Scientific Paper Entitled: Anesthesia Considerations In Neurosurgical Emergencies: A Narrative Review

Ali Abdulrman Alzurayq*[1], Ahmad Hmdan Alonazi[2], Ghanim Mohammed Sayer Alshammry[3], Yahya Nasser Bakeet[4], Mansour Abdullah Ali Alqahtani[5], Omar Mawqad Almutairi[6], Mohmmed Mushafil Mohmmed Hazazi[7], Abdulrahman Eid Alonazi[8], Bader Muslih Alharbi[9], Duaa Mohammed Alkhudaimi[10], Mohammed Abdullah Alredini [11], Mohammed Saad Almutairi[12], Ali Ahmad Ali Alsahabi[13], Muhannad Abdulmohsen Aljammaz[14], Majed Hajij Alkhaldi[15], Mohmmad Ali Nuwayl Almajanuni[16], Break Hamad Al Omari[17], Mohammed Rasheed Alharbi[18]

1. *Ali Abdulrman Alzurayq, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. aalzurayq@hotmail.com
2. Ahmad Hmdan Alonazi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. Ahmed.h.alonazi@gmail.com
3. Ghanim Mohammed Sayer Alshammry, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. qqqccc8@gmail.com
4. Yahya Nasser Bakeet, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. bakheetyahya9@gmail.com
5. Mansour Abdullah Ali Alqahtani, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. manssor.q1415@gmail.com
6. Omar Mawqad Almutairi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. omar.mutairii@gmail.com
7. Mohmmed Mushafil Mohmmed Hazazi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. MH618823@GMAIL.COM
8. Abdulrahman Eid Alonazi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. ksa-2002@live.com

9. Bader Muslih Alharbi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. Bder.t0t@gmail.com
10. Duaa Mohammed Alkhudaimi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. duaa5895@hotmail.com
11. Mohammed Abdullah Alredini, Anestheia Technicain, Al-Iman General Hospital, Ministry of Health, Kingdom of Saudi Arabia. Malrdeni@gmail.com
12. Mohammed Saad Almutairi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. o2x6@hotmail.com
13. Ali Ahmad Ali Alsahabi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. ali-alsahabi@outlook.sa
14. Muhannad Abdulmohsen Aljammaz, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. muhannad700@gmail.com
15. Majed Hajij Alkhaldi, Anestheia Technicain, Al-Iman General Hospita, Ministry of Health, Kingdom of Saudi Arabia. majedalkhaldi9111@gmail.com
16. Mohmmad Ali Nuwayl Almajanuni, Anesthesia Technician, Shqra General Hospital, Ministry of Health, Kingdom of Saudi Arabia. mohhmmadali@gmail.com
17. Break Hamad Al Omari, Anesthesia Technician, Shqra General Hospital, Ministry of Health, Kingdom of Saudi Arabia. BuAlamri@moh.gov.sa
18. Mohammed Rasheed Alharbi, Anesthesia Technician, Shqra General Hospital, Ministry of Health, Kingdom of Saudi Arabia. MAlharbi565@moh.gov.sa

Summary

Anesthesiologists play a crucial role in preserving neuronal homeostasis during neurosurgical emergencies by ensuring that patients have enough oxygen, ventilation, and hemodynamic conditions. A systematic approach may be helpful in diagnosis and therapy. Understanding the different aspects of airway management, breathing tactics, and anesthetic considerations in this review is crucial since these decisions have the potential to impact cerebral physiology and ultimately patient outcomes. When intubating a patient, RSI is typically the recommended method. Ventilation should be modified to preserve eucapnia and maximize brain

perfusion. During neurosurgical emergencies, neuroprotective measures that provide sufficient perfusion can lead to better outcomes. Cerebral perfusion pressure is disrupted and intracranial pressure is raised in many intracranial situations, such as trauma and intracranial vascular emergencies. A key component of anesthetic therapy for spinal traumas is preventing secondary injury following traumas to the spine and ensuring adequate perfusion to the spinal cord.

Introduction

Upper airway obstruction and respiratory failure are common unfortunate sequela among neurosurgical emergencies, especially if they have progressed to life-threatening complications. Although neurons are renowned to be extraordinarily prone to ischemia and metabolic insults, they may be restored if their homeostasis is reestablished rapidly, but a delay of even just a few seconds can cause irremediable cell damage. Most of these patients will require general anesthesia to allow mechanical ventilation and endotracheal intubation . In addition, pharmacologic treatments such as sedation to endure essential diagnostic procedures or urgent surgical treatments caused by their neurosurgical issues or confusional states include agitation, anxiety, or delirium. As a rule, general anesthesia is a necessary component of urgent medical and surgical intervention in the form of craniotomies, endovascular procedures for ischemic strokes, and osmotic medication or surgical interventions intended to lower the intracranial-pressure . Furthermore, sedation is common for patients with neurosurgical emergencies who are presented in the ER and intensive care units. For this reason, it is vital to understand the physiologic effects, pharmacologic characteristics, and typical adverse effects of these medications. In this review, we discuss vital and frequently specific issues for anesthesia and airway care in patients having neurosurgical emergencies.

Approach to a Patient with Altered Mental Status.

Neurosurgical procedures are distinct in that an intact mental status is the most significant indicator and the neurologic examination is the best monitoring tool. The neurosurgery team can be informed of the urgency of a patient's care by an altered mental state (AMS).

A useful tool for evaluating damage at various levels of the central nervous system (CNS) in accordance with reaction to certain stimuli is the Glasgow Coma Scale **(GCS; Table 1).** It uses a 15-point rating system to classify and estimate the severity and results of brain damage (1,2). When the GCS is 8 or

lower, or when there is a noticeable fall in the GCS, intubation, and airway protection are advised. In general, brain damage is classified as follows:

- Severe, GCS <8 to 9
- Moderate, GCS 8 to 12
- Mild, GCS 13 to 15

Testing the verbal and eye responses is challenging when there is tracheal intubation and significant face and ocular edema. Under these conditions, the score is assigned a modifier (either "t" for intubation or "c" for closed eyes) and is set at one.

Recently, the GCS Pupils Score was clarified. To get the total score, the estimated GCS is subtracted from the Pupil Reactivity Score **(Table 2).** The evaluation of emergent intervention's nature necessitates a combination of these two scores (1).

A shift in the mental state may occur during treatment or may be the first indication of a CNS pathologic condition. Following treatment for a subarachnoid hemorrhage (SAH), a shift in mental state may indicate a rebleeding or vasospasm, both of which require rapid intervention for diagnosis and treatment, such as angiography or neurosurgical intervention. Intracranial pressure (ICP) reduction measures are started right away and will be covered in more detail later. Acute exacerbation of specific medical diseases can also manifest as AMS change, with a range of underlying reasons including pituitary tumors, hypothyroidism, substance addiction, and prescription errors (3).

Following a craniotomy, a patient's delayed emergence is regarded as a shift in mental status since it indicates a potential surgical problem because the patient does not return to normal consciousness. The following are contributing factors: hyponatremia, hypoglycemia, cerebral hemorrhage, subclinical seizures, poor reversal of neuromuscular blockade, ventilation and oxygenation issues, and electrolyte imbalances. Reviewing anesthetic elimination and muscle relaxation reversal, arterial blood gas (ABG) data, and, if necessary, a computed tomography (CT) scan, can all help with an appropriate diagnosis.

A space-occupying lesion (such as a tumor, hemorrhage, or hydrocephalus) or an elevated ICP might also first manifest as AMS. In a later debate, management is described.

To identify medication-related AMS, including polypharmacy, a review of the patient's prescription regimen is crucial. Numerous anticonvulsants have the potential to produce drowsiness, which could be

mistaken for altered consciousness. The prognosis of an intracranial pathologic disease already present can deteriorate due to the influence of metabolic causes of AMS. Both hypoglycemia and hyperglycemia can significantly worsen outcomes, thus they should be addressed right away. Iatrogenic causes of electrolyte imbalances include the use of diuretics, mannitol, and hypertonic saline in the treatment of elevated intracranial pressure. Sepsis, systemic infections, and localized central nervous system infections are the three categories of infectious causes of AMS. Results are directly impacted by the rapid detection and management of these infectious processes.

The anesthesiologist should be aware of potential iatrogenic causes of AMS even though the neurosurgical team is primarily responsible for its investigation. This is because treating and managing AMS will require numerous, urgent interventions, including making sure the airway is protected at all times. The patient is more likely to aspirate, thus intubation and mechanical breathing may be necessary immediately. Once hypoxia and hypercapnia have been corrected, a thorough review of history and physical examination can aid in the diagnosis and, if required, the course of treatment.

Airway Management

Neurosurgical emergencies may result in respiratory failure or airway obstruction that requires ventilator support. Brain perfusion and physiology are directly impacted by techniques used for airway management, intubation, ventilator support, and sedative selection (4). Patients with a minimal risk of aspiration may benefit from noninvasive positive pressure breathing, but patient selection and monitoring must be extremely careful. Intubation is usually necessary to maintain airway patency in patients with neurosurgical emergencies. When there is a risk of cardiopulmonary decline and trouble breathing or oxygenating, intubation may be necessary. Failure of airway protection (due to decreased awareness and/or oropharyngeal poor coordination), neuromuscular respiratory exhaustion, and/or predicted cognitive deterioration are particularly concerning in neurosurgical emergencies (4). Although the gag reflex has been used historically, it is not a reliable sign of airway protection in patients experiencing neurosurgical emergencies, since up to 25% of healthy people do not have the reflex (5). The capacity to swallow, the amount and quality of secretions secreted, and the strength of the cough reflex are all factors that impact airway protection (6). Intubation has been linked to a decreased death rate in individuals with Glasgow Coma Scale scores of around 8, which is also symptomatic of impaired protective airway reflexes (7). When feasible, a focused

neurological examination should take place concurrently with immediate airway care. An assessment for intubation and bag-mask ventilation difficulties should be part of the first evaluation. Some useful mnemonics are "MOANS" **(table 3)** for difficulty with bag-mask ventilation and "LEMON" **(table 4)** to forecast problematic endotracheal intubation.

Anesthetic Induction

According to the Emergency Neurologic Life Support (ENLS) algorithm, rapid sequence intubation (RSI) is the preferred technique to reduce the risks of hemodynamic instability, worsening of neurological damage, respiratory complications, and gastric content aspiration (4). Patients experiencing neurosurgical emergency should be especially concerned about the sympathetic reflex caused by laryngoscopy, Since the associated hypertension may be deleterious. Pharmacologic pretreatment, with a rapid intravenous lidocaine bolus, provided 60 to 90 seconds before laryngoscopy(8)(9)(10), has been reported to reduce this hemodynamic response and associated intracranial pressure (ICP) effect,(8) however, data are limited. It also should be considered to give a short-acting opioid, such as fentanyl, which can be given 30 to 60 seconds before a laryngoscopy at a dose of 2 to 3 mg/kg (4). However, due to the possibility of hypotension, this should not be done in patients who have severe hemodynamic instability or who are dependent on sympathetic tone (11).

To prevent hypotension during peri-intubation, it is crucial to monitor hemodynamics closely. Vasopressors should ideally be placed at the patient's bedside and connected via an intravenous line. If the ICP is known, a cerebral perfusion pressure (CPP) of more than 60 mm Hg or a mean arterial pressure (MAP) of 80 to 110 mm Hg should be maintained (12). The ENLS recommendations state that etomidate (0.3 mg/kg) or ketamine (2 mg/kg) can be used for induction (4). For the induction of anesthesia in these individuals, propofol (1 to 2 mg/kg) is still often utilized. Lower induction dosages may be required for a large number of patients with neurosurgical emergencies to reduce the hemodynamic adverse effects. Either rocuronium or succinylcholine are typically used to achieve paralysis. After induction, sedation and analgesia should be easily accessible at the patient's bedside. Reduce the number of noxious stimuli, such as tracheal suctioning, as these may raise ICP.

Mechanical Ventilation

The maintenance of physiologic homeostasis in intubated patients requires careful adjustment of mechanical ventilation, given that Pa CO2 is a powerful acute modulator of cerebral vascular tone and cerebral blood flow. Given the

potential of cerebral vasoconstriction and reduced cerebral blood flow (CBF), hyperventilation should generally be avoided (13)(14). Hyperventilation may be temporarily employed in patients with acute ICP and potential herniation while other treatment procedures are being implemented. Nonetheless, it is crucial to take into account the dangers of severe vasoconstriction and the major CBF drop brought on by hyperventilation (15). While final treatment is provided, a brief course of hyperventilation lasting less than two hours to a PaCO2 of 30 to 35 mm Hg may be recommended. Long-term hyperventilation is neither safe nor effective because, among other things, cerebrospinal fluid buffering prevents it (16)(17). In central neurogenically hyperventilator patients who are at high risk of exacerbation of brain damage caused by ischemia, Targeted therapies (e.g., intravenous opioids, benzodiazepines, or even general anesthetics) may be considered to suppress the respiratory drive in addition to therapies aimed at treating the underlying pathophysiology (18). Additionally, hypoventilation can be dangerous since it can lead to detrimental cerebral vasodilation, especially in those with intracranial hypertension. Similarly, in cases of metabolic acidosis, the metabolic disturbance may worsen if the compensatory respiratory effort is suppressed through sedation or neuromuscular blockade (4).

While it is well known that hypoxia can result in brain damage, hyperoxia (Pa O2 >300 mm Hg) can also induce injury to the reperfusion system because of reactive oxygen species, which can worsen the prognosis of patients suffering from traumatic brain injury (TBI) and global hypoxic-ischemic injury following cardiac arrest (19). Weaning oxygen to the lowest fraction of inspired oxygen (Fi O2) that keeps the O2 saturation over 94% is the appropriate course of action after a successful intubation.

It has been demonstrated that moderate positive end-expiratory pressure (PEEP) (<12 cmH2O) doesn't significantly affect the ICP or CPP in individuals with acute ischemic stroke; this likely applies to the majority of people with acute neurologic damage (20). Because high PEEP affects intrathoracic and venous pressures, it may exacerbate intracranial hypertension (21). Depending on the clinical situation, caution should be used, keeping in mind that impaired ventilation or oxygenation due to insufficient PEEP can also have negative effects. For patients who require high levels of PEEP to maintain appropriate oxygenation and are at risk for rising ICP, ICP monitoring is recommended.

Anesthesia and Sedation

For ventilator synchronization, endotracheal tube tolerance, and ICP minimization in intubated patients with neurologic emergencies, sedation and analgesia may be required. The goal is to avoid oversedation and

preserve the capacity to clinically assess a patient's neurologic status by using the lowest dose of sedative/analgesic that preserves comfort and ventilator synchronization. Propofol is a preferred medication in neurology intensive care units (ICUs) because of its efficiency and very brief duration of action. Also often used and generally well tolerated by patients is dexmedetomidine, a centrally acting alpha 2 adrenoceptor agonist. It is important to use caution when administering dexmedetomidine because it can cause dose-dependent and idiosyncratic bradycardia and hypotension. This is particularly concerning when administering bolus doses (22).

Additionally, weaning needs to be done cautiously because of its prolonged context-sensitive half-life, even if some patients may also experience agitation, hypertension, and tachycardia as a result of dexmedetomidine withdrawal. Despite having a longer duration of action and a correlation with delirium and cognitive impairment, benzodiazepines are not the best sedatives for these patients; nonetheless, their anticonvulsant qualities make them useful for individuals experiencing seizures. This is especially crucial because delirium is frequently seen in individuals experiencing neurologic problems and has been linked to abnormalities in the nervous system (23). Consequently, it is preferable to provide benzodiazepines in single boluses as opposed to continuous infusions when not using them to treat seizures.

Targeted sedation and delirium scores, daily sedation interruption or minimization, linked spontaneous awakening and breathing trials, and early mobilization of ICU patients are a few structured and customized sedation strategies that have been linked to improved patient outcomes and can lower the risk of excessive or prolonged sedation (24). Nonetheless, these crucial actions must be customized to the unique requirements and capacities of the patient with a neurological injury. For example, spontaneous breathing trials are not recommended in patients with active seizures or increased ICP, and early mobilization following a stroke may be detrimental. The use of inhaled anesthetics for ICU sedation is a new trend in ICU sedation; nevertheless, further research is required to determine the safety of breathed drugs for extended periods (25).

Acute Increase in Intracranial Pressure.

Because the cranium is a tight space with poor compliance, even a slight volume change can increase intracranial pressure. a rise in ICP results in ischemia and edema due to a decrease in blood flow. The brain, intracranial blood, and cerebrospinal fluid (CSF) are the three components of the cranium. The Monroe-Kelly doctrine states that a rise in any one of these factors will raise the ICP unless it is followed by a decrease in the others.

A rise in ICP can be preceded by symptoms such as AMS, restlessness, irritability, and disorientation. Voluntary emotions, feelings, and extraocular movements will slow down as ICP continues to rise. T-wave alterations will also appear on the ECG as a result of the sympathetic surge. The patient may have projectile vomiting and cardiac arrhythmia ranging from supraventricular tachycardia to severe bradycardia, that develop to unconsciousness when pressure around the medulla rises. A brain herniation that is about to happen will cause the pupils to dilate, become unresponsive, and turn outward, impairing extraocular movement. If time permits, a CT scan can be used to diagnose midline shift, herniation, and the degree of brain injury or bleeding in addition to clinical signs and symptoms (26).

Both epidural and subdural hematomas, as well as other related injuries, are linked to traumatic brain injury (TBI). A fracture of the temporal bone can cause an epidural hematoma, which lacerates the middle meningeal artery beneath it. There is a brief "lucid interval" after the initial loss of consciousness. If treatment is not received, signs and symptoms of an imminent brain herniation and elevated ICP will eventually appear. The results of the CT scan show a convex lens-shaped hyperdensity, which is the result of blood clotting between the dura and the skull bone. On the other hand, there is no clear "lucid interval" when subdural bleeding happens between the dura and arachnoid.

A true neurosurgical emergency is when there is an abrupt rise in intracranial pressure and a possible brain herniation. It is required to receive concurrent medical treatment in addition to a surgical decompression. The objective is to maintain cerebral perfusion pressure, which can be characterized as the differential between ICP and mean arterial pressure (MAP). While elevating MAP can enhance cerebral blood flow, it may have adverse effects if intracranial hemorrhage or hypertensive crisis are the underlying causes of elevated intracranial pressure. Anesthesiologists are essential to the medical care of these patients because they carry out brain protective procedures, maintain cerebral perfusion, and lower intracranial pressure. The latter is accomplished by lowering the oxygen requirement and cerebral metabolic rate. When a patient is unconscious, it's critical to protect their airway against aspiration by performing endotracheal intubation and anesthetic induction.

In addition to regulating end-tidal carbon dioxide, mechanical ventilation aids in preventing further spikes in intracranial pressure caused by hypercarbia.

The following is a summary of the induction-based anesthetic goals:

- Reduce the amount of hemodynamic reaction to intubation and laryngoscopy.
- Maintain the oxygen delivery to neurons and the cerebral perfusion pressure.
- Reduce movement of the Cervical spine during intubation and laryngoscopy.
- Quickly and securely fasten the endotracheal tube.

Given the urgent nature of the procedure and the potential for insufficient airway assessment due to AMS, it is advised to have challenging airway equipment on hand. Depending on the patient's hemodynamic state, any induction drug can be given at the right dose, although ketamine may raise ICP even higher. Hypotension should be avoided at all costs as it is harmful to TBI patients. When a patient is uncooperative, rapid sequence intubation is preferred. intubation can be done with manual in-line stabilization to protect the airway from potential gastric contents aspiration. Because there may be underlying skull base fractures in individuals with traumatic brain injury, nasotracheal intubation should be avoided.

Invasive blood pressure (BP) monitoring is advised in addition to normal American Society of Anesthesiologists (ASA) monitors since it allows the serial ABG to be checked for hemoglobin, glucose, electrolytes, and other parameters. Urine output monitoring calls for the placement of a Foley catheter, but only after it has been determined that there has been no damage to the bladder or ureter. In certain situations, advanced monitoring methods like transcranial Doppler, which measures cerebral blood flow (CBF) and velocities, and near-infrared spectroscopy (NIRS), which continually measures regional cerebral tissue oxygenation, may be employed. Venous air embolism can be quickly diagnosed with transthoracic echocardiography and/or Doppler to determine the underlying heart functional state.

Adequate intravenous access is necessary for the quick delivery of fluids and drugs, including vasoactive agents, as well as the transfusion of blood products, just like in any major emergency operation. A central venous catheter (CVC) may be necessary for several reasons, such as inadequate peripheral vascular access, the need for hypertonic saline (3% or higher concentrations) or vasoactive agent infusion, the necessity for central venous blood pressure measurements (such as large fluid shifts) or blood draws (such as mixed venous oxygen saturation), the necessity for a jugular bulb or pulmonary vein access path, the risk of venous air embolism, and the need for long-term venous access in the intensive care unit (ICU) (eg, total parenteral nutrition). Aside from the time constraints, moving the cervical spine is nearly definitely necessary for placing a jugular line, which is

something that patients with traumatic brain injury should probably avoid. If ICP is high, Trendelenburg's positioning for CVC implantation may be harmful. Pneumothorax is a danger associated with subclavian line placement that can quickly raise PaCO2 and ICP. Therefore, it is important to carefully weigh the benefits and hazards. The implantation of these lines shouldn't be postponed due to surgical decompression. A femoral CVC may occasionally be the only choice available, even though it can raise the risk of infection. Another good substitute is a CVC that is implanted peripherally.

ICP can be monitored via the external ventricular drain (EVD) transducer if the patient already has one in place. The EVD helps with CSF draining to lower ICP and helps assess cerebral perfusion pressure. In individuals with elevated ICP, lumbar drain placement, lumbar CSF collection, and subarachnoid access should be avoided as they can result in brain herniation.

To optimize venous outflow from the head, patients with excessive ICP should be positioned head up. It's crucial to stay away from tight neck tape, severe neck flexion or rotation, and stimuli like endotracheal suctioning that might cause Valsalva responses. ICP can be lowered during anesthesia by keeping patients comfortably sedated and calm. This is achieved by decreasing metabolic demand, ventilator desynchrony, venous congestion, and sympathetic reactions.

It has been demonstrated that maintaining mechanical ventilation to lower Pa CO2 to 26 to 30 mm Hg will quickly lower ICP by vasoconstriction and lower the amount of intracranial blood; a 1-mm Hg change in Pa CO2 is linked to a 3% change in CBF (27). In the first 24 hours following a traumatic brain injury, hyperventilation is not advised, according to several recent studies (28). Even slight hyperventilation during this time will exacerbate neurologic results by causing vasoconstriction and ischemia in damaged brain regions. Hyperventilation should only be considered in the first 24 hours if there is a serious risk of an imminent herniation and the ICP is high. The episode of hyperventilation should be extremely brief, even in this case (28).

By avoiding all free water (including D5W [5% dextrose in water], 0.45% saline, and enteral-free water) and utilizing only isotonic or slightly hypertonic fluids (such as normal saline), patients with elevated ICP should be kept euvolemic and normo-osmolar to hyperosmolar. It is recommended to maintain serum osmolality levels above 280 mOsm/L, often within the range of 295 to 305 mOsm/L. Especially when combined with SAH, hyponatremia is frequently observed in the context of increased ICP.

Studies on the effectiveness of colloid versus crystalloid fluid resuscitation in patients with increased ICP have yielded conflicting results. One big study's subgroup analysis revealed that using albumin for fluid resuscitation during traumatic brain injury (TBI) patients was linked to a greater death rate than using normal saline (29).

Anticoagulation and Neurosurgical Emergencies

The number of patients requiring emergency neurosurgical intervention who are on anticoagulant or antiplatelet therapy has increased due to the growing number of patients being treated with oral anticoagulation for many other reasons. Clinical scenarios frequently involve a patient who has acute AMS following a fall while taking anticoagulant medication.

To remove the hematoma, these individuals need both depressive neurosurgery and an immediate reversal of anticoagulation. As previously mentioned, anesthesiologists are involved in the management of elevated ICP and the reversal of anticoagulation.

The prognosis for cerebral hematomas associated with anticoagulants is particularly bad; after three months, case fatality rates might reach 50% (30). Before stopping the patient's anticoagulation, it's critical to go over their prescriptions and their indications. Prothrombin complex concentrate (PCC) must be administered right away to maximize the therapeutic impact on these patients' anticoagulation due to the significant risk of hematoma expansion in the first few hours after onset. Recently, a large observational study of 853 patients with vitamin K antagonist-intracerebral hemorrhage receiving PCC at 19 German centers was published. It showed that treatment administered within 4 hours of hospital admission is associated with a significant reduction in hematoma enlargement (19.8% vs. 41.5%) (31).

Carotid Artery Stenosis

Neurologic signs and symptoms of insufficient intracranial hypoperfusion, such as transient ischemic attack or stroke, are indicative of carotid artery stenosis and require immediate, if not urgent, medical attention. Endovascular stenting or open endarterectomy are the two possible courses of treatment. There might not be enough time in either case for a thorough assessment and patient optimization (32).

The anesthesiologist should take into account any related comorbidities, such as diabetes mellitus, hyperlipidemia, and smoking history, that may have an impact on the vascular supply in other organs. Patients with these comorbidities may be at an increased risk of developing

heart failure, hypertension, chronic obstructive pulmonary disease, renal insufficiency or failure, and coronary artery disease. Although carotid stenosis can be treated intravascularly without MAC, complications can always arise and need an instant switch to general anesthetic and intubation. The abrupt bradycardia that can occur with carotid stent deployment is usually self-limited, but if symptoms persist, medical attention may be necessary. Regional anesthesia is another option for the open carotid endarterectomy, which allows for an awake patient to undergo continuous neurologic examination during the carotid artery cross-clamping procedure. On the other hand, most neurosurgeons would rather operate under general anesthesia. When assessing the collateral blood supply during a carotid cross-clamp, electroencephalography (EEG) is typically the preferred method due to its ability to detect a slowing of the EEG waveform in the event of insufficient perfusion. NIRS is another tool the anesthesiologist can use to verify that the perfusion is sufficient. It is recommended to use invasive hemodynamic monitoring to identify hypotension and induced hypertension to improve collateral flow during a cross-clamp procedure, regardless of the chosen course of treatment. Additionally, cautious hemodynamic monitoring is required following an endarterectomy or the implantation of a carotid stent to prevent hypertension, which may result in hyperperfusion injury to the brain (33) (34).

Intracranial Aneurysms and Arteriovenous Malformations

In most cases of SAH, the cause is the rupture of an intracranial aneurysm. The symptoms can range from focal neurologic deficits and/or AMS (see earlier discussion) to headaches, which are typically referred to as "the worst headache" of the patient's life, neck stiffness, or pain. Treatment options include open microsurgical clipping or endovascular coiling.

When to operate on an aneurysm depends on the patient's clinical condition and other related considerations. Candidates in good health or those with unstable blood pressure, seizures, thrombus mass effect, significant volumes of blood, or signs of aneurysm expansion or rebleeding are preferred for early surgery (i.e., <48–96 hours after SAH). While there is a significant decrease in the risk of vasospasm and rebleeding, there is still an increased operative morbidity associated with early surgery. Patients with poor clinical status or large aneurysms in challenging locations may be candidates for delayed surgery (10–14 days following SAH). Patients without significant age or extenuating contraindications should consider surgery for ruptured or symptomatic aneurysms. In general, poor clinical state (equivalent to Hunt and Hess grade 4 or 5) precludes surgery (35).

Nonetheless, regardless of the time, size of the aneurysm, or length of operation, a retrospective analysis analyzing 105 patients, 23 found that younger patients with high clinical grade were associated with a favorable neurologic outcome both at discharge and at 6 months following surgery (36). Continuous communication with the surgeon is required during open microsurgical clipping to initiate burst suppression and protect the brain, induce hypotension (during surgical dissection around the aneurysm), and induce hypertension (when a temporary clip is required) to allow collateral perfusion of the brain areas affected by the temporary clip.

Cerebral vasospasm is one of the potential outcomes of SAH, requiring urgent interventional angioplasty and/or vasodilator injection. Anesthesiologists should focus on hemodynamic objectives, such as sustaining a sufficient intravascular volume and inducing hypertension. During angiography, selective intraarterial nicardipine administration might result in substantial hemodynamic instability, necessitating supportive anesthesiologist care (37).

AVM rupture may require immediate surgical intervention and can result in SAH with or without elevated ICP. Anesthesia treatment is comparable to that for intracranial aneurysms. After removing an AVM, there is a chance that backflow of pressure will occur from cutting off the low-pressure venous flow of a high-pressure arterial anomaly, which could cause cerebral edema. To see bleeding during AVM removal, the surgeon may need to temporarily clamp feeder arteries. The operator can detect and manage arterial bleeding with the use of strict blood pressure management. Short-acting medications like nitroprusside or esmolol should be taken into consideration to achieve this. The benefits of blood pressure regulation on vasogenic edema and bleeding risk exceed the theoretical danger of vasodilator-induced steal. Induced hypotension might need to be maintained during the postoperative phase to reduce the possibility of cerebral edema from a normal perfusion pressure breakthrough (38).

Management of Patients with Ischemic Stroke

Individuals who show signs of an acute ischemic stroke must have CBF replacement as soon as possible to preserve any remaining ischemic brain tissue. When administered within 4.5 hours of the onset of symptoms, intravenous tissue plasminogen activator (alteplase), the first line of therapy for reperfusion, improves functional outcomes at 3 to 6 months. 18 Up to 24 hours after the onset of symptoms, a mechanical thrombectomy and the removal of the triggering clot can be carried out. These patients are being managed by neuro-anesthesiologists as they undergo endovascular therapy, which is used for both diagnosis and treatment (39).

General anesthesia or monitored anesthesia care (MAC)/sedation can be used for therapy. To reduce treatment delays, a brief and targeted pre-anesthesia evaluation must be carried out while the patient is being readied for the procedure.

The use of sedation instead of general anesthesia during thrombectomy has not been demonstrated to produce improved results in recent research. 150 patients with acute ischemic stroke in the anterior circulation were included in the SIESTA trial (40), and during stroke thrombectomy, the patients were randomized to two groups: one under intubated general anesthesia and the other under non-intubated conscious sedation. The main result, early neurologic improvement at 24 hours, did not differ statistically between the two groups; therefore, the results do not suggest that conscious sedation is advantageous.

In the General or Local Anesthesia in Intra Arterial Therapy experiment, which compared conscious sedation and general anesthesia for anterior circulation ischemic stroke, there was no discernible difference in the tissue or clinical outcomes following general anesthesia (41).

The authors advise appropriate patients who can maintain their airways and who are compliant to use MAC as the first line of anesthetic therapy. It is important to remember that a quick switch to general anesthesia may be necessary. MAC offers several benefits, including facilitating immediate neurologic assessment, enabling a rapid start of treatment, and reducing hemodynamic fluctuations compared to general anesthesia. Standard ASA monitors as well as emergency medications and supplies must always be on hand. Although an invasive arterial blood pressure monitor can help maintain strict blood pressure management, the treatment shouldn't be postponed if the arterial line placement takes longer than expected.

The following are additional crucial intraoperative factors to take into account:

- Blood pressure control: Prolonged hypotension exacerbates neurological consequences and should be avoided. Vasopressors, such as alpha agonists like phenylephrine or norepinephrine infusions, may be used to treat hypotension. Because there is a chance of hemorrhagic change in the reperfused tissue, severe hypertension should be avoided, especially after reperfusion. Labetalol and/or niacin can be used to treat hypertension.
- Anticoagulation: To reduce additional thrombotic events, intravenous heparin is given during these procedures. Heparin

activity should be tracked by measuring active clotting time (ACT), with a target ACT of 250 to 300 seconds (42).

- Postoperative ICU admission and continued monitoring are necessary for these patients.

Acute Cervical Spinal Cord Injury

Like the brain, the spinal cord is made up of neurons that are extremely susceptible to ischemia and hypoperfusion. Acute interruption of the spinal cord's vascular supply is the primary cause of most spinal cord emergencies. Emergencies include tumors or degenerative diseases generating unstable alterations and pressure to the spinal cord, abscesses, trauma, and epidural or subdural hematomas. Clinically, these may show up as an abrupt loss of motor and/or sensory abilities both above and below the site of the lesion.

Two posterior and one anterior spinal artery supply the spinal cord with blood, and specific low-flow or watershed locations are more vulnerable to ischemia-induced injury. Maintaining the spinal cord perfusion pressure is the aim, regardless of the pathologic condition, to prevent neurologic symptoms from getting worse and to prevent secondary injuries from spreading and potentially transecting the spinal cord or severely decreasing its blood supply.

Traumatic spinal cord injuries (SCI) affecting the cervical spine put patients at very high risk for respiratory failure because of several factors, such as loss of strength in the chest and abdominal walls, trauma-related airway edema, and diaphragmatic innervation loss (C3, C4, and C5), which inhibits ventilation. Early, elective intubation and artificial breathing should be explored for all patients with a complete cervical traumatic SCI (12). The ability of patients with partial or lower injuries to maintain appropriate breathing and oxygenation will vary. Breathlessness, a vital capacity of less than 10 mL/kg, or a declining vital capacity are factors to take into account with these patients. Additionally, the occurrence of "quad breathing," which is characterized by a sudden exhalation of air from the abdomen, indicates a necessity for intubation. When in doubt, elective intubation of a patient with a cervical traumatic SCI is preferable to waiting until an emergency occurs (4).

In most cases, a skilled practitioner should use a fiberoptic technique to intubate patients with cervical traumatic SCI (43) (44). Alternatives such as video or direct laryngoscopy are appropriate in emergencies or when fiberoptic equipment is unavailable (45). It is crucial to maintain cervical stability irrespective of the intubation technique selected. Always be careful for aspirating, and take RSI into account. Be cautious when selecting an

induction regimen because sympatholytic drugs may produce new or worsening bradycardia and hypotension in these patients, who typically suffer loss of vasomotor tone due to SCI. It is important to use caution while assessing for coexisting conditions such as pulmonary contusions pneumothorax, and rib fractures that could affect intubation and mechanical ventilation. Since the inability to cough and clear secretions raises the danger of aspiration, noninvasive ventilation might not be the best option (12).

A kind of distributive shock known as neurogenic shock, which is characterized by bradycardia and hypotension, frequently develops in patients with severe SCI above T6. Patients may have dry, heated skin and hypotension as a result of diminished sympathetic tone. Other possible causes of hypotension in traumatic injury patients, such as bleeding, need to be carefully considered. Targeting a MAP of 85 to 90 mm Hg for the first seven days of hypotension management is widely acknowledged, despite the absence of compelling evidence to support this strategy (12). The first step in this process is to provide appropriate volume resuscitation using intravenous fluids, colloids, and blood transfusions as needed. After that, try adding second-line therapy with inotropes and vasopressors if there is ongoing hypotension. Because it has both beta and alpha action, norepinephrine is the ideal medication for treating bradycardia and hypotension. Because it is simple to administer and titrate, phenylephrine is frequently used; nevertheless, care should be taken because it does not treat bradycardia and could make it worse through reflexive processes (43). Since corticosteroids have been linked to an increased risk of complications without neurologic benefit, they are often avoided in cases with traumatic SCI (46).

The following are some of the objectives of anesthesia and airway treatment in these patients:

- Evaluation of respiratory function because airway preservation from high cervical spinal cord injury may need intubation due to compromised breathing. To reduce cervical spine movement during intubation, extreme caution and attention must be used. The best way to properly secure the airway is still with an awake fiber-optic intubation with the right topicalization if the patient can handle it. Manual inline stabilization safeguards the cervical spine during intubation in certain severe cases requiring prompt care.
- Large-bore intravenous access is necessary, particularly during the first spinal shock, for fluid resuscitation. The internal jugular vein may not be preferred over the subclavian vein in cases where central venous access is needed.

- Intravascular catheterization and hemodynamic monitoring.
- Preventing hypotension is important since it can exacerbate spinal cord damage. To preserve SCPP, it is advised to keep MAP higher than 85 to 90 mm Hg.
- Maintaining C-spine immobility during patient transportation, transfer to the operating room table, and patient placement requires constant attention.

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