The residual effects of anesthesia, intraoperative positioning, and surgical trespass in combination with pre-existing co-morbidity create a variety of physiological disturbances that may become evident in the Post-Anesthesia Care Unit (PACU) or the early post-operative period. While cardiac and/or respiratory problems are most frequently encountered after surgery, complications may arise from any organ system or even from the limitations within the healthcare system itself as clinicians from differing disciplines share responsibility for postoperative care. Postoperative care is by necessity multidisciplinary, and it is of critical importance that clinicians work in cooperative fashion. Errors of judgment or communication are the most commonly implicated factors contributing to PACU complications. Since complications may arise from factors simultaneously within the medical, surgical, and anesthetic domains, it is important to establish and maintain effective lines of communication among healthcare providers who may view clinical problems from the differing perspectives of their respective areas of expertise.

Residual effects of anesthesia

The goals of general anesthesia including amnesia, akinesis, anxiolysis, analgesia, and autonomic stability are achieved in modern anesthetic practice by the judicious application of polypharmacy with vigilant monitoring. These medications often include volatile gases, neuromuscular blocking agents, opioids, benzodiazepines, anticholinergics, anticholinesterases, local anesthetics, and barbiturate or non-barbiturate induction agents as well as other medications administered during surgery. Each has profound physiological action, narrow therapeutic windows, and the potential to cause postoperative complications, either in isolation or in combination.

Respiratory disturbances are common after surgery and may be divided into three broad categories: disturbances in the mechanics of breathing, disturbances in ventilatory drives, and disturbances in pulmonary function.

1. Mechanics of breathing: Disturbances in the mechanics of breathing arise from dysfunction of either the pharyngeal muscles maintaining airway patency or the actual muscles of ventilation. Many of the drugs administered during the course of anesthesia may negatively impact pharyngeal muscle activity, leading to obstruction. Opioids and neuromuscular blocking agents are most commonly implicated, but residual effects of inhaled or intravenous agents may also contribute. Some residual
neuromuscular blockade is common after general anesthesia, affecting 31% of patients admitted to the PACU in one small observational study.\cite{2} Mild weakness is usually well-tolerated by most patients, but more profound degrees of neuromuscular blockade have been associated with both upper airway obstruction and severe hypoxia after surgery.\cite{3} The diaphragm is the major respiratory muscle and recovers relatively quickly from neuromuscular blockade, but the intercostal muscles are vulnerable to residual blockade or dysfunction after spinal or epidural anesthesia. The intercostal muscles assume a greater importance in ventilation in the supine position and in patients with lung disease including chronic obstructive pulmonary disease. Impairment of function of these muscles may place some patients at risk for postoperative hypoxic events.

2. **Ventilatory drives:** Disturbances in ventilatory drives also occur commonly after surgery. In a retrospective study of 198,103 surgical procedures, respiratory depression was the most common cause of postoperative mortality.\cite{4} Normally, ventilation may be controlled consciously or by intrinsic ventilatory drives, of which the hypercarbic and the hypoxic drives are the most important. Consciousness may be impaired after surgery, as may the intrinsic ventilatory drives. Inhaled anesthetics have profound effects on ventilatory drives, altering both rate and tidal volume, and may exert some effect after surgery, but opioids have the most profound effect on ventilation. The magnitude of the effect of opioids is both dose-dependent and idiopathic depending upon the tolerance of the patient, and will also attenuate the hypercapnic drive. Some degree of respiratory acidosis is virtually ubiquitous after surgery owing to the use of opioids. The hypoxic drive is less reliable after the administration of opioids, so the sedated postoperative patient may become profoundly hypoxic because of the lack of ventilatory effort. The respiratory depressant effect of opioids is synergistic with other medications, especially benzodiazepines. Hypoxia therefore occurs after surgery among patients making adequate respiratory attempts but unable to maintain patent upper airways, and among patients not making adequate respiratory effort. A weaker ventilatory drive arises from pulmonary stretch receptors whose neuronal input may be lost during spinal or epidural anesthesia. This may lead to the interesting opposite phenomenon of patients who are making perfectly adequate respiratory effort and are neither hypoxic nor hypercarbic, but who may be extremely concerned that they perceive they are not breathing since they can no longer feel it.

3. **Pulmonary function:** Disturbances in pulmonary function also occur after general anesthesia. Atelectasis may occur because of effects on pulmonary surfactants, high concentrations of inspired oxygen, and mechanical ventilation. Surgical positioning in head-down position, placing the weight of abdominal organs on the diaphragm and shifting it towards the head, may further exacerbate atelectasis. Functional residual capacity is reduced under general anesthesia. The resultant small airway closure both increases the metabolic expenditure for the work of breathing, while expanding areas of poor ventilation contributing to pulmonary shunt. Ventilatory dead space is increased in the postoperative period. Volatile agents inhibit hypoxic pulmonary vasoconstriction, which may additionally contribute to postoperative hypoxia. In susceptible individuals, volume resuscitation may contribute to pulmonary edema, or airway manipulation may cause bronchospasm, further compromising pulmonary function.
Cardiac disturbances are also common after surgery and are observed with approximately the same frequency as respiratory complications. Electrocardiogram (ECG) changes in axis, intraventricular conduction delays, T-wave changes, and QT prolongation may be observed. Pharmacological alterations in both sympathetic and parasympathetic nervous system activity may also alter cardiac rate and activity. Reductions in cardiac preload occur during general anesthesia because of the venous dilatation coupled with intravascular volume depletion due to hemorrhage and insensible fluid loss from evaporative loss through surgical incisions. As these effects dissipate after surgery, fluctuations in cardiac preload may occur from changes in venous tone, intraoperative volume resuscitation, and third-spacing. Pharmacological depression of cardiac contractility may also occur from medications administered during surgery.

Hepatic blood flow is decreased during general anesthesia, but a greater demand is placed upon the liver to metabolize and detoxify the myriad of medications administered. Inadequacies in hepatic function may be revealed after surgery. Volume shifts and fluid administration may stress renal function, but urine concentrating ability may be impaired from the metabolism of fluorinated hydrocarbons used in anesthesia into renal toxic fluoride ions.

Residual effects of intraoperative positioning

Positioning of the patient for surgery is primarily determined by the need for surgical exposure. All patients are at increased risk for compression neuropathy or skin breakdown from prolonged immobility during the procedure. If surgical positioning places the incision above the level of the heart, there is also the risk of venous air embolism. However, apart from the discrete complications that may be seen after surgical positioning, there are transient physiological effects that may manifest in the postoperative period.

Many of the effects of intraoperative positioning are caused by the exertion of gravity on the body and circulation. For example, “head-down tilt” position (Trendelenburg position) is associated with visceral force against the diaphragm (see Figure 1.1). In this position, functional residual capacity is reduced, atelectasis is promoted, and intrathoracic pressure during mechanical ventilation is increased. These factors increase the risk of pulmonary
barotrauma. Atelectasis may persist after return to supine positioning, and central obesity and longer duration in this position may exacerbate this problem.

Gravity also exerts hemodynamic effects related to positioning. Some of these effects may be clinically beneficial. For example, the sitting position may be used for posterior fossa craniotomy or high cervical spine surgery, providing good surgical access while promoting gravity-dependent venous and cerebral spinal fluid drainage and lowering of intracranial pressure. Position in lateral decubitus positioning for thoracotomy promotes pulmonary blood flow to the dependent (non-operative) lung, minimizing shunt fraction and improving oxygenation during one lung ventilation. However, some of the gravitational effects on hemodynamics may produce untoward effects. These include edema formation in body parts positioned below the level of the heart and arterial ischemia in body parts above the level of the heart.

Procedures performed in extreme head-down tilt position, such as robot-assisted prostatectomy, may be associated with edema accumulation in the head and torso. Sclera edema may be seen after prolonged duration in this position. Although usually benign and self-limited, when this position has been prolonged and large amounts of crystalloid have been infused, delayed extubation of the trachea may be prudent to allow for edema resolution to avoid potential airway compromise. Increases in periocular venous pressure from face-down (prone) positioning have been implicated in postoperative visual loss from posterior ischemic optic neuropathy, although the etiology of this complication is not fully understood and causality has not been proven.

Systemic arterial pressure is much higher than normal pulmonary artery or venous pressures and therefore less affected by gravity. However, intraoperative positioning may still contribute to tissue ischemia. Mean arterial pressure falls by 2 mmHg for each inch above the level of the heart.\[5\] When portions of the body are placed high above the level of the heart, the risk for arterial ischemia increases. In high lithotomy position, the feet may be 36 inches or more above the level of the heart, greatly reducing pedal perfusion pressure. Limb ischemia and lower extremity compartment syndromes have been observed postoperatively, if higher mean arterial pressures are not maintained during surgery. Similarly, “beach chair” position, which places the head above the level of the heart, may predispose to cerebral ischemia by a similar mechanism. It is important to note that great care is taken to mitigate this risk and cerebral ischemia in the beach chair position is no more common than in other surgical positions.\[6\] However, anatomical variations may place some patients at unrecognized increased risk.\[7\]

**Physiological effects of surgical trespass**

The “stress” of surgery is a systemic physiological load above and beyond basal metabolic activities. This global burden has a multifactorial etiology including previously discussed pharmacological effects of the anesthetic drugs, as well as the systemic release of inflammation modulating hormones from the site of surgery, and the macroscopic, mechanical alterations in function from regional structures affected by surgical manipulation.

**Local inflammatory responses with global, systemic effects:** Varying body tissues respond differently to physical surgical manipulation. In a similar fashion to a surgeon’s hand causing release of vaso active substances from a pheochromocytoma, surgical manipulation in any of the body’s tissues can cause local release of inflammatory cytokines that may gain access to the systemic circulation. This systemic inflammatory response
syndrome (SIRS) can be initiated anywhere in the body and can be accentuated by pre-existing conditions, especially infection.

1. **Postoperative fever:** Fever in the first 24 hours following surgery is not uncommon. Clinically, early postoperative fever is often attributed to atelectasis, but there is little evidence to support the role of atelectasis in the etiology of postoperative fever. Atelectasis resolves rapidly and spontaneously with innate mechanisms such as coughing and sighing, which were suppressed during general anesthesia, but occur once emergence from anesthesia is complete. This process occurs in 1 to 2 days, and fever that persists beyond 24 to 48 hours should not be attributed to atelectasis.

The astute clinician must be aware of the less common but much more pathological causes of fever in the postoperative period. An acute necrotizing skin and soft tissue infection at the surgical site must always be considered, and direct examination of the surgical wound should be performed in all febrile patients. If this requires removal of the surgical dressing, then that should be done. Erythema, acute edema, and crepitus should all prompt immediate surgical consultation and evaluation.

Fever may also represent transient bacteremia, especially in a clean-contaminated, contaminated, or dirty surgical field. As long as no major source of infection remains present in the patient, this is generally spontaneously cleared in the immune-competent patient. In the face of immunosuppression or altered immunity, antibiotics should be considered more strongly, and close observation for subsequent development of SIRS or frank sepsis should be performed. This may include subsequent monitoring in a higher level of care post the PACU stay.

2. **Postoperative hypotension:** There are many causes of postoperative hypotension, most of which will be addressed in other sections. These include acute coronary syndrome, SIRS, sepsis, and basic hypovolemia. However, hypovolemia from acute hemorrhagic shock must be considered in all post-surgical patients. Prompt evaluation for surgical hemorrhage should involve interrogation of the surgical site dressing for saturation, and measurement and character evaluation of any surgical drain output. Additionally, the surgical site should be examined for hematoma formation. If direct examination of the surgical space is not possible (deeper cavity not palpable transdermally), then other means of imaging or reexploration should be considered. Site-specific signs and symptoms of hemorrhage will be further addressed in the following section on the systems-based evaluation of surgical trespass.

3. **Secondary (direct-inflammatory) end-organ injury:** Surgical manipulation can cause direct inflammation and injury to structures physically handled by the surgical team. These same structures, on a cellular level, have the potential to release cytokines systemically and drive a global response to local stimulation. This secondary inflammatory process can take place throughout the body, but those organs most susceptible include the lungs, kidneys, liver, heart, and brain.

An acute lung injury can manifest as interstitial edema causing pulmonary edema and hypoxia; acute kidney injury causes acute tubular necrosis with or without oliguria; acute liver injury can manifest as cholestasis; cardiac injury via cardiomyopathy of critical illness; and injury in the brain as delirium. All of these secondary injuries are treated with supportive care and alleviation, if possible, of the inflammatory cytokine source. Of note, these SIRS reactions can occur in 7% to 44% of operative cases, and occur with greater frequency in longer procedures.
Macroscopic, mechanical alterations caused by surgical manipulation: It is important to be familiar with surgical disease processes and how surgical intervention functions to resolve the condition. Knowledge of this pathophysiology allows for the intuitive comprehension of potential complications, not only intraoperative, but those that present in the post-anesthesia period.

1. **Neurological and spine surgery:** The brain and spinal cord are within an enclosed space. Any fluid build-up, including blood, can cause an acute deterioration in central nervous system (CNS) function. Further, vascular manipulation leading to or within the CNS can be complicated by thrombosis or an acute embolic event. Therefore, any change in neurological exam postoperatively needs to be evaluated urgently. This includes lethargy or a depressed mental state and any lateralizing signs such as an unequal pupillary exam, facial asymmetry, or discrepancy in extremity motor/sensory exam. In this setting, an acute intervention is needed to prevent permanent injury; therefore, prompt recognition is imperative.

2. **Maxillofacial and neck surgery:** Maxillofacial and neck surgery either involves or is closely related to the airway. Airway obstruction is therefore the most dreaded complication and can occur secondary to infection, edema, or hematoma. The first step of treatment involves rapidly establishing a patent airway, usually via reintubation. This intubation can be complicated by abnormal anatomy from the inciting factor. Opening the surgical wound can often decompress the airway obstruction and allow for intubation, but should only be done when major bleeding and exsanguination are less likely. This is based on the type of surgery; thyroid surgery or mass excisions are much less likely to cause exsanguination than a carotid endarterectomy with a fresh major vascular anastomosis. The presence of the surgeon is of paramount importance when deciding to open an incision.

   Creation of a myocutaneous free tissue flap in the head and neck area has become very prevalent in the past several years to allow for coverage after major tumor resections. Along with hematoma formation, these procedures are also complicated by flap ischemia. A PACU practitioner must be able to evaluate the blood flow to and from the flap. Often, a site for Doppler scanning is marked by the surgeon, but flap color, capillary refill, and temperature should be a part of ongoing flap assessments in the PACU.

3. **Thoracic surgery:** An acute hemothorax in the postoperative period can cause several problems. The hemothorax can occupy thoracic cavity volume that would otherwise allow for lung expansion and gas exchange. With a large enough hemothorax, tension physiology may occur if the blood not only affects the unilateral lung but places pressure on the mediastinal structures. Tension on the mediastinum can affect venous blood return to the heart and cause hypotension as well. Finally, global hemorrhagic shock can occur from the blood loss, and it must be treated with intravascular volume resuscitation.

   Most thoracic surgery procedures result in the placement of a chest tube, which can drain some of the accumulating hemothorax and clue the practitioner to an ongoing bleed. It should be noted that the chest tubes themselves can become non-functional from clot and therefore should never used as a reason to rule out a hemothorax. Other signs include hypoxia, tachycardia, shortness of breath, and anxiety. The diagnosis can usually be confirmed via chest X-ray. After thoracic surgery, a low threshold for an interval chest X-ray should be considered if any issues arise in the PACU.
In a similar fashion to hemothorax, an acute pneumothorax can obstruct lung function and similarly place tension on the mediastinum. Tension pneumothorax can occur quite acutely. Classic signs include decreased breath sounds, tracheal deviation away from the side of the pneumothorax, hypoxia, and hypotension. The diagnosis here can be made either by recognizing the clinical presentation or confirmed by chest X-ray, if the diagnosis is in doubt. Treatment involves placing an additional chest tube with or without needle decompression first. If an indwelling chest tube is already present, it is prudent to search for the cause for its malfunction. Loss of the tidal movement of the fluid in the tubing may be an indicator of a non-functional tube. Kinking and occlusion of the tubing, as well as malfunction of the chest tube drain itself should be sought out.

Intrathoracic manipulation, even exterior to the pericardium, can cause cardiac irritability and result in dysrhythmias. These dysrhythmias may start as tachycardia and degenerate to more malignant rhythms. Atrial fibrillation is the most common dysrhythmia after thoracic surgery. First-line treatment involves the use of β-blockers, both in a prophylactic and therapeutic manner. Second-line therapies include calcium channel blockers, amiodarone, or digoxin. Cardioversion should be reserved for hemodynamically unstable patients.

Treatment of additional underlying contributing factors of the dysrhythmia should be investigated. This differential should include endotracheal tube obstruction, electrolyte abnormalities, intravascular volume abnormalities, and myocardial ischemia/infarction.

Lung and pleural cavity manipulation can also result in direct trauma and subsequent atelectasis. Segmental hypoventilation during surgery can also result in pooling of bronchial secretions causing subsequent mucous plugging that can become clinically significant in the postoperative setting. Finally, pulmonary mobilization, even to small degrees, can allow lobar torsion and result in acute decompensation. Diagnosis is made on chest X-ray and treatment is emergent operative de-torsion.

4. Intra-abdominal surgery: Intra-abdominal hypertension (IAH) is caused by increased pressure in the abdominal cavity. If left untreated, abdominal compartment syndrome can result. The patients at most risk for IAH are those who required large amounts of volume in the operating room or postoperatively and patients who underwent a hernia repair with a loss of domain for the abdominal viscera. This loss of domain comes from returning the viscera back into the formal peritoneal cavity from the hernia sac or actual contraction of the peritoneal cavity via the hernia repair process itself. Besides abdominal pain, IAH also places increased pressure on the diaphragm, causing respiratory difficulty, and occlusive pressure on the renal veins, causing an acute kidney injury.

The bedside clinician should maintain a high index of suspicion for IAH based on abdominal exam along with respiratory status and kidney function. The diagnosis is usually confirmed indirectly by measuring the pressure in the bladder via a commercial bladder pressure kit that gets connected to the Foley catheter. If close observation of IAH is warranted, the patient should transition to an intensive care setting for monitoring. If abdominal compartment syndrome is imminent or present, immediate surgical decompression of the abdominal wall should be performed.
Postoperative intra-abdominal hemorrhage can be difficult to recognize as the abdomen can accommodate a fair volume of blood before distension ensues. Abdominal pain is generally the earliest sign, although not specific. Any abdominal drains such as Jackson–Pratt drains should be interrogated. Ultimately, physiology suspicious for bleeding (tachycardia and hypotension) is usually the determining factor in persuading the surgeon to re-explore the abdomen.

5. **Vascular surgery**: Vascular surgery can be complicated by surgical site bleeding, which is generally quite obvious. More conspicuously, vascular anastomoses can thrombose and place downstream organs and/or extremities at risk of ischemia. Neurovascular evaluation at least hourly is warranted in the postoperative period following vascular surgery, whether open or endovascular. Any change in examination should prompt evaluation by the vascular surgeon; this includes pulse, pallor, paresthesias, pain, or paralysis.\[14\]

6. **Orthopedic surgery**: Acute hardware failure following orthopedic surgery is generally visible as a deformity at the surgical site. If this site is wrapped, pain out of proportion to postoperative recovery is usually the first sign. Diagnosis is made on radiological imaging.

   Surgical manipulation can also result in an extremity compartment syndrome. Pain is again the first sign. A thorough neurovascular exam should be performed and if there is any question, compartment pressures should be measured by the orthopedic surgeon.\[15\] Vascular compromise can also be direct following orthopedic surgery if the vessels were injured or kinked during the surgery. A change or abnormal vascular exam should prompt early evaluation by a vascular surgeon.

7. **General issues**: Hypovolemia will be addressed in several locations throughout this book. For the purposes of surgical trespass, it should be noted that generally, during an exploratory laparotomy, the patient does have evaporative losses that equate to approximately one liter per hour. This certainly changes based on patient demographics, and other physiological measures should be used to maintain euvoemia.

   Despite optimal resuscitation, acute kidney injury may still present in the PACU. Those at greatest risk are those with pre-existing dysfunction, those undergoing surgery involving the aorta, and those with predisposing conditions such as sepsis or shock.\[16\] Evaluation of oliguria should be algorithm-based and treated based on underlying cause. This will be addressed in further detail elsewhere in this book.

   Hypothermia can result from any surgery but abdominal surgery is specifically prone to heat losses due to the exposed surface area. Both active and passive rewarming postoperatively should be initiated in hypothermic patients as hypothermia can result in increased blood loss and increased transfusion requirements.\[17\]

**References**


The function of the Post-Anesthesia Care Unit (PACU) is to monitor and care for patients of all ages that are admitted following procedures where general anesthesia, sedation, or regional anesthesia has been administered. These patients may be discharged home on the day of surgery or admitted for continuing care and treatment depending upon need. The PACU performs a vital role in promoting patient health and recovery after the surgical procedure. The primary goals are patient safety, recovery from anesthesia, and treatment of postoperative complications.

Each institution should develop policies and standards for recovery from anesthesia. In the United States, the guidelines from the American Society of Anesthesiologists (ASA)\textsuperscript{[1]} and the American Association of PeriAnesthesia Nurses (ASPAN)\textsuperscript{[2]} serve as the foundation of institutional policies. Oversight and maintenance of policies should be established jointly with the anesthesia medical director and the nurse manager and educator of PACU. All policies and procedures should be reviewed annually to ensure that clinical or institutional changes are reflected in accurate and up-to-date information and guidelines for staff.

Recovery after anesthesia may be divided into three phases, which require different levels of care which may or may not be delivered in geographically distinct locations within the institution. Phase One is for patients who require intense respiratory and hemodynamic monitoring as well as management of pain and nausea, and fluid management. Phase Two occurs when the patient no longer requires intensive monitoring and the attention shifts towards planning for discharge from the unit or home readiness. Phase Three, or Extended Care, may occur for patients who otherwise complete Phase Two, but are awaiting discharge to another area of the hospital.