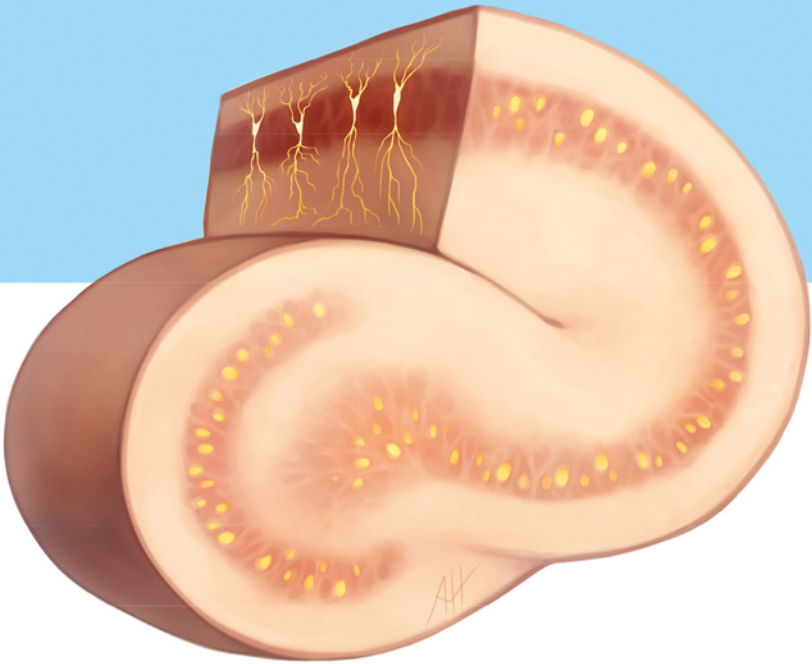
Neurosurgical Intensive Care

Second Edition

Javed Siddiqi



Thieme

Neurosurgical Intensive Care

Second Edition

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This second edition of Neurosurgical Intensive Care is dedicated to my wife, Seema, and to my children Amman and Saira, who inspire me to keep doing my best. It is also dedicated to my sisters, Shahina and Zarina, who always kept me on track, and represent the earliest and most enduring source of support in my life.

Contents

Foreword

Neurosurgical intensive care is a multispecialty and multidisciplinary field dedicated to improving the care and outcomes of patients with neurologic conditions. Over the past 20 years I have watched the field grow in terms of perceived need, knowledge, and acceptance across a growing number of medical specialties and disciplines. It is clearly evident in this text, which includes contributors from the specialties of neurology, vascular neurology, neurosurgery, interventional neuroradiology, anesthesiology, and critical care as well as the disciplines of nutrition and advanced practice nursing. This change has been driven as much by advances in medical knowledge and techniques as by the vision of its practitioners, such as the editor and contributors to this second edition of *Neurosurgical Intensive Care.*

By bringing together this breadth of expertise to update this concise and focused handbook, Javed Siddiqi has created a tool for practitioners from a wide range of specialties and disciplines who take care of critical neurological patients.

The format of this handbook lends itself to being easy to read, concise, and to the point. While it is not meant to be comprehensive, it captures the most important key points that are necessary for thoughtful clinical decision making. The tables and figures provide easy-to-use tools that facilitate rapid evaluation and decision making, both for trainees in neurocritical care as well as for experienced practitioners in related fields.

Dr. Siddiqi's *Neurosurgical Intensive Care* is a succinct and highly practical handbook for understanding the basics of patient management in the neurosurgical intensive care unit setting. Whether the reader is a nurse, medical student, resident, fellow or an attending physician, it is the best quick reference for managing critically ill neurosurgical patients.

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Preface

With the obvious exception of primary brain trauma, cerebral ischemia appears to be the final common pathway for most brain damage, whether from stroke, vasospasm, secondary brain injury, or tumor proliferation. Interestingly, the cornu ammonis 1 (CA1) pyramidal neurons of the hippocampus appear to be the most vulnerable to global cerebral ischemia, being the first to die, whereas motor cortex neurons appear to be relatively resistant to the same ischemia.[1] The fact that CA 1 neurons are essential for cognitive ("higher") functions, such as spatial learning and memory, versus motor cortex neurons serving movement, raises interesting questions about the evolutionary priority of memory over muscle, and learning over locomotion. While evolutionary biologists theorize about the fragility of higher brain functions over gross motor ability, the neurointensivist understands very clearly what is at stake—the patient who is physically intact, but cannot formulate new memories, concentrate, or analyze complex situations is also devastated in a way not always clinically self-evident. In short, if we can rescue CA1 neurons from the shadow of death, perhaps we stand a chance to preserve all other brain functions.[2]

The critical care of neurosurgical patients has evolved also over the recent two decades from an emphasis on pulmonary care (ventilation and oxygenation) to a more nuanced understanding of cerebral protection measures necessary to manage a disrupted blood–brain barrier. The advances in neuromonitoring methods are leading the way toward a more directed and individualized care plan for the neurocritical care patient. For example, the use of intracranial pressure monitors, external ventricular devices, brain tissue oxygen monitoring devices, and cerebral microdialysis are opening up a new frontier for tailoring interventions to the uniqueness of each patient's condition; the increasing use of continuous electroencephalography in the intensive care unit (ICU) has also rendered the invisible, silent status epilepticus, visible. Now, the traditional ICU management of fluid and electrolyte correction, as well as ventilator manipulation, has become dramatically more complex.

For the neurosurgeon involved in neurocritical care, decompression is still the mainstay of surgical intervention. For example, we still evacuate traumatic hematomas, perform hemicraniectomies for malignant edema from ischemic stroke, and resect malignant brain tumors causing mass effect; however, decompression is often insufficient as a stand-alone measure, and a large proportion of neurocritical care patients never go to the operating room. Whether our patients need open surgery or not, a nuanced understanding of neurophysiology is the best approach to neuron rescue. An evolution away from a mechanistic approach has rendered neurocritical care a multidisciplinary effort.

In this age of cerebral monitoring, after the ABCs of resuscitation, the key principles to neurocritical care follow age-old maxims of sound clinical practice: prompt diagnosis; compassionate communication with the patient and family; frequent neurologic exams to guide care; high-quality nursing; multidisciplinary collaboration; titration of therapies to avoid over- or undershooting desired goals; prudent use of blood work and neuroimaging to determine etiology of any neurological fluctuation or decline; and aggressive and early use of surgical intervention when necessary.

Finally, there is an ongoing debate among intensivists whether, when compared to general medical intensive care units, dedicated neurocritical care units improve outcomes for typical neurologic and neurosurgical patients with head injury, hydrocephalus, ischemic or

hemorrhagic stroke, status epilepticus, intracranial hypertension, and the like. Although the answer to this question may be intuitive to neurointensivists, the evidence for this conclusion is becoming increasingly clear if we consider individual categories of diseases or conditions treated in a typical neurocritical care unit. For example, in their study of outcome after intracerebral hemorrhage, Diringer and Edwards reported that treatment in dedicated neurocritical care units was associated with a 3.4-fold reduction in hospital mortality rate compared with management of similar patients in general ICUs.[3] Other authors have shown similar findings for traumatic brain injury.[4,5] Perhaps the best example of the advantage of dedicated neurocritical care units comes from the management of ischemic stroke in the United States, where the highest level of national stroke accreditation for any hospital, "comprehensive stroke center," is not permitted without a dedicated neurocritical care unit. Certainly, the neurocritical care unit is the hub of stroke care in any comprehensive stroke center, involving real-time collaboration between stroke neurologists, neurointerventionalists, neurosurgeons, and neurointensivists.

Every credible trauma or stroke center should aspire to establishing a high-caliber neurocritical care unit, which is the ideal place for protocol-driven treatment of neurologic and neurosurgical diseases and conditions that may otherwise suffer from "ad hocery"; they are also uniquely equipped with specialty trained neurospecialists who can work and learn together. Another clear value of dedicated neurocritical care units is their ability to advance the frontiers of clinical neuroscience research and training in a way not hitherto possible in other venues. The presence of dedicated neurocritical care units attracts talented neurosurgeons and neurologists interested in advancing the frontiers of this expanding specialty. Luckily for the neurologic and neurosurgical patients who are their greatest beneficiaries, neurocritical care units are here to stay.

Javed Siddiqi, MD, DPhil (Oxon)

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1 Bedside Neurologic Exam

Robert Dahlin, Dan E. Miulli, and Javed Siddiqi

Abstract
The bedside neurologic exam is the most sensitive test to determine the condition of the patient in the neurosurgical intensive care unit. It should be conducted frequently and consistently, whether the person is in a coma or not. Each part of the neurologic exam, from higher mentation to the cranial nerve exam and motor, sensory, and reflex exam, pinpoints the astute clinician to a specific pathology and anatomy.

Keywords: aphasia, coma, cortical exam, cranial nerve exam, eye exam, Glasgow Coma Scale, motor testing, verbal testing

Case Presentation

A 46-year-old woman presented to the emergency room with a spontaneous unilateral third nerve palsy manifested as a large, nonreactive pupil and minimal or no eye movement abnormalities. The patient had had a severe, and uncharacteristic, headache 2 days earlier, and she came to the emergency room only because of blurred vision. Other than the eye finding, the patient was awake and alert and in no acute distress, with a normal neurologic exam.
See end of chapter for Case Management.

1.1 Introduction

Patients admitted to the neurosurgical intensive care unit (NICU) are among the most critically ill and unstable. Many are admitted for traumatic brain injury, aneurysmal subarachnoid hemorrhage, spinal cord injury, postoperative craniotomies, stroke, and much more. With the advent of improved laboratory data and advanced imaging techniques, the physical examination has become less emphasized in training. While imaging and laboratory data augment our clinical decision making, the decision to order these tests and their interpretation should be influenced by the patient's physical exam. A detailed physical examination, with attention to all of the subtleties, is necessary to guide the treatment of a patient and the decision to order tests. Knowledge of the physical examination and its terminology also allows for more effective communication among health care workers whose clinical decision making will rely on the information passed down to them.

1.2 The Power of Observation

In the NICU, as elsewhere, the art of medicine should never be underestimated. While in many NICU patients, the neurologic exam is rendered more difficult by sedation, intubation, and paralytics, leaving the neurosurgeon and his or her team to rely on invasive monitoring data, serial neuroimaging, and intermittently withholding sedation to assess the patient, observation is still an important component of the patient's examination. For example, the obtunded or comatose patient breathing rhythmically in a specific pattern offers important lesion-localizing clues to the astute neurosurgeon (▶ Table 1.1). Observation of asymmetric spontaneous movements of the extremities, or change in their frequency, can be another clue to evolving brain or spinal cord lesions.

In the smaller number of awake patients in the NICU, the neurosurgeon has greater leeway to observe and converse with the patient. In these patients, observing whether they show any subtle localizing signs may betray early and enlarging focal lesions in the brain. For example, the patient who complains of

Table 1.1 Breathing patterns in brain injury

Breathing with rostral to caudal progression of lesions	Pattern	Location of lesion
Cheyne–Stokes	Periodic crescendo–decrescendo amplitude longer than variable pause, then repeat, yielding respiratory alkalosis	Generalized cerebral forebrain or midbrain lesion, metabolic encephalopathy without brainstem injury; impending herniation, congestive heart failure
Reflex hyperventilation	Hyperventilation with hypocapnia	Pons tegmentum, midbrain, reticular formation; psychiatric, metabolic acidosis, pulmonary congestion, hepatic encephalopathy
Apneustic	Irregular full inspiration then irregular pause	Pons, dorsal medulla, metabolic coma, transtentorial herniation
Cluster	Rapid irregular then pause	Pons, upper medulla, posterior fossa lesion; greatly increased intracranial pressure
Ataxic	No pattern	Medulla, acute posterior fossa lesion
No autonomic respiration (Ondine's curse)	Loss of autonomic respirations—awake normal breathing, no breathing during sleep or distraction	Reticular nucleus of medulla (respiratory center)
Apnea	No breathing	Bilateral damage to caudal medulla reticular nucleus
Kussmaul	Deep regular inspiration	Metabolic acidosis

a focal headache and who holds his hand over his head in the same area repeatedly, and on request is able to point to a specific area of his head as the site of most discomfort, may be helping the neurosurgeon with localization of an existent or developing lesion (tumor, hematoma, abscess, edema, etc.). This ability of the awake patient to localize the lesion for the neurosurgeon by effectively putting a finger on where it hurts the most, or showing the "Siddiqi sign," constitutes an observation and communication component of the neurologic exam with significant interobserver reliability (in patients without the confounding variables of recent soft tissue bruising or incision on the head). The value of focality of the Siddiqi sign is lost when the patient complains of a global headache, or pain "all over." A conscious attempt should be made in the NICU to not dismiss the initial observation of the patient in favor of exhaustive analyses of numerous data sets generated by an ever-increasing number of invasive monitoring techniques.

1.3 Coma

1.3.1 Glasgow Coma Scale

First published in 1974 by Graham Teasdale and Bryan Jennett, the Glasgow Coma Scale (GCS) has become a universally used tool for measuring a patient's overall state of alertness. Its score often guides medical decision making in neurointensive care. Despite its seeming simplicity there exists significant interrater variability. In an attempt to decrease this variability, a discussion will follow detailing the subtleties of this scale.

The GCS is calculated by adding up the points from each category, with motor receiving 6 points, verbal receiving 5, and eyes receiving 4. During an examination, the best scores for all three categories will be added together (▶ Table 1.2, ▶ Table 1.3). Patients are considered to be in a coma with a GCS of 8 or less.

Table 1.2 Glasgow Coma Scale (age 4 or more)

Points	Motor best response	Verbal/speech best response	Eyes best response
6	Obeys commands	–	–
5	Localizes to pain	Oriented conversation	–
4	Withdraws from pain	Confused conversation	Opens spontaneous
3	Decorticate—flexes abnormally	Inappropriate words	Opens to name or verbal stimuli
2	Decerebrate—rigid extension	Incomprehensible sounds	Opens to pressure/pain
1	No movement	No speech response	No eye opening
3–15	Add the totals of best responses from each column		

Table 1.3 Children's Coma Scale (age less than 4)

Points	Motor best response	Verbal/speech best response	Eyes best response
6	Obeys commands	–	–
5	Localizes to pain	Oriented, smiles, follows objects	–
4	Withdraws from pain	Confused but consolable	Opens spontaneous
3	Decorticate—flexes abnormally	Inappropriate, moaning	Opens to name or verbal stimuli
2	Decerebrate—rigid extension	Incomprehensible sounds, inconsolable	Opens to pressure/pain
1	No movement	No speech response	No eye opening
3–15	Add the totals of best responses from each column		

1.3.2 Motor Score

The motor score is measured out of a total of 6 points. A score of 6 points is given when a patient follows commands. Standard commands include asking patients to give a thumbs up, show two fingers, stick out their tongue, or wiggle their toes. Caution should be given when asking a patient to squeeze the examiner's hand during this part of the exam because this can often represent a frontal release sign in patients with lesions of the frontal lobe and may not indicate true command following.[1] Because of this, it is not recommended to use hand squeeze as a command. A score of 5 is given for localization. This can be interpreted as any purposeful movement performed by the patient, such as a limb crossing the midline to reach for painful stimuli or an endotracheal tube, to scratch an itch, or to fix the blanket. A score of 4 is given for withdrawal from painful stimuli. This can be given for a patient who tries to move away from a painful stimulus or who grimaces with pain. It is important not to confuse withdrawal with a spinal reflex. Withdrawal is held, whereas spinal reflexes return to a normal position while the stimulus is still being applied. The best location to perform a painful stimulus to determine withdrawal versus reflex is on the inner aspect of the upper arm. In withdrawal, the patient will move the arm away from the torso, or abduct the arm. In a reflex response, the patient will bring the arm closer to the torso, or adduct the arm. A score of 3 is given for abnormal flexion in response to painful stimuli. This is called the decorticate response and can include flexion at the biceps, wrist, or thighs or dorsiflexion without localizing to the stimuli. Decorticate posturing localizes the lesion in the brain to be above the red nucleus. Special attention should be given to the triple flexion response, which is often misinterpreted. It occurs in response to painful stimuli of the toes when a patient will dorsiflex the foot, flex at the knee, and flex at the thigh and is a form of decorticate posturing. A

score of 2 is given for extensor posturing, also known as decerebrate posturing. In decerebrate posturing, there is disruption between the superior colliculi or the decussation of the rubrospinal pathway and the rostral portion of the vestibular nuclei. Decerebrate posturing consists of extension of the upper or lower extremities in response to painful stimuli. A score of 1 is given for no motor response to stimulation.

1.3.3 Verbal Score

The verbal score is measured with a total of 5 points. A score of 5 is given to patients who are oriented to name, where they are at, the date or year, and the reason they are in the hospital. Inability to answer these questions reliably constitutes a disoriented patient. A score of 4 is given when a patient is unable to answer all questions regarding orientation. However, the patient must be able to attempt to answer questions with a response that is appropriate to the question being asked. A score of 3 is given when a patient's response to a question is inappropriate to what was asked. A score of 2 is given when a patient's verbal responses are inaudible. This is constituted by mumbling, grunts, or other produced sounds. A score of 1 is given when a patient is nonverbal despite stimulation or questioning. All intubated patients receive a score of 1. However, to signify that their poor GCS score is a reflection of intubation and not neurologic injury a *T* will be placed at the end of the score to signify the patient cannot receive those points.

1.3.4 Eye Score

The eye score is measured with a total of 4 points. A score of 4 is given to patients who can open their eyes spontaneously or who, after being awakened, continue to keep their eyes open. It is important that patient not be given a score of 4 if the patient's eyes are stuck open. A score of 3 is given to patients who are able to open their eyes to voice or their name. A score of 2 is given to patients who are able to open their eyes only when stimulated with pain. A score of 1 is given to patients who do not open their eyes despite the level of stimulation given.

1.4 Cranial Nerves

The neurosurgical patient's cranial nerves may be examined quickly at bedside. Cranial nerve examination is important in identifying and localizing lesions. Multiple pathologies result in cranial neuropathies, including stroke, Chiari malformations with or without syringobulbia, fungal meningitis, posterior

fossa surgery, cerebellopontine angle surgery, microvascular decompression of the trigeminal nerve, glomus jugulare tumors, and leptomeningeal carcinomatosis.

1.4.1 Cranial Nerve I: Olfactory Nerve

Olfaction is tested by supplying the patient with different odors and asking the patient to identify them. Each nostril should be tested individually. Allergic rhinitis is the most common cause of a loss or a decrease in smelling capacity. The most common neurologic cause results from significant head trauma that causes shearing of the olfactory bulb/fibers off the cribriform plate. Other causes include congenital diseases, such as Kallman syndrome, or tumors causing local compression.

1.4.2 Cranial Nerve II: Optic Nerve

Pupillary Light Reflex

The pupillary light reflex is perhaps the single most important neurologic reflex and the quickest way to get a neurosurgeon's attention. The reflex arc involved in the pupillary light reflex starts as light enters the retina and is transmitted along the optic nerve and synapses in the pretectal nucleus. Fibers from the pretectal nucleus then travel bilaterally to each Edinger–Westphal nucleus. From there, preganglionic parasympathetic fibers arise and travel with the oculomotor nerve and synapse in the ciliary ganglion and then travel in the short ciliary nerve to the sphincter muscle of the iris, leading to constriction of the pupil.

Afferent Pupillary Defect: Marcus Gunn Pupil

Afferent pupillary defect is caused by damage to the optic nerve. It can be identified by using the swinging light test. When light is directed toward the functional optic nerve the contralateral pupil will constrict normally. However, when the light is switched to the affected optic nerve, the pupil will dilate due to the pretectal nucleus receiving less light input from the damaged optic nerve.

Anisocoria

Differing pupillary diameters is termed anisocoria and is defined as a difference in pupillary size of at least 0.4 mm.[2] Anisocoria is present in 20% of the population and is generally not pathological until the difference is greater than 1 mm.[3]

Papilledema

Papilledema can be best viewed by funduscopic exam with pupillary dilation and is graded from 0 to 5 using the Frisen scale. Papilledema is characterized by haloing around the optic disc, elevation of the borders of the optic disc, and, at more severe stages, obscuration of the vessels at the optic disc. In studies it has been found to have a sensitivity of 100% and specificity of 98% for elevated intracranial pressure. However, this finding is age dependent, and in patients younger than 8 years old it indicates increased intracranial pressure in only 22% of patients.[4]

Visual Fields

Checking the visual fields is part and parcel of the cranial nerve II examination (see the discussion later in this chapter on this topic).

1.4.3 Cranial Nerve III: Oculomotor Nerve

The oculomotor nerve originates in the midbrain just anterior to the periaqueductal gray matter. The oculomotor nerve is responsible for innervation of the levator palpebrae superioris, medial rectus, inferior rectus, superior rectus, inferior oblique, and iris sphincter muscles. Loss of function of the oculomotor nerve can lead to pupillary dilation and an eye that deviates downward and laterally.

Pupil-Sparing Third Nerve Palsy

The nerve fibers that innervate the extraocular muscles travel on the periphery of the nerve and are subject to damage by microvascular pathology, such as hypertension, diabetes, or dyslipidemia. These palsies are typically incomplete and temporary, usually resolving within 3 months.[5]

Non-Pupil-Sparing Third Nerve Palsy

Multiple etiologies exist that result in unilateral dilation of a pupil, ranging from benign to life threatening and requiring emergent intervention. Although it is more typical of a neurocritical care patient to have malignant underlying pathologies, knowledge of the other causes is important to keep in mind when composing a differential diagnosis.

Mass Lesion

A mass lesion that results in uncal herniation and compression of the oculomotor nerve will lead first to a dilated pupil, followed by a nonreactive pupil.

Causes include intracerebral hemorrhage, cytotoxic edema from a stroke, subdural or epidural hematomas, and tumor. A special circumstance involving unilateral pupil dilation includes a posterior communicating artery aneurysm exerting local pressure on the oculomotor nerve.

Traumatic Mydriasis

Traumatic mydriasis arises from traumatic injury to the globe and results from either tearing of the iris sphincter muscle fibers or its nerve fibers innervating it.

Horner's Syndrome

Horner's syndrome consists of unilateral miosis, ptosis, enophthalmos, and anhidrosis. This is caused by a disruption at any point in the sympathetic innervation to the eye. Ptosis and enophthalmos are due to paralysis of Müller's muscles of the tarsal plates. Anhidrosis is due to sympathetic chain disruption in the carotid sheath.[6] Etiologies are numerous, but include Pancoast's tumor, lower cervical cord lesion, carotid injury/dissection, posterior inferior cerebellar artery occlusion (as a part of Wallenberg's syndrome), syringobulbia, and others.[7]

Adie's Pupil

Adie's pupil presents as a dilated pupil that is slow to react with light, but with almost normal response to accommodation. Adie's tonic pupil is thought to be caused by either a viral or a bacterial infection that leads to damage of the ciliary ganglion. Because patients with an Adie's pupil have damage only to the ciliary ganglion, the pupil will respond to parasympathomimetics, such as pilocarpine.

1.4.4 Cranial Nerve IV: The Trochlear Nerve

The trochlear nerve (CN IV) supplies the superior oblique muscle of the eyeball. It completely decussates in the superior medullary velum at the level of the inferior colliculus before exiting the brainstem posteriorly. Patients with trochlear nerve palsy will complain of double vision with downward gaze. On exam, the patient's pathological eye will be slightly more superior on downward medial gaze. Asking the patient to tilt the head to the contralateral side will improve the diplopia, whereas tilting the head to the ipsilateral side will worsen the diplopia.[8]

1.4.5 Cranial Nerve V: Trigeminal Nerve

The trigeminal nerve (CN V) is the largest nerve and exits the midlateral pons to supply sensation to the face and dura of the anterior and middle fossae (portio major nervi trigemini). Motor function is subserved by the motor root (portio minor nervi trigemini) supplying the muscles of mastication, tensor veli palatini, tensor tympani, and anterior belly of the digastric and mylohyoid muscles.

Corneal Reflex

The corneal reflex is elicited by stimulation of the cornea or eyelid with reflexive blinking of the eye. The afferent limb is conducted by the trigeminal nerve to the spinal trigeminal nucleus. The efferent limb is conducted by the facial nerve to elicit blinking. The V1 distribution of the trigeminal nerve can be tested by brushing either the upper eyelid or the sclera. The V2 distribution can be tested by brushing the lower eyelid.

Sensation

To test sensation each distribution of the trigeminal nerve should be checked for intact light touch, pinprick, and temperature. Each side should be compared to the patient's contralateral side because this can elucidate subtle deficiencies in sensation.

Motor

Motor can be tested by touching the cheeks and asking the patient to bite down and the examiner feels for the strength of the muscles of mastication.

1.4.6 Cranial Nerve VI: Abducens Nerve

The abducens nerve (CN VI) supplies the lateral rectus muscle of the eye. Malfunction of this cranial nerve can lead to double vision for the patient, with the affected eye being incapable of moving laterally past midline, resulting in the two eyes becoming dysconjugate.

1.4.7 Cranial Nerve VII: Facial Nerve

The facial nerve (CN VII) provides innervation to the facial muscles, platysma, and taste sensation to the anterior tongue. Motor function of CN VII to the face is tested by testing the platysma, smiling, pursing the lips, closing the eyes, and raising the eyebrows. Distinction of whether unilateral facial weakness is

forehead sparing is pertinent to localization of the lesion. The cortex supplies innervation to the frontalis muscle bilaterally, and a stroke or mass lesion of the primary motor cortex will lead to a forehead-sparing paralysis on the contralateral side. Destruction of the facial nucleus or nerve will lead to complete paralysis of the ipsilateral side. Sensation of the facial nerve is assessed by taste to the anterior two-thirds of the tongue.

1.4.8 Cranial Nerve VIII: Vestibulocochlear Nerve

The vestibulocochlear nerve (CN VIII) provides sensation for sound and balance. Testing these nerves in an awake patient requires detailed assessment of each testable division of each nerve.

Oculocephalic Reflex: Doll's Eye Reflex

The oculocephalic reflex is important for the stabilization of images on the retina as the body and head move through space. As the semicircular canals change in orientation, signals are sent to the vestibular nuclei and through the medial longitudinal fasciculus to stabilize the eyes. Movement of the head in a patient with a functional reflex will cause the eyes to move to the contralateral side. Absence of this will result in no reflexive movement of the eyes. The absence of movement of the eyes is referred to as the doll's eye reflex because, at the time when the reflex was discovered, the eyes of dolls were painted on and would hence not move with movement of the head.

Vestibulo-ocular Reflex

The vestibulo-ocular reflex is tested by instilling approximately 30 to 100 mL of ice water into the external auditory canal (with an intact tympanic membrane) with the head of the bed at approximately 30 degrees. Patients with intact function will have a slow, tonic gaze toward the side of stimulation. Those without this reflex continue to stare ahead. This test evokes severe nausea and vomiting in an awake patient. This is most often performed on deeply comatose patients or those with suspected brain death.

1.4.9 Cranial Nerve IX: Glossopharyngeal Nerve

Gag Reflex

Glossopharyngeal (CN IX) and vagus (CN X) nerve function can be assessed with the gag reflex. This may be performed in an awake patient by stimulating the posterior pharynx with a tongue blade. The afferent sensory limb of the

reflex stems from the glossopharyngeal nerve, and the motor limb from the vagus nerve.

1.4.10 Cranial Nerve X: Vagus Nerve

Cough Reflex

This reflex involves deep bronchial suctioning of an intubated patient. Sensory nerves within the bronchi respond to mechanical stimulation, sending afferents along the superior laryngeal nerve to the medulla. The efferents travel back along the vagus nerve to initiate a cough.

1.4.11 Cranial Nerve XI: Accessory Nerve

The accessory nerve is responsible for innervation of the sternocleidomastoid and trapezius muscles. The sternocleidomastoid can be tested by having patients rotate their head against the examiner's hand. The trapezius can be tested by having patients shrug their shoulders against resistance.

1.4.12 Cranial Nerve XII: Hypoglossal Nerve

The hypoglossal nerve innervates the muscles of the tongue and can be tested by having a patient stick the tongue out. Injury to this nerve can be visualized when a patient's tongue deviates to the ipsilateral side of injury. Caution should be exerted in a patient with facial paralysis because it can give the illusion of tongue deviation. To avoid this, the physician can ask the patient to touch the tongue to the nose.

1.5 Cortical Examination

1.5.1 Broca's Aphasia

Broca's aphasia results from damage to the dominant hemisphere's pars opercularis and pars triangularis. It is an expressive aphasia in which patients can comprehend what is said and they know what they would like to say in return, but they are unable to form the words. Patients are often able to use simple words, such as *yes* or *um*, or their name. Patients are often visibly frustrated. Patients with this aphasia are said to not have fluent speech.

1.5.2 Wernicke's Aphasia

Wernicke's aphasia results from damage to the dominant hemisphere's superior frontal gyrus. In this aphasia, patients are unable to comprehend what is

heard and are unable to respond appropriately. They are unaware of their deficit and produce speech that either is inappropriate to the question or is composed of nonsensical words. This is considered an aphasia with fluent speech.

1.5.3 Conduction Aphasia

Conduction aphasia results from damage to the arcuate fasciculus, which is involved in direct transfer of information from the Wernicke's to the Broca's area. Patients with conduction aphasia are able to comprehend speech and string together novel sentences but are unable to repeat a phrase given to them. A way to test this would be to ask a patient to repeat "no ifs, ands, or buts."

1.5.4 Gaze Deviation

Prévost's Sign

Also known as Vulpian's sign, this refers to the acute and transient gaze palsy in a frontal lesion (e.g., infarct), which is toward the side of the lesion and away from the concurrent hemiparesis. The eyes can be brought to the other side with the oculocephalic maneuver or caloric testing. In contrast, thalamic and basal ganglia hemorrhages produce forced deviation of the eyes to the side contralateral to the lesion (wrong-way eyes).

Setting Sun Sign

The setting sun sign is defined as tonic downward deviation of the eyes and may include downbeating nystagmus. This results from midbrain compression of the interstitial nucleus of Cajal. It may exist with a constellation of other symptoms in Perinaud's syndrome. Perinaud's syndrome results from significant midbrain compression and presents with the setting sun sign, loss of pupillary light reflex, loss of convergence, and upper eyelid retraction.

1.5.5 Visual Fields

In order to understand deficits to the visual fields and how to localize the lesion within the brain, knowledge of the anatomy is crucial. The retina can be divided into the nasal hemiretina and the temporal hemiretina. Once light impacts the retina, impulses travel down the optic nerve where nerve fibers from the nasal hemiretina decussate at the optic chiasm to become the optic tract. Nerves of the optic tract synapse at the lateral geniculate ganglion. Nerve impulses then travel via the optic radiations to the primary visual cortex. Nerve

fibers pertaining to the contralateral superior quadrant travel to the visual cortex via Meyer's loop, which travels in the temporal lobe.

The visual fields can be tested by either kinetic or static perimetry at an ophthalmologist's office or at the bedside by a confrontational field exam. A bedside confrontational examination is difficult to perform and must be carried out in a specific way, and in experienced hands it is capable of detecting only approximately 40% of lesions.[9] First a patient must cover one eye, then the physician will place a number of fingers in the right and left superior fields. A patient can then either state the number of fingers seen or note which ones appear blurrier or dimmer. The physician then can move to the inferior visual fields for testing. This is repeated for the contralateral eye and then without either eye being covered.

Monocular Blindness

Monocular blindness occurs from damage to the visual pathway anterior to the optic chiasm, which includes damage to the retina or the optic nerve. This typically occurs as a result of an embolic phenomenon from atherosclerotic disease. Temporary loss of vision as a result of embolic disease is termed amaurosis fugax.

Bitemporal Hemianopsia

Bitemporal hemianopsia results from damage to the central portion of the optic chiasm. This leads to bilateral loss of the temporal visual fields. The classic cause of this is a sellar mass, typically a pituitary adenoma.

Homonymous Hemianopsia

Homonymous hemianopsia results from damage to the visual pathway posterior to the optic chiasm. A complete lesion to the pathway leads to complete loss of vision of the contralateral visual fields. Typical causes include hemorrhagic stroke, ischemic stroke, or a mass lesion.

1.5.6 Hemineglect

Tactile Extinction

Tactile extinction is tested by applying a tactile stimulation bilaterally at the same time and asking the patient which side the stimulus occurred on. This can detect even subtle neglect at times. This can localize the lesion to the contralateral hemisphere.

Visual Extinction

Visual extinction is tested by asking a patient to add up the number of fingers simultaneously displayed bilaterally. The number of fingers on the side of neglect will often not be counted or recognized as present. This can localize the lesion to the contralateral hemisphere.

Hippus

This exam finding is characterized by irregular rhythmic dilation and contraction of the pupillary sphincter muscles. Hippus is often a normal phenomenon and may be seen in recovery from oculomotor nerve injury. It has been suggested by studies as an indication of underlying nonconvulsive status epilepticus.[10,11,12]

1.6 Cerebellar Examination

1.6.1 Dysdiadochokinesia

Dysdiadochokinesia represents a patient's difficulty with performing rapid alternating movements. This can be assessed by either rapid pronation/supination of the arms or hands, or by having patients tap their foot on the floor as fast as possible. This can localize the lesion to the ipsilateral cerebellar hemisphere.

1.6.2 Dysmetria

Dysmetria is also referred to as past-pointing sign. Patients are asked to touch their index finger to the examiner's finger, which is placed at the far end of the patient's reach. Patients with dysmetria will reach for a point that exists past the examiner's finger. This can localize the lesion to the ipsilateral cerebellar hemisphere.

1.6.3 Heel to Shin

This maneuver is performed by asking a patient to run the heel up and down the contralateral shin. Jerky performance or poor coordination can localize the lesion to the ipsilateral cerebellar hemisphere.

1.7 Spinal Cord Examination

Patients presenting to the emergency room or trauma bay with a spinal cord injury require special consideration in their initial management. This initial

management and the potential need for immediate versus delayed surgical management will depend on the neurological findings on presentation and subsequent examinations during the patient's hospital stay.

The first indication that a patient may have a spinal cord injury occurs during the primary survey with the assessment of the ABCs. A patient presenting with hypotension and bradycardia may be the first indication of spinal cord injury and is suggestive of the patient being in either spinal shock or neurogenic shock.

Spinal shock must be differentiated from neurogenic shock during the initial evaluation to guide further management. Neurogenic shock results from a disruption of the autonomic pathways of the spinal cord above the level of T6[13] and can last from 24 hours to 6 weeks. Neurogenic shock presents as a distributive shock with warm extremities, hypotension, and often bradycardia. Spinal shock is a complete loss of all spinal cord functions, reflexes, and autonomic support. Therefore all patients with spinal shock are also in neurogenic shock. The end of spinal shock is heralded by the return of spinal cord reflexes, with the deep plantar response typically being the first to return, followed by the bulbocavernosus reflex, the cremasteric reflex, the ankle jerk, Babinski's reflex, and finally the knee jerk.[14]

The degree of injury is classified using the American Spinal Injury Association (ASIA) grading system. Ten muscle groups (the deltoid or biceps, wrist extensors, triceps, flexor digitorum profundus, hand intrinsics, iliopsoas, quadriceps, tibialis anterior, extensor hallucis longus, and gastrocnemius) are tested individually and graded on a scale of 0 to 5, and a detailed sensory exam with a pin must be conducted to define the sensory level of the patient.

Injuries that lead to spinal cord injury typically require significant forces and high mechanisms of injury. As such it is not uncommon to have patients with concurrent brain injury who may be unresponsive or unable to participate in an examination. In such patients, as long as they are not in spinal shock, localization of the level of injury by physical exam will rely heavily on the spinal reflex portion of the exam because this will be unaffected by level of consciousness.

1.7.1 Strength Exam

Muscle strength is graded using the Royal Medical Research Council of Great Britain Scale. It is scored on a range from 0 to 5 (▶ Table 1.4), and muscle groups that are unable to be assessed secondary to casting or other immobilization must be recorded as not testable.

A knowledge of which nerves innervate a muscle or at which level they leave the spinal canal is also necessary for localization of a lesion and can be seen in ▶ Table 1.5.

Table 1.4 Muscle strength grading

The most caudal segment of the spinal cord with *normal* sensory and motor function on both sides of the body denotes the intact level.	
Motor level is the caudal key muscle with at least grade 3 provided the key muscles above that level are judged to be normal	
0	Total paralysis
1	Palpable or visible contraction
2	Active movement, gravity eliminated
3	Active movement against gravity
4– 4 4 +	Active movement against less resistance Active movement against some resistance Active movement against more resistance
5	Active movement against full resistance
NT	Not testable

Table 1.5 Muscle innervation

Dermatome	Nerve	Action	Muscle	Reflex
XI	Spinal accessory	Shoulder shrug	Trapezius	
C2–C4		Neck flexion	Sternocleidomastoid	
C3–C4	Spinal accessory	Fixes scapula	Trapezius	
C3–C5	Phrenic	Inspiration, tV, FEV1	Diaphragm	
C4–C5	Dorsal scapular	Hand behind back and palm resistance	Rhomboids	
C5	Suprascapular	Lateral rotate arm at shoulder	Infraspinatus	
C5	Suprascapula	Arm abduction 0–15 degrees	Supraspinatus	
C5	Axillary	Arm abduction > 90 degrees	Deltoid	
C5	Musculocutaneous	Flex supinated elbow	Biceps, brachialis	Biceps C5
C5–C6	Subscapular	Medial rotate arm at shoulder	Subscapularis	
C6–C7	Posterior interosseus	Supination	Supinator	
C5–C7	Long thoracic	Push at wall, scapula and back	Serratus anterior	

Table 1.5 continued

Dermatome	Nerve	Action	Muscle	Reflex
C6	Radial	Flex 1/2 supinated elbow	Brachioradialis	Brachioradialis C6
C6	Radial	Wrist extension 2–3	Extensor carpi radialis brevis/longus	
C5–T1	Anterior thoracic	Adduct arm	Pectoralis major	Pectoral
C6–C7	Median	Pronation	Pronator teres	
C6–C7	Median	Flex palm at wrist and hold 2–3 digit	Flexor carpi radialis	
C5–C7	Subscapular	Adduct horizontal arm	Teres major	
C7	Thoracodorsal	Adduct horizontal arm cough scapula > contract	Latissimus dorsi	
C7	Radial	Extend forearm	Triceps	Triceps C7
C7	Interosseous	Thumb away 1st digit in plane of palm	Abductor pollicis longus	
C7	Posterior interosseus	Finger extension at MP joint	Extensor digitorum	
C7	Posterior interosseus	Extend thumb	Extensor pollicis	
C7–C8	Ulnar	Flex palm at wrist and hold 4–5 digit	Flexor carpi ulnaris	
C8	Median	Flex fingers at DIP, 2–3 digit	Flexor digitorum profundus	
C8	Ulnar	Flex fingers at DIP, 3–4 digit	Flexor digitorum profundus	
C8	Median	Flex fingers at MP	Flexor digitorum superficial	
T1	Median	Extend fingers at PIP	Lumbricals	
T1	Median	Thumb at little finger	Opponens pollicis	
T1	Median	Thumb away (MC) from index plane palm	Abductor pollicis brevis	

(continued)

Table 1.5 continued

Dermatome	Nerve	Action	Muscle	Reflex
T1	Median	Flex first phalanx thumb	Flexor pollicis brevis	
C8–T1	Ulnar	Thumb at palm	First interossei	
C8–T1	Anterior interosseus	Flex distal phalanx thumb	Flexor pollicis longus	
T1	Ulnar	Fingers apart	Palmar interossei	
T1	Ulnar	Fingers together	Dorsal interossei	
T1	Ulnar	Thumb at index, in plane palm	Adductor pollicis	
T1	Ulnar	Abduct and flex 5 digit	Hypothenar (abductor digiti quinti)	
T5–L2	Intercostal	Lift trunk umbilicus deviation	Rectus abdominis, I/E oblique	Abdominal cutaneous
L2	Femoral	Hip flexion	Iliopsoas	Cremasteric
L2–L3	Obturator	Adduct thigh	Adductor magnus, brevis/longus	
L3–L4	Femoral	Knee extension	Quadratus femoris	Patella L3–L4
L4	Deep peroneal	Dorsiflexion	Tibialis anterior	
L4–L5	Tibial/peroneal	Foot inversion	Tibialis anterior/ posterior	Medial hamstring
L4–L5	Superior gluteal	Abduct thigh medial rotation leg	Gluteus medius	
L4–L5	Superior gluteal	Flex thigh	Tensor fasciae latae	
L4–S3	Sciatic	Adducts thigh	Adductor magnus	Gluteal
L5	Deep peroneal	Great toe extension	Extensor hallucis longus	
L5–S2	Inferior gluteal	Hip extension	Gluteus maximus	
S1	Sciatic	Knee flexion	Hamstrings bicep/ semitendinosus, semimembranosus and biceps femoris	
L5–S1	Superficial peroneal	Foot eversion	Peroneus longus/ brevis	
L5	Deep peroneal	Toe 2–5 extension	Extensor digitorum longus/brevis	

Table 1.5 continued

Dermatome	Nerve	Action	Muscle	Reflex
S1–S2	Tibial	Plantar flex foot	Gastrocnemius/ soleus	Achilles S1–S2
S1–S2	Tibial	Plantar flex toes	Flexor hallucis longus	
S2–S4		Clamp during rectal	Bladder, bowel, sphincter	Bulbocavernosus

Abbreviations: DIP, distal interphalangeal joints; I/E, internal/external; FEV1, forced expiratory volume in 1 second; MC, metacarpal; MP, metacarpophalangeal joints; PIP, proximal interphalangeal joint; tV, tidal volume.

Table 1.6 Reflex grading scale

Score	Description
0	Absent reflex
1+	Trace, or seen only with reinforcement
2+	Normal
3+	Brisk
4+	Nonsustained clonus (i.e., repetitive vibratory movements)
5+	Sustained clonus

1.7.2 Reflex Exam

Reflexes are graded as well on a scale of 0 to 5 as described in ▶ Table 1.6. During the exam it is important to note not only abnormal reflexes but also asymmetry between reflexes because this too will help in localization of a lesion. In the setting of spinal cord injury, reflexes will be normal above the level of injury, absent or hypoactive at the level of injury, and hyperactive below the lesion. In cases of malingering, when there is a question of an acute spinal cord injury, normal and symmetrical reflexes throughout would lead to questioning the validity of no movement and no response to painful stimuli in the awake patient.

1.7.3 Sensory Exam

Pinprick

Sensory examination using pinprick is the best way to localize a lesion—better than a motor or reflex exam. The assessment of fine touch checks for integrity

of the posterior column–medial lemniscus pathway that transmits the sense of touch to the ipsilateral spinal cord's posterior column, decussates in the medulla, synapses in the thalamus, and ends in the postcentral gyrus. The sensory examination should be carried out by checking each dermatomal distribution in sequence (▶ Fig. 1.1). The subjective feeling of one dermatome should be compared to the contralateral side to detect subtle differences. Examination can be initiated with light touch but must be performed with pinprick to determine the specific dermatome.

Proprioception

The assessment of proprioception also checks for integrity of the posterior column–medial lemniscus pathway. It can be checked by movement of a patient's digits either upward or downward and asking the patient to identify the direction. Impaired proprioception is also suggested by a positive Romberg's sign.

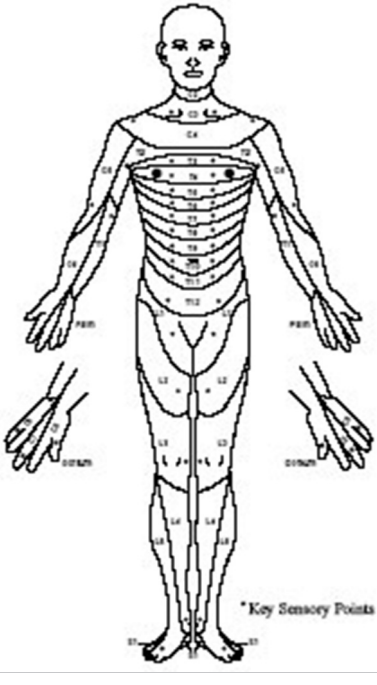

Fig. 1.1 Dermatomal and sensory nerve distribution. (Reproduced with permission from the American Spinal Injury Association.)

Temperature

The sensation of temperature first enters the spinal cord, where it immediately decussates before ascending in the anterolateral spinothalamic tract and synapses in the contralateral thalamus before traveling to the postcentral gyrus. It can be assessed by running an alcohol swap or cold utensil submerged in ice water over the dermatome of interest.

Hoffmann's Sign

This maneuver is performed by flicking the distal phalanx of the middle finger with reflexive flexion of either the index finger or the thumb. The presence of this finding is suggestive of a pyramidal tract lesion about the level of C5–C6. This sign should be interpreted with caution because it may be a normal finding in patients with hyperreflexivity, anxiety, or hyperthyroidism. Studies vary in terms of its accuracy, with sensitivity ranging from 58 to 94% and specificity ranging from 74 to 78%.[15,16,17]

Clonus Maneuver

The clonus maneuver is performed by rapid dorsiflexion of the foot with maintenance of the foot in the dorsiflexed position. The number of beats elicited from this maneuver is counted and is considered pathological if five or more beats are present. The presence of this exam finding is highly suggestive of pyramidal tract dysfunction.[18]

Babinski Maneuver

The Babinski maneuver is performed by scratching the lateral plantar surface with continuation to the transverse arch, lasting 5 to 6 seconds. Initial *extension* of the great toe with subsequent downward fanning of the other toes is the classic abnormal extensor response. The presence of this exam finding indicates pyramidal tract dysfunction. While Babinski's sign has a low sensitivity (~ 50%), it has a specificity for pyramidal tract dysfunction of 99%.[19]

Priapism

Priapism is sustained, unintended penile erection. In the context of spinal cord injury it is an ominous sign and is strongly associated with complete ASIA classification A (ASIA A) spinal cord injury. It is also associated with spinal shock. Priapism occurs immediately after injury and will not occur in a delayed fashion.

1.7.4 Rectal Exam

During the rectal examination of a patient five things should be noted in regard to spinal cord injury and can be recorded as present or absent. Pinprick sensation, active and passive rectal tone, anal wink reflex, and bulbocavernosus reflex (BCR). The BCR is a test of integrity of the S2, S3, and S4 nerve roots and involves contraction of the anal sphincter in response to squeezing of the glans penis or clitoris; it is the first reflex to return as spinal shock starts to recede, and as such is critical to determining the validity of the neurological exam after spinal cord injury. In a patient with a Foley catheter, this can be stimulated by gentle tugging on the catheter. The anal wink reflex tests the integrity of the S4, S5 nerve roots and is elicited by scratch or pinprick to the perianal region with reflexive contraction of the external sphincter ani muscle.

1.8 Malingering

Defined by the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV) as "the intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs."[20] DSM-IV also recommends suspicion of malingering for patients who present with two or more of the following: medicolegal issues, disagreement between objective and subjective stress, noncompliance with evaluation or treatment, or antisocial personality disorder. Testing reflexes will help confirm malingering. An acute spinal cord injury will not have equal and symmetric reflexes; if this occurs additional methods must be used to confirm malingering. There are many physical exam tests to help support the diagnosis; however, evidence supporting it is lacking and is class C at best.

For the Hoover test, the physician places a hand under the thigh of the "paralyzed" leg and asks the patient to flex the contralateral thigh. A patient with true paralysis will not exert a downward force with the affected leg; however, patients with a nonorganic cause of paralysis should exert a downward force. One study tested a group of normal controls, patients with true hemiparesis, and patients suspected of feigning paralysis. A strain gauge was used under the affected leg to detect the downward force. The results of the study accurately distinguished between each group. However, in the study the strain gauge results were not compared to results of a physician-performed Hoover exam.[21]

Case Management

The mere presence of a spontaneous CN III palsy, especially one involving a large pupil unilaterally, should be considered an expanding or ruptured posterior communicating artery aneurysm until proven otherwise. The transient history of severe headache in this patient is suspicious for a sentinel bleed from a posterior communicating artery aneurysm. This patient needs a workup for subarachnoid hemorrhage, and at the minimum a computed tomographic angiogram to rule out an aneurysm. Should the patient turn out to have a ruptured aneurysm, or even an enlarging nonruptured aneurysm, she should be admitted to the NICU, with planned surgical or endovascular treatment of the aneurysm, because she has a high risk of catastrophic bleed.

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2 Altered Mental Status and Coma: Pathophysiology and Management

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Abstract
Coma is not a lack of function but should be considered a derangement of function and possibly a disruption in neuroanatomy. When there is a change in the mental status usually there are lateralizing signs, but the brain's environment can be effected by seizures, metabolic changes, and other pathologies; it is the physician's responsibility to evaluate these processes.

Keywords: altered mental status, coma, consciousness, delirium, dementia, lateralizing signs, nonconvulsive status epilepticus, sundowning

Case Presentation

A 57-year-old woman was brought to the emergency room after being found on the kitchen floor in their home after the husband returned from the store. She was lethargic and confused, and this lasted about 5 hours in the emergency room. Her deficits included some slurred speech and mild left-sided weakness, but these symptoms seemed to resolve within the hour. She was in good health and took no medication other than hormone replacement for postmenopausal symptoms. The patient reported worsening early morning mild headaches over the last 2.5 years. Upon further workup, a computed tomographic (CT) scan demonstrated a 4 cm homogeneously enhancing left frontal-parietal mass with edema and 4 mm of midline shift.

See end of chapter for Case Management.

2.1 Altered Mental Status

2.1.1 Introduction

Before discussing the causes, categories, workup, or treatment of altered mental status (AMS), it is necessary to give a brief description of the term *consciousness*. Although this subject has philosophical, religious, and ethical connotations, simply stated, consciousness is the awareness of one's self and of one's environment, which includes people, places, and things. Consciousness also includes a multitude of higher mental functions, such as concept formation and the ability to manipulate these concepts. It is the physician's

responsibility to evaluate these mental processes and compare them within the context of the patient's age, medical condition, baseline level of mental functioning, and numerous other factors, including comparison to the average mental functioning of the general population. This chapter discusses the evaluation process, differential diagnosis, and initial management of these patients in the neurosurgical intensive care unit (NICU).

2.1.2 Definitions

There are three major categories of AMS that should be defined: delirium, dementia, and coma. Dementia is a progressive and persistent loss of cognitive function, where both short- and long-term memory are impaired. These are typically associated with disorders such as aphasia, apraxia, and agnosia, and impairments of personality, planning, and critical and cognitive thinking.[1] It is critical to understand that dementia is a diagnosis of exclusion when other behavioral manifestations, such as delirium and other psychiatric diseases, have been ruled out.

Delirium, by definition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM) 5th Edition*, consists of four key features[2]:

1. Disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and orientation to the environment.
2. Disturbance develops over a short period of time (usually hours to a few days) and represents an acute change from baseline that is not solely attributable to another neurocognitive disorder and tends to fluctuate in severity during the course of a day.
3. A change in an additional cognitive domain, such as memory deficit, disorientation, or language disturbance, or perceptual disturbance that is not better accounted for by a preexisting, established, or evolving other neurocognitive disorder.
4. Disturbances in numbers 1 and 3 must not occur in the context of a severely reduced level of arousal, such as coma.

The distinction between dementia and delirium is based on etiology and the time course of the disease process. Delirium is generally due to more acute, reversible processes, whereas dementia tends to be due to chronic, irreversible diseases. The following table summarizes and provides an overview of the characteristics of each (▶ Table 2.1).[3] Coma is a more severe depressed state, which will be further discussed later in this chapter.

Table 2.1 Comparison of altered states of consciousness[3]

Consciousness state	Definition	Pathophysiology	Time course	Disposition
Delirium	Acute confusional state, with impaired attention, perception, thinking, and memory	Always has an organic cause -Primary intracranial disease -Systemic disease -Exogenous toxins -Drug withdrawal	Acute	Often reversible when underlying etiology is addressed
Dementia	Implies a loss of mental capacity, short-term memory is particularly affected as well as cognitive abilities	Most are idiopathic (e.g., Alzheimer's, Parkinson's, vascular dementia)	Usually chronic and progressive	Usually not reversible, but can be treated symptomatically to a limited extent; only 10–20% have a reversible condition
Coma	Reduced state of alertness and responsiveness in which the patient cannot be aroused	Complex with many different sources (see below)	Usually acute or subacute	May be reversible if source is rapidly identified. The more time a patient spends in a coma the less favorable the prognosis

2.1.3 Approach to the Patient with Altered Mental Status

History

Because the patient most often will not be able to provide an accurate history, when evaluating the AMS patient for the first time, most of the history can be obtained from interviewing relatives or caregivers. A thorough history may reveal a recent illness, a history of drug abuse or alcoholism, depression, or a current medication list. If preceded by a trauma, mechanisms of insult and on-scene reports may help guide differential diagnoses and general approach to the examination.

Physical Exam

Within the NICU, the most common method for evaluating the patient's altered level of consciousness and confusional states is the Glasgow Coma Scale (GCS) (▶ Table 2.2). It is the most widely accepted score among physicians secondary to its high level of interexaminer and intraexaminer reliability. If the patient is uncooperative or confused, we should focus on the patient's vital signs, fluid balance, and general appearance. One can suspect hepatic failure if the patient is jaundiced, possible recreational drug abuse if needle tracks are noted, or possible seizure in a postictal state if there are signs of a bitten tongue. Any change in the GCS score of 2 points or more should be taken seriously and not dismissed as artifact.

The neurological exam in the NICU can reveal lateralizing signs of possible intracranial pathologies. Careful attention should be given to visual fields, cranial nerves, and motor deficits. It is also prudent to notice any changes from the initial presentation. Please see Chapter 1 for an overview of the neurological exam. However, it is important to note that absence of any focal findings does not exclude the possibility of focal neurologic lesions as the cause of the patient's delirium. If the patient is awake and alert with stable vital signs and no focal neurologic deficit, an assessment of mental status should be performed. The Mini Mental Status Examination (▶ Table 2.3) evaluates the patient's overall appearance, attitude, disorders of thought or perception, mood, insight, and judgment, as well as sensorium and intelligence. Points are allotted, with scores greater than 27, between 19 and 24, between 10 and 18, and less than 9 indicating normal, mild, moderate, and severe cognitive impairment, respectively (▶ Table 2.2, ▶ Table 2.3, ▶ Table 2.4).[1,5,6]

Table 2.2 Glasgow Coma Scale (3–15 points, higher points better)[4]

Score	Best motor response	Best verbal response	Best eye response
1	No response	No response	No response
2	Decerebrate posturing (extensor)	Incomprehensible sound	Open eyes to pain
3	Decorticate posturing (flexor)	Inappropriate speech	Open eyes to voice
4	Withdraws to pain	Confused (not oriented)	Opens spontaneously
5	Localizes to pain	Oriented	
6	Follows commands		

Table 2.3 Mini Mental Status Exam (0–30 points, higher points better)[4]

Total points	Test
5	What is the date, month, year, day of the week and season of the year (1 point each)
5	What is the hospital name, city, county, state, country (1 point each)
3	Name three objects after examiner, eg, apple, envelope, pen, eyeglasses (1 point each)
3	Recall the names of the three objects at 5 minutes (1 point each)
5	Serial 7's: Count back from 100 in 7's (1 point each correct answer up to 5)
2	Name a pencil and a watch
1	Repeat the phrase "No ifs, ands, or buts"
3	Three-stage command: e.g., "Take the paper in your right hand, fold it in half, and put it on the floor"
1	On a piece of paper or screen, read and obey: "Close your eyes"
1	Write a sentence
1	Copy the diagram shown to the right

2.1.4 Causes of Altered Mental Status

Careful attention must be given to the time of onset and the course of cognitive decline. It is prudent always to entertain delirium as a part of the working diagnosis and to rule out medical etiologies. The causes of altered mentation in the NICU are often different than those in the emergency room. NICU patients have head trauma, strokes, intracranial surgery, or other known sources of intracranial insult that could lead to an altered level of consciousness. Many times the neurointensivist is faced with a patient that was previously alert and aware prior to deterioration. The challenge to the neurointensivist is to identify the cause of the altered level of consciousness and institute the appropriate intervention for life-threatening conditions if need be. Acute neurologic disorders can include delayed presentations of subdural or epidural hematomas or seizures with the postictal state. Other common causes include the following:

- Drug or alcohol toxicity, including withdrawal syndromes (e.g., chronic alcoholics)
- Metabolic disorders (e.g., hypoglycemia, thyrotoxicosis)
- Infections (e.g., urinary tract infections, respiratory tract infections)
- Fluid and electrolytes (e.g., hyponatremia, hypernatremia)
- Cardiovascular issues (e.g., heart failure, acute myocardial infarction)
- Postoperative states (more common in the elderly)

Table 2.4 National Institutes of Health Stroke Scale (0–67 points)[4]

Item (score)	Item (score)	Item (score)
1a. Level of consciousness • Alert (0) • Drowsy (1) • Stuporous (2) • Coma (3)	4. Facial Paresis • Normal (0) • Flat nasolabial fold (1) • Partial paralysis (2) • Complete paralysis (3)	7. Limb ataxia • No ataxia (0) • Present in one limb (1) • Present in two limbs (2)
1b. Month and patient age • Both correct (0) • One correct (1) • None correct (2)	5a. Motor right arm • Normal (extends to 90 degrees without drift × 10 s) (0) • Drift (1) • Some effort against gravity (2) • No effort against gravity (3) • No movement (4) • Not testable (9)	8. Sensory • Normal (0) • Mild to moderate decrease (1) • Severe to total decrease (2)
1c. Open and close eyes or Squeeze and let go • Both (0) • One (1) • None (2)	5b. Motor left arm • Normal (extends to 90 degrees without drift × 10 s) (0) • Drift (1) • Some effort against gravity (2) • No effort against gravity (3) • No movement (4) • Not testable (9)	9. Best language • Normal (0) • Mild to moderate aphasia (1) • Severe aphasia (2)
2. Best horizontal eye movement • Normal (0) • Partial gaze palsy (1) • Forced deviation (2)	6a. Motor right leg • Normal (leg to 30 degrees for 5 s) (0) • Drift (1) • Some effort against gravity (2) • No effort against gravity (3) • No movement (4) • Not testable (9)	10. Dysarthria • Normal articulation (0) • Mild to moderate slurring (1) • Near complete dysarthria (2) • Intubated/not testable (9)
3. Visual field testing • Normal (0) • Partial hemianopia (1) • Complete hemianopia (2) • Bilateral hemianopia (blind) (3)	6b. Motor left leg • Normal (leg to 30 degrees for 5 s) (0) • Drift (1) • Some effort against gravity (2) • No effort against gravity (3) • No movement (4) • Not testable (9)	11. Inattention or extinction • Normal (0) • Extinction to one sensory modality (1) • Severe hemi-neglect (2)

It is beyond the scope of this book to give an exhaustive list of the possible causes of AMS. ▶ Table 2.5 presents the categories of causes and some of the most common etiologies of AMS in each category.

Table 2.5 Common causes of altered level of consciousness

Drugs and toxins: Prescription medications; nonprescription medications; drugs of abuse; withdrawal states, including delirium tremens, medication side effects, poisons (e.g., carbon monoxide, cyanide)
Infections: Sepsis, systemic infections, pneumonia, urinary tract infections, fever-related delirium
Metabolic derangements: Electrolyte disturbances (e.g. sodium, calcium, magnesium, phosphate), endocrine disturbances, hypercarbia, hyperglycemia, hypoglycemia, hyperosmolar and hypo-osmolar states, hypoxemia, Wernicke's encephalopathy, vitamin B12 deficiency, folate deficiency, niacin deficiency
Intracranial disorders: Encephalitis, meningitis, brain abscesses, epidural abscesses, epileptic seizures, nonconvulsive epilepticus, traumatic brain injury, increased intracranial pressure, hydrocephalus, hypertensive encephalopathy, psychiatric disorders
Systemic organ failure: Cardiac failure, acute myocardial infarction, hematologic disorders (e.g., thrombocytosis, hypereosinophilia, leukemic blast cell crisis, polycythemia), pulmonary disorders, pulmonary embolisms, renal failure
Physical disorders: Burns, electrocution, hyperthermia, hypothermia, polytrauma

Sundowning

This is a frequent, though poorly understood, symptom complex that generally occurs in patients with dementia or cognitive impairment, and usually manifests around sunset. Sundown syndrome refers to the emergence of neuropsychiatric symptoms, such as agitation, confusion, anxiety, and aggressiveness in the evening or at night.[7] It is thought to be associated with impaired circadian rhythmicity, and it appears to be mediated by degeneration of the suprachiasmatic nucleus of the hypothalamus and decreased production of melatonin.[7] Studies have shown that patients wakened from sleep during darkness experienced agitation, with a trend indicating the apparent worsening of agitation during the winter. This may suggest involvement of the circadian timing system.[2,8]

The diagnosis is clinical. There have been no laboratory values or imaging studies associated in the literature review. Management of sundowning includes encouraging increased activity, having the patient ambulate out of bed to the chair, exposure to light therapy during the day, and keeping a quiet and a dark environment during the night. It has been shown that bright light therapy has helped with agitated patients and restlessness in the elderly and patients with dementia.[9,10,11]

Nonconvulsive Status Epilepticus

Nonconvulsive status epilepticus (NCSE) can be underrecognized and can be a fairly common cause of altered mental status in the NICU. Once structural, metabolic, and iatrogenic causes of coma have been excluded, and NCSE workup should be the next step. Some clinical signs that may suggest NCSE include prominent bilateral facial twitching, unexplained nystagmus in obtunded patients, and unexplained automatisms, such as lip smacking, chewing, swallowing movements, acute aphasia, or neglect without a structural lesion. Continuous electroencephalographic (EEG) monitoring is necessary for the diagnosis and management of NCSE.

2.1.5 Workup for Altered Mental Status

The workup for AMS is driven mainly by the clinician's suspicion for a certain etiology. For example, a patient that has sudden onset of AMS with tonico-clonic-type movements would lead the physician toward a workup and intervention for a seizure. In a patient who has a known small epidural hematoma with a GCS score of 15 upon admission and deteriorates to a GCS score of 12, a stat CT should be ordered to further evaluate for possible hematoma expansion and the possible need for surgical intervention. If a postoperative craniotomy patient has a decreasing level of consciousness and fever, the suspicion of a postop infection should be entertained and a CT scan with contrast and a lumbar puncture will likely need to be performed. The noncontrast CT scan is used in many situations as the initial study for AMS because it gives information quickly as to whether there is a need for immediate surgical intervention. Once an anatomical source is ruled out, further testing should be ordered to find an etiology for the AMS. ▶ Table 2.6 gives common interventions for each major category of disease process.[4,12,13,14,15,16]

Table 2.6 Testing for altered level of consciousness for patients in the neurosurgical intensive care unit[16]

Category	Most common diagnostic modalities
1. Trauma	CT without contrast, ICP monitoring, brain metabolic monitoring
2. Epileptic	EEG, continuous EEG, MRI
3. Cerebrovascular	CT without contrast, carotid duplex, MRA, four-vessel angiography
4. Infectious	CT/MRI with and without contrast if warranted, blood cultures, sputum cultures, urine cultures, immunocompromised workup if warranted
5. Toxic/drug	CT (rule out anatomical disease), drug screen, toxin screen, EtOH level

Table 2.6 continued

Category	Most common diagnostic modalities
6. Metabolic	CT (rule out anatomical disease), metabolic profile (serum electrolytes, creatinine, glucose, calcium, complete blood count, urinalysis, urine cultures), liver function, Schilling test, EEG, folate, B12, thyroid function
7. Cardiopulmonary	EKG, cardiac enzymes, ABG, spiral CT, CT (rule out anatomical disease) if warranted
8. Psychiatric	CT (rule out anatomical disease), psychiatric consult, review medications and side effects

Abbreviations: ABG, arterial blood gas; CT, computed tomography; EEG, electroencephalography; EKG, electrocardiography; EtOH, ethanol; ICP, intracranial pressure; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

2.1.6 Interventions for Altered Mental Status in the Neurosurgical Critical Care Unit

Any particular intervention for AMS will be dependent upon the disease process. Processes that cause anatomical derangement should be managed in an emergent fashion. Subdural hematomas, epidural hematomas, contusions, and the like, are all disease processes that, if severe enough, will require emergent surgical intervention. A seizure will require anticonvulsant therapy, and if prolonged, in the case of status epilepticus or multiple seizures, may require emergent benzodiazepines. Cerebrovascular accidents of the ischemic type may be treated with clot-busting drugs or other interventional techniques if the time period is within certain guidelines. A large middle cerebral artery territory ischemic stroke may require a lifesaving decompressive craniectomy. Hemorrhagic strokes may require surgical intervention for evacuation of clot if the patient meets certain criteria (see Chapter 7). ▶ Table 2.7 shows some of the common interventions in each category. Many of these treatments and their specific implementation are covered in more detail in Chapters 26 and 27 of this book.

Table 2.7 Intervention for altered level of consciousness[16]

Category	Most common treatment modalities
1. Trauma	Craniotomy for evacuation of hematoma, craniectomy for ICP control, CSF drainage, osmotic diuretics
2. Epileptic	Anticonvulsants; benzodiazepines for status epilepticus, future vagal nerve stimulation, future epilepsy surgery
3. Cerebrovascular	Clipping/coiling of aneurysms, clot lyses, Merci extractor, stenting, endarterectomy, hematoma evacuation, craniectomy for ICP control in MCA strokes

(continued)

Table 2.7 continued

Category	Most common treatment modalities
4. Infectious	IV antibiotics, surgical debridement of infected hardware or abscess, sepsis management
5. Toxic/drug	Symptomatic treatment of drug and EtOH withdrawal
6. Metabolic	Lactulose, glycemic control, electrolyte correction
7. Cardiopulmonary	Anticoagulation, cardiology consult
8. Psychiatric	Psychiatric consult, antipsychotics, review of medication and side effects

Abbreviations: CSF, cerebrospinal fluid; EtOH, ethanol; ICP, intracranial pressure; IV, intravenous; MCA, middle cerebral artery.

2.2 Coma

Plum and Posner defined the normal wakeful conscious state in humans as serially time-ordered, organized, restricted, and reflective awareness of self and the environment and an experience of graded complexity and quantity.[16] Coma is a more severe form of depressed consciousness in which the person is incapable of perceiving self or environment, and the brain is not able to receive stimuli from the environment without aggressive stimulation. The patient's interaction with the environment during coma is at best reflexive.

Brain function has been found to show that cerebral oxygen had declined to 20% below normal levels in hepatic encephalopathy, lethargy, and global confusion. Other studies have shown deficiency in cholinergic function and excess release of dopamine, norepinephrine, and glutamate. The cholinergic system has been shown to provide the main input relay and the reticular nuclei of the thalamus from the upper brainstem. In fact, some studies have related coma to sleep states, where both conditions are due ultimately to the lack of activity of the ascending arousal system and where both impaired states of consciousness and non–rapid eye movement sleep are characterized by EEG patterns that include high-voltage slow waves.[17]

The GCS is the most commonly used grading system of consciousness (or coma) within the trauma setting. Coma is usually defined as a person who has a GCS score of 8 or less. These patients, at best, may open their eyes to painful stimulation and will localize to pain. Anatomically, coma can be caused by diffuse cortical dysfunction or by a dysfunction of the reticular activating system located in the brainstem (midbrain) (▶ Table 2.8).[18] Comatose patients remain motionless in an eyes-closed state without spontaneous eye movement, with total absence of patterned behavioral arousal or EEG features of the normal sleep–wake cycle. By definition, coma implies the state has endured for at least

1 hour, while eventually progressing through functional stages of the vegetative state (VS) and minimally conscious state (MCS).

The VS was first discussed by Jennett and Plum in 1972 and was associated with alternating periods of eye opening and closing in patients who do not show any signs of awareness of their environment. The most common causes of the VS are traumatic brain injury and cardiac arrest. Postmortem autopsies of patients who had been in VS show a loss of thalamic neurons near the thalamic intralaminar nuclei. Although diffuse axonal injury and hypoxic brain injury can cause severe loss of thalamic neurons, significant brainstem damage is not commonly found, emphasizing that VS is primarily a disorder of the corticothalamic system.

A VS is labeled as persistent once it lasts more than a month. It is considered permanent after 3 or 12 months, depending on the nature of the initial injury.[20] MCS is the first level of behavior recovery beyond VS, characterized by evidence of bedside responses to environmental stimuli. Such behavior can be visual tracking or fixation. Autopsies of patients who have died while in the MCS show no significant evidence of thalamic cell loss or severe diffuse axonal injury but show overall reduced cerebral cell death. Giacino et al[21] have suggested demonstration of the following behaviors in order to make the diagnosis of MCS:

1. Following simple commands.
2. Gestural or verbal yes/no responses (regardless of accuracy).
3. Intelligible verbalization.
4. Purposeful behavior, such as the following:
   a) Appropriate emotional responses to the linguistic or visual but not to neutral topics or stimuli.
   b) Vocalizations or gestures that occur in direct response to the linguistic content of questions.

Table 2.8 Glasgow Coma Scale[19]

Score	Best motor response	Best verbal response	Best eye response
1	No response	No response	No response
2	Decerebrate posturing (extensor)	Incomprehensible sound	Opens eyes to pain
3	Decorticate posturing (flexor)	Inappropriate speech	Opens eyes to voice
4	Withdraws to pain	Confused (not oriented)	Opens spontaneously
5	Localizes to pain	Oriented	
6	Follows commands		

c) Reaching for objects, demonstrating a relationship between object location and direction of reach.
d) Touching or holding objects, accommodating the size and shape of the object.
e) Pursuit eye movement or sustained fixation that occurs in direct response to moving or salient stimuli.

2.2.1 Initial Care of the Comatose Patient

There are often three types of comatose and obtunded patients a neurosurgical consultant will encounter. The first type is a patient with overwhelming structural brain injury with a poor prognosis for persistent VS. The second type is a patient who initially shows an early recovery. The third type of patient has a mix of both structural brain injuries with diffuse metabolic alterations. Most neurosurgical services do not become involved in the initial evaluation of a patient presenting to the emergency room in a coma of unknown etiology; however, it is useful to review the proper initial management of a comatose patient who has not received any laboratory or radiographic workup (▶ Table 2.9).

Table 2.9 Initial management of comatose patient[18,19]

Ensure patent airway and adequate oxygenation	Start mechanical ventilation or O_2 by mask if patient breathing on own
Protect C-spine	Immobilize C-spine with cervical collar
Maintain MAP above 100	Use fluids and vasopressors as necessary
Treat possible metabolic issue after initial blood draw	Thiamine 100 mg IV then glucose 25 g IV (d50)
Treat increased ICP if there is strong suspicion	Mannitol 0.25–1 g/kg bolus
Treat seizures	Benzodiazepine IV (lorazepam 2 mg IV)
Restore acid–base balance	Judicious use of fluids (0.9% saline preferred)
Treat any suspected drug overdose	Naloxone 0.2 mg IV and repeat; physostigmine 1 mg IV; Flumazenil 0.2 mg IV
Rule out space-occupying lesion	Stat CT scan of head
Normalize body temperature	Warm fluids and warming blankets
Treat any suspected meningitis or systemic infection	Wide spectrum antibiotics
Specific therapy ASAP	

Abbreviations: ASAP, as soon as possible; CT, computed tomography; ICP, intracranial pressure; IV, intravenous; MAP, mean arterial pressure.

2.2.2 Examination of a Comatose Patient

The exam for a comatose patient is much simpler than that for the awake patient, similar to the general format previously in this chapter. An advantage is the short length of the exam that must be performed within an emergent situation to yield critical information for patient care. The following exam items will allow specific localizations in unresponsive patients:

1. Mental status: document the level of consciousness with details of the specific responses along with the GCS. The subtle examination findings may be interpreted differently in a comatose patient when the scale is vague. In addition, this will allow repeating exams to be followed in a more detailed manner.
2. Cranial nerve exam.
   a) Ophthalmoscopic exam (CN II).
   b) Vision (CN II): blink to threat.
   c) Pupillary responses (CN II, III) (see oculomotor responses).
   d) Extraocular movements and vestibulo-ocular reflexes (CN III, IV, VI, VIII)
      1. Spontaneous ocular movements, nystagmus, dysconjugate gaze, oculocephalic maneuvers, caloric testing (brainstem reflexes).
   e) Corneal reflex, facial asymmetry, grimace response (CN V, VII).
   f) Gag reflex (CN IX, X).
3. Sensory and motor exam.
   a) Spontaneous movements.
   b) Withdrawal form painful stimuli.
4. Reflexes.
   a) Deep tendon reflexes.
   b) Plantar response.
   c) Posturing reflexes.
   d) Special reflexes suspected spinal cord lesions.

2.2.3 Causes of Coma

Causes of coma can be broken down into four main categories: structural, metabolic, electrical, and self-induced or iatrogenic. Structural lesions cause coma by physically interfering with nervous system pathways, either by trauma, compression by tumor, or increased pressure. Structural brain dysfunction is due to anatomical derangement of pathways due to physical phenomena. Metabolic causes of coma result from chemical derangements leading to improper functioning of the nervous system or some of its components (▶ Table 2.10).[18] Irrespective of specific etiology, for coma to occur there are limited final common pathways of damage necessary, including the following:

1. Diffuse impairment of both cerebral hemispheres.

Table 2.10 Structural and metabolic causes of coma[16]

Structural coma	Metabolic coma	
Hematoma	Hypoglycemia	Hypo-/hyperthermia
Trauma	Adrenal failure	Hypo-/hyperosmolality
Tumor	Liver disease	Diabetic ketoacidosis
Hydrocephalus	Renal disease	Encephalopathy
Abscess	Pulmonary disease	Drugs
	Dialysis dysequilibrium	Toxins

2. Impairment of the midline and paramedian upper brainstem and basal forebrain regions containing nuclei associated with the ascending reticular activating system.

Structural lesions can cause coma through three general mechanisms: compression of the brainstem, direct damage to the brainstem, or diffuse dysfunction of bilateral cerebral hemispheres. Damage to the brainstem can occur through a primary effect on the brainstem, such as a tumor, hemorrhage, or infarct of the brainstem, or it can be due to external pressure on the brainstem by another part of the brain that contains the pathology. This can be due to pathology that causes transtentorial herniation of the diencephalon or medial temporal lobe, or it can be due to a posterior fossa lesion causing compression of the brainstem. On the other hand, unilateral hemispheric lesions or lesions of the brainstem at the level of the midpons or below, should not cause coma.[17]

Supratentorial pathology tends to lead to one of the herniation syndromes as listed here. The herniation syndromes, especially uncal and central herniation, pass through four general stages: the diencephalic stage, the mesencephalic–pontine stage, the pontomedullary stage, and the medullary stage. The relay of the thalamic nuclei provides the largest source of input to the cerebral cortex; therefore, thalamic lesions that are sufficiently large can produce the same result as bilateral hemispheric cortical injury. For example, the "tip of the basilar" syndrome can cause bilateral thalamic infarction. The tonsillar and cingulate herniation syndromes may be progressive, but their progression is less well defined. Posterior fossa lesions may also cause supratentorial type herniation by causing an obstructive hydrocephalus, which can also lead to herniation (▶ Table 2.11, ▶ Table 2.12, ▶ Table 2.13).[17,19]

Table 2.11 Supratentorial herniation syndromes[22]

	Central herniation	Uncal herniation	Tonsillar herniation	Cingulate herniation
Lesion location	Diffuse supratentorial increase in intracranial pressure with no pressure gradient from right to left	Usually unilateral lesions, especially those located in the temporal lobes	Posterior fossa space-occupying lesions	Usually unilateral lesions, especially those located above the temporal lobes
Structures involved	Diencephalic compression progressing to pressure on the reticular activating system	Ipsilateral cranial nerve III, ipsilateral posterior cerebral artery, contralateral cerebral peduncle	Medullary respiratory center	Anterior cerebral artery
Signs/symptoms	Altered level of consciousness	Ipsilateral pupil dilation, ipsilateral hemiparesis (Kernohan's notch phenomenon)	Respiratory arrest	Leg weakness

Table 2.12 Stages of herniation[22]

Central herniation	Diencephalic stage	Mesencephalic-pontine stage	Pontomedullary stage	Medullary stage
Consciousness	Agitation or drowsiness	Comatose	Comatose	Comatose
Respiration	Sighs and yawns	Cheyne–Stokes respiration or tachypnea	Regular or shallow and rapid	Slow, irregular rate and depth and possible hyperpnea with apneic periods
Systemic responses	Diabetes insipidus (DI)	Hypothalamic dysfunction (DI, poikilothermia)		Fluctuating pulse, drop in blood pressure
Pupils	Small (1–3 mm) reactive	Midposition (3–4 mm), fixed	Small-midposition and fixed	Dilated and fixed
Eye movements	Roving eye movements	Vestibulo-ocular reflex im-	No vestibulo-ocular reflex	No vestibulo-ocular reflex

(continued)

Table 2.12 continued

Central herniation	Diencephalic stage	Mesencephalic-pontine stage	Pontomedullary stage	Medullary stage
	Vestibulo-ocular reflex may be weak or brisk No caloric response No vertical eye movement	paired with possible dysconjugate response	No oculocephalic response	No oculocephalic response
Motor	Worsening of existing hemiplegia Decorticate posturing	Decerebrate posturing	Flaccid flexor response	Flaccid, no deep tendon reflexes

Table 2.13 Stages of uncal herniation[19]

	Early third nerve stage	Late third nerve stage
Consciousness	Agitated or drowsy	Obtunded
Respiration	Normal	Hyperventilation
Pupils	Relative dilation of ipsilateral pupil	Fully dilated pupil
Eye movements	Oculocephalic normal or dysconjugate	
Motor	Appropriate to pain (localizes), contralateral Babinski's sign	Possible ipsilateral hemiplegia (Kernohan's notch) decerebrate

Metabolic causes of coma can include respiratory changes leading to acid–base changes, hyperventilation, and metabolic encephalopathy.[17] Mechanisms that can cause irreversible anoxic-ischemic brain damage, disorders of glucose (including both hypoglycemia and hyperglycemia), diseases of the liver, kidneys, or pancreas, and adrenal disorders can cause changes in mental status and coma. Diabetes is the most common endocrine disorder presenting as undiagnosed stupor and coma where most diabetic patients are prone to nonketotic hyperglycemic hyperosmolar coma, ketoacidosis, lactic acidosis, hyponatremia, hypophosphatemia, uremia-hypertensive encephalopathy, hypotension, or even sepsis. Other endocrine disorders that one may see in the neurosurgical setting include thyrotoxicosis in an elderly patient, where the usual signs of hypermetabolism are masked with depression and apathy. ▶ Table 2.10 can be reviewed for common causes of coma.[18]