Complications of Neuroanesthesia

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30.1 Introduction

Complications may occur during neurologic surgery. Anesthesiologists need to be aware of the various issues that can arise, potential approaches to prevention, and effective management strategies. This chapter is intended to describe the most common complications encountered by neuroanesthesiologists in the neurosurgical operating room and the neurological ICU.

30.2 Venous Air Embolism

A venous air embolism (VAE) occurs when atmospheric air is entrained into the vascular system. Subatmospheric venous pressure occurs when a venous opening or wound is elevated above the heart. The pressure differential can pull air into the venous system. The incidence increases with the height of the surgical field as compared to the heart. Air entry is increased by the presence of noncollapsable veins such as in the dural sinuses. VAE is most common during posterior fossa operations in the sitting position (Fig. 30.1). Fathi et al. demonstrated a 39% incidence (range 7–76%) in a pooled analysis of posterior fossa surgery. VAE occurred in 11% (range 2–35%) of cervical spine surgeries [1]. Other neurosurgical procedures with an increased risk of VAE include craniosynostosis repair, spinal fusion, and deep brain stimulator placement [2].

Classically, air enters the right ventricle and pulmonary arteries via the superior vena cava. This leads to elevated pulmonary artery pressures due to direct obstruction of pulmonary arterioles as well as reflex pulmonary vasoconstriction [3]. Air embolism increases alveolar dead space resulting in decreased end-tidal CO_2 and increased $PaCO_2$. As the air diffuses across the alveolar-capillary membrane, the nitrogen is exhaled.

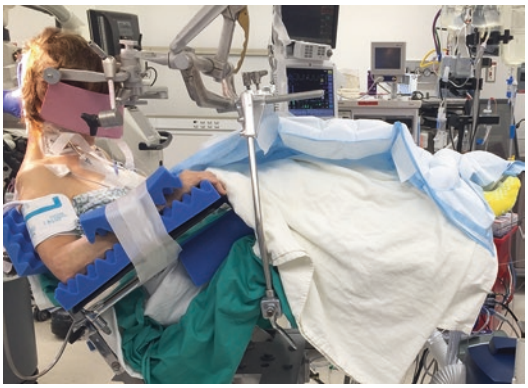

Fig. 30.1 Neurosurgical patient in the sitting position for a posterior fossa craniotomy

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Alternatively, air may cross a patent foramen ovale resulting in a paradoxical air embolism. In this setting, air enters the arterial system and may result in mesenteric, myocardial, extremity, or cerebral ischemia. Probe patent foramen ovale is estimated to occur in 20–30% of adults, and in many centers, a PFO is a contraindication to neurosurgery in the sitting position [3].

Multiple monitors function to detect VAE. The most sensitive monitor, transesophageal echocardiography (TEE), can detect volumes as small as 0.02 mL/kg. However, due to clinical inexperience and the invasiveness of this monitoring technique, TEE is not utilized routinely in the neurosurgical suite. Precordial Doppler ultrasound is the second most sensitive monitor after TEE. The device is placed over the SVC-RA junction just right of the sternum at the third or fourth intercostal space. Correct positioning is confirmed with the injection of aerated saline [3]. Proper positioning may be difficult in obese patients, and interference can occur with sound artifacts and electrocautery. End-tidal CO_2 and nitrogen are not as sensitive as precordial Doppler, and the majority of institutions lack the capabilities to monitor end-tidal nitrogen. Pulmonary artery catheters are no more sensitive than end-tidal CO_2 and introduce the dangers inherent in pulmonary artery catheter placement [3].

Clinical presentation depends on the volume of air and the rate of air entrainment. Small volumes (<0.5 mL/kg) may cause decreased end-tidal carbon dioxide, increased end-tidal nitrogen, and mild oxygen desaturations. Moderate volumes (0.5–2.0 mL/kg) may lead to pulmonary hypertension, right heart strain, arrhythmias, hypotension, myocardial ischemia, and bronchoconstriction. Large volumes (>2.0 mL/kg) can cause an airlock with complete right ventricular outflow tract obstruction leading to cardiopulmonary collapse [2]. Lethal volumes of air have been reported to be 200–300 mL or 3–5 mL/kg.

The mainstay of treatment is to stop the entrainment of air. The neurosurgeon should immediately be notified to flood the field with saline and apply bone wax. Air and nitrous oxide (if used) should be discontinued and 100% oxygen instituted. Jugular venous compression has been shown to raise the pressure in the dural sinus which can reduce entrainment of air; however, this technique may cause increased intracranial pressure and cerebral edema [4]. Also, activation of carotid baroreceptors may produce bradycardia. Positive end-expiratory pressure (PEEP) should not be used to decrease the rate of air entry. PEEP can increase right atrial pressure and may theoretically increase the risk of paradoxical air embolism. In addition, the PEEP required to reduce venous return from the head would considerably impede venous return from the SVC and therefore decrease cardiac output [5]. If present, immediate aspiration of a multi-orifice right atrial catheter can be attempted. This maneuver has been shown to reduce morbidity from VAE [6]. Additional maneuvers include lowering the head, fluid administration, and vasopressor initiation. Changing position to left lateral decubitus with Trendelenburg may help reposition air to the right ventricle but is not helpful with continuous entrainment of air. Complete cardiovascular collapse requires advanced cardiac life support.

30.3 Intracranial Hypertension

The cranial vault is a rigid structure containing three main components: brain tissue (1400 mL), cerebrospinal fluid (CSF, 150 mL), and blood (150 mL). The Monro-Kellie doctrine describes the relationship between intracranial pressure (ICP) and volume of these three components; the total volume of these components must remain constant due to the constraint of the cranium, such that an increase in volume of any one component must be accompanied by a decrease in the volume of another component or an increase in ICP. The ICP elastance curve, a plot of intracranial pressure over volume, is a biphasic, exponential curve. In the lower-volume portion of the curve, the slope is relatively flat (meaning changes in pressure are minimal with changes in volume) due to compensatory mechanisms including displacement of CSF into the spinal compartment. After this phase of compensation, small increases in the volume of intracranial components lead to large

increases in ICP, steepening the slope of the elastance curve.

Normal adult ICP is between 5 and 15 mmHg. Intracranial hypertension is defined as an ICP at or above 20 mmHg, at which pressure neurologic changes such as decreased level of consciousness, nausea, and vomiting will be observed. Methods of monitoring ICP include ventriculostomies/external ventricular drains (EVDs), which can be used for therapeutic CSF removal in addition to ICP monitoring, parenchymal manometers ("subarachnoid bolts") placed directly into brain tissue, and epidural manometers. The most recent guidelines from the Brain Trauma Foundation recommend ICP monitoring in all salvageable patients with severe TBI (GCS 3–8) and an abnormal CT brain [7].

Treatment of elevated ICP consists of reducing the volume of intracranial contents while maintaining cerebral blood flow and reducing cerebral oxygen consumption. Excessive CSF can be removed via extraventricular drainage (or, in chronic conditions, ventriculoperitoneal or ventriculoatrial shunting). Brain parenchyma may be removed in the case of brain tumor or abscess, but more commonly treatment is targeted at reduction of cerebral edema using hyperosmotic therapy with mannitol or hypertonic saline. Hyperventilation-induced hypocapnia results in cerebral alkalosis and vasoconstriction which decreases ICP. However, this effect dissipates over a period of hours as the CSF pH returns to normal. Due to the fleeting effect and potential for vasoconstriction-induced ischemia, this technique is particularly useful in the emergent treatment of intracranial hypertension [8]. Finally, cerebral blood volume reduction is achieved by evacuation of hematoma, avoidance of venous congestion (elevation of the head, avoidance of neck compression by excessive flexion or rotation, endotracheal tube ties, monitoring cables, or internal jugular central venous catheters, and avoidance of excessive positive intrathoracic pressure or PEEP), and careful regulation of cerebral blood flow with avoidance of cerebral vasodilating stimuli including hypoxia, hypercarbia, hyperthermia, and systemic hypertension in the setting of failed autoregulation [9]. With treatment to reduce cerebral blood volume, cerebral perfusion pressure, defined as the difference between mean arterial pressure and ICP (or CVP, if this value exceeds ICP), should be maintained over 60 mmHg, as reduction below this is associated with decreased brain tissue oxygenation [10].

With failure to treat elevated ICP, end-stage intracranial hypertension manifests as brain herniation. Impending herniation is classically heralded by Cushing's triad of hypertension with wide pulse pressure, bradycardia, and irregular Cheyne-Stokes respirations. If medical therapy has been exhausted, decompressive craniectomy to relieve near terminal intracranial pressure may be attempted.

30.4 Pneumocephalus

Pneumocephalus represents air trapped in the cranial vault (Fig. 30.2). During neurosurgical procedures, air can enter the cranium in a similar mechanism as an inverted soda bottle. This phenomenon occurs most frequently after craniotomy or craniectomy but may also occur with endoscopic sinus or transsphenoidal surgery, burr hole decompression, or shunt placement. In addition,

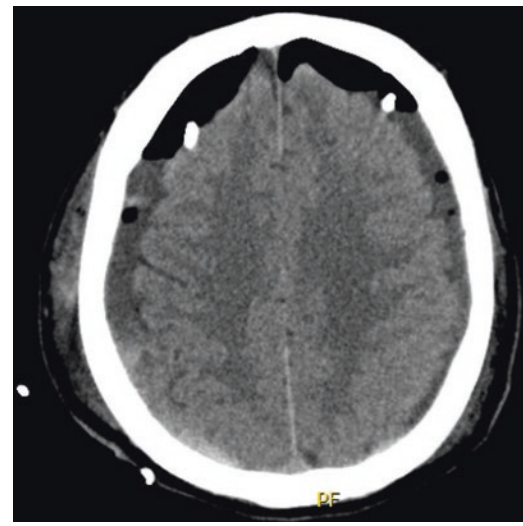

Fig. 30.2 CT scan demonstrating pneumocephalus following craniotomy evacuation of chronic subdural hematoma

pneumocephalus may occur following trauma, infection, barotrauma, or spontaneously [11]. Risk factors for the development of pneumocephalus include head position, duration of surgery, use of nitrous oxide, hydrocephalus, intraoperative osmotic diuresis, hyperventilation, spinal anesthesia, barotrauma, continuous CSF drainage, epidural anesthesia, infections, and neoplasms [11].

The majority of cases are asymptomatic; however, symptoms can include headache, nausea and vomiting, dizziness, seizures, and altered mental status [12]. Pneumocephalus may present as delayed emergence following neurologic surgery. Postoperative CT scans have demonstrated that almost 100% of patients have some degree of pneumocephalus in the first 2 days following surgery, which may persist for over a week. A retrospective study of 240 patients revealed a 26.3% incidence of pneumocephalus 3 weeks following craniotomy [13].

Tension pneumocephalus is an accumulation of air that behaves as a mass lesion in the brain requiring immediate decompression. Air can enter the cranium when hyperventilation, osmolar therapy, CSF loss, and venous drainage have reduced the volume of the intracranial contents. Following dural closure and resumption of normocapnia and normovolemia, the air can become compressed and result in a mass effect. Nitrous oxide can produce a tension pneumocephalus if used in the presence of trapped air and closed dura. Two CT signs have been described which suggest tension pneumocephalus. The Mount Fuji sign occurs when subdural air separates the frontal lobes resulting in a distribution of air resembling Mount Fuji. The air bubble sign demonstrates multiple small air bubbles inside the subarachnoid cisterns [14].

There is little in the anesthesiologist's armamentarium capable of preventing pneumocephalus. Normovolemia and normocapnia should be established toward the end of the procedure before dural closure. Nitrous oxide use is controversial; however, discontinuation of nitrous oxide prior to dural closure is recommended [15]. BiPAP or CPAP should not be used following transsphenoidal surgery due to the risk of dural suture disruption resulting in asymptomatic or tension pneumocephalus [16].

30.5 Delayed Emergence

Delayed emergence or delayed awakening occurs when a patient fails to regain an appropriate level of consciousness within an expected period (20–60 min) following cessation of a general anesthetic [17, 18]. Multiple risk factors exist which may predict delayed emergence including the extremes of age (geriatrics, neonates), obesity, obstructive sleep apnea, and preoperative cognitive dysfunction, seizures, or stroke [17]. The type of procedure may contribute as well. Patients undergoing spinal surgery or craniotomy with small mass excision have been shown to awaken faster than those with large mass excisions [19].

The primary causes of delayed emergence are due to residual drug effect, metabolic factors, and neurological disorders, among others. Residual sedation is the most common mechanism of delayed emergence. Volatile and intravenous anesthetics can take time to eliminate, particularly following prolonged operations. Benzodiazepines can produce prolonged sedation, especially when combined with opioids, which are known to reduce the respiratory rate and the ventilatory response to carbon dioxide. Opioid overdose demonstrates pinpoint pupils on neurological exam. Rarely, liberal use of local anesthetics can cause toxicity which may manifest as delayed emergence. Central anticholinergic syndrome can be caused by the use of antihistamines, anticholinergics, and even anesthetic medications such as volatile anesthetic and can present with seizure, tachycardia, mydriasis, coma, and respiratory depression [20]. In addition to sedative medications, neuromuscular blocking agents can result in delayed emergence if significant blockade persists or if the blockade has not been reversed. Succinylcholine and mivacurium use in a patient with undiagnosed pseudocholinesterase deficiency maintains neuromuscular blockade for 4–8 h [21]. Train-of-four monitoring is

instrumental in evaluating the neuromuscular blockade. Drug-drug interactions may contribute, and serotonin syndrome has been reported as a cause of delayed emergence [22].

On the other hand, non-pharmacologic causes of delayed emergence can result in serious morbidity and should be excluded. Metabolic factors such as hyper- and hypoglycemia should be ruled out, particularly in the diabetic patient. Hyperglycemia in the diabetic patient can result in diabetic ketoacidosis or hyperosmolar coma which can result in profound sedation and require urgent management. Hypothyroidism or myxedema coma may contribute as well as other metabolic factors such as hypercarbia or acidosis. Electrolyte abnormalities such as hyper- or hyponatremia should also be excluded. Hypothermia frequently occurs in the operating room and has been shown to contribute to prolonged recovery from anesthesia [23].

Neurologic disorders can result in catastrophic morbidity and mortality if left undiagnosed. Intraoperative hemorrhagic or an ischemic cerebral vascular accident can present as delayed emergence. The neurologic exam will be limited and may or may not demonstrate signs of stroke. Seizures and status epilepticus can delay emergence and occur in up to 4.3% of neurosurgical patients in the first 24 h following surgery [24]. Local anesthetic infiltration with accidental introduction into the cerebral spinal fluid may result in a total spinal with brainstem anesthesia.

Delayed emergence has many etiologies. Thus, it is essential to develop a stepwise approach to rule out each potential cause (Table 30.1). Airway, breathing, and circulation should take precedence with evaluation of airway, oxygenation, ventilation, and vital signs [18]. A review of the patient's medical and medication history as well as intraoperative medication administration may reveal potential causes. Considering residual drug effect represents the most common cause of delayed emergence, it is appropriate to continue evaluation by ensuring all anesthetic agents have been discontinued and allowed adequate time for elimination. If the patient is unresponsive, residual muscle paralysis can be evaluated with train-of-four monitoring and reversed if required. If the patient is following commands but appears profoundly weak, sugammadex administration should be considered. Opioid reversal with 40 mcg of naloxone can be initiated and repeated every 2 min up to a dose of 200 mcg [18]. Benzodiazepine reversal with 0.2 mg of flumazenil can be administered every minute up to a total dose of 1 mg [18]. The acetylcholinesterase inhibitor physostigmine (up to 2 mg) can be used to treat anticholinergic syndrome and may be useful for the reversal of postoperative somnolence [25]. After eliminating pharmacologic mechanisms, metabolic causes should be evaluated. Finger-stick glucose testing can assess for hypo- or hyperglycemia. Capnography should demonstrate normocapnia. An arterial blood gas will reveal any electrolyte or acid-base abnormalities, and the patient should be rewarmed if hypothermic. Following evaluation of metabolic causes, neurologic disorders should be immediately assessed with a neurologic exam, computed tomography scan, and electroencephalography. If no cause is uncovered, the patient should be monitored in an intensive care setting with neurology consultation and frequent neurologic exams.

Table 30.1 Checklist for delayed emergence

Residual drug effect
– Evaluate neuromuscular blockade with train-of-four
– Reverse neuromuscular blockade if present
– Consider naloxone (40 mcg, up to 400 mcg) for opioid reversal
– Consider flumazenil (0.2 mg up to 1 mg) for benzodiazepine reversal
– Consider physostigmine (up to 2 mg) to aid arousal
Metabolic
– Check finger-stick glucose
– Ensure normocapnia
– Evaluate patient temperature and rewarm if required
– Arterial blood gas
Neurologic causes
– Neurologic physical examination
– CT scan
– EEG
If patient fails to emerge, he/she may need to be monitored in ICU setting for 24–48 h

30.6 Postoperative Seizures

There is an increased risk of seizure activity following neurosurgical procedures. Kvam et al. reported a 4% incidence of seizures in the first 24 h following neurologic surgery, and up to 20% may experience seizures in the first postoperative week [26, 27]. Intraoperative hypoxia, electrolyte derangements, acid-base abnormalities, neurologic insults, vascular abnormalities, tumors, and epilepsy may contribute to postoperative seizures. Intraoperatively, hematoma formation, extensive retractor use, edema, and manipulation of brain tissue can lead to seizures as well [27]. Any intracranial mass lesions such as abscesses, hematomas, tumors, arteriovenous malformations, and aneurysms can act as epileptogenic foci. Skardelly et al. demonstrated an age greater than 60 years, a total tumor/edema volume ≤ 0.64 cm^3, and the size of resection as risk factors for postoperative seizures following tumor resection. The extent of resection was the primary determinant in the development of seizures [28]. Zheng et al. found a new neurologic deficit postoperatively to be a risk factor for seizure development following meningioma resection [22].

Pharmacologic prophylaxis of perioperative seizure following neurologic surgery is controversial. A 2015 Cochrane review evaluated the use of prophylactic antiepileptic drug (AED) treatment with non-trauma neurosurgical patients. Eight randomized controlled trials were evaluated, and only one trial showed a statistically significant advantage of prophylactic AED treatment in the prevention of postoperative seizures. Regarding head-to-head AED comparisons, one trial demonstrated a reduction in seizures with levetiracetam when compared to phenytoin. Other head-to-head trials failed to show significance. In their conclusions, the authors found insufficient evidence to recommend either strategy over the other [29]. On the other hand, patients with preoperative epilepsy should continue their AEDs throughout the perioperative period. When AEDs are used perioperatively, it is essential to monitor the neuromuscular blockade as these medications are known to decrease the duration of action of commonly used paralytics.

Postoperative seizures are typically easy to diagnose in awake, unanesthetized patients. Patients under general anesthesia with neuromuscular blockade will not demonstrate classic signs of seizure activity and will need electroencephalography for diagnosis. Upon recognition, the clinician should assess the patient's airway, breathing, and circulation. Oxygen should be applied, and benzodiazepines should be administered. Refractory status epilepticus may require endotracheal intubation and general anesthesia or barbiturate coma. Following resolution of seizure activity, the patient's electrolytes and acid-base balance should be assessed and corrected. AED serum drug levels should be evaluated in patients with a history of epilepsy to confirm therapeutic dosing. Dosages may need to be altered. Seizures in the immediate postoperative period may indicate a severe neurologic insult, and computed tomography scanning may be required to rule out a new or evolving intracranial process.

30.7 Cardiac Dysfunction Following Neurologic Insult

The heart-brain connection was first described in the medical literature in 1903 by Cushing, who noted a hypertensive response in cases of intracranial hemorrhage with acute cerebral compression. The phenomenon of reversible, nonobstructive cardiac dysfunction after neurologic injury, known as neurogenic stunned myocardium, is best studied in the current literature in subarachnoid hemorrhage (SAH), although this phenomenon has been observed in other central nervous system insults including status epilepticus, meningitis/encephalitis, traumatic brain injury, ischemic stroke, intracranial mass lesions, and brain death. In a literature review by Sakr, the incidence of EKG abnormalities, predominantly ST-segment changes (Fig. 30.3), ranged from 40 to 100% in patients with SAH and was associated with an elevation in cardiac troponin levels in 20–40% and regional wall motion abnormalities (RWMAs) in 9–31% [30]. In an observational

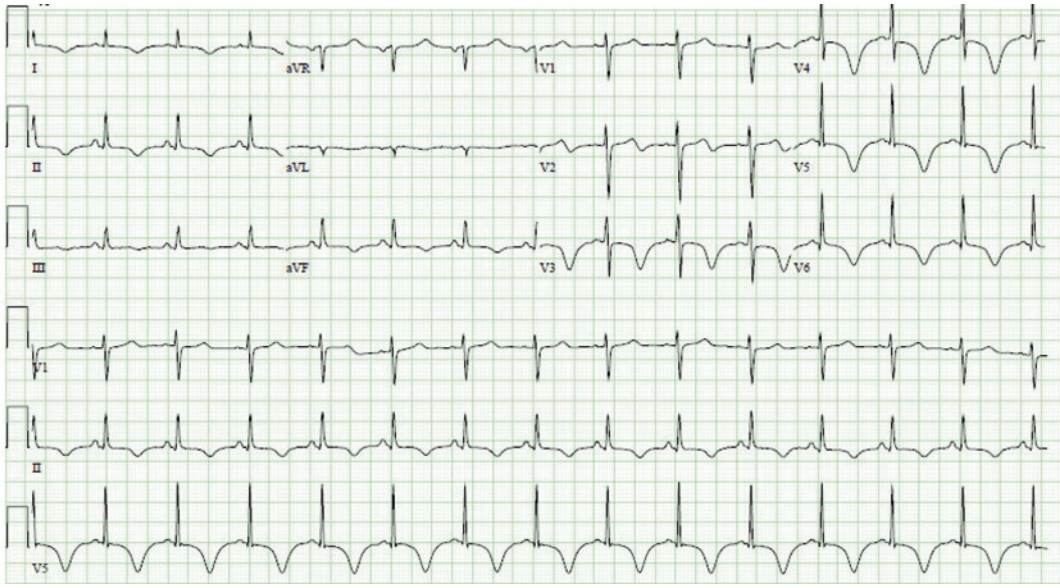

Fig. 30.3 EKG demonstrating the electrical changes of stress cardiomyopathy in a patient with subarachnoid hemorrhage. This patient had an elevated troponin (0.268 ng/mL)

study by Kuroiwa of 23 patients presenting with ST-segment elevation after cerebral aneurysm rupture, all had RWMAs; subsequent cardiac catheterization of 8 of these patients revealed no obstructive coronary artery disease [31]. In a case study by Naidech, 69% of patients presenting with SAH had abnormal EKG findings, prompting cardiac enzyme evaluation [32]. Troponin elevation above normal lab values was present in 98% of patients tested or 68% of the cohort, and abnormal left ventricular function was noted on echocardiography in 55%. Interestingly, increased peak cardiac troponin (cTnI) levels were related to higher Hunt-Hess clinical grade, intraventricular hemorrhage or global cerebral edema on neuroimaging, and loss of consciousness at ictus after an associated seizure, all features associated with inferior neurologic outcomes. In this study, higher peak troponin levels were associated with increased risk of left ventricular dysfunction, pulmonary edema, hypotension requiring vasopressor treatment, and delayed cerebral ischemia from vasospasm or cerebral infarction from any cause. Elevated peak troponin was also associated with increased all-cause mortality and increased disability on the modified Rankin score at 3-month follow-up, highlighting the potential prognostic significance of cTnI measurements after SAH [32]. This potential was reiterated by Tanabe in a case series of 103 patients with SAH. In this series, 52% of patients had a positive cTnI, with 23% having a "highly positive" cTnI of over 1.0 ng/ml. This subset of patients had a higher mean Hunt-Hess grade and a higher incidence of relatively depressed left ventricular function, ventricular diastolic dysfunction, left ventricular wall motion abnormalities, and pulmonary congestion on chest radiography than patients with no troponemia or with "mildly positive" cTnI. The degree of myocardial dysfunction in this subset of patients was mild and transient, with no incidence of cardiogenic shock and 71% improvement of ventricular function on repeat echocardiogram 5–10 days after SAH; however, a highly positive cTnI had an association with both clinical severity of SAH and significantly longer ICU length of stay [33].

The mechanism of injury proposed in neurogenic stunned myocardium is that of catecholamine excess, in which a sympathetic surge initiates a cascade of cellular events ultimately leading to myocardial damage. Anatomically, the heart is innervated by noradrenergic fibers

from the sympathetic nervous system traveling in the intermediolateral gray column of the spinal cord in the cervical and upper thoracic (T1–T4) levels and by cholinergic fibers from the parasympathetic nervous system via the vagus nerve. After brain injury, an increase in both systemic and local catecholamine release at the myocardium from sympathetic nerve terminals is observed. This leads to prolonged opening of beta-1 receptor-controlled calcium channels on myocardial cells with rapid ATP depletion and contraction band necrosis, in which myocardiocytes die in a contracted state following excessive calcium influx. This is histologically distinct from the coagulation necrosis seen in myocardiocytes after ischemic cell death. In a histopathological study by Greenhoot, autopsy examination of myocardium from three patients who expired after intracranial hemorrhage revealed areas of subendocardial hemorrhage, cytoplasmic banding of myocardial cells, and acute inflammatory response interspersed with large numbers of unremarkable myocardial cells. In the same study using a cat model of neurologic injury, histologic examination of feline myocardium revealed a similar pattern of injury, and electron microscopy detailed areas with the most injury immediately adjacent to intracardiac nerves, with changes less apparent at a distance from the nerve [34]. This pattern of injury, which does not correspond to coronary artery distributions, suggests direct neural insult to the heart.

30.8 Cardiopulmonary Resuscitation in the Prone Position

In the neurosurgical operating suite, both cranial and spinal procedures are frequently performed in the prone position, for surgical access to posterior anatomic structures, which can pose significant challenges to the anesthesiologist. Physiologic changes associated with the prone position include increased intrathoracic pressure, decreased respiratory compliance, increased peak airway pressures, and decreased ventricular compliance with a resultant decrease in venous return and preload and an increase in central venous pressure. The accompanying reduction in stroke volume combined with peripheral vasodilation in the anesthetized patient can result in severe hypotension. Paired with increased intrathoracic pressure, increased intra-abdominal and pelvic pressure causes venous pooling which contributes to surgical bleeding from vertebral and epidural veins; estimated blood loss exceeding a liter is not uncommon in multilevel complex spine surgery. Additionally, patients undergoing posterior fossa or prone spinal operations are at risk of air embolism causing cardiovascular collapse as the operative site is above the level of the heart and the veins encountered with decompression of the spinal column and cranium are scaffolded in bone matrix and non-compressible. Laceration of the aorta, vena cava, or iliac vessels during lumbar discectomy can result in complete cardiovascular collapse [35]. Unstable cardiac arrhythmias can be provoked by brainstem manipulation in posterior fossa surgery. Respiratory arrest is also a risk in the prone patient due to the hazard of endotracheal tube kinking or dislodgement with poor access to the airway, in addition to aforementioned alterations in respiratory compliance that can compromise adequate ventilation and oxygenation. These cardiovascular and respiratory changes associated with the prone position elevate the risk of intraoperative cardiac arrest.

Repositioning the prone patient into the supine position for classical CPR requires additional time, personnel, and equipment (stretcher) and risks loss of the airway, intravenous access, and invasive monitors vital to the resuscitation effort. Furthermore, supine positioning obscures surgical access to the posterior anatomic structures and prevents securement in the event of surgical bleeding contributing to arrest. Supinating the intraoperative patient with an unstable spinal column also carries the risk of devastating neurologic injury as well as contamination of the surgical site and potential infection. Prone ACLS is not currently taught in BLS or ACLS courses, and the AHA 2015 guidelines only recommend that ACLS in the prone position may be reasonable when the patient cannot be safely placed in the supine position.

Although no guidelines exist for optimal performance of prone CPR, it has been described in case reports and small trials addressing feasibility in the ICU setting. Prone CPR was first described as the "modified Schafer method" in 1989. Schaefer's method was initially proposed in the 1940s as a mechanism of providing artificial respiration to the near-drowning victim via lower thoracic compressions in the prone position. McNeil suggested that this method also provided circulatory assistance while protecting against aspiration of vomitus and encouraging lay bystanders to perform resuscitation without fear of infectious disease transmission via mouth to mouth [36]. In the hospital setting, a successful case of prone defibrillation during a thoracolumbar decompression was described by Brown, with paddle placement at the right axilla and left apex; no compressions were administered, but the author's experience prompted a systematic review of the literature which identified 22 case reports of CPR on prone patients. Several techniques were described depending on patient characteristics and the surgical site [37]. These include two patients described by W. Sun who suffered cardiac arrest in the prone position and were successfully resuscitated using the "reverse precordial compression maneuver." The first patient arrested during a posterior fossa craniotomy for occipital fracture, after surgical brain retraction to address a dural venous sinus tear, led to severe bradycardia. The patient's head was fixed in the Mayfield head clamp whose safe removal for repositioning would have delayed initiation of chest compressions, and the surgeon had yet to secure the bleeding dural sinus and required continued posterior access. Compressions were performed by a single provider, the staff anesthesiologist, who placed the left hand in a fist against the lower third of the sternum for counter-pressure while compressing the mid-thoracic spine with the right hand. The second case arrested after airway obstruction during cervical laminectomy and fusion for fracture with spinal cord compression. As the spine was not yet stabilized, compressions were carried out in the prone position with a two-provider technique in which the surgeon compressed the thoracic spine with both hands at the level of T7, while the anesthesiologist provided counter-pressure on the lower third of the sternum with a clenched fist [38]. A case of successful prone CPR on a pediatric patient who arrested during thoracic spinal fusion for scoliosis was reported by Tobias. Using a technique in which the surgeon placed both hands at the mid-thoracic level on either side of the incision for compressions, with the thoracic support of the Gardener frame providing counter-pressure, the patient achieved return of spontaneous circulation (ROSC) in 7 min. Effective cardiac output was confirmed throughout CPR via both invasive and noninvasive blood pressure monitoring, and postoperative examination revealed unchanged neurologic status from baseline as well as no evidence of end-organ hypoperfusion injury (Fig. 30.4) [39].

In addition to case reports, two small trials have demonstrated the physiologic adequacy of prone CPR in generating cardiac output. Mazer investigated the utility of "reverse CPR" in a crossover design study on six ICU patients who sustained cardiac arrest and failed to achieve ROSC after 30 min of ACLS. Enrolled patients received an additional 15 min of supine CPR, were repositioned prone with a sandbag on the CPR underbody board as a sternal counter-pressure device, and received another 15 min of CPR. All patients had the airway secured with an endotracheal tube prior to prone positioning. ACLS protocol guided medication administration and defibrillation in both positions. During both supine and prone CPR, systolic and diastolic arterial blood pressures were recorded. The authors found a statistically

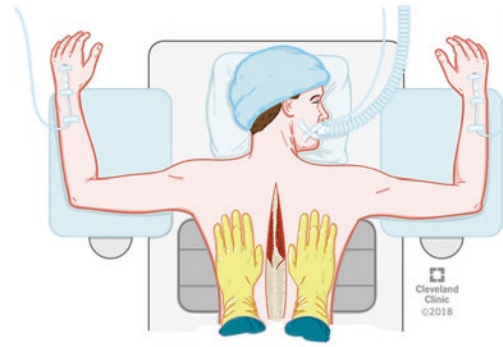

Fig. 30.4 Illustration depicting technique of prone CPR

significant improvement in systolic arterial pressures and calculated mean arterial pressures during prone CPR [40]. A similar study by Wei enrolled five patients who had a cardiac arrest in the ICU and failed to achieve ROSC with ACLS. After a decision by the staff physician to abandon the resuscitation attempt, the patients were turned prone, and compressions continued with the recording of arterial blood pressure. Prone CPR generated a mean systolic pressure of 95 mmHg and diastolic pressure of 25 mmHg [41]. Though small, these studies corroborate operating room case reports that prone CPR can be hemodynamically effective, and, importantly, neither found inferior hemodynamic parameters to supine CPR. As it is established that early continuous CPR improves outcomes in cardiac arrest, and current ACLS guidelines do note the reasonable nature of prone compressions in specialized settings, the anesthesia team should be aware of the feasibility and techniques of prone CPR when providing care to the prone neurosurgical patient.

30.9 Airway Management Pitfalls in the Unstable or Postoperative Cervical Spine Patient

Cervical spine injuries are the most common spinal injuries and occur in 2–4% of adult blunt trauma patients, with an increased incidence in the presence of head trauma with GCS < 8 [42]. Not only should the anesthesiologist be concerned regarding airway securement of the trauma victim due to mental status, hemodynamic instability, or planned operative intervention but must also consider protection of neural structures from secondary injury. As cervical spine injuries at or above C5 can lead to diaphragmatic weakness and respiratory dysfunction, securement of the airway with mechanical ventilation prevents hypoxia which itself can exacerbate neurologic injury by decreased oxygen delivery to the spinal cord. However, airway maneuvers including chin lift, bag-mask ventilation, LMA placement, both direct and indirect laryngoscopy, and flexible fiber-optic techniques have been shown to cause movement of the cervical spine, and the optimal manner of securing the airway in the potentially cervical spine-injured patient remains controversial [43].

Many anesthesiologists prefer awake flexible fiber-optic intubation in trauma patients, although the awake patient who is inadequately topicalized can displace an unstable cervical spine by coughing, and uncooperative or unconscious trauma patients are not candidates for awake intubation. Asleep flexible fiber-optic intubation is an option, provided the anesthesiologist is cognizant of the forces associated with bag-mask ventilation if the patient is not adequately preoxygenated prior to the intubation attempt. In the event of airway bleeding, fiber-optic techniques may not be possible. In a randomized, controlled crossover trial, Houde et al. fluoroscopically compared occiput –C5 segmental motion during flexible fiber-optic intubation with luminous stylette intubation and found no significant difference in mean maximum motion (11° with the flexible fiber-optic scope versus 12° with the luminous intubating stylette, both values including necessary jaw-lift maneuvers) and significantly less time to secure the airway with the luminous intubating stylette [44]. A similarly designed randomized crossover trial by Robitaille compared maximum segmental cervical spine motion during intubation using indirect videolaryngoscopy and direct laryngoscopy with a Macintosh blade, both with manual in-line stabilization. Not only did this trial find no significant difference in cervical motion between direct and indirect laryngoscopy, but the mean maximum cervical motion was between 8° and 11°, slightly less than motion observed in the aforementioned trial of the flexible fiber-optic scope and the luminous intubating stylette [45].

Using more standard intubating equipment, a cadaver model of injury at C5–C6 compared relative safety, based on fluoroscopic degree of axial distraction, anteroposterior (AP) displacement, and angular rotation, of two immobilization techniques as well as three laryngoscope blades, and concluded that manual in-line stabilization (MILS) resulted in less AP displacement than rigid cervical collar immobilization during laryngoscopy.

The Cormack-Lehane grade obtained was superior with MILS, and all intubations were endotracheal on the first attempt with no esophageal intubations; the Miller laryngoscope blade was noted to allow less axial distraction than the Macintosh or McCoy blades [46]. Manual in-line stabilization, a two-person technique in which the non-intubating provider places one hand on either side of the patient's head with the index finger on the mastoid process and the thumb just anterior to the external auditory meatus, with remaining digits posteriorly supporting the neck and maintaining cervical alignment without axial traction, is recommended by ATLS for orotracheal intubation attempts on any patient suspected of having a cervical spine injury, regardless of the method chosen for intubation [47].

The routinely scheduled case of anterior cervical discectomy and fusion (ACDF) can likewise present an anesthetic challenge. As ACDF is performed for the indication of painful, symptomatic herniated cervical disk, the preoperative examination should assess for associated cervical myelopathy that may be exacerbated by airway maneuvers including bag-mask ventilation and laryngoscopy. Postoperative airway obstruction due to wound hematoma, though rare with a reported incidence from 0.2% to 1.9%, can lead to devastating consequences if not quickly and effectively acted upon. The surgical exposure for ACDF is carried out through a plane between the carotid sheath laterally and the midline viscera (esophagus and trachea) medially to expose the ventral surface of the cervical vertebrae. This dissection creates a potential space posterolaterally on one side of the trachea, and hematoma development in this space can cause airway compression both extrinsically from the hematoma itself distorting the trachea and from occlusion of venous and lymphatic drainage of the neck at even lower pressures leading to venous congestion and laryngeal edema. In vitro studies of external pressure applied to the pig trachea up to 257 mmHg (well above systolic blood pressure, the maximum possible pressure achieved due to a hematoma as blood flows down a pressure gradient) resulted in only 20% compression of the trachea, suggesting that the latter mechanism of airway edema due to impaired venous outflow contributes most to the loss of the airway in the setting of postoperative neck hematoma. Therefore, the anesthesiologist should be aware that a distorted airway can still be encountered even if surgical hematoma evacuation is performed [48].

Rapid diagnosis is paramount and should be considered when respiratory distress—heralded with subtle voice changes and restlessness, followed by dyspnea, tachypnea, poor management of secretions, hypoxia, and inspiratory stridor—is noted in the immediate postoperative period, generally within the first 12 h of surgery. Delayed airway obstruction beyond this time is also observed, but more commonly due to prevertebral edema possibly due to spinal construct failure (Fig. 30.5) or the development of CSF leak or retropharyngeal abscess; regardless, the goal is to secure the

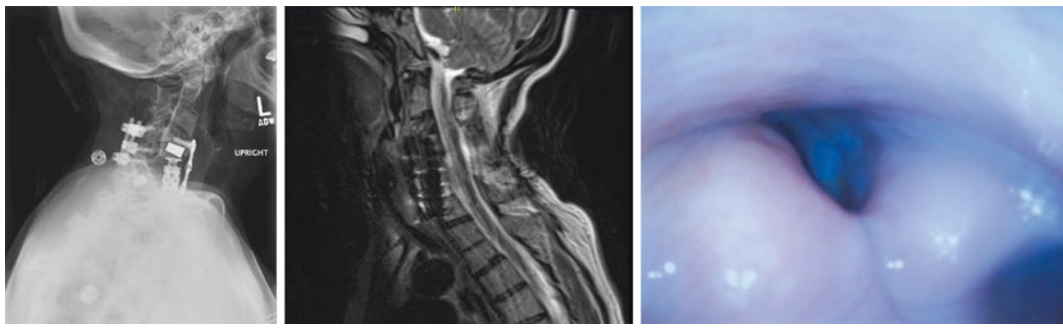

Fig. 30.5 (**a**) Lateral C-spine X-ray demonstrating construct failure in a patient presenting with delayed stridor after ACDF. Note widened prevertebral space and laryngeal edema suggested by soft tissue shadowing in the airway. (**b**) MRI C-spine in the same patient demonstrating airway narrowing and laryngeal edema. (**c**) Fiber-optic bronchoscope image of patient's airway with edema of the epiglottis, arytenoids, and vocal cords

airway via placement of an endotracheal tube. Palumbo et al. suggest a multidisciplinary management approach involving a spine surgeon, anesthesiologist, and ENT surgeon, as follows. If the airway is not severely compromised and the patient is stable for transfer, the optimal setting for securing the airway is in the operating room, via an awake technique. If anatomy is too distorted by an apparent space-occupying hematoma, it is suggested that the spine surgeon open the incision under local anesthesia prior to the subsequent intubation attempt [49]. It is worthwhile to note again that laryngeal edema will likely still be present after hematoma decompression and the airway should still be treated as "difficult" after opening the surgical incision. If the awake intubation attempt fails despite hematoma decompression, a surgical airway should be established via cricothyroidotomy, which can be performed with midline extension of the neck incision. With any postoperative ACDF patient, a plan such as that outlined above should be preemptively considered, so it can be rapidly executed in the event of postoperative airway compromise.

30.10 Sodium Disorders

Hyponatremia, defined as serum sodium less than 135 mmol/L, is reported in up to 50% of neurosurgical patients and has been demonstrated to be an independent risk factor for all-cause morbidity and mortality [50]. In SAH patients, hyponatremia has been associated with a twofold increase in the incidence of cerebral ischemia, even if fluid restriction is avoided [51]. As correction of serum sodium may improve mortality, expeditious diagnosis and treatment of hyponatremia are key. The two most common etiologies of hyponatremia in the neurosurgical population are the syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW). These pathologies are both characterized by hyponatremia and low serum osmolarity (<285 mOsm/L) with high urine osmolarity (>200 mOsm/L) and elevated urine sodium levels (>25 mmol/L) but can be distinguished based on the evaluation of the extracellular fluid status. This distinction has significant treatment implications. In SIADH, excessive ADH from the posterior pituitary leads to increased free water reabsorption from the collecting ducts of the nephron with a resultant euvolemic or hypervolemic dilutional hyponatremia; the perturbation is one of excessive water retention and should be treated with fluid restriction. A notable exception to this is in SAH patients at risk of vasospasm in which euvolemia should be maintained. In CSW, excessive renal sodium losses, thought to be due to natriuretic peptide release from the injured brain, are accompanied osmotically by water loss leading to a hypovolemic hyponatremia. Treatment is based on fluid replacement therapy and sodium supplementation.

While hyponatremia can lead to cerebral edema, vasospasm after SAH, and increased mortality, overly rapid correction of hyponatremia can also have deleterious results. Osmotic demyelination syndrome or central pontine myelinolysis describes the neurologic sequelae of encephalopathy or coma, spastic paralysis, and pseudobulbar palsy after rapid correction of hyponatremia due to noninflammatory demyelination in the brainstem [52]. To prevent this complication, expert opinion suggests gentle sodium correction, not more than 1 mmol/L/h up to 10 mmol/L/day.

Hypernatremia is defined as serum sodium >145 mmol/L and is less commonly seen than hyponatremia in hospitalized patients. Central or neurogenic diabetes insipidus, in which the posterior pituitary fails to release ADH leading to lack of water reabsorption by the distal collecting tubule and collecting duct of the nephron, is the most common cause of hypernatremia in the neurosurgical patient. It is characterized by high-volume, dilute urine and is most commonly seen after pituitary surgery, traumatic brain injury, and SAH, as well as after brain death [53]. Treatment consists of fluid replacement with or without exogenous ADH supplementation with DDAVP.

30.11 Conclusion

No anesthesiologist or intensivist desires complications following neurologic surgery. However, they do occur, and it is essential for providers to

remain vigilant in the identification and management of these potentially catastrophic situations. As with any emergency, airway, breathing, and circulation should be assessed and optimized first. With the knowledge of potential complications, clinicians can identify the most likely diagnosis and intervene prior to patient deterioration.

Key Points

- A venous air embolism occurs most commonly during posterior fossa and cervical spine operations. This event, characterized by decreased end-tidal CO_2, oxygen desaturation, and hypotension, should be treated immediately by notifying the surgeon to flood the field with saline.
- Delayed emergence is caused by residual drug effect, metabolic factors, and neurologic disorders. It is essential to follow a stepwise approach during diagnosis to rule out each potential cause.
- Nonobstructive, reversible stress cardiomyopathy is common after subarachnoid hemorrhage and may manifest as troponemia, ST-segment changes, and regional wall motion abnormalities. Treatment is supportive with management of the underlying neurologic insult.
- In the neurosurgical operating suite, CPR may need to be performed on the prone patient to prevent neurologic injury and delays in care with repositioning. Hemodynamic efficacy of prone CPR has been demonstrated in small trials and case studies.
- There is no standard optimal manner of securing the airway in the patient with a cervical spine injury; however, manual in-line stabilization (MILS) is recommended for any orotracheal intubation attempt when cervical spine injury is suspected.

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