Dedication

This book is dedicated to my parents, my family, my teachers and the ones who mean everything to me – Amyra, Anavi, Anav, Avi and Aishwarya

COMPLICATIONS IN NEUROANESTHESIA

Edited by

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Foreword

Until about two decades ago, neuroanesthesia practice was not deemed any different from general anesthesia practice. Neuroanesthesia has of recent emerged as a challenging subspecialty of anesthesia because of the absence of leeway to its practitioners. Therefore, the anesthesia provider has to be extra vigilant during the intraoperative and postoperative periods. Owing to the vulnerability of the brain to suffer from irreversible damage following an even innocuous looking insult, failure to anticipate complication/s could make or mar the outcome of a neurosurgical patient. How to recognize complications quickly and manage them efficiently, therefore, is central to the practice of neuroanesthesia.

There are quite a few popular textbooks on neurosurgical anesthesia practice by eminent authors from the Western world but they all have mentioned complications in a perfunctory manner. The editor of this book realized this shortcoming and took it upon himself to write comprehensively in *Complications in Neuroanesthesia*.

The editor has ensured this book is written in a very simple language, which readers will find easy to read and assimilate. Under his guidance, he has compiled the various topics penned by distinguished neuroanesthesiologists. He has tried to include all the possible complications that a practitioner of neuroanesthesia may come across at some point in time. This book is meant for both the occasional neuroanesthesiologist and those physicians who are regular providers of neuroanesthesia. Furthermore, this book will be handy not only for students of neuroanesthesia but also for the consultants of neuroanesthesia too because, it is a kind of ready reckoner. The book would prove equally useful for trainees and specialists of allied branches, such as neurointensivists, neurosurgery, neurology and neuroradiology. The simple and easy-to-understand language makes it accessible to technical and nursing staff as well. Efficient management of the complications in neuroanesthesia is not determined entirely by the experience of the anesthesia provider. It requires an in-depth knowledge of pathophysiology behind it and understanding of the surgical procedure. A half-baked knowledge may thus result in irrational management, spelling doom for the patient.

Written in a very comprehensive manner, this book is sure to find its place in the bookshelves of all neuroanesthetists.

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Preface

This book is a compilation of possible complications one can encounter during the practice of neuroanesthesia. Each chapter reviews challenges we face in our day-to-day practice. Readers will get a comprehensive insight of various complications one can possibly confront while managing neurosurgical cases in the operation theater and in the intensive care units. Familiarity with these complexities is essential for successful management and a good outcome of patients.

I am grateful to all the authors who believed in me and my proposed format of this book. Above all, I am also thankful to the patients who actually taught and made us aware of these complications. I'm sure readers will benefit from the cognizance of the renowned neuroanesthetists. The vignettes described toward the end of the book will help readers stimulate their neurons, searching for the right answer and also testing their knowledge.

The purpose of this book will be truly accomplished if we are able to improve the clinical conditions of our patients by providing better care.

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CHAPTER

1

Brain Herniation

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OUTLINE

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OVERVIEW

Brain herniation is the displacement of brain tissue through the rigid dural folds (i.e., falx and tentorium) or skull openings (e.g., foramen magnum).[1] Although patients with chronic brain herniation associated with developmental defects, such as Arnold–Chiari malformation, may remain asymptomatic for many years,[2] acute brain herniation following neurosurgery is a catastrophic event that results in mechanical and vascular damage of the brain. In many circumstances, brain herniation is often regarded as a terminal event.

MECHANISM OF BRAIN HERNIATION

From a mechanistic point of view, brain herniation is the result of a pressure gradient that squeezes the vulnerable brain matter from one compartment in the brain to another through various anatomical channels. In general, any pathologic process that increases intracranial pressure provides the driving pressure for brain herniation.[1] It should be clear that the pressure gradient appears to be the most important factor, and brain herniation may occur regardless of the size of the opening.[3] In the perioperative setting, hemorrhage, cerebral swelling associated with perioperative stroke, and hydrocephalus are the common causes for intracranial hypertension after neurosurgery (Table 1). In a systematic review of patients with clinical deterioration following intracranial surgery, 0.8–6.9% of cases were thought to be due to postoperative hemorrhage.[4] In patients who received regular imaging surveillance, up to 50% of cases had evidence of significant intracranial hemorrhage following neurosurgery.[4,5] It is reassuring that few patients with postoperative intracranial hematoma actually end up with brain herniation. However, even a small amount of blood may produce

TABLE 1 Common Causes of Intracranial Hypertension Following Neurosurgery that May Lead to Brain Herniation

1. Postoperative intracranial hemorrhage (extradural, subdural, intracerebral, or intraventricular hematoma) due to surgical bleeding or patients with bleeding tendency.
2. Brain contusion related to primary traumatic brain injury or instrumental damage (e.g., brain retraction injury).
3. Cerebral swelling due to:
   a. Perioperative stroke
   b. Exacerbation of peritumor or periabscess edema
   c. Cerebral venous thrombosis
   d. Hyperemia (e.g., hyperperfusion syndrome following carotid endarterectomy)
   e. Metabolic causes (e.g., diabetic ketoacidosis, hyponatremia, liver failure)
4. Hydrocephalus

sufficient pressure to produce significant brain herniation. This is especially important in patients who already have limited intracranial compliance.

It should be noted that not all cases of brain herniation are related to intracranial hypertension. In patients who had decompressive craniectomy, acute drainage of cerebrospinal fluid, upright posture, or hyperventilation may produce a transient negative pressure gradient between the atmosphere and intracranial compartments. This extra intracranial pressure gradient across the skull defect may be large enough to push the brain matter down into the tentorial notch or the foramen magnum, resulting in a rare phenomenon known as paradoxical brain herniation.[6,7]

CLASSIFICATION OF BRAIN HERNIATION

The brain can be broadly divided into a number of compartments, with boundaries formed by the falx, the tentorium, and the foramen magnum. When the pressure within a compartment is increased, its contents will be pushed toward the adjacent compartments. The directions of displacements are shown in Figure 1.[1] Briefly, the inner part of the temporal lobe (uncal herniation), the entire diencephalon (central/downward transtentorial herniation), and the frontal lobe (cingulate or subfalcine herniation) are common areas for herniation within the supratentorial compartment.

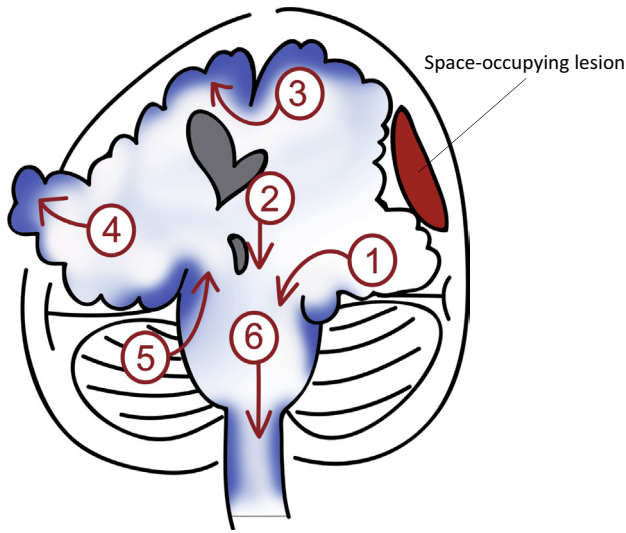

FIGURE 1 Coronal view of brain herniations. (1) uncal herniation, (2) central transtentorial herniation, (3) subfalcine/cingulate herniation, (4) transcalvarial herniation, (5) reverse transtentorial herniation, and (6) tonsillar herniation. Red arrows indicate the direction of brain displacement. Noted that reverse transtentorial herniation is due to the effect of infratentorial lesion and not related to the space occupying lesion shown (red).

In the infratentorial compartment, cerebellar tonsils may be squeezed down through the foramen magnum (tonsillar herniation or coning). In contrast, the cerebellum in the posterior fossa may also be pushed upward when the infratentorial pressure exceeds that in the supratentorial compartment (reverse transtentorial herniation). Finally, the part of brain matter that is adjacent to a craniectomy wound or site of fracture may be herniated out of the skull (transcalvarial herniation).

CLINICAL FEATURES OF BRAIN HERNIATION

The clinical presentation of brain herniation depends largely on the underlying lesion in the brain, the manifestations of intracranial hypertension, and the function of specific part of the brain that is being compressed. Table 2 summarizes the mechanisms and common clinical signs associated with different forms of brain herniations.

TABLE 2 Clinical Presentations of Brain Herniation

Type of herniation	Mechanisms	Clinical presentations
Uncal herniation	**1.** Compression on parasympathetic followed by somatic component of oculomotor nerve **2.** Infarction of ipsilateral visual cortex **3.** Lateral displacement of brain stem to compress the contralateral corticospinal tract **4.** Distortion of the ascending arousal system **5.** Duret hemorrhage	**1.** Dilated pupils, ptosis, and "down and out position" of the ipsilateral eye **2.** Contralateral homonymous hemianopia **3.** Ipsilateral hemiparesis or hemiplegia **4.** Unconsciousness **5.** Decorticate posture, respiratory depression, and death
Central transtentorial herniation	**1.** Early stage **2.** Late stage **3.** Compression of pituitary stalk	**1.** Agitation and drowsiness; pupils are small but reactive **2.** Decorticate, decerebrate posture **3.** Diabetes insipidus
Subfalcine herniation	Compression of cingulate gyrus and intracranial hypertension	Nonspecific signs
Transcalvarial herniation	Compression against external wound	Physical sign depends on the part and extent of brain herniation
Tonsillar herniation	Brain stem compression	**1.** Unconsciousness **2.** Respiratory depression and cardiovascular instability

Uncal (Transtentorial) Herniation

The uncus is the most medial part of the temporal lobe. When it is squeezed against the tentorium, it exerts pressure on the third cranial (oculomotor) nerve as it leaves the midbrain and travels along the free edge of the tentorium. In addition, there is pressure against the ipsilateral brain stem. Not surprisingly, the earliest clinical signs associated with an uncal herniation are those due to ipsilateral third nerve palsy. It is often suggested that the parasympathetic input to the eye, lying in the outermost part of the nerve, is first affected. This will result in an ipsilateral fixed and dilated pupil. As the pressure on the third cranial nerve is further increased, the somatic component is also affected, leading to complete ptosis and deviation of the eye to a "down and out" position. However, the motor functions of the eye cannot be easily tested in an unconscious patient. Therefore, a fixed and dilated pupil may become the only physical sign of an uncal herniation. As lateral displacement becomes more severe, the brain stem is being compressed against the contralateral tentorium leading to injury of the contralateral corticospinal tract with ipsilateral hemiparesis.[8] This is obviously a false localizing sign. In addition, the contralateral posterior cerebral artery may be compressed leading to ipsilateral visual cortex infarction and giving rise to contralateral homonymous hemianopia.

When both hemispheres are under pressure, the diencephalon and parts of the temporal lobes are squeezed through the tentorial notch, resulting in central transtentorial herniation. In the early stage, the pupils are small and reactive. Interestingly, they dilate briskly in response to pinching of the neck (ciliospinal reflex). Oculocephalic reflexes and plantar responses are intact. In patients who are breathing spontaneously, yawning with occasional pauses are often observed. This may progress to Cheyne–Stokes breathing in the later stage. Further compression on the brain stem will result in decorticate posture, respiratory depression, and death.

Radiologically, one should observe the general features of intracranial hypertension in the computed tomography (CT) scans. These include midline shift, obliteration of ventricles, and effacement of sulci and cisterns. Specifically, uncal herniation produces a duret hemorrhage that appears as flame or linear-shaped hyperintensities in the brain stem. This is due to tearing of small vessels. In addition, notching of the contralateral midbrain (Kernohan's notch)[8] indicates severe lateral displacement of the brain stem (Figure 2).

Subfalcine/Cingulate Herniation

Subfalcine herniation is the displacement of the medial frontal lobe (cingulate gyrus) underneath the free edge of the falx cerebri. Symptoms

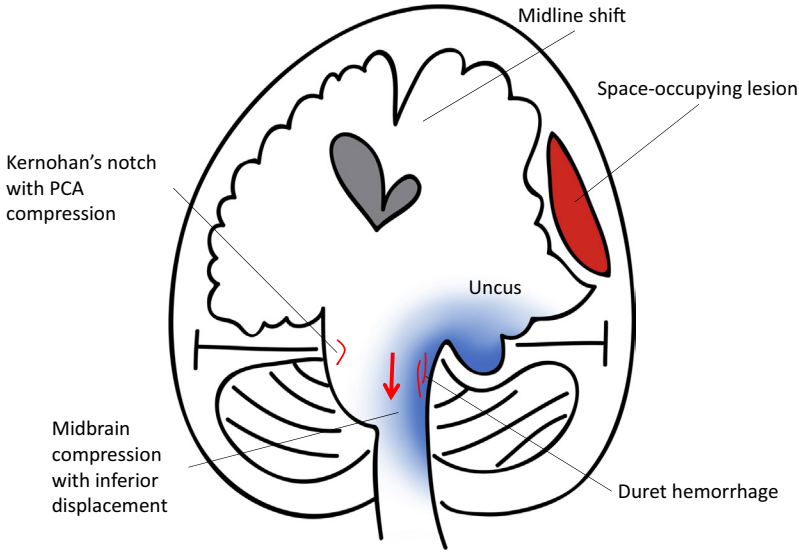

FIGURE 2 Important radiological features seen in uncal and central transtentorial herniation. Red arrows indicate the direction of brain displacement.

are generally nonspecific. On the axial image of a CT scan, subfalcine shift can be demonstrated by drawing a straight line from the anterior to the posterior aspect of the falx, highlighting the deviation of the septum pellucidum from its expected midline position (Figure 3). This may result in cerebral infarction over the ipsilateral anterior cerebral artery territory. One may also note the dilatation of the contralateral ventricle due to the obstruction of the foramen of Monro.

Transcalvarial Herniation

This is also known as "external herniation," where part of the brain is displaced out of the cranium because intracranial pressure is much larger than that of the atmosphere (Figure 4). Obviously, clinical presentation depends on the part and extent of brain matter that is herniated through the skull defect.

Reverse Transtentorial Herniation

Reverse transtentorial herniation occurs when pressure in the posterior fossa exceeds that of the supratentorial compartment. The cerebellum is therefore pushed superiorly between the free edges of the tentorium (Figure 5). Clinically, bilateral fixed dilated pupils may be the only physical sign that can be elicited. This is due to injury to the third cranial nerves as the midbrain is being compressed and the tentorium is stretched. Reverse transtentorial herniation should be suspected when there is rapid postoperative deterioration in patients with unrelieved pressure in the posterior

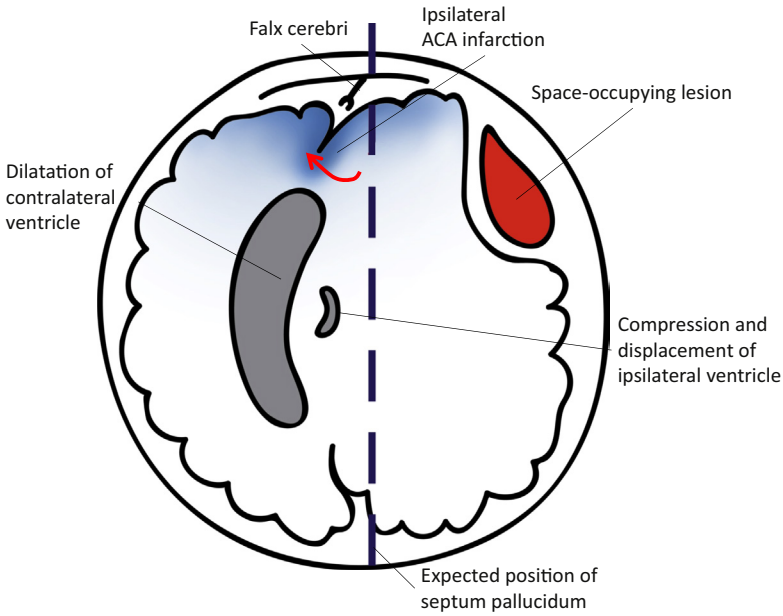

FIGURE 3 Subfalcine/cingulate herniation. Dotted line indicate the expected position of septum pellucidum. Red arrow indicates the direction of brain displacement. ACA, anterior cerebral artery.

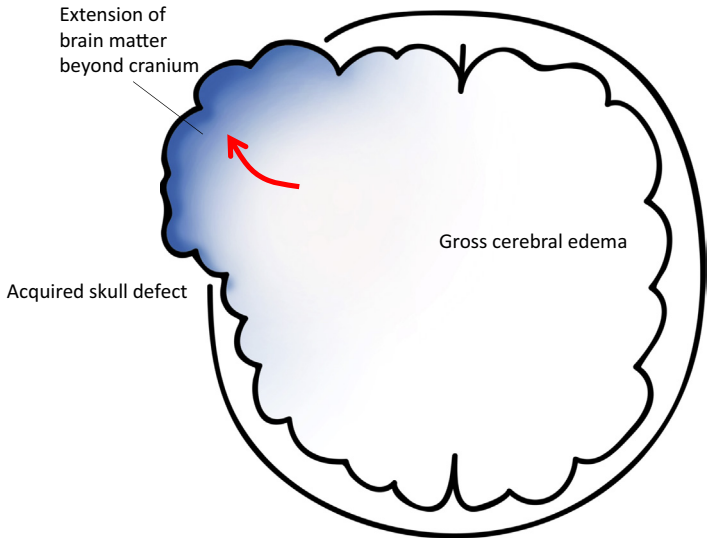

FIGURE 4 Transcalvarial herniation through acquired skull defect. Red arrow indicates the direction of brain displacement.

fossa (e.g., residual cerebellar tumor). In this setting, acute drainage of hydrocephalus produces sufficient negative pressure gradient from infra- to supratentorial compartments for brain herniation.

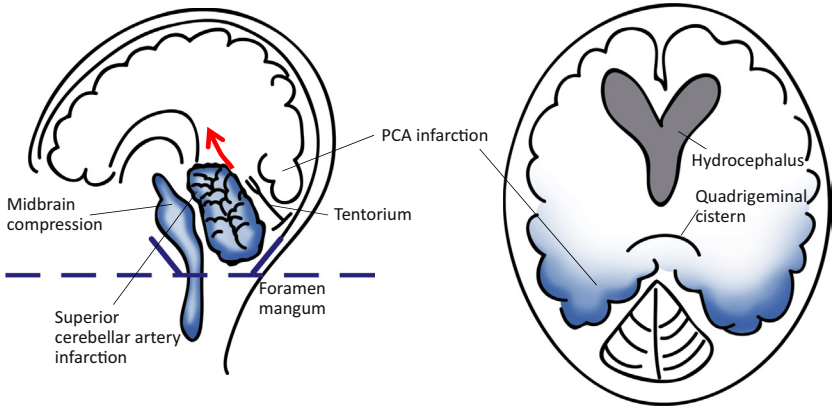

FIGURE 5 Reverse transtentorial herniation. Sagittal view (left) showing upward displacement of cerebellum toward supratentorial compartment. Transverse view (right) showing reverse and flattening of quadrigeminal cistern (frown-shaped appearance). Red arrow indicates the direction of brain displacement. PCA, posterior cerebral artery.

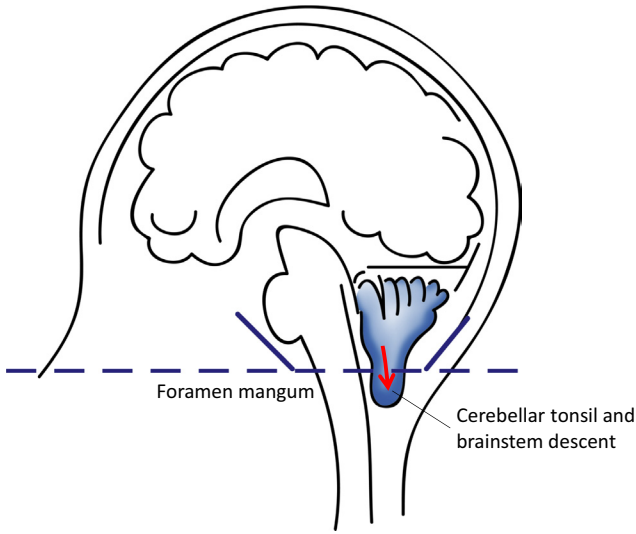

FIGURE 6 Tonsillar herniation—descent of cerebellar tonsils and brain stem beyond foramen magnum. Red arrow indicates the direction of brain displacement.

In the CT scans, flattening of the superior cerebellar cistern may be observed. The usual "smile-shaped" quadrigeminal cistern is reversed to produce a "frown-shaped" appearance (Figure 5). One should also look for infarction in areas supplied by the posterior cerebral and superior cerebellar arteries. Obstructive hydrocephalus due to compression of the cerebral aqueduct may also occur.

Tonsillar Herniation

This is commonly known as coning, when the cerebellar tonsils move downward through the foramen magnum (Figure 6). Tonsillar herniation exerts pressure over the lower brain stem and the upper cervical spinal cord against the narrow foramen magnum. In the postoperative setting, brain stem compression results in unconsciousness, flaccid paralysis, and respiratory and cardiac depression.

In sagittal scans, an inferior descent of the cerebellar tonsils, >5 mm in adults and >7 mm in children, below the foramen magnum is considered significant. The cerebrospinal fluid cisterns around the brain stem may become effaced as well.

PREVENTION OF BRAIN HERNIATION

Little is known about prevention of brain herniation after neurosurgery. Nevertheless, appropriate preoperative preparation for patients with a bleeding diathesis (e.g., desmopressin administration for von Willebrand's disease), stopping aspirin, other antiplatelets, and anticoagulants ahead of scheduled surgery or prior embolization of a vascular tumor may avoid massive bleeding during surgery and may prevent postoperative hematoma.[9] Meticulous surgery to avoid retraction injury and inappropriate coagulation of perforators will prevent postoperative ischemic brain swelling. Other maneuvers include administration of corticosteroid for brain tumor and prophylactic anticonvulsant therapy may also decrease the risk of postoperative malignant brain swelling. Despite success of endovascular treatment for acute stroke in the general population,[10] it is unclear whether the treatment for perioperative stroke could produce a similar benefit after neurosurgery.

TREATMENT OF BRAIN HERNIATION

Brain herniation following neurosurgery is a life-threatening neurosurgical emergency and requires immediate resuscitation to prevent irreversible injury and death. Treatment strategies should be directed to control intracranial pressure, so that an intracranial pressure ≤20 mm Hg and a cerebral perfusion pressure ≥70 mm Hg are the therapeutic targets. General measures include maneuvers to ensure good oxygenation, appropriate head positioning to facilitate cerebral venous drainage, osmotherapy, sedation to decrease cerebral metabolic rate, and hyperventilation to reduce cerebral blood volume (Table 3).[11,12] In addition, it is important to correct any underlying pathology. This may

TABLE 3 General Management for Control of Intracranial Pressure

1. Ensure airway patency, adequate breathing, and circulation.
   a. Tracheal intubation for airway protection and invasive mechanical ventilation.
   b. Ensure oxygenation with hemoglobin saturation ≥92%.
   c. Limit positive end-expiratory pressure <12 cm H_2O.
   d. Maintain cerebral perfusion pressure (50–70 mm Hg).
2. Positioning to facilitate cerebral venous drainage with head of bed elevated to 30°–45° in neutral position.
3. Hyperventilation to maintain arterial carbon dioxide tension of 30–35 mm Hg.
4. Correct hyponatremia and maintain normoglycemia.
5. Antipyretics for fever.
6. Use of prophylactic anticonvulsants and treat seizure promptly.
7. Administer sedation and analgesia, when appropriate.
8. Administer dexamethasone for brain tumor–related vasogenic edema.
9. Osmotherapy using mannitol (0.25–2 g/kg over 10–15 min) or hypertonic saline[a] (but keeping plasma sodium <160 mmol/L).
10. Careful drainage of cerebrospinal fluid if an external ventricular drainage device is available.

[a] *Common regimens include 3% solution at 5 ml/kg over 15 min; 7.5% solution at 2.5 ml/kg over 15 min; 23.4% solution at 30 ml over 20–30 min.*

include replacement of clotting factors to correct coagulopathy, relief of hydrocephalus, drainage of intracranial hematoma, and surgical excision of contused brain tissue. In patients who suffer from hyperperfusion after neurosurgery, aggressive control of arterial pressure using labetalol or barbiturate infusion to reduce mean flow velocity <120 cm/s of the proximal cerebral artery on transcranial Doppler monitoring may be useful.[13]

In patients with malignant brain swelling following neurosurgery, either due to infarction or hyperemia, early decompressive craniectomy may be lifesaving. Based on the experience from acute ischemic stroke, a large hemicraniectomy performed within 48 h after onset of stroke has been shown to provide survival benefit as well as functional improvement.[14,15]

CONCLUSIONS

Brain herniation is a devastating complication after neurosurgery. Early recognition of brain herniation based on clinical signs, such as failure to arouse and dilated pupils after surgery, should alert clinicians that brain herniation may have occurred. Radiological assessment should be performed to identify the underlying pathology. Timely resuscitation to decrease intracranial pressure is the key to minimize damage.

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CHAPTER

2

Delayed Emergence

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OUTLINE

DEFINITION

Emergence from general anesthesia is defined as the return of neuromuscular conduction, airway protective reflexes, and appropriate level of consciousness following discontinuation of anesthetic agents at the end of the surgery or intervention. Failure to regain the expected level of consciousness within 20–30 min of cessation of anesthetic agent administration is termed as delayed emergence.[1]

Time to emergence is variable and depends upon various patient related factors, the type of anesthetic given, and the length of surgery. A pertinent review of the medical and drug history of the patient is important, prior to considering the perioperative anesthetic management, as

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various medical illnesses predispose a patient to delayed awakening or prolonged paralysis. The differential diagnosis of delayed emergence includes residual drug effects, metabolic derangements, or neurologic disorders. All these should be excluded in a stepwise manner, and supportive ventilation continued till the patient becomes fully responsive.

CAUSES[2]

1. Residual drug effects may be because of several reasons:
   - **a.** Drug overdose: usually hypnotics or opioids: pinpoint pupils and slow, deep respiration are signs of opioid narcosis.
     Patients at extremes of age (pediatric and geriatric patients) show an unusual sensitivity to anesthetic drugs.
     Patients with preoperative cognitive and psychiatric disorders, or patients who were anesthetized while intoxicated with alcohol or recreational drugs, may be more difficult to arouse.
   - **b.** Residual neuromuscular blockade: may occur secondary to overdose, or incomplete reversal of nondepolarising muscle relaxants, or in a patient with suxamethonium apnea (due to an abnormal or absent plasma cholinesterase enzyme), or in patients with myasthenia gravis and muscular dystrophies.
   - **c.** Reduced drug metabolism/excretion: seen in liver disease, renal disease, severe hypothyroidism, hypoalbuminemia. Patients with low cardiac output may have delayed absorption of intramuscular injections.
   - **d.** Potentiation by other drugs[3]:
     - Reserpine and methyldopa decrease minimum alveolar concentration and predispose to anesthetic overdose
     - Acute ethanol intoxication decreases barbiturate metabolism
     - Antiparkinsonian drugs and tricyclic antidepressants augment sedation produced by scopolamine by their anticholinergic effects.
2. Respiratory failure: Patients with underlying respiratory disease, like chronic obstructive pulmonary disease or obstructive sleep apnea (with CO_2 retention preoperatively), or who may have received high doses of narcotics, may hypoventilate in the postoperative period, and may be drowsy because of CO_2 retention.
3. Metabolic disorders:
   - **a.** Hypoglycemia: may occur in
     - Neonates (most common in premature babies) and infants
     - Liver failure
     - Patients on insulin or oral hypoglycemic drugs
     - Sepsis

- Malaria
- Alcohol intoxication

b. Hyperglycemia: may occur in decompensated diabetics, presenting as hyperosmotic hyperglycemic nonketotic coma, or diabetic ketoacidosis

c. Electrolyte imbalance: hyponatremia, hypernatremia, hypo- or hypercalcemia may all present with delayed emergence

d. Hypothermia: decreases minimum alveolar concentration of inhalational agents, antagonizes muscle relaxation reversal, and limits drug metabolism. A core temperature of less than 33 °C has a marked anesthetic effect itself and will potentiate the central nervous system depressant effects of anesthetic drugs.[4]

e. Central anticholinergic syndrome: is a rare entity and is precipitated by drugs including antihistamines, atropine, scopolamine, and antidepressants. It is thought to be due to a decrease in inhibitory anticholinergic activity in the brain, and may manifest as confusion, restlessness, hallucinations, convulsions, and coma, and therefore as delayed awakening from anesthesia. Treatment is with physostigmine 0.04 mg/kg slowly, intravenously.[5]

4. Neurologic disorders: Certain surgical procedures, like carotid endarterectomy, cardiopulmonary bypass, and intracranial procedures, are associated with an increased incidence of postoperative neurological deficits.

a. Cerebral hypoxia due to any cause may result in delayed awakening.
Patients with chronic hypertension have an altered cerebral blood flow autoregulation, and any episode of intraoperative hypotension is poorly tolerated and may lead to hypoxia.

b. Intracerebral hemorrhage, embolism, or thrombosis

c. Seizures

DIAGNOSIS

If the patient does not wake up from anesthesia, a few rapid tests may aid in diagnosing the underlying issue. These include:

- Patients' vitals, including temperature
- Train of four (TOF) monitoring for residual neuromuscular paralysis
- Arterial blood gas (ABG) analysis with electrolytes
- Blood glucose
- Neurological examination—pupils, cranial nerves, reflexes, response to pain
- Computed tomography (CT) scan, if indicated

MANAGEMENT

Prompt, efficient assessment and treatment of delayed awakening are key to minimizing potential catastrophes. The priority should always be ABC, i.e., airway, breathing, and circulation, which should be reevaluated throughout the course of assessment. Airway obstruction, hypoxia, and hypercarbia should be excluded with the assessment of vital signs, especially the rate and character of spontaneous breathing and oxygen saturation, and physical examination. An ABG analysis aids in diagnosing any respiratory or metabolic derangements.

The patient's medical history and the history of drug intake should be reascertained in cases of delayed emergence to look for any potential associations. It is prudent that if the patient is unable to protect airway reflexes, then it is best to maintain a secure airway (keep them intubated) until fully awake. The anesthetic chart should be looked into and the perioperative management reassessed.

- Confirm that all inhalational and intravenous anesthetic agents are off.
- Check for residual muscular paralysis with TOF if patient is asleep, and reverse accordingly.
  If the patient follows commands, but is still paralysed, it is advisable to sedate the patient and ventilate till adequate recovery from neuromuscular blockade is achieved. An additional dose of reversal agent (neostigmine 2.5 mg/glycopyrrolate 0.5 mg) may be tried. Patients with suxamethonium apnea may need prolonged ventilation.
- Consider:
  - Narcotic reversal: 40 μg IV naloxone; repeat every 2 min up to 0.2 mg
  - Benzodiazepine reversal: 0.2 mg flumazenil every 1 min; max dose = 1 mg
- Measure the patient's temperature and warm if hypothermic.
- Check blood glucose and treat hypo-/hyperglycemia.
- Measure and correct dyselectrolytemia–hyponatremia correction should be performed slowly so as to avoid complications (like central pontine myelinolysis or cardiac failure).
- After all possible drug effects and metabolic causes have been ruled out, consider a thorough neurological examination of the patient to exclude any intracerebral event (ischemic or hemorrhagic stroke). A CT scan may aid diagnosis and guide further course of action.

PREVENTION

With pharmacological advancement and advent of sophisticated monitoring, the incidence of delayed emergence has decreased considerably. Careful titration of drugs to their effects, use of intermediate acting relaxants (like vecuronium and atracurium), use of a nerve stimulator to guide dosing and reversal, and careful evaluation of serum chemistries during the peri and postoperative periods, help prevent delayed awakening from anesthesia. The occurrence of an acute intracerebral event must always be kept in mind while evaluating a patient with an unexplained prolonged emergence.

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CHAPTER

3

Hydrocephalus

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OUTLINE

DEFINITION

Hydrocephalus (derived from Greek words *hydros* meaning water and *cephalus* meaning head) is defined as the excessive accumulation of cerebrospinal fluid (CSF) in the ventricular system and cisterns of the brain, leading to an increase in intracranial pressure (ICP) and its sequelae. Considering the hydrodynamics of the CSF, Rekate defined hydrocephalus as an active distension of the ventricular system of the brain resulting from

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