postoperative delirium should be identified in the preoperative period using patient history, physical examination, and a cognitive screening tool such as the Mini-Cog.[36] Early identification of patients at risk for delirium can help guide management in the preoperative, intraoperative, and postoperative settings.

In addition, the workup of postoperative delirium must include evaluation to exclude arterial hypoxemia, hypercapnia, pain, sepsis, and electrolyte abnormalities (Box 39.9). Clinical evaluation of a delirious patient in the PACU includes a thorough evaluation of any underlying disease and metabolic derangements, such as hepatic- and renal-related encephalopathy.

Box 39.8 Risk Factors for Delirium

Predisposing
Reduced cognitive reserve: dementia, depression, advanced age
Reduced physical reserve: atherosclerotic disease, renal impairment, pulmonary disease, advanced age, preoperative β-blockade
Sensory impairment (vision, hearing)
Alcohol abuse
Malnutrition
Dehydration

Precipitating
Medications or medication withdrawal: anticholinergics, muscle relaxants, antihistamines, GI antispasmodics, opioid analgesics, antiarrhythmics, corticosteroids, more than six total medications, more than three new inpatient medications
Pain
Hypoxemia
Electrolyte abnormalities
Malnutrition
Dehydration
Environmental change (e.g., ICU admission)
Sleep-wake cycle disturbances
Urinary catheter use
Restraint use
Infection
Psychotropic medications: antidepressants, antiepileptics, antipsychotics, benzodiazepines

GI, Gastrointestinal; *ICU*, intensive care unit.

Box 39.9 Differential Diagnosis of Postoperative Delirium in the Postanesthesia Care Unit

Arterial hypoxemia
Preexisting cognitive disorder—Parkinson disease, baseline dementia
Hypoventilation with hypercapnia
Metabolic derangements—renal, hepatic, endocrine
Drugs—anticholinergics, benzodiazepines, opioids, β-blockers
Drug or ETOH withdrawal
Electrolyte abnormalities
Incomplete muscle relaxant reversal
Acute CNS event—hemorrhage, ischemic stroke
Infection
Seizures

CNS, Central nervous system; *ETOH*, ethanol.

Management

Management of postoperative delirium begins with nonpharmacologic treatments including withdrawal of any inciting stimulus and environmental modifications such as frequent reorientation. Pharmacologic treatment may be necessary for severely agitated patients, and the typical antipsychotic haloperidol (0.5 to mg IV) is considered first-line therapy if

no contraindications exist. Severely agitated patients may require restraints and additional personnel to control their behavior and avoid self-inflicted injury or dislodgement of intravascular catheters and the endotracheal tube.

Because the elderly population (also see Chapter 35) may become delirious from both pain and sedating drugs such as opioids, a multimodal pain relief strategy utilizing nonopioid medications may be beneficial in reducing the likelihood of postoperative delirium in this population. On the contrary, patients with tolerance to opioids may require increased opioid doses to treat pain and anxiety and avoid the onset of withdrawal.

Emergence Agitation

Emergence agitation is a transient period of excitation characterized by inconsolable crying, agitation, and delirium that is associated with emergence from general anesthesia. Emergence agitation is common in children, with more than 30% experiencing agitation or delirium at some period during their PACU stay (also see Chapter 34). The peak age of emergence agitation in children is between 2 and 4 years.

Unlike delirium, emergence agitation typically resolves quickly and is followed by uneventful recovery. Emergence excitement is more frequent with rapid "waking up" from inhaled anesthetics. In children, preoperative medication with midazolam may increase the incidence and duration of postoperative delirium, but whether midazolam is an independent factor or merely a reflection of other preoperative variables remains unclear.

RENAL DYSFUNCTION

The risk of postoperative acute kidney injury (AKI) ranges from 5% to 10%.[37] The differential diagnosis of postoperative renal dysfunction includes preoperative, intraoperative, and postoperative causes (Box 39.10) (also see Chapter 28). Frequently, the cause is multifactorial, with a preexisting renal insufficiency that is exacerbated by an intraoperative insult. For example, preoperative or intraoperative angiography can result in ischemic injury secondary to renal vasoconstriction and direct renal tubular injury. Intravascular volume depletion can exacerbate hepatorenal syndrome or acute tubular necrosis caused by sepsis. In the PACU, diagnostic efforts should focus on identification and treatment of the readily reversible causes of oliguria (urine output < 0.5 mL/kg/h). For example, urinary catheter obstruction or dislodgement is easily remedied and often overlooked.

Box 39.10 Causes of Postoperative Renal Dysfunction

Prerenal
Hypovolemia (bleeding, sepsis, third-space fluid loss, inadequate volume resuscitation)
Hepatorenal syndrome
Low cardiac output
Renal vascular obstruction or disruption
Intra-abdominal hypertension

Renal
Ischemia (acute tubular necrosis)
Radiographic contrast dyes
Rhabdomyolysis
Tumor lysis
Hemolysis

Postrenal
Surgical injury to the ureters
Obstruction of the ureters with clots or stones

Other
Mechanical (urinary catheter obstruction or malposition)

Oliguria

Postoperative Urinary Retention

The reported incidence of urinary retention in the PACU is between 5% and 70%. Clinical postoperative urinary retention is defined as the inability to void despite a bladder volume of more than 500 to 600 mL. Risk factors include age older than 50 years, male gender, volume of intraoperative intravascular fluid infusion, duration of surgery, and bladder volume on admission. Type of surgery is also predictive, with urinary retention occurring most commonly in anorectal and joint replacement surgery. Commonly used perioperative medications such as anticholinergics, β-blockers, and narcotics also contribute to urinary retention. Diagnosis can be made by clinical examination, bladder catheterization, or ultrasound assessment. Bladder volumes measured by ultrasound imaging correlate well with volumes obtained by urinary catheterization, an uncomfortable procedure that can be complicated by catheter-related infections and urethral trauma. Bladder ultrasound is an efficient and accurate method to evaluate patients at risk for oliguria.[38]

Decreased Intravascular Volume

The most common cause of oliguria in the immediate postoperative period is a decrease of intravascular volume. An intravascular fluid challenge (500 to 1000 mL of crystalloid) is usually effective in restoring urine output. The hematocrit should be measured when surgical blood loss is suspected and repeated intravascular boluses of fluid are required to maintain urine output. Resuscitation by intravenous administration of fluids to maximize renal perfusion is particularly important in order to prevent ongoing ischemic injury and the development of acute tubular necrosis.

If an intravascular fluid challenge is contraindicated or oliguria persists, assessment of intravascular volume or cardiac function is indicated to differentiate hypovolemia from sepsis and low cardiac output states. Fractional excretion of sodium can be useful in determining the adequacy of renal perfusion (assuming that diuretics have not been given), but the diagnosis of prerenal azotemia

will not differentiate between hypovolemia, congestive heart failure, or hepatorenal syndrome. In these cases evaluation with central venous monitoring or echocardiography may facilitate the diagnosis.

Intra-Abdominal Hypertension

Intra-abdominal hypertension (IAH) is a sustained measured intra-abdominal pressure higher than 12 mm Hg and should be considered as a cause of oliguria in patients following abdominal surgery, major trauma, or burns, and in those who are critically ill. Abdominal compartment syndrome is defined as sustained intra-abdominal pressure higher than 20 mm Hg that is associated with new organ dysfunction or failure.[39] In addition to cardiovascular effects, IAH may impede renal perfusion and lead to renal ischemia and postoperative renal dysfunction. Intra-abdominal pressure should be measured (via bladder pressure) in patients in whom intra-abdominal hypertension is suspected so that prompt intervention can be initiated to relieve intra-abdominal pressure and restore renal perfusion.

Rhabdomyolysis

Rhabdomyolysis is a possible cause of postoperative renal insufficiency in patients who have suffered major crush or thermal injury as well as with patients undergoing elective surgery. The incidence is increased in morbidly obese patients, particularly those having bariatric surgery. Risk factors include increased body mass index (BMI), prolonged duration of surgery, male gender, and patient positioning (lithotomy and lateral decubitus).[40] Patient history and the operative course should guide the decision to measure creatinine phosphokinase in the PACU. Severe postoperative pain is characteristic of myonecrosis and rhabdomyolysis, often in the areas of contact with the operating room table such as the gluteal, lumbar, and shoulder muscles. Volume loading, mannitol, and alkalinization of urine may help prevent rhabdomyolysis from progressing to AKI. Loop diuretics can be used to maintain urine output and avoid fluid overload.

Contrast Nephropathy

Angiography with intravascular stent placement is replacing open procedures to treat carotid stenosis, aortic aneurysms, and peripheral vascular disease. Patients undergoing these procedures often have chronic renal insufficiency and are at risk for developing renal failure secondary to intravenous contrast infusion. Management of these patients in the PACU includes particular attention to intravascular volume status in order to prevent AKI. Although aggressive hydration with normal saline provides the single most effective protection against contrast nephropathy, alkalinization with bicarbonate has been shown to provide additional protection. If bicarbonate is used for renal protection in this setting, 154 mEq/L should be infused at a rate of 1 mL/kg/h for 6 hours after the procedure. Mucomyst can be given and is a relatively inexpensive and easily administered medication (single oral dose before and after procedure) that may also provide renal protection.[27]

BODY TEMPERATURE AND SHIVERING

Postoperative shivering is a dramatic consequence of general and epidural anesthesia. The incidence of postoperative shivering may be as high as 65% (range 5% to 65%) after general anesthesia and 33% after epidural anesthesia. Identified risk factors include male gender and the choice of drug for induction of anesthesia (i.e., more likely with propofol than thiopental).

Mechanism

Postoperative shivering is usually associated with a decrease in the patient's body temperature. Although thermoregulatory mechanisms can explain shivering in a hypothermic patient, a separate mechanism has been proposed to explain shivering in normothermic patients. The proposed mechanism is based on the observation that the brain and spinal cord do not recover simultaneously from general anesthesia. The more rapid recovery of spinal cord function results in uninhibited spinal reflexes manifested as clonic activity. This theory is supported by the fact that doxapram, a central nervous system stimulant, is somewhat effective in abolishing postoperative shivering.

Treatment

Intervention includes the identification and treatment of hypothermia if present. In addition to shivering, mild to moderate hypothermia (33° to 35° C) inhibits platelet function, coagulation factor activity, and drug metabolism. It exacerbates postoperative bleeding, prolongs neuromuscular blockade, and may delay awakening. Shivering also increases oxygen consumption and is potentially detrimental in the postoperative patient with history of cardiac disease or limited reserve. Accurate core body temperatures can be most quickly and easily obtained using a temporal artery thermometer.[41] Forced air warmers can be used preoperatively to prevent hypothermia as well as to actively warm the hypothermic patient in the PACU.[42] A number of opioids and α_2-agonists are effective in abolishing shivering once it starts, but meperidine (12.5 to 25 mg IV) is the most effective treatment.

POSTOPERATIVE NAUSEA AND VOMITING

The consequences of PONV in the PACU include delayed discharge from the PACU, unanticipated hospital admission,

increased incidence of pulmonary aspiration, and significant postoperative discomfort and patient dissatisfaction. Therefore, the ability to identify high-risk patients for prophylactic intervention can significantly improve the quality of patient care and satisfaction in the PACU (also see Chapter 37).

High-Risk Patients

Risk factors for PONV can be grouped into three categories: patient, anesthetic, and surgery-related factors. A subset of factors has been established for each. The most significant patient-related factors include female gender (postpuberty), nonsmoking status, age less than 50 years, and history of motion sickness or PONV. Anesthesia-related factors include the use of volatile anesthetics or nitrous oxide and the administration of large doses of neostigmine and perioperative opioids. The most significant surgical risk factor is duration of surgery (Box 39.11).

Box 39.11 Factors Associated With Increased Incidence of Postoperative Nausea and Vomiting (PONV)

History of PONV or motion sickness
Female gender
Age less than 50 years
Postoperative opioids
Nonsmoking status
Type of surgery—eye muscle surgery, middle ear surgery, cholecystectomy, gynecologic surgery—laparoscopic approach
Duration of surgery
Anesthetic drugs—opioids, nitrous oxide, volatile anesthetics
Gastric distention—swallowed blood

High-risk patients can be identified by a simplified risk score consisting of four mostly patient-related factors: (1) female gender, (2) history of motion sickness or PONV, (3) nonsmoking, and (4) the use of postoperative opioids. The incidence of PONV correlates with the number of these factors present: zero, one, two, three, and four factors correspond to an incidence of 10%, 21%, 39%, 61%, and 79%, respectively.

Cost-effective management of PONV takes into consideration the patient's underlying risk. A single intervention in a patient with four risk factors will result in an absolute risk reduction of 21% compared with a 3% risk reduction in a patient with an initial risk of only 10%. These numbers correlate to a number of 5 and 40, respectively, needed to treat.[43]

Prevention and Treatment

Prophylactic measures against PONV include modification of the anesthetic technique and pharmacologic intervention. Strategies to reduce baseline risk include avoidance of general anesthesia by the use of regional anesthesia, preferential use of propofol infusions, avoidance of nitrous oxide and volatile anesthetics, minimization of giving postoperative opioids, and adequate hydration.[44] Although prophylactic measures to prevent PONV are clearly more effective than rescue, a subset of patients will require treatment in the PACU even after receiving appropriate prophylactic treatment. When choosing an antiemetic for these patients, both the class of drug and the timing of administration are important factors (Box 39.12). For instance, dexamethasone is effective when given prophylactically at

Box 39.12 Commonly Used Antiemetics, With Adult Doses

Anticholinergics
Scopolamine: transdermal patch, 1.5 cm^2
Apply to a hairless area behind the ear before surgery; remove 24 hours postoperatively

Antihistamines
Hydroxyzine: 12.5-25 mg IM

Phenothiazines
Promethazine: 12.5-25 mg IV/IM
Prochlorperazine: 2.5-10 mg IV/IM

Butyrophenones
Droperidol: 0.625-1.25 mg IV
See black box warning regarding torsades de pointes: monitor the ECG for prolongation of the QT interval for 2-3 hours after administration—preoperative 12-lead ECG recommended

Nk-1 Receptor Antagonists
Aprepitant: 40 mg PO prior to induction of anesthesia

Prokinetic
Metoclopramide: 10-20 mg IV
Minimal antiemetic properties, avoid in patients with any possibility of gastrointestinal obstruction

Serotonin Receptor Antagonists
Ondansetron: 4 mg IV 30 minutes before conclusion of surgery
Granisetron: 0.35-3 mg IV near the conclusion of surgery
Tropisetron: 2 mg IV near the conclusion of surgery
Palonsetron: 0.075 mg IV with induction of anesthesia
Dolasetron: 12.5 mg IV 15-30 minutes before conclusion of surgery (no longer marketed in the United States due to risk of QTc prolongation and torsades de pointes)

Corticosteroids
Dexamethasone: 4-8 mg IV with induction of anesthesia
Methylprednisolone: 40 mg IV with induction of anesthesia

Other Antiemetics
Propofol: subhypnotic doses such as 20 μg/kg/min IV infusion intraoperatively

ECG, Electrocardiogram; *IM*, intramuscularly; *IV*, intravenously; *PO*, by mouth.

the start of surgery, whereas serotonin receptor antagonists are effective when given near the end of anesthesia administration.

Upon admission to the PACU, the patient's risk profile and anesthetic technique should be noted, along with whether a prophylactic antiemetic was administered intraoperatively. If an adequate dose of antiemetic given at the appropriate time proves ineffective, simply giving more of the same class of drug in the PACU is unlikely to be of significant benefit. If no prophylactic drug was given, the recommended treatment is a low dose 5-HT_3 antagonist.

DELAYED AWAKENING

Even after prolonged surgery and anesthesia, a response to stimulation in 60 to 90 minutes should be expected. When delayed awakening occurs, the vital signs (systemic blood pressure, arterial oxygenation, ECG, body temperature) should be evaluated and a neurologic examination performed. Monitoring with pulse oximetry and analysis of arterial blood gases should be used to rule out hypoxemia and hypoventilation. Additional studies may be indicated to evaluate possible electrolyte derangements, metabolic disturbances, and hypoglycemia. Rarely, computed tomographic imaging is indicated to rule out an acute intracerebral event.

Treatment

Residual sedation from drugs used during anesthesia is the most frequent cause of delayed awakening in the PACU. If residual effects of opioids are a possible cause of delayed awakening, carefully titrated doses of naloxone (20- to 40-μg IV increments in adults) should be given, while keeping in mind that this treatment will also antagonize opioid-induced analgesia. Physostigmine may be effective in reversing the central nervous system sedative effects of anticholinergic drugs (especially scopolamine). Flumazenil is a specific antagonist for the residual depressant effects of benzodiazepines. In the absence of pharmacologic effects to explain delayed awakening, other causes, such as hypothermia (especially <33° C) and hypoglycemia, should be considered.

DISCHARGE CRITERIA

Specific PACU discharge criteria may vary, but certain general principles are universally applicable (Box 39.13) (also see Chapter 37). For example, a mandatory minimum stay in the PACU is not required. Patients must be observed until they are no longer at risk for ventilatory depression and their mental status is clear or has returned to baseline. Hemodynamic criteria are based on the patient's baseline hemodynamics without specific systemic blood pressure and heart rate requirements (Table 39.1).[2]

Box 39.13 General Principles for Discharge From the Postanesthesia Care Unit

Patients should be routinely required to have a responsible person accompany them home.
Requiring patients to urinate before discharge should not be part of a routine discharge protocol and may be necessary only in selected patients.
The demonstrated ability to drink and retain clear fluids should not be part of a routine discharge protocol but may be appropriate for selected patients.
A minimum mandatory stay in the unit should not be required.
Patients should be observed until they are no longer at increased risk for cardiorespiratory depression.

Table 39.1 Criteria for Determination of Discharge Score for Release From the Postanesthesia Care Unit

Variable Evaluated	Score
Activity	
Able to move four extremities on command	2
Able to move two extremities on command	1
Able to move no extremities on command	0
Breathing	
Able to breathe deeply and cough freely	2
Dyspnea	1
Apnea	0
Circulation (systemic blood pressure)	
Within 20% of the preanesthetic level	2
20% to 49% of the preanesthetic level	1
≥50% of the preanesthetic level	0
Consciousness	
Fully awake	2
Arousable	1
Not responding	0
Oxygen Saturation (pulse oximetry)	
>92% while breathing room air	2
Needs supplemental oxygen to maintain saturation >90%	1
<90% even with supplemental oxygen	0

Adapted from Aldrete JA. The post anaesthesia recovery score revisited. *J Clin Anesth*. 1995;7:89-91.

Table 39.2 Criteria for Determination of Discharge Score for Release Home to a Responsible Adult

Variable Evaluated	Score[a]
Vital signs (stable and consistent with age and preanesthetic baseline)	
Systemic blood pressure and heart rate within 20% of the preanesthetic level	2
Systemic blood pressure and heart rate 20% to 40% of the preanesthetic level	1
Systemic blood pressure and heart rate >40% of the preanesthetic level	0
Activity level	
Steady gait without dizziness or meets the preanesthetic level	2
Requires assistance	1
Unable to ambulate	0
Nausea and vomiting	
None to minimal	2
Moderate	1
Severe (continues after repeated treatment)	0
Pain (minimal to no pain, controllable with oral analgesics)	
Yes	2
No	1
Surgical bleeding (consistent with that expected for the surgical procedure)	
Minimal (does not require dressing change)	2
Moderate (up to two dressing changes required)	1
Severe (more than three dressing changes required)	0

[a]Patients achieving a score of at least 9 are ready for discharge.
Modified from Marshall SI, Chung F. Discharge criteria and complications after ambulatory surgery. *Anesth Analg*. 1999;88:508-517.

To facilitate PACU discharge, discharge scoring systems have been developed and modified over time to reflect current technology and anesthesia practice (Tables 39-1 and 39-2).[45] The ASA Standards of Care require that a physician accept responsibility for discharge of patients from the unit (Standard V). This is the case even when the decision to discharge the patient is made by the bedside nurse in accordance with the hospital-sanctioned discharge criteria or scoring system. If discharge scoring systems are to be used in this way, they must first be approved by the department of anesthesia and the hospital medical staff. A responsible physician's name must be noted on the record.

QUESTIONS OF THE DAY

1. What are the most likely causes of upper airway obstruction for a patient in the postanesthesia care unit (PACU)? What steps can be taken to differentiate the causes?
2. What are the potential manifestations of residual neuromuscular blockade for a patient who has just arrived in the PACU?
3. A patient who had prolonged surgery in the prone position arrives to the PACU with the trachea intubated. What steps can be taken to determine the presence of significant upper airway edema prior to extubation?
4. A patient with coronary artery disease is recovering in the PACU after noncardiac surgery. What monitoring should be done to evaluate for postoperative myocardial ischemia or infarction?
5. What factors can predict the risk of postoperative nausea and vomiting (PONV)? How does the degree of risk affect the approach to preventing and treating PONV?
6. What criteria can be used to determine whether a patient is ready for discharge from the PACU? What is the utility of scoring systems in making a discharge decision?

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