CHAPTER

56

Postanesthesia Care

KEY CONCEPTS

1. Formerly anesthetized patients should not leave the operating room unless they have a patent airway, have adequate ventilation and oxygenation, and are hemodynamically stable; qualified anesthesia personnel must also be available to attend the transfer.
2. Before the recovering patient is fully responsive, pain is often manifested as postoperative restlessness. Serious systemic disturbances (eg, hypoxemia, respiratory or metabolic acidosis, or hypotension), bladder distention, or a surgical complication (eg, occult intraabdominal hemorrhage) must also be considered in the differential diagnosis of postoperative agitation.
3. Intense shivering causes precipitous rises in oxygen consumption, CO_2 production, and cardiac output. These physiological effects are often poorly tolerated by patients with preexisting cardiac or pulmonary impairment.
4. Respiratory problems are the most frequently encountered serious complications in the postanesthesia care unit (PACU). The overwhelming majority are related to airway obstruction, hypoventilation, and/or hypoxemia.
5. Hypoventilation in the PACU is most commonly due to the residual depressant effects of anesthetic agents on respiratory drive.
6. Obtundation, circulatory depression, or severe acidosis (arterial blood pH <7.15) is an indication for immediate and aggressive respiratory and hemodynamic intervention, including airway and inotropic support as needed.
7. Following naloxone administration, patients should be observed closely for recurrence of opioid-induced respiratory depression ("renarcotization"), as naloxone has a shorter duration than do most opioids.
8. Increased intrapulmonary shunting from a decreased functional residual capacity relative to closing capacity is the most common cause of hypoxemia following general anesthesia.
9. The possibility of a postoperative pneumothorax should always be considered following central line placement, intercostal blocks, abdominal or chest trauma (including rib fractures), neck dissections, tracheostomy, nephrectomies, or other retroperitoneal or intraabdominal procedures (including laparoscopy), especially if the diaphragm may have been penetrated or disrupted.
10. Hypovolemia is by far the most common cause of hypotension in the PACU.
11. Noxious stimulation from incisional pain, endotracheal intubation, or bladder distention is usually responsible for postoperative hypertension.

Historically, emphasis on specialized nursing care during the immediate postoperative period was prompted by the realization that many early postoperative deaths occurred immediately after anesthesia and surgery and that many of these deaths were preventable. A nursing shortage in the United States following World War II, as well as the experience of providing surgical care to large numbers of battle casualties during the war, contributed to the postwar trend of centralization of immediate postoperative care in the form of recovery rooms, where one or more nurses could pay close attention to several acute postoperative patients at one time. Over the past two decades, the accelerating practice of caring for selected postoperative patients overnight in a postanesthesia care unit (PACU), or the equivalent, has been a response to increasingly complex surgical procedures performed on higher-acuity patients, often in the setting of a shortage of surgical intensive care beds. The success of PACUs in decreasing postoperative morbidity and mortality has been a major influence on the evolution of modern surgical intensive care units.

Another recent transformation in postanesthesia care is related to the shift from inpatient to outpatient surgery. It is estimated that more than 70% of all surgical procedures in the United States are now performed on an outpatient basis. Two phases of recovery may be recognized for outpatient surgery. *Phase 1* is the immediate intensive care level recovery that cares for patients during emergence and awakening from anesthesia and continues until standard PACU criteria are met (see Discharge Criteria below). *Phase 2* is a lower-level care that ensures that the patient is ready to go home. "Fast-tracking" of selected outpatients may allow them to safely bypass phase 1 recovery and go directly to the phase 2 level of care.

In many institutions, the PACU also commonly functions as a more intensely monitored location for perioperative and chronic pain patients undergoing procedures such as single-shot nerve blocks and placement of epidural and peripheral nerve catheters, and for patients undergoing other procedures such as central line placement, electroconvulsive therapy, and elective cardioversion. The PACU must be appropriately staffed and equipped to routinely manage these patients and their potential procedure-related complications. For example, in areas where regional and epidural blocks are administered, Intralipid® should be stocked in anticipation of treating local anesthetic toxicity.

This chapter discusses the essential components of a modern PACU, the general care of patients acutely recovering from anesthesia and surgery, and the respiratory and circulatory complications most commonly encountered in the PACU.

THE POSTANESTHESIA CARE UNIT

At the conclusion of any procedure requiring anesthesia, anesthetic agents are discontinued, monitors are disconnected, and the patient emerging from sedation or anesthesia is taken to the PACU. Following general anesthesia, if an endotracheal tube or laryngeal mask airway (LMA) was utilized, and if ventilation is judged adequate, the endotracheal tube or LMA is usually removed prior to transport. Patients are also routinely observed in the PACU following regional anesthesia and monitored anesthesia care (local anesthesia with sedation). Most procedure guidelines require that a patient be admitted to the PACU following any type of anesthesia, except by specific order of the attending anesthesiologist. After a brief verbal (and in some cases written) "hand off" report to the PACU nurse, the patient is left in the PACU until the major effects of anesthesia have worn off. This period is characterized by a relatively high incidence of potentially life-threatening respiratory and circulatory complications.

The delivery of anesthesia services in areas remote from the main operating room, such as endoscopy, interventional radiology, and magnetic resonance imaging suites is increasingly common. Patients recovering from anesthesia delivered in these areas must receive the same standard of care as patients recovering from anesthesia received in the main operating room. Some institutions have developed "satellite" PACUs to serve each of these remote areas individually, and others have combined their procedural areas into one centralized procedural suite served by a single PACU.

Design

The PACU should be located near the operating rooms and off-site invasive procedure areas. A central location in the operating room area itself is desirable, as it ensures that the patient can be rushed back to surgery, if needed, or that members of the operating room team can quickly respond to urgent or emergent patient care issues. Proximity to radiographic, laboratory, and other intensive care facilities on the same floor is also advantageous. The transfer of critically ill patients in elevators or through long corridors can jeopardize their care because urgent problems may arise along the way.

An open-ward design facilitates observation of multiple patients simultaneously. However, an appropriate number of individually enclosed patient care spaces is required for patients needing isolation for infection control. A ratio of 1.5 PACU beds per operating room is customary, although this number will vary depending on the respective operating room suite's case volume, variety of surgical procedures, and patient acuity. Each patient space should be well-lighted and large enough to allow easy access to patients in spite of poles for intravenous infusion pumps, a ventilator, or radiographic equipment; construction guidelines dictate a minimum of 7 ft between beds and 120 sq ft/patient. Multiple electrical outlets, including at least one with backup emergency power, and at least one outlet each for oxygen and suction, should be present at each bed space.

Equipment

Many PACU incidents leading to serious morbidity or mortality are related to inadequate monitoring. Pulse oximetry (Spo_2), electrocardiogram (ECG), and automated noninvasive blood pressure (NIBP) monitors are mandatory for each space. Although ECG, Spo_2, and NIBP must be utilized for every patient in the initial phase of recovery from anesthesia (phase 1 care), decreased monitoring may be adequate thereafter. Appropriate equipment must be available for those patients requiring invasive arterial, central venous, pulmonary artery, or intracranial pressure monitoring. Capnography is useful for intubated patients and is increasingly employed for extubated patients as well. Temperature-sensitive strips may be used to measure temperature in the PACU but are not sufficiently accurate to document the results of treatment for hypothermia or hyperthermia; mercury or electronic thermometers must be used if an abnormality in temperature is suspected. A forced-air warming device, heating lamp, and/or a warming/cooling blanket should be available.

The PACU must have its own supplies of basic and emergency equipment, separate from that of the operating room, based on the needs of the patient population. This includes airway equipment and supplies, such as oxygen cannulas, a selection of masks, oral and nasal airways, laryngoscopes, endotracheal tubes, LMAs, a cricothyrotomy kit, and self-inflating bags for ventilation. A readily available supply of catheters for vascular cannulation (venous, arterial, central venous) is mandatory. A defibrillation device with transcutaneous pacing capabilities, and an emergency cart with drugs and supplies for advanced life support (see Chapter 55) and infusion pumps, must be present and periodically inspected. Transvenous pacing catheters; pulse generators; and tracheostomy, chest tube, and vascular cut-down trays are typically present, depending on the surgical patient population.

Respiratory therapy equipment for aerosol bronchodilator treatments, continuous positive airway pressure (CPAP), and ventilators should be in close proximity to the recovery room. Immediate availability of a bronchoscope is desirable.

Staffing

Inadequate staffing is often cited as a major contributing factor in PACU mishaps. The PACU should be staffed by nurses specifically trained in the care of adult and/or pediatric patients emerging from anesthesia. They should have expertise in airway management and advanced cardiac life support, as well as problems commonly encountered in surgical patients relating to wound care, drainage catheters, and postoperative bleeding.

Patients in the PACU should be under the medical direction of an anesthesiologist, who must be immediately available to respond to urgent or emergent patient care problems. High-volume tertiary care surgical institutions often have an anesthesiologist assigned full time to the PACU. The management of

the patient in the PACU should reflect a coordinated effort involving anesthesiologists, surgeons, nurses, respiratory therapists, and appropriate consultants. The anesthesia team emphasizes management of analgesia, airway, cardiac, pulmonary, and metabolic problems, whereas the surgical team generally manages any problems directly related to the surgical procedure itself. Based on the assumptions that the average PACU stay is 1 hr and that the average inpatient procedure lasts 2–3 hr, a ratio of one recovery nurse for two patients is generally satisfactory. However, staffing for nursing care should be tailored to the unique caseload requirements of each facility. If the operating room schedule regularly includes pediatric patients or frequent short procedures, a ratio of one nurse to one patient is often needed. A charge nurse should be assigned to ensure optimal staffing at all times, including the appropriate response to urgent or emergent patient care problems.

Care of the Patient

EMERGENCE FROM GENERAL ANESTHESIA

Recovery from general or regional anesthesia is a time of great physiological stress. Emergence from general anesthesia should ideally be characterized by a smooth and gradual awakening in a controlled environment. However, problems such as airway obstruction, shivering, agitation, delirium, pain, nausea and vomiting, hypothermia, and autonomic lability are frequently encountered. Patients receiving spinal or epidural anesthesia may experience decreases in blood pressure during transport or recovery; the sympatholytic effects of major conduction blocks prevent compensatory reflex vasoconstriction when patients are moved or when they sit up.

Following an inhalational-based anesthetic, the speed of emergence is directly proportional to alveolar ventilation, but inversely proportional to the agent's blood solubility (see Chapter 8). As the duration of anesthesia increases, emergence also becomes increasingly dependent on total tissue uptake, which is a function of agent solubility, the average concentration used, and the duration of exposure to the anesthetic. Hypoventilation delays emergence from inhalational anesthesia.

Emergence from an intravenous anesthetic is a function of its pharmacokinetics. Recovery from most intravenous anesthetic agents is dependent primarily on redistribution rather than metabolism and elimination. As the total administered dose increases, however, cumulative effects become clinically apparent in the form of prolonged emergence; the termination of action becomes increasingly dependent on the metabolism or elimination. This is the basis for the concept of a context-sensitive half-time (see Chapter 7). Advanced age or renal or hepatic disease can prolong emergence (see Chapter 9). Short and ultrashort-acting anesthetic agents, such as propofol and remifentanil, significantly shorten emergence, time to awakening, and discharge. Some studies show that the use of a Bispectral Index Scale (BIS) monitor (see Chapter 6) may reduce total drug dosage and shorten recovery and time to discharge. LMA (rather than an endotracheal tube) use may also allow lighter levels of anesthesia that could speed emergence.

The speed of emergence can also be influenced by preoperative medications. Premedication with agents that outlast the procedure (eg, lorazepam) may be expected to prolong emergence. The short duration of action of midazolam makes it a suitable premedication agent for short procedures. The effects of preoperative sleep deprivation or drug ingestion (alcohol, sedatives) can also be additive to those of anesthetic agents and can prolong emergence.

Delayed Emergence

The most frequent cause of delayed emergence (when the patient fails to regain consciousness 30–60 min after general anesthesia) is residual anesthetic, sedative, and analgesic drug effect. Delayed emergence may occur as a result of absolute or relative drug overdose or potentiation of anesthetic agents by prior drug or alcohol ingestion. Naloxone (in 80 mcg increments in adults) and flumazenil (in 0.2 mg increments in adults) will readily reverse the effects of an opioid and benzodiazepine, respectively. Physostigmine (1–2 mg) may partially reverse the effect of other agents. A nerve stimulator can be used

to exclude persisting neuromuscular blockade in poorly responsive patients on a mechanical ventilator who have inadequate spontaneous tidal volumes.

Less common causes of delayed emergence include hypothermia, marked metabolic disturbances, and perioperative stroke. A core temperature of less than 33°C has an anesthetic effect and greatly potentiates the actions of central nervous system depressants. Forced-air warming devices are most effective in raising body temperature. Hypoxemia and hypercarbia are readily excluded by pulse oximetry, capnography, and/or blood gas analysis. Hypercalcemia, hypermagnesemia, hyponatremia, and hypoglycemia and hyperglycemia are rare causes of delayed emergence that require laboratory measurements for diagnosis. Perioperative stroke is rare, except after neurological, cardiac, and cerebrovascular surgery (see Chapter 28); diagnosis is facilitated by neurological evaluation and radiological imaging.

TRANSPORT FROM THE OPERATING ROOM TO THE PACU

This seemingly short period may be complicated by the lack of adequate monitoring, medication access, or resuscitative equipment. Formerly anesthetized patients should not leave the operating room unless they have a patent airway, have adequate ventilation and oxygenation, and are hemodynamically stable; qualified anesthesia personnel must attend the transfer. Supplemental oxygen should be administered during transport to patients at risk of hypoxemia. Some studies suggest that transient hypoxemia (Spo_2 <90%) may develop in as many as 30% to 50% of otherwise "normal" patients during transport while breathing room air; supplemental oxygen may therefore be advisable for all transported patients, especially if the PACU is not in immediate proximity to the operating room. Unstable patients should remain intubated and should be transported with a portable monitor (ECG, Spo_2, and blood pressure) and a supply of emergency drugs.

All patients should be taken to the PACU on a bed or gurney that can be placed in either the head-down (Trendelenburg) or back-up position. The head-down position is useful for hypovolemic patients, whereas the back-up position is useful for patients with underlying pulmonary dysfunction (see Chapters 20 and 23). Patients at increased risk of vomiting or upper airway bleeding (eg, following tonsillectomy) should be transported in the lateral position. This position also helps prevent airway obstruction and facilitates drainage of secretions.

ROUTINE RECOVERY

General Anesthesia

Airway patency, vital signs, oxygenation, and level of consciousness must be assessed immediately upon PACU arrival. Subsequent blood pressure, heart rate, and respiratory rate measurements are routinely made at least every 5 min for 15 min or until stable, and every 15 min thereafter. Pulse oximetry should be monitored continuously in all patients. The occurrence of hypoxemia does not necessarily correlate with the level of consciousness. Neuromuscular function should be assessed clinically (eg, head-lift and grip strength). At least one temperature measurement must also be obtained. Additional monitoring includes pain assessment (eg, numerical or descriptive scales); the presence or absence of nausea or vomiting; and fluid input and output, including urine flow, drainage, and bleeding. After initial vital signs have been recorded, the anesthesia provider should give a brief report to the PACU nurse that includes (1) the preoperative history (including mental status and any communication problems, such as language barriers, deafness, blindness, or mental disability); (2) pertinent intraoperative events (type of anesthesia, the surgical procedure, blood loss, fluid replacement, antibiotic and other relevant medication administration, and any complications); (3) expected postoperative problems; (4) anticipated need for PACU medication administration, such as antibiotics; and (5) postanesthesia orders (analgesia and nausea/vomiting therapy; epidural or perineural catheter care; including the need for acute pain service involvement, administration of fluids or blood products, postoperative ventilation, chest x-ray for follow-up of central venous catheterization, etc.).

All patients recovering from general anesthesia must receive supplemental oxygen and pulse oximetry monitoring during emergence because transient hypoxemia can develop even in healthy patients. A rational decision regarding continuation of supplemental oxygen therapy at the time of PACU discharge can be made based on Spo_2 readings on room air. Arterial blood gas measurements may be obtained to confirm abnormal oximetry readings, but are not necessary in most patients. Oxygen therapy should be carefully controlled in patients with chronic obstructive pulmonary disease and a history of, or potential for, CO_2 retention. Patients should generally be nursed in the back-up position, whenever possible, to optimize oxygenation. However, elevating the head of the bed before the patient is responsive can lead to airway obstruction. In such cases, the oral or nasal airway should be left in place until the patient is awake and able to maintain airway. Deep breathing and coughing should be encouraged periodically.

Regional Anesthesia

Patients who are heavily sedated or hemodynamically unstable following regional anesthesia should also receive supplemental oxygen in the PACU. Sensory and motor levels should be periodically recorded following regional anesthesia to document regression of the block. Precautions in the form of padding or repeated warning may be necessary to prevent self-injury from uncoordinated arm movements following brachial plexus blocks. Blood pressure should be closely monitored following spinal and epidural anesthesia. Bladder catheterization may be necessary in patients who have had spinal or epidural anesthesia for longer than 4 hr.

Pain Control

Moderate to severe postoperative pain is most commonly treated with oral or parenteral opioids. However, perioperative opioid administration is associated with side effects (nausea and vomiting, respiratory depression, pruritis, ileus, and urinary retention) which may have significant adverse effects on postoperative convalescence. In response to this problem, a variety of *opioid sparing* strategies have been increasingly embraced over the past two decades to decrease opioid requirements, and thus opioid-related side effects, while maintaining satisfactory analgesia (see Chapter 47). Preoperative oral administration of nonsteroidal antiinflammatory drugs (NSAIDs), acetaminophen, and gabapentin or pregabalin may significantly reduce postoperative opioid requirements, and these medications may be resumed postoperatively if the patient can continue oral medication. Additional analgesic modalities utilizing local anesthetics, such as intraoperative wound infiltration, postoperative wound catheter infusions, single-shot and continuous catheter peripheral nerve blocks, and continuous epidural infusions, also reduce postoperative opioid analgesic requirements, and thus also reduce opioid-related side effects.

Mild to moderate postoperative pain can be treated orally with acetaminophen, ibuprofen, hydrocodone, or oxycodone. Alternatively, ketorolac tromethamine (15–30 mg in adults) or acetaminophen (15 mg/kg, or 1 g if patient >50 kg) may be administered intravenously.

In situations where moderate to severe postoperative pain is present, or oral analgesia is not possible, parenteral or intraspinal opioids, single-shot or continuous nerve blocks, and continuous epidural analgesia are used, often in combination techniques. Parenteral opioids are most safely administered by titration of small doses. Considerable variability in opioid requirements should be expected in surgical patients recovering in the PACU, and adequate analgesia must be balanced against the risk of excessive sedation and respiratory depression. Opioids of intermediate to long duration, such as hydromorphone 0.25–0.5 mg (0.015–0.02 mg/kg in children) or morphine 2–4 mg (0.025–0.05 mg/kg in children), are most commonly used. Meperidine is most often used in small doses to treat postoperative shivering. Opioid requirements are often markedly increased in patients with a history of chronic pain and chronic opioid therapy, because of opioid tolerance, and in patients with a history of opioid addiction, because of opioid tolerance and psychological dependence. Consultation with a pain specialist is often extremely helpful in these situations.

Analgesic effects of parenteral opioids usually peak within minutes of administration. Maximal

respiratory depression, particularly with morphine and hydromorphone, may not occur until 20–30 min later. When the patient is fully awake, patient-controlled analgesia can be instituted for inpatients. Intramuscular administration of opioids is discouraged because delayed and variable onset (10–20 min or longer) and delayed respiratory depression (up to 1 h).

When an epidural catheter is used, epidural bolus administration of fentanyl (50–100 mcg) or sufentanil (20–30 mcg) with 5–10 mL of 0.1% bupivacaine can provide excellent pain relief in adults. Epidural morphine (3–5 mg) may also be used, but delayed respiratory depression with epidural administration of this opioid mandates close monitoring for 24 hr afterward (see Chapter 48).

Agitation

2 Before the recovering patient is fully responsive, pain is often manifested as postoperative restlessness. Serious systemic disturbances (such as hypoxemia, respiratory or metabolic acidosis, or hypotension), bladder distention, or a surgical complication (such as occult intraabdominal hemorrhage) must also be considered in the differential diagnosis of postoperative agitation. Marked agitation may necessitate arm and leg restraints to avoid self-injury, particularly in children. When serious physiological disturbances have been excluded in children, cuddling and kind words from a sympathetic attendant or the parents often calms the pediatric patient. Other contributory factors include marked preoperative anxiety and fear, as well as adverse drug effects (large doses of central anticholinergic agents, phenothiazines, or ketamine). Physostigmine 1–2 mg intravenously (0.05 mg/kg in children) is most effective in treating delirium due to atropine and scopolamine. If serious systemic disturbances and pain are excluded, persistent agitation may require sedation with intermittent intravenous doses of midazolam 0.5–1 mg (0.05 mg/kg in children).

Nausea & Vomiting

Postoperative nausea and vomiting (PONV) is common following general anesthesia, occurring in 30% to 40% of all patients. Moreover, PONV occurs at home within 24 hr of an uneventful discharge (postdischarge nausea and vomiting) in a significant number of ambulatory surgery patients. The etiology of PONV is usually multifactorial and associated with anesthetic and analgesic agents, the type of surgical procedure, and intrinsic patient factors, such as a history of motion sickness. It is also important to recognize that nausea is a common complaint reported at the onset of hypotension, particularly following spinal or epidural anesthesia.

TABLE 56–1 Risk factors for postoperative nausea and vomiting.

Patient factors
Young age
Female gender, particularly if menstruating on day of surgery or in first trimester of pregnancy
Large body habitus
History of prior postoperative emesis
History of motion sickness
Anesthetic techniques
General anesthesia
Drugs
Opioids
Volatile agents
Nitrous oxide
Surgical procedures
Strabismus surgery
Ear surgery
Laparoscopy
Orchiopexy
Ovum retrieval
Tonsillectomy
Breast surgery
Postoperative factors
Postoperative pain
Hypotension

Table 56–1 lists commonly recognized risk factors for PONV. An increased incidence of nausea and vomiting is reported following opioid administration and intraperitoneal (especially laparoscopic), breast, and strabismus surgery. The greatest incidence seems to be in young women; nausea may be more common during menstruation. Increased vagal tone manifested as sudden bradycardia commonly precedes, or coincides with, emesis. Propofol anesthesia decreases the incidence of PONV, and a preoperative history of smoking lessens the

likelihood of PONV. Selective 5-hydroxytryptamine (serotonin) receptor 3 (5-HT_3) antagonists, such as ondansetron 4 mg (0.1 mg/kg in children), granisetron 0.01–0.04 mg/kg, and dolasetron 12.5 mg (0.035 mg/kg in children), are effective in preventing PONV, and, to a lesser extent, in treating established PONV. It should be noted that unlike ondansetron, which is usually effective immediately, dolasetron requires 15 min for onset. An orally disintegrating tablet preparation of ondansetron (8 mg) may be useful for treatment and prophylaxis against postdischarge nausea and vomiting. Metoclopramide, 0.15 mg/kg intravenously, is a less effective alternative to 5-HT_3 antagonists. 5-HT_3 antagonists are not associated with the acute extrapyramidal (dystonic) manifestations and dysphoric reactions that may be encountered with metoclopramide or phenothiazine-type antiemetics. Transdermal scopolamine is effective, but can be associated with side effects, such as sedation, dysphoria, blurred vision, dry mouth, urinary retention, and exacerbation of glaucoma, particularly in elderly patients. Dexamethasone 4–10 mg (0.10 mg/kg in children), when utilized as an antiemetic, has the additional advantages of providing a varying degree of analgesia and a sense of patient well-being. Moreover, it seems to be effective for up to 24 hr, and, thus, may be useful for postdischarge nausea and vomiting. Oral aprepitant (Emend®) 40 mg may be administered within 3 hr prior to anesthesia induction. Intravenous droperidol 0.625–1.25 mg (0.05–0.075 mg/kg in children), when given intraoperatively, significantly decreases the likelihood of PONV. Unfortunately, droperidol carries a US Food and Drug Administration "black box" warning, indicating that large (5–15 mg) doses can prolong the QT interval and have been associated with fatal cardiac arrhythmias. Nonpharmacological prophylaxis against PONV includes ensuring adequate hydration (20 mL/kg) after fasting, and stimulation of the P6 acupuncture point (wrist). The latter may include application of pressure, electrical current, or injections.

Controversy exists regarding routine PONV prophylaxis for all patients. Because of the cost of treatment of established PONV, it may be cost-effective to provide prophylaxis to all patients in certain populations (eg, outpatients). Clearly, patients with multiple risk factors should receive prophylaxis. In addition, the use of two or three agents that act on differing receptors is more effective than single-agent prophylaxis.

Shivering & Hypothermia

Shivering can occur in the PACU as a result of intraoperative hypothermia or the effects of anesthetic agents, and it is also common in the immediate postpartum period. The most important cause of hypothermia is a redistribution of heat from the body core to the peripheral compartments (see Chapter 6). A relatively cool ambient operating room temperature, prolonged exposure of a large wound, and the use of large amounts of unwarmed intravenous fluids or high flows of unhumidified gases can also be contributory. Nearly all anesthetics, particularly volatile agents and spinal and epidural anesthesia, decrease the normal vasoconstrictive response to hypothermia by decreasing sympathetic tone. Although anesthetic agents also decrease the shivering threshold, shivering commonly observed during or after emergence from general anesthesia represents the body's effort to increase heat production and raise body temperature and may be associated with intense vasoconstriction. Emergence from even brief general anesthesia is sometimes also associated with shivering, and although the shivering can be one of several nonspecific neurological signs (posturing, clonus, or Babinski's sign) that are sometimes observed during emergence, it is most often due to hypothermia. Regardless of the mechanism, its incidence seems to be related to the duration of surgery and the use of a volatile agent. Shivering may occasionally be sufficiently intense to cause hyperthermia (38–39°C) and significant metabolic acidosis, both of which promptly resolve when the shivering stops. Other causes of shivering should be excluded, such as bacteremia and sepsis, drug allergy, or transfusion reaction.

Hypothermia should be treated with a forced-air warming device, or (less satisfactorily) with warming lights or heating blankets, to raise body temperature to normal. Intense shivering causes precipitous rises in oxygen consumption, CO_2 production, and cardiac output. These physiological effects are often poorly tolerated by patients with preexisting

cardiac or pulmonary impairment. Hypothermia has been associated with an increased incidence of myocardial ischemia, arrhythmias, increased transfusion requirements due to coagulopathy, and increased duration of muscle relaxant effects. Small intravenous doses of meperidine (10–25 mg) can dramatically reduce or even stop shivering. Intubated and mechanically ventilated patients can also be sedated and given a muscle relaxant until normothermia is reestablished by active rewarming and the effects of anesthesia have dissipated.

Discharge Criteria

A. PACU

All patients must be evaluated by a qualified anesthesia provider prior to discharge from the PACU unless strict discharge criteria are adopted. Standards for discharging patients from the PACU are established by the department of anesthesiology and the hospital medical staff. They may allow PACU nurses to determine when patients may be transferred without the presence of a qualified anesthesia provider if all PACU discharge criteria have been met. Criteria can vary according to whether the patient is going to be discharged to an intensive care unit, a regular ward, the outpatient department (phase 2 recovery), or directly home.

Before discharge, patients should have been observed for respiratory depression for at least 20–30 min after the last dose of parenteral opioid. Other minimum discharge criteria for patients recovering from general anesthesia usually include the following:

1. Easy arousability
2. Full orientation
3. The ability to maintain and protect the airway
4. Stable vital signs for at least 15–30 min
5. The ability to call for help, if necessary
6. No obvious surgical complications (such as active bleeding)

Postoperative pain and nausea and vomiting must be controlled, and normothermia should be reestablished prior to PACU discharge. Scoring systems are widely used. Most assess Spo_2 (or color), consciousness, circulation, respiration, and motor activity (Table 56–2). The majority of patients can meet discharge criteria within 60 min from the time of PACU arrival. Patients to be transferred to other intensive care areas need not meet all requirements.

In addition to the above criteria, patients receiving regional anesthesia should also be assessed for regression of both sensory and motor blockade. Complete resolution of the block prior to PACU dismissal avoids inadvertent injuries due to motor weakness or sensory deficits; however,

TABLE 56–2 Postanesthetic Aldrete recovery score.[1,2]

Original Criteria	Modified Criteria	Point Value
Color	**Oxygenation**	
Pink	Spo_2 > 92% on room air	2
Pale or dusky	Spo_2 > 90% on oxygen	1
Cyanotic	Spo_2 < 90% on oxygen	0
Respiration		
Can breathe deeply and cough	Breathes deeply and coughs freely	2
Shallow but adequate exchange	Dyspneic, shallow or limited breathing	1
Apnea or obstruction	Apnea	0
Circulation		
Blood pressure within 20% of normal	Blood pressure ± 20 mm Hg of normal	2
Blood pressure within 20% to 50% of normal	Blood pressure ± 20–50 mm Hg of normal	1
Blood pressure deviating >50% from normal	Blood pressure more than ± 50 mm Hg of normal	0
Consciousness		
Awake, alert, and oriented	Fully awake	2
Arousable but readily drifts back to sleep	Arousable on calling	1
No response	Not responsive	0
Activity		
Moves all extremities	Same	2
Moves two extremities	Same	1
No movement	Same	0

[1]Data from Aldrete JA, Kronlik D: A postanesthetic recovery score. Anesth Analg 1970;49:924 and Aldrete JA: The post-anesthesia recovery score revisited. J Clin Anesth 1995;7:89.

[2]Ideally, the patient should be discharged when the total score is 10, but a minimum of 9 is required.

many institutions have protocols that allow earlier discharge to appropriately monitored areas, and patients may be discharged with peripheral nerve blocks from single-shot or continuous perineural catheter infusions for the purpose of regional analgesia. Documenting regression of a block is important. Failure of a spinal or epidural block to resolve 6 hr after the last dose of local anesthetic raises the possibility of spinal subdural or epidural hematoma, which should be excluded by prompt radiological imaging and neurologic evaluation.

In some centers, outpatients who meet the above discharge criteria when they come out of the operating room may be "fast-tracked," bypassing the PACU and proceeding directly to the phase 2 recovery area. Similarly, inpatients who meet the same criteria may be transferred directly from the operating room to their ward, if appropriate staffing and monitoring is present.

B. Outpatients

In addition to emergence and awakening, recovery from anesthesia following outpatient procedures includes two additional stages: home readiness (phase 2 recovery) and complete psychomotor recovery. A scoring system has been developed to help assess home readiness discharge (Table 56–3). Recovery of proprioception, sympathetic tone, bladder function, and motor strength are additional criteria following regional anesthesia. For example, intact proprioception of the big toe, minimal orthostatic blood pressure and heart rate changes, and normal plantar flexion of the foot are important signals of recovery following spinal anesthesia. Urination and drinking or eating before discharge are usually no longer required; exceptions include patients with a history of urinary retention and those with diabetes.

All outpatients must be discharged home in the company of a responsible adult who will stay with them overnight (the latter is required if they have received an anesthetic). Patients must be provided with written postoperative instructions on how to obtain emergency help and to perform routine follow-up care. The assessment of home readiness is the responsibility of the qualified anesthesia provider, preferably one who is already familiar with the patient, although authority to discharge a patient home can be delegated to a nurse, if approved discharge criteria are applied.

Home readiness does not imply that the patient has the ability to make important decisions, to drive, or to return to work. These activities require complete psychomotor recovery, which is often not achieved until 24–72 hr postoperatively. All outpatient centers must use some system of postoperative follow-up, preferably phone contact the day after discharge.

TABLE 56–3 Postanesthesia discharge scoring system (PADS).[1,2]

Criteria	Points
Vital signs	
Within 20% of preoperative baseline	2
Within 20% to 40% of preoperative baseline	1
>40% of preoperative baseline	0
Activity level	
Steady gait, no dizziness, at preoperative level	2
Requires assistance	1
Unable to ambulate	0
Nausea and vomiting	
Minimal, treated with oral medication	2
Moderate, treated with parenteral medication	1
Continues after repeated medication	0
Pain: minimal or none, acceptable to patient, controlled with oral medication	
Yes	2
No	1
Surgical bleeding	
Minimal: no dressing change required	2
Moderate: up to two dressing changes	1
Severe: three or more dressing changes	0

[1]Modified from Marshall SI, Chung F: Discharge criteria and complications after ambulatory surgery. Anesth Analg 1999;88:508.

[2]Score ≥ 9 is required for discharge.

Management of Complications

RESPIRATORY COMPLICATIONS

4 Respiratory problems are the most frequently encountered serious complications in the PACU. The overwhelming majority are related to

airway obstruction, hypoventilation, or hypoxemia. Because hypoxemia is the final common pathway to serious morbidity and mortality, routine monitoring of pulse oximetry in the PACU leads to earlier recognition of these complications and fewer adverse outcomes.

Airway Obstruction

Airway obstruction in unconscious patients is most commonly due to the tongue falling back against the posterior pharynx (see Chapter 19). Other causes include laryngospasm, glottic edema, secretions, vomitus, a retained throat pack or blood in the airway, or external pressure on the trachea (most commonly from a neck hematoma). Partial airway obstruction usually presents as sonorous respiration. Near-total or total obstruction causes cessation of airflow and an absence of breath sounds and may be accompanied by paradoxic (rocking) movement of the chest. The abdomen and chest should normally rise together during inspiration; however, with airway obstruction, the chest descends as the abdomen rises during each inspiration (paradoxic chest movement). Patients with airway obstruction should receive supplemental oxygen while corrective measures are undertaken. A combined jaw-thrust and head-tilt maneuver pulls the tongue forward and opens the airway, and insertion of an oral or nasal airway often alleviates the problem. Nasal airways may be better tolerated than oral airways by patients emerging from anesthesia and may decrease the likelihood of trauma to the teeth when the patient bites down.

If the above maneuvers fail to reestablish an open airway, laryngospasm should be considered. Laryngospasm is usually characterized by high-pitched crowing noises, but may be silent with complete glottic closure. Spasm of the vocal cords is more apt to occur following airway trauma, repeated instrumentation, or stimulation from secretions or blood in the airway. The jaw-thrust maneuver, particularly when combined with gentle positive airway pressure via a tight-fitting face mask, usually breaks laryngospasm. Insertion of an oral or nasal airway is also helpful in ensuring a patent airway down to the level of the vocal cords. Any secretions or blood in the hypopharynx should be suctioned to prevent recurrence. Refractory laryngospasm should be treated with a small dose of intravenous succinylcholine (10–20 mg in adults) and positive-pressure ventilation with 100% oxygen. Endotracheal intubation may occasionally be necessary to reestablish ventilation; cricothyrotomy or transtracheal jet ventilation is indicated if intubation is unsuccessful in such instances.

Glottic edema following airway instrumentation is an important cause of airway obstruction in infants and young children because of the relatively small airway lumen. Intravenous corticosteroids (dexamethasone, 0.5 mg/kg, 10 mg dose maximum) or aerosolized racemic epinephrine (0.5 mL of a 2.25% solution with 3 mL of normal saline) may be useful in such cases. Postoperative wound hematomas following thyroid, carotid artery, and other neck procedures can quickly compromise the airway, and opening the wound immediately relieves tracheal compression in most cases. Rarely, gauze packing may be unintentionally left in the hypopharynx following oral surgery and can cause immediate or delayed complete airway obstruction, especially in patients with intermaxillary fixation.

Accidental or intentional decannulation of a fresh tracheostomy is hazardous because the various tissue planes have not yet organized into a well-formed track, thereby often making recannulation very difficult or impossible. In cases of tracheostomy performed within the previous 3–4 weeks, intentional replacement of a tracheostomy cannula should only be performed with a qualified surgeon at the bedside and a surgical tracheostomy instrument set, along with other appropriate airway equipment, immediately available.

Hypoventilation

Hypoventilation, which is generally defined as a $Paco_2$ >45 mm Hg, is common following general anesthesia. In most instances, the hypoventilation is mild, and most cases are undiagnosed. Significant hypoventilation is usually clinically apparent when the $Paco_2$ is >60 mm Hg or arterial blood pH is <7.25. Signs are varied and include excessive somnolence, airway obstruction, slow respiratory rate, tachypnea with shallow breathing, or labored breathing. Mild to moderate respiratory acidosis

may cause tachycardia, hypertension, and cardiac irritability (via sympathetic stimulation), but more severe acidosis produces circulatory depression (see Chapter 50. If significant hypoventilation is suspected, assessment and management is facilitated by capnography and/or arterial blood gas measurement.

5 Hypoventilation in the PACU is most commonly due to the residual depressant effects of anesthetics on respiratory drive. Opioid-induced respiratory depression characteristically produces a slow respiratory rate, often with large tidal volumes. Excessive sedation is usually present, but the patient is often responsive and able to breathe on command. Delayed occurrence of respiratory depression have been reported with all opioids. Proposed mechanisms include variations in the intensity of stimulation during recovery and delayed release of the opioid from peripheral compartments, such as skeletal muscle (or possibly the lungs with fentanyl), as the patient rewarms or begins to move.

Causes of residual muscle paralysis in the PACU include inadequate reversal, pharmacological interactions, altered pharmacokinetics (due to hypothermia, altered volumes of distribution, and renal or hepatic dysfunction), and metabolic factors (such as hypokalemia or respiratory acidosis). Regardless of the cause, generalized weakness, dyscoordinated movements ("fish out of water"), shallow tidal volumes, and tachypnea are usually apparent. The diagnosis can be made with a nerve stimulator in unconscious patients; head lift and grip strength can be assessed in awake patients. The ability to sustain a head-lift for 5 sec may be the most sensitive test for assessing the adequacy of reversal.

Splinting due to incisional pain, diaphragmatic dysfunction following upper abdominal or thoracic surgery, abdominal distention, and tight abdominal dressings are other factors that can contribute to hypoventilation. Increased CO_2 production from shivering, hyperthermia, or sepsis can also increase $Paco_2$, even in normal patients recovering from general anesthesia. Marked hypoventilation and respiratory acidosis can result when these factors are superimposed on an impaired ventilatory reserve due to underlying pulmonary, neuromuscular, or neurological disease.

Treatment of Hypoventilation

Treatment should generally be directed at the underlying cause, but marked hypoventilation always requires assisted or controlled ventilation until causal

6 factors are identified and corrected. Obtundation, circulatory depression, and severe acidosis (arterial blood pH <7.15) are indications for immediate and aggressive respiratory and hemodynamic intervention, including airway and inotropic support as needed. Antagonism of opioid-induced depression with large doses of naloxone often results in sudden pain and marked increase in sympathetic tone. The latter can precipitate a hypertensive crisis, pulmonary edema, and myocardial ischemia or infarction. If naloxone is used to reverse opioid-induced respiratory depression, titration in small increments (80 mcg in adults) usually avoids complications by reversal of hypoventilation without sig-

7 nificant reversal of analgesia. Following naloxone administration, patients should be observed closely for recurrence of opioid-induced respiratory depression ("renarcotization"), as naloxone has a shorter duration than most opioids. If residual muscle paralysis is present, additional cholinesterase inhibitor may be given. Residual paralysis in spite of a full dose of a cholinesterase inhibitor necessitates controlled ventilation under close observation until spontaneous recovery occurs. Hypoventilation due to pain and splinting following upper abdominal or thoracic procedures should be treated with intravenous or intraspinal opioid administration, intravenous ketorolac, epidural anesthesia, or intercostal nerve blocks.

Hypoxemia

Mild hypoxemia is common in patients recovering from anesthesia when supplemental oxygen is not given. Mild to moderate hypoxemia (Pao_2 50–60 mm Hg) in young healthy patients may be well tolerated initially, but with increasing duration or severity, the initial sympathetic stimulation often seen is replaced with progressive acidosis and circulatory depression. Obvious cyanosis may be absent if the hemoglobin concentration is reduced. Hypoxemia may also be suspected from restlessness, tachycardia, or cardiac irritability (ventricular or atrial). Obtundation, bradycardia, hypotension, and cardiac arrest are late

signs. Pulse oximetry facilitates early detection of hypoxemia and must be routinely utilized in the PACU. Arterial blood gas measurements may be performed to confirm the diagnosis and guide therapy.

Hypoxemia in the PACU is usually caused by hypoventilation, increased right-to-left intrapulmonary shunting, or both. A decrease in cardiac output or an increase in oxygen consumption (as with shivering) will accentuate hypoxemia. Diffusion hypoxia (see Chapter 8) is an uncommon cause of hypoxemia when recovering patients are given supplemental oxygen. Hypoxemia due exclusively to hypoventilation is also unusual in patients receiving supplemental oxygen, unless marked hypercapnia or a concomitant increase in intrapulmonary shunting is present. Increased intrapulmonary shunting from a decreased functional residual capacity (FRC) relative to closing capacity is the most common cause of hypoxemia following general anesthesia. The greatest reductions in FRC occur following upper abdominal and thoracic surgery. The loss of lung volume is often attributed to microatelectasis, as atelectasis is often not identified on a chest radiograph. A semi-upright position helps maintain FRC.

Marked right-to-left intrapulmonary shunting ($\dot{Q}s/\dot{Q}T$ >15%) is usually associated with radiographic findings, such as pulmonary atelectasis, parenchymal infiltrates, or a large pneumothorax. Causes include prolonged intraoperative hypoventilation with low tidal volumes, unintentional endobronchial intubation, lobar collapse from bronchial obstruction by secretions or blood, pulmonary aspiration, or pulmonary edema. Postoperative pulmonary edema most often presents as wheezing within the first 60 min after surgery, and, to a lesser extent, pink frothy fluid in the airway, and may be due to left ventricular failure (cardiogenic), acute respiratory distress syndrome, or relief of prolonged airway obstruction (negative pressure pulmonary edema). In contrast to wheezing associated with pulmonary edema, wheezing due to primary obstructive lung disease, which also often results in large increases in intrapulmonary shunting, is not associated with edema fluid in the airway or infiltrates on the chest radiograph. The possibility of a postoperative pneumothorax should always be considered following central line placement, supraclavicular or intercostal blocks, abdominal or chest trauma (including rib fractures), neck dissection, tracheostomy, nephrectomy, or other retroperitoneal or intraabdominal procedures (including laparoscopy), especially if the diaphragm may have been penetrated or disrupted. Patients with subpleural blebs or large bullae can also develop pneumothorax during positive-pressure ventilation.

Treatment of Hypoxemia

Oxygen therapy with or without positive airway pressure is the cornerstone of treatment for hypoxemia. Routine administration of 30% to 60% oxygen is usually enough to prevent hypoxemia with even moderate hypoventilation and hypercapnia (conversely, clinical signs of hypoventilation and hypercapnia may be masked by routine oxygen administration). Patients with underlying pulmonary or cardiac disease may require higher concentrations of oxygen; oxygen therapy should be guided by Spo_2 or arterial blood gas measurements. Oxygen concentration must be closely controlled in patients with chronic CO_2 retention to avoid precipitating acute respiratory failure. Patients with severe or persistent hypoxemia should be given 100% oxygen via a nonrebreathing mask or an endotracheal tube until the cause is established and other therapies are instituted; controlled or assisted mechanical ventilation may also be necessary. The chest radiograph (preferably with the patient positioned sitting upright) is valuable in assessing lung volume and heart size and in demonstrating a pneumothorax or pulmonary infiltrates. However, in cases of pulmonary aspiration, infiltrates are usually initially absent. If pneumothorax is suspected, a chest radiograph taken at end-expiration helps highlight the pneumothorax by providing the greatest contrast between lung tissue and adjacent air in the pleural space. In the situation of an intubated patient with hypoxemia, a chest radiograph provides additional usefulness to breath sound assessment in verifying endotracheal tube position, especially when the tube is inadvertently positioned immediately above the carina, with resultant intermittent migration into a main bronchus.

Additional treatment of hypoxemia should be directed at the underlying cause. A chest tube or Heimlich valve should be inserted for any

symptomatic pneumothorax or one that is greater than 15% to 20%. An asymptomatic pneumothorax may be aspirated using a intercostal catheter or followed by observation. Bronchospasm should be treated with aerosolized bronchodilator therapy. Diuretics should be given for circulatory fluid overload and cardiac function should be optimized. Persistent hypoxemia in spite of 50% oxygen generally is an indication for positive end-expiratory pressure ventilation or CPAP. Bronchoscopy is often useful in reexpanding lobar atelectasis caused by bronchial plugs or particulate aspiration. In the setting of an intubated patient, secretions or debris must be removed by suction and also by lavage, if necessary, and a malpositioned endotracheal tube must be appropriately repositioned.

CIRCULATORY COMPLICATIONS

The most common circulatory disturbances in the PACU are hypotension, hypertension, and arrhythmias. The possibility that the circulatory abnormality is secondary to an underlying respiratory disturbance should always be considered before any other intervention, especially in children.

Hypotension

Hypotension is usually due to relative hypovolemia, left ventricular dysfunction, or, less commonly, excessive arterial vasodilatation. Hypovolemia is by far the most common cause of hypotension in the PACU. Absolute hypovolemia can result from inadequate intraoperative fluid replacement, continuing fluid sequestration by tissues ("third-spacing"), wound drainage, or hemorrhage. Vasoconstriction during hypothermia with central sequestration of intravascular volume may mask hypovolemia until the patient's temperature begins to rise again; subsequent peripheral vasodilation during rewarming unmasks the hypovolemia and results in delayed hypotension. Relative hypovolemia is often responsible for the hypotension associated with spinal or epidural anesthesia (especially in the setting of concomitant general anesthesia), venodilators, and α-adrenergic blockade: the venous pooling reduces the effective circulating blood volume, despite an otherwise normal intravascular volume (see Chapter 45). Hypotension associated with sepsis and allergic reactions is usually the result of both hypovolemia and vasodilation. Hypotension from a tension pneumothorax or cardiac tamponade is the result of impaired venous return to the right atrium.

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Left ventricular dysfunction in previously healthy persons is unusual, unless it is associated with severe metabolic disturbances (hypoxemia, acidosis, or sepsis). Hypotension due to ventricular dysfunction is primarily encountered in patients with underlying coronary artery or valvular heart disease or congestive heart failure and is usually precipitated by fluid overload, myocardial ischemia, acute increases in afterload, or arrhythmias.

Treatment of Hypotension

Mild hypotension during recovery from anesthesia is common and typically does not require intensive treatment. Significant hypotension is often defined as a 20% to 30% reduction in blood pressure below the patient's baseline level and usually requires correction. Treatment depends on the ability to assess intravascular volume. An increase in blood pressure following a fluid bolus (250–500 mL crystalloid or 100–250 mL colloid) generally confirms hypovolemia. With severe hypotension, a vasopressor or inotrope (dopamine or epinephrine) may be necessary to increase arterial blood pressure until the intravascular volume deficit is at least partially corrected. Signs of cardiac dysfunction should be sought in patients with heart disease or cardiac risk factors. Failure of a patient with severe hypotension to promptly respond to initial treatment mandates invasive hemodynamic monitoring, or, better still, echocardiographic examination; manipulations of cardiac preload, contractility, and afterload are often necessary. The presence of a tension pneumothorax, as suggested by hypotension with unilaterally decreased breath sounds, hyperresonance, and tracheal deviation, is an indication for immediate pleural aspiration, even before radiographic confirmation. Similarly, hypotension due to cardiac tamponade, usually following chest trauma or thoracic surgery, often necessitates immediate pericardiocentesis or surgical exploration.

Hypertension

Postoperative hypertension is common in the PACU and typically occurs within the first 30 min after admission. Noxious stimulation from incisional pain, endotracheal intubation, or bladder distention is usually responsible. Postoperative hypertension may also reflect the neuroendocrine stress response to surgery or increased sympathetic tone secondary to hypoxemia, hypercapnia, or metabolic acidosis. Patients with a history of hypertension are likely to develop hypertension in the PACU, even in the absence of an identifiable cause. Fluid overload or intracranial hypertension may also occasionally present as postoperative hypertension.

Treatment of Hypertension

Mild hypertension generally does not require treatment, but a reversible cause should be sought. Marked hypertension can precipitate postoperative bleeding, myocardial ischemia, heart failure, or intracranial hemorrhage. Although decisions to treat postoperative hypertension should be individualized, in general, elevations in blood pressure greater than 20% to 30% of the patient's baseline, or those associated with adverse effects such as myocardial ischemia, heart failure, or bleeding, should be treated. Mild to moderate elevations can be treated with an intravenous β-adrenergic blocker, such as labetalol, esmolol, or metoprolol; an angiotensin-converting enzyme inhibitor, such as enalapril; or a calcium channel blocker, such as nicardipine. Hydralazine and sublingual nifedipine (when administered to patients not receiving β-blockers) may cause tachycardia and myocardial ischemia and infarction. Marked hypertension in patients with limited cardiac reserve requires direct intraarterial pressure monitoring and should be treated with an intravenous infusion of nitroprusside, nitroglycerin, nicardipine, clevidipine, or fenoldopam. The end point for treatment should be consistent with the patient's own normal blood pressure.

Arrhythmias

Respiratory disturbances, particularly hypoxemia, hypercarbia, and acidosis, will commonly be associated with cardiac arrhythmias. Residual effects from anesthetic agents, increased sympathetic nervous system activity, other metabolic abnormalities, and preexisting cardiac or pulmonary disease also predispose patients to arrhythmias in the PACU.

Bradycardia often represents the residual effects of cholinesterase inhibitors, opioids, or β-adrenergic blockers. Tachycardia may represent the effect of an anticholinergic agent; a β-agonist, such as albuterol; reflex tachycardia from hydralazine; and more common causes, such as pain, fever, hypovolemia, and anemia. Anesthetic-induced depression of baroreceptor function makes heart rate an unreliable monitor of intravascular volume in the PACU.

Premature atrial and ventricular beats often represent hypokalemia, hypomagnesemia, increased sympathetic tone, or, less commonly, myocardial ischemia. The latter can be diagnosed with a 12-lead ECG. Premature atrial or ventricular beats noted in the PACU without discernable cause will often also be found on the patient's preoperative ECG, if one is available. Such patients with a preexisting history of extrasystoles may or may not have a history of palpitations or other symptoms, and previous cardiology evaluation often has found no definitive cause. Supraventricular tachyarrhythmias, including paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation, are typically encountered in patients with a history of these arrhythmias and are more commonly encountered following thoracic surgery. The management of arrhythmias is discussed in Chapters 20 and 55.

CASE DISCUSSION

Fever & Tachycardia in a Young Adult Male

A 19-year-old man sustains a closed fracture of the femur in a motor vehicle accident. He is placed in traction for 3 days prior to surgery. During that time, a persistent low-grade fever (37.5–38.7°C orally), mild hypertension (150–170/70–90 mm Hg), and tachycardia (100–126 beats/min) are noted. His hematocrit remains between 30% and 32.5%. Broad-spectrum antibiotic coverage is initiated. He is scheduled for open reduction and internal fixation of the fracture. When the patient is brought into the operating room, vital

signs are as follows: blood pressure 162/95 mm Hg, pulse 150 beats/min, respirations 20 breaths/min, and oral temperature 38.1°C. He is sweating and is anxious in spite of intravenous premedication with fentanyl 50 mcg and midazolam 1 mg. On close examination, he is noted to have a slightly enlarged thyroid gland.

Should the surgical team proceed with the operation?

The proposed operation is elective; therefore, significant abnormalities should be diagnosed and properly treated preoperatively, if possible, to make the patient optimally ready for surgery. If the patient had an open fracture, the risk of infection would clearly mandate immediate operation. Even with a closed femoral fracture, cancellations or delays should be avoided because nonoperative treatment potentiates the risks associated with bed rest, including atelectasis, pneumonia, deep venous thrombosis, and potentially lethal pulmonary thromboembolism. In deciding whether to proceed with the surgery, the anesthesia provider must ask the following questions:

1. What are the most likely causes of the abnormalities based on the clinical presentation?
2. What, if any, additional investigations or consultations might be helpful?
3. How would these or other commonly associated abnormalities affect anesthetic management?
4. Are the potential anesthetic interactions serious enough to delay surgery until a suspected cause is conclusively excluded? The tachycardia of 150 beats/min and the low-grade fever therefore require further evaluation prior to surgery.

What are the likely causes of the tachycardia and fever in this patient?

These two abnormalities may reflect one process or separate entities (Tables 56–4 and 56–5). Moreover, although multiple factors can often be simultaneously identified, their relative contribution is often not readily apparent. Fever commonly follows major trauma; contributory factors can include the inflammatory reaction to

TABLE 56–4 Perioperative causes of tachycardia.

Anxiety
Pain
Fever (see Table 56–5)
Respiratory
Hypoxemia
Hypercapnia
Circulatory
Hypotension
Anemia
Hypovolemia
Congestive heart failure
Cardiac tamponade
Tension pneumothorax
Thromboembolism
Drug-induced
Antimuscarinic agents
β-Adrenergic agonists
Vasodilators
Allergy
Drug withdrawal
Metabolic disorders
Hypoglycemia
Thyrotoxicosis
Pheochromocytoma
Adrenal (addisonian) crisis
Carcinoid syndrome
Acute porphyria

TABLE 56–5 Perioperative causes of fever.

Infections
Immunologically mediated processes
Drug reactions
Blood reactions
Tissue destruction (rejection)
Connective tissue disorders
Granulomatous disorders
Tissue damage
Trauma
Infarction
Thrombosis
Neoplastic disorders
Metabolic disorders
Thyroid storm (thyroid crisis)
Adrenal (addisonian) crisis
Pheochromocytoma
Malignant hyperthermia
Neuroleptic malignant syndrome
Acute gout
Acute porphyria

the tissue trauma, superimposed infection (most commonly wound, pulmonary, or urinary), antibiotic therapy (drug reaction), or thrombophlebitis. Infection must be seriously considered in this patient because of the risk of bacteria seeding and infecting the metal fixation device placed during surgery. Although tachycardia is commonly associated with a low-grade fever, it is usually not of this magnitude in a 19-year-old patient. Moderate to severe pain, anxiety, hypovolemia, or anemia may be other contributory factors. Pulmonary fat embolism should also be considered in any patient with long bone fracture, particularly when hypoxemia, tachypnea, or mental status changes are present. Lastly, the possibly enlarged thyroid gland, sweating, and anxious appearance, together with both fever and tachycardia, suggest thyrotoxicosis.

What (if any) additional measures may be helpful in evaluating the fever and tachycardia?

Arterial blood gas measurements and a chest film would be helpful in excluding fat embolism. A repeat hematocrit or hemoglobin concentration measurement would exclude worsening anemia; significant tachycardia may be expected when the hematocrit is below 25% to 27% (hemoglobin <8 g/dL) in most patients. The response to an intravenous fluid challenge with 250–500 mL of a colloid or crystalloid solution may be helpful; a decrease in heart rate after the fluid bolus is strongly suggestive of hypovolemia. Similarly, response of the heart rate to sedation and additional opioid analgesia can be helpful in excluding anxiety and pain, respectively, as causes. Although a tentative diagnosis of hyperthyroidism can be made based on clinical grounds, confirmation requires measurement of serum thyroid hormones; the latter usually requires 24–48 hr in most hospitals. Signs of infection—such as increased inflammation or purulence in a wound, purulent sputum, an infiltrate on the chest film, pyuria, or leukocytosis with premature white cells on a blood smear (shift to the left)—should prompt cultures and a delay of surgery until the results are obtained and correct antibiotic coverage is confirmed.

The patient is transferred to the PACU for further evaluation. A 12-lead ECG confirms sinus tachycardia of 150 beats/min. A chest film is normal. Arterial blood gas measurements on room air are normal (pH 7.44, $Paco_2$ 41 mm Hg, Pao_2 87 mm Hg, and HCO_3^- 27 mEq/L). The hemoglobin concentration is found to be 11 g/dL. Blood for thyroid function tests is sent to the laboratory. The patient is sedated intravenously with midazolam (2 mg) and fentanyl (50 mcg) and is given 500 mL of 5% albumin. He seems to be relaxed and pain free, but the heart rate decreases only to 144 beats/min. The decision is made to proceed with surgery using continuous lumbar epidural anesthesia with 2% lidocaine. Esmolol is administered slowly until his pulse decreases to 120 beats/min, and a continuous esmolol infusion is administered at a rate of 300 mcg/kg/min.

The procedure is completed in 3 1/2 hr. Although the patient did not complain of any pain during the procedure and was given only minimal additional sedation (midazolam, 2 mg), he is delirious upon admission to the PACU. The esmolol infusion is proceeding at a rate of 500 mcg/kg/min. He has also received propranolol, 24 mg intravenously. Estimated blood loss was 500 mL, and fluid replacement consisted of 2 units of packed red blood cells, 1000 mL of hetastarch, and 9000 mL of lactated Ringer's injection. Vital signs are as follows: blood pressure 105/40 mm Hg, pulse 124 beats/min, respirations 30 breaths/min, and rectal temperature 38.8°C. Arterial blood gas measurements are reported as follows: pH 7.37, $Paco_2$ 37 mm Hg, Pao_2 91 mm Hg, and HCO_3^- 22 mEq/L.

What is the most likely diagnosis?

The patient is now obviously in a hypermetabolic state manifested by excessive adrenergic activity, fever, markedly increased fluid requirements, and a worsening mental status. The absence of major metabolic acidosis and lack of exposure to a known triggering agent exclude malignant hyperthermia (see Chapter 52). Other possibilities include a transfusion reaction, sepsis, or an undiagnosed pheochromocytoma. The sequence of events makes the first two unlikely, and the

decreasing prominence of hypertension (now replaced with relative hypotension) and increasing fever also make the latter unlikely. The clinical presentation now strongly suggests thyroid storm. He has also received a very large dose of esmolol for several hours and this may be contributing to the relatively low blood pressure despite aggressive fluid administration.

Emergency consultation is obtained with an endocrinologist, who concurs with the diagnosis of thyroid storm and assists with its management. How is thyroid storm managed?

Thyroid storm (crisis) is a medical emergency that carries a 10% to 50% mortality rate. It is usually encountered in patients with poorly controlled or undiagnosed Graves disease. Precipitating factors include (1) the stress of surgery and anesthesia, (2) labor and delivery, (3) severe infection, and, rarely, (4) thyroiditis 1–2 wk following administration of radioactive iodine. Manifestations usually include mental status changes (irritability, delirium, or coma), fever, tachycardia, and hypotension. Both atrial and ventricular arrhythmias are common, particularly atrial fibrillation. Congestive heart failure develops in 25% of patients. Hypertension that often precedes hypotension, heat intolerance with profuse sweating, nausea and vomiting, and diarrhea may be prominent initially. Hypokalemia is present in up to 50% of patients. Levels of thyroid hormones are high in plasma, but correlate poorly with the severity of the crisis. The sudden exacerbation of thyrotoxicosis may represent a rapid shift of the hormone from the protein-bound to the free state or increased responsiveness to thyroid hormones at the cellular level.

Treatment is directed toward reversing the crisis and its complications. Large doses of corticosteroids inhibit the synthesis, release, and peripheral conversion of thyroxine (T_4) to the more active triiodothyronine (T_3). Corticosteroids also prevent relative adrenal insufficiency secondary to the hypermetabolic state. Propylthiouracil is administered to inhibit synthesis of thyroid hormone, and iodide is given to inhibit release of thyroid hormones from the gland. Propranolol not only antagonizes the peripheral effects of the thyrotoxicosis, but may also inhibit the peripheral conversion of T_4. Combined β_1- and β_2-blockade is preferable to selective β_1-antagonism (esmolol or metoprolol) because excessive β_2-receptor activity is responsible for the metabolic effects. β_2-Receptor blockade also reduces muscle blood flow and may decrease heat production. Supportive measures include surface cooling (cooling blanket), acetaminophen (aspirin is not recommended because it may displace thyroid hormone from plasma carrier proteins), and generous intravenous fluid replacement. Vasopressors are often necessary to support arterial blood pressure.

Ventricular rate control is indicated in patients with atrial fibrillation. Transthoracic echocardiography, transesophageal echocardiography, and hemodynamic monitoring may facilitate management of patients with signs of congestive heart failure or persistent hypotension. β-Adrenergic blockade is contraindicated in patients with congestive heart failure.

Propranolol, dexamethasone, propylthiouracil, and sodium iodide are given; the patient is admitted to the intensive care unit, where treatment is continued. Over the next 3 days, his mental status markedly improves. The T_3 and total thyroxine levels on the day of surgery were both elevated to 250 ng/dL and 18.5 ng/dL, respectively. He was discharged home 6 days later on a regimen of propranolol and propylthiouracil, with a blood pressure of 124/80 mm Hg, a pulse of 92 beats/min, and an oral temperature of 37.3°C.

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