Clinical Examination of Critically Ill Patients

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Chapter 8

Central Nervous System Examination in a Critically Ill Patient: Part 1 Brain

George John

ABSTRACT

Clinical examination of the brain is an essential component of the clinical evaluation of a critically ill patient. It should be done systematically taking into consideration the constraints of examination in a critically ill patient in an intensive care unit setting.

CASE SCENARIO 1

A 20-year-old girl from a village was brought to the emergency department of a tertiary care center with a history of being unarousable after going to sleep on the verandah of her house in the village. There was no history of ingestion of any poison. The patient was deeply cyanosed, had total ophthalmoplegia, dilated pupils, and absent pupillary, vestibulo-ocular, and corneal reflexes. There were no facial movements or limb movements in response to pain. Deep tendon reflexes were not elicitable.

INTRODUCTION

The most important organ which is being supported in any intensive care unit (ICU) is the brain. To give an analogy, in a chess game, if the king is lost, the game is lost, even if all the other pieces are on the board. Similarly, in any clinical scenario, if a person's brain is irreversibly damaged, all is lost in terms of the meaningful survival of the person.

This section will focus on the examination of the brain and cranial nerves relevant to the ICU patient. In this context, the evaluation of focal cortical deficits such as aphasia, agnosia, or apraxia are only discussed in so far as that it affects the performance of the clinical examination and its interpretation.

The clinical neurological evaluation, to be useful, must be done meaningfully (understanding what is being tested) rather than mechanically. To obtain meaningful information, it must be systematically done and documented. Performing a minutely detailed neurological examination without a purpose is a waste of time and can yield minor findings, which are red herrings and cloud the picture.

All details of scoring systems in this discussion are given at the end of the section to preserve the continuity of one's flow of thought while reading.

The key to neurological evaluation is the *observation*.

- *Presence or absence* of a response—*spontaneous* or *induced* with a stimulus
- *Compare: Asymmetry* of a response is the key to recognizing subtle and early signs—see both sides; observe for unilateral/asymmetrical response
- *Serial evaluations over time* are essential to recognize trends and pick up early deterioration
- *Confounders* such as sedation, analgesia, neuromuscular paralyzing agents, metabolic disturbances, and *constraints* to examination such as injuries, intubation, and bandages must be factored in the assessment.

Patients in an ICU with a neurological problem may have the following scenarios:

- Admitted with a specific critical neurological problem:
  - Depressed sensorium/coma
  - Cerebrovascular accident
  - Head injury
  - Postcardiac arrest
  - Seizures
- Develop a neurological problem as a result of being critically ill due to other organ system failures:
  - Delirium
  - Critical illness neuromuscular abnormalities

Examination of the brain and cranial nerves is important in all the above scenarios for:

- *Diagnosis:*
  - What is the lesion?
  - Where is the lesion?
  - Brain death: change the focus of support
    - Withdrawing care
    - Organ donation/harvesting
- *Prognosis:*
  - Is ICU care likely to be beneficial?
  - Family counseling regarding cost, palliative mode of care, and long-term care
- *Change in status recognition/documentation:*
  - Monitor and document any improvement or deterioration

Basic rules:

- *Restlessness in any critically ill patient should be assumed to be due to cerebral hypoxia unless proven otherwise. Rule it out before proceeding to a formal evaluation.*
- *Altered sensorium in the ICU may be medication related: Check the medications before interpretation.*

The examination of the brain and cranial nerves should be systematic and sequential. A common template that can be followed is to assess the brain function into the following subdivisions:

- Consciousness
- Thought content
- Cranial nerves

ASSESSMENT OF CONSCIOUSNESS

The assessment of consciousness may differ depending on the patient's status:

- *Altered sensorium:*
  - What is the *level* of consciousness?
  - What is the *content* of consciousness?
- *Normal sensorium:*
  - Focal deficits with impaired communication: *Aphasia/agnosia/apraxia*
  - Focal deficits with normal communication: *What and where is the lesion?*

Patients with Altered Sensorium

There are two functioning neurological structures needed for consciousness—*the brain stem* and *one cerebral cortex.*

Consciousness has two components:

1. *Arousal* (wakefulness, level of consciousness) is the *overall level of alertness* to environmental stimuli (*wakefulness*). Arousal is a set of changes that occurs when the person transits to alertness—the most obvious sign being the opening of the eyes. It depends on an intact, functioning ascending reticular activating system (ARAS) passing through the *thalamus* to the *cerebral cortices*. It is a subcortical phenomenon and needs a ***functioning brainstem***. *Bilateral thalamic lesions* can also cause coma because they interrupt the ARAS fibers passing through them to the cortex **(Table 1)**.

   The capacity of lesions in the brainstem to cause coma depends on its site, size, and speed of progression. Brainstem infarctions and hemorrhage commonly cause coma, whereas slower lesions such as multiple sclerosis or slowly infiltrating neoplasms rarely do so. *Lesions below the pons rarely cause coma.*

2. *Awareness* is the ability to perceive specific environmental stimuli in different visual, auditory, and somatosensory domains. It is the ability to be aware of one's environment and understand one's relationship to one's surroundings (*responsiveness*). This depends on the *content* of consciousness and is the summation of a person's cognitive and affective (emotional) mental functions. *Awareness* (the content of consciousness) depends on the integrity of the *cortex* and the *limbic* system (basal forebrain, amygdala, hippocampus, hypothalamus, and cingulum; **Table 1**).

In a nutshell, the state of arousal is easily confirmed by the opening of the eyes, and awareness is inferred from the ability to follow commands.

TABLE 1: Classification of conscious states.

Aroused, aware	*Aroused, unaware*	*Unaroused, unaware*
Normal	Delirium: hypoactive, hyperactive, mixed	Sleep, deep sedation
Anxiety, depression	Psychosis, dementia	Coma
Lethargy	Vegetative state	Brain dead

Comatose Patient

The cortex of a comatose patient is, by definition, dysfunctional. This may be either primary or secondary because of a dysfunctional ARAS. Drugs and metabolic states induce coma by depressing both the ARAS and the cerebral cortex.

If the brainstem function is intact, then the coma is due to a *primary cerebral cortical dysfunction*. If one or more

levels of the brainstem are damaged, then the *cerebral cortical dysfunction* is *secondary*, and the cause of coma is at the brainstem level.

Examination of a comatose patient is first focused on assessing the brainstem function. This is carried out in a craniocaudal format, level by level. A *rapid 4-layer functional examination* and interpretation is useful in the ICU. Level-by-level testing of the brainstem involves testing the brainstem at each level with at least one neurological reflex. If the response at a particular level is abnormal, the corresponding brainstem level is considered dysfunctional.

If all 4 levels of the brainstem are functioning normally, then the coma is cortical (the cortical dysfunction is primary). *A unilateral primary cortical dysfunction does not cause coma unless there is pre-existent dysfunction in the other cortex or there is a secondary brain stem dysfunction (herniation).*

Levels

First step: Response to verbal instructions
Instruct the patient to open the eyes and look up and down and side to side; if the patient opens the eyes and follows all verbal commands appropriately as requested, the *level and content* of consciousness are *normal*.

If the response is not as expected, check the following described further.

Level 1: Thalamus—Response to Pain

- *Stimulus site:* Supraorbital, nail bed, and sternum
- *Observe:* Presence of response, nature of the response, and symmetry of response
- *Appropriate:* Push away stimulus or withdraw from it
- *Inappropriate responses and interpretation:*
  - *Decorticate:* Flexion of upper limbs and extension with internal rotation of lower limbs
    - *Interpretation: Subcortical/thalamic dysfunction*
  - *Decerebrate:* Extension and internal rotation of both upper and lower limbs
    - *Interpretation: Upper brainstem (midbrain or upper pons) dysfunction*
  - No response or extension of the upper limbs, bending of knees
    - *Interpretation:* Reflex response at the spinal level.

Level 2: Midbrain—Pupillary Reflex

- *Stimulus:* Light is shown into the pupil and response is observed; one can use an ophthalmoscope to magnify the pupil and see minimal responses.
- *Observe:* Pupillary constriction on both ipsilateral and contralateral pupils in response to light
- *Appropriate:* Details are given in the section on the examination of cranial nerves under ocular nerves.
- *Inappropriate responses and interpretation: Lesions above the thalamus and below the pons spare pupillary responses*

Level 3: Pons

- *Doll's eye reflex (oculocephalic/oculovestibular reflex)*
- *Corneal reflex*
- *Respiratory pattern (pons and medulla)*

If cervical spine injury is suspected, do not turn your head.

Details are given in the section on the examination of cranial nerves under ocular and trigeminal nerves.

Level 4: Medulla

Spontaneous cardiorespiratory changes:

- Apnea
- Unstable blood pressure
- Irregularities in heart rate and rhythm.

Coma Look-Alikes (Differential Diagnosis)

Locked-in Syndrome

This is due to a focal brainstem lesion usually involving the junction between the upper third and lower two-thirds of the pons. There is enough ARAS in the upper third of the pons to *preserve wakefulness and content of consciousness*. However, all functions below the pontine level are lost. These patients can move muscles controlled by cranial nerves III and VI—ocular movements are preserved but extremities cannot be moved. They can see and hear the examiner but sense neither touch nor pain. The eyes are open (in a true coma they are closed) and they can track the examiner. They may communicate using eye movement.

Vegetative State

These patients can be aroused (wakefulness) but lack content of consciousness. There is *awareness-arousal dissociation*. It has been named by some as a *"locked-out" syndrome* in that the cortex is unaware of the external world and so is disconnected from it. This can be due to widespread cerebral cortical damage (hypoxia or ischemia), diffuse damage to subcortical structures, or bilateral thalamic damage.

Psychological Dysfunctions

Hysteric (Conversion) reaction: This can mimic coma. One common clinical test used is to raise the upper limb above the face and let it drop. In conversion reaction, the face will be protected by the patient by allowing the upper limb to fall slowly to the side.

Catatonia: The patient is awake; the eyes are open but immobile. Transient improvement with sedation may occur.

The *level of consciousness* can be objectively assessed and documented by *any* of the following methods:

- *Alert, Voice, Pain, Unresponsive (AVPU) score*
- *Full Outline of Unresponsiveness (FOUR)*
- *Glasgow Coma Scale (GCS) score*
- *Richmond Agitation Sedation Scale (RASS)*
- *Ramsay Sedation Scale (RSS)*

Noncomatose Patient

Mentation, Emotions, Thought Content: Remember the acronym—*EMOJI*

- *Emotions:* Observe and evaluate whether the emotions exhibited are *appropriate/inappropriate and if appropriate, proportionate* to the situation.
- *Memory: Immediate, recent,* and *remote* memory can be tested
- *Orientation:* Tested with reference to *time, place, and person*
- *Judgment:* Ability to make a judgment in ambiguous situations
- *Intellect:* Test with spelling 5 letters *words* forward or back, serial *calculation* backward or forward.

The following conditions can interfere with neurological evaluation but are not discussed further:

- *Aphasia:* Acquired disorder in *using language* due to a lesion in the *dominant cerebral cortex.*
- *Apraxia:* Difficulty in performing a *complex motor task* despite normal primary motor functions. It occurs due to lesions in the *association areas* of the *frontal cortex* in either *dominant* or *nondominant hemispheres.*
- *Agnosia:* Difficulty in recognizing a *complex sensory stimulus* in the presence of normal primary sensations. It occurs due to lesions in the *association cortex in either dominant* or *nondominant hemispheres* in the *parietal* or *temporal cortices.*

THOUGHT CONTENT

Delirium is an *acute disturbance of cognition* that develops over a short timeline. It manifests itself by a clouding of consciousness (decreased clarity of awareness of the environment) and fluctuates with time. It manifests itself as confusion in the patient's thought process. Its characteristic time profile is its *acute onset* and *fluctuating clinical course.* It can manifest in the following three forms:

1. *Hypoactive:* Lethargic, decreased alertness and activity
2. *Hyperactive:* Restless, agitated, pull out tubes and lines
3. *Mixed:* Fluctuates between the two states

Validated instruments to identify delirium in the ICU are available. The two which are commonly used are as follows:

1. *Confusion Assessment Method for the ICU (CAM-ICU)*
2. *Intensive Care Delirium Screening Checklist (ICDSC)*

These scores can only be used if the patient is not comatose (RASS score must be ≥ –3).

CRANIAL NERVES

The clinical evaluation of the 12 cranial nerves is done in sequential order. In critically ill patients, it may not be possible to test all of them in every patient. Sensory components of the cranial nerves are difficult to interpret in those who are not fully conscious, but a normal reflex implies that the sensory component is intact. The technique of testing, interpretation of the response, its significance in the critically ill patient, and the constraints to testing are discussed here.

- Olfactory
- Optic
- *Ocular nerves:* Oculomotor, trochlear, and abducens
- Trigeminal
- Facial
- Vestibulocochlear
- Glossopharyngeal, vagus
- Accessory
- Hypoglossal

Most cranial nerves are *bilaterally controlled* via *upper motor neurons (UMNs)* originating in *both cerebral cortices.* The notable exceptions are:

- Lower part of facial nerve nucleus (only *contralateral* UMN control in *all* persons)
- Trigeminal and hypoglossal (only *contralateral* UMN control in *some* individuals)

Olfactory Nerve: I

The sensation of smell can be tested in each nostril using familiar substances (coffee powder). However, diminished or altered sensorium, presence of a nasogastric (NG) tube, or blood in the nasal cavity preclude testing. Testing should not be done with irritant substances because they stimulate the pain fibers of the trigeminal sensory component—the pain sensation is being tested, not olfaction. This fact is useful and can be used to test the olfaction of a person who is suspected to be malingering, stating that he/she has lost olfaction after trauma. The olfactory fibers traverse the cribriform plate of the ethmoid bone and can be genuinely damaged in head trauma. However, in a malingering patient, both sensation of smell and noxious substances when tested are stated to be lost—though they traverse different pathways. This has medicolegal consequences with respect to compensation.

Optic Nerve: II

The testing components are:

- Visual acuity
- Visual fields
- Color vision
- Pupillary reflexes
  - Light
  - Accommodation

In the ICU, vision can be grossly tested by the *menace response*. The menace response involves cerebral cortical integration and is not a reflex response. This test evaluates the behavioral response to a visual threat. It is a cortically mediated closure of both eyelids produced by a threatening or unexpected gesture or action suddenly appearing in the near visual field. The response consists of the afferent pathway (optic nerve, optic tract, lateral geniculate body, and optic radiation), occipital cortex (integration), and facial nerve (efferent). Care must be taken not to create too much air turbulence, which will elicit the corneal reflex. One eye is covered, and a hand is then moved rapidly toward the open eye stopping prior to causing physical contact. The normal response seen is the reflex bilateral blinking that occurs in response to the rapid approach of an object. The test is then repeated for the other eye.

Visual field and color vision cannot be adequately tested in the ICU and are not useful. The pupillary reflex (especially the light reflex) is very important and is given in detail in the section on ocular nerves.

Ocular Nerves: III, IV, VI

The following are important to observe:

- Eyelids
- Eye movements
- Pupils

1. The oculomotor nerve has two nuclei in the midbrain:
   a. *Oculomotor nucleus (somatic and motor)*
   b. *Edinger-Westphal nucleus (parasympathetic nucleus for pupillary control)*
2. The trochlear nerve nucleus is also in the midbrain.
3. The abducens nerve nucleus is in the upper pons.

In a patient with orbital trauma, the findings are difficult to observe and interpret.

Eyelids

- Observe for ptosis and eye opening
- Neuromuscular causes of ptosis are as follows:
  - Oculomotor lesion
  - *Sympathetic lesion:* Horner syndrome
  - Muscle diseases, for example, myasthenia (usually bilateral)

Eye Movements

Remember that the contralateral trochlear nerve [lower motor neuron (LMN)] innervates the eye being examined (in contrast to all the other cranial nerves where the ipsilateral cranial nerve lower motor neuron innervates the relevant muscles) **(Figs. 1A and B)**.

In a person with delirium, eye movements *cannot be tested meaningfully*.

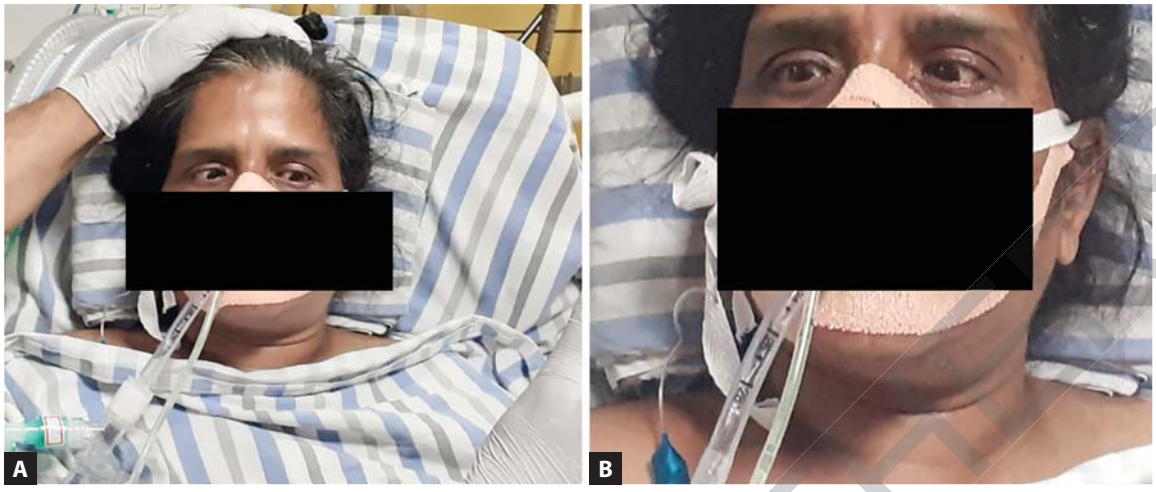
In an *unconscious patient*, the *oculocephalic* and *oculovestibular reflexes* are tested to evaluate the integrity of the vestibular system, brain stem, and ocular nerves. They test the same neural pathways.

Pathway: Afferent receptors in the inner ear (semicircular canals and utriculus)

The stimulus can be:

- Mechanical (Rotation of the head in the horizontal plane) or
- Thermal (Ice cold water into the external auditory canal with head up 30° from horizontal)

Central: Afferent signals via the eighth cranial nerve to the brainstem. The connections between the third and sixth nerve nuclei occur via the *medial longitudinal fasciculus*



Figs. 1A and B: Examination of eye movements. The examination of eye movements is performed when the patient is conscious and can follow commands. In patients who are unconscious, a full range of eye movements cannot be tested. Only horizontal movements may be tested if there are horizontal roving movements. Explain to the patient and ensure the patient is comfortable. Ask the patient to look straight, and keep your hand on the head to prevent head movements. Instruct the patient to follow your finger (torch light can also be used instead of a finger). Move the finger in all directions and note down the eyeball movements. Figures A and B show the restriction of left eye lateral movement in this patient.

(MLF) embedded in the ARAS. The response of conjugate movement of the eyes thus tests the integrity of the *ARAS* (in which the MLF is embedded).

Efferent is via the third and sixth cranial nerves.

Technique:

Oculocephalic reflex ("dolling"): This test will also aggravate a pre-existing cervical spinal injury and should *not be done in case of any suspected injury to the cervical spine.* It may be necessary to disconnect the patient from the ventilator for 20–30 s (after preoxygenation with 100% oxygen and on a pulse oximeter) to be able to rotate the head adequately.

The caloric stimulation provides a greater stimulus than head rotation as the stimulus is more sustained.

If the test can be done safely,

Hold the patient's eyes open. The head is held in the neutral starting position and is turned briskly to one side. It is held in this position for 3–4 s and is then turned 180° briskly to the opposite side. The head is turned approximately once per second and remains in the new position for 3–4 seconds. Eye positions and movements are observed in each steady position.

Positive doll's eyes reflex: Reflex conjugate deviation of the eyeballs in the direction *opposite to turning the head* ***(Figs. 2A to C)***.

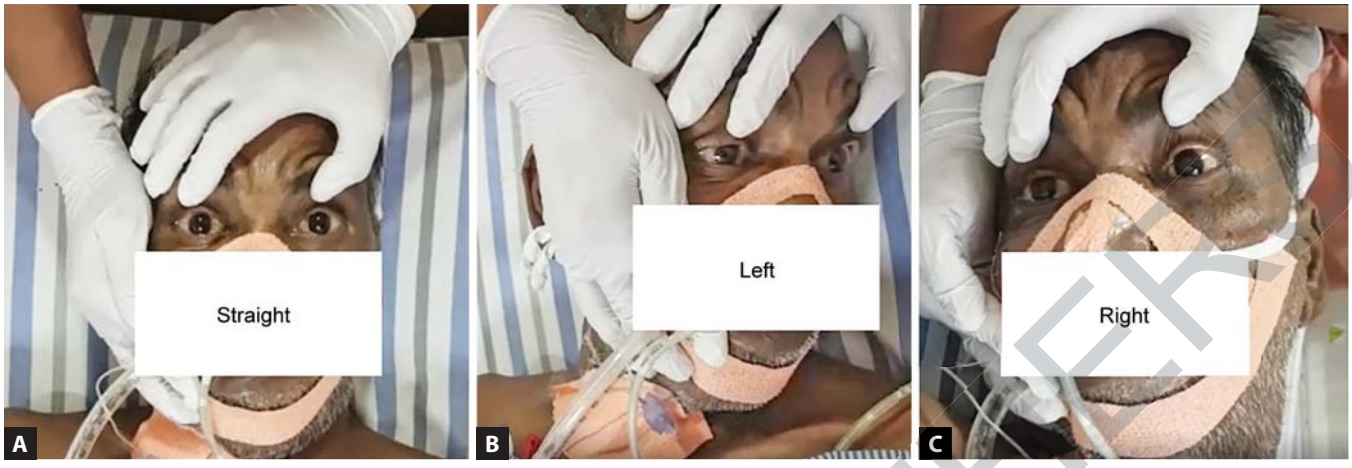
In an *alert individual*, the eyes will, within a fraction of a second, orient with the head (the oculocephalic reflex is suppressed in order for the eyes to see where the head is pointing). *A negative oculocephalic reflex occurs in a person who is awake because cerebral cortical activity suppresses it so that the eyes look where the head is turned.*

In a *comatose patient with damaged cerebral hemispheres* (*causing the coma*) but with a *functioning brainstem*, release phenomena can be seen. Passive rotation of the head will cause a conjugate deviation of the eyes to the opposite side. The eyes do not stay fixed (they move as in some dolls). The eyes are "out of phase" with the head. The reflex is *positive.*

In a *brainstem injury causing the coma*, this *reflex is lost* (*not suppressed* as in an alert person) and the head and eyes will move together (as if painted on). The reflex is *negative.*

Interpretation of response:

- *Positive:*
  - *Preserved brainstem:* Movement of eyes to opposite side. If this occurs, the brainstem is functioning, and the coma is cortical.
- *Negative:*
  - *Brainstem damage:* No movement—eyes move with the head as if they were painted eyes.
  - *Patient feigning coma* due to malingering or psychiatric illness (intact cortex): The cerebral



Figs. 2A to C: Doll's eye. Hold the head and neck by placing a hand over the back of the neck. Hold the endotracheal tube with another hand. The second operator will open both eyes and observer for eye movements. (A) Step 1—keep the head and neck in a neutral position (Rule out contraindications for the testing, e.g., unstable cervical spine); (B) Step 2—rotate the neck toward one side 90°. Observe for eye movements and patient for any discomfort; (C) Step 3—rotate 180° to the opposite side rapidly and observe for eye movements.

cortex suppresses contralateral eye movement (so that eyes can see where head is turning), eyes turn with head (as in brain stem dysfunction). However, other brain stem reflexes will reveal the true picture.

Thermal oculocephalic reflex (caloric test)

- A thermal stimulus is used for this test. An otoscopic examination should be done prior to testing to view the external auditory canal and ensure that the eardrum is visible. If there is wax (cerumen) in the external canal, it must be gently cleared. If there is a chronically perforated tympanic membrane, cold air (not water) may be used as a stimulus. *The presence of a ruptured eardrum does not invalidate the test.* Fractures to the base of the skull or petrous temporal bone may obliterate the response on that side.
- The patient's head is placed at 30° elevation from the supine position ensuring that the horizontal semicircular canal is horizontal (thus maximizing the stimulus). A soft catheter is introduced into the external auditory canal (a scalp vein needle with the butterfly flaps and metal needle cut off is a useful device). A volume of about 50 mL of iced water (up to 200 mL can be given) is given by slow injection into the external auditory canal with the eyes held open by an assistant. The eyes should be observed for at least a minute after the injection is completed. A period of 5 minutes should be allowed between tests before repeating the test on the opposite side to allow re-equilibration. Cold water (≤30°C) *mimics a head turn to the contralateral side.*
- With cold stimulation, the *normal response with intact brainstem and cerebral cortex* is that both the eyes turn toward the ipsilateral ear *(tonic conjugate deviation to ipsilateral ear), with compensatory horizontal nystagmus* (corrective component by the cortex) to the contralateral ear.
- In comatose patients *with an intact brainstem* (implying that the coma is due to bilateral cerebral cortical dysfunction), tonic deviation of the eyes occurs to the irrigated side *(tonic conjugate deviation of eyes to ipsilateral ear)*. However, as both the cerebral cortices are dysfunctional (causing the coma) the fast, corrective component (nystagmus) controlled by the cerebral cortex disappears.
- In a *comatose patient with brain stem dysfunction*, there is *no eye movement.*

  During testing for brain death, *any movement of either eye* (whether conjugate or not) *excludes* brain death.

  Warm water (≥44°C) *mimics a head turn to the ipsilateral side.* If it is used as the stimulus, the response will be the opposite of the above.

The acronym "COWS" (Cold opposite, Warm same) or the phrase "cold repels while warm attracts" summarizes the direction of the nystagmus (conventionally named as per fast component direction) not the tonic deviation.

In a *conscious patient*, eye movements can be deliberately tested as for a *standard neurological examination* **(Figs. 1A and B)**.

The following points described further are useful:
Gaze palsies: This may be due to weakness of muscles in one eye or due to a defect in the conjugate movement.

Monocular gaze palsies:
- The muscle affected in monocular gaze palsies can be picked up by requesting the patient to carry out the cardinal eye movements.
- The abducent nerve innervates the ipsilateral lateral rectus and hence its lesion would cause a loss of abduction in the same eye (ipsilateral).
- The trochlear nerve controls the superior oblique on the opposite side and hence it causes difficulty in looking down with the eye in adducted position (difficulty walking down the stairs due to diplopia). This is in the contralateral eye.
- All other eye movements are controlled by the oculomotor nerve of the same (ipsilateral) side.

Conjugate gaze palsies:
- *Horizontal gaze palsy:*
  - The frontal gaze center forces the eyes to look to the opposite side. Hence, an irritative frontal focus (seizure) will push the eyes to gaze away from the side of the lesion while a destructive lesion will prevent a conjugate movement to the contralateral side (the eyes at rest will look at the side of the lesion; **Fig. 3**).
  - The pontine gaze center does the opposite. It forces the eyes to look to the same side and hence a destructive pontine gaze center lesion will prevent conjugate movement to same side—so the eyes will look away from the lesion. An irritative lesion will have the opposite effect.
  - *Internuclear ophthalmoplegia:*
    - This is caused by a lesion in the MLF. The MLF contains fibers that connect the abducens nucleus to the contralateral oculomotor nucleus to perform horizontal conjugate lateral gaze.
    - It is not a peripheral nerve lesion as the MLF is in the brainstem. Unilateral lesions in the MLF disconnect the two nuclei. When the patient is asked to look laterally, the abducting eye is able to move but the *adducting eye ipsilateral to the abnormal MLF is unable to move medially. Nystagmus is often present in the abducting eye.* This symptom complex, which may be unilateral or bilateral, is termed *internuclear ophthalmoplegia (INO).*
    - *It is distinguished from a lesion of the medial rectus because the patient retains the ability to converge.*
- *Vertical gaze palsy:*
  - Vertical gaze becomes more limited with aging. It usually results from the midbrain lesions. There may be associated pupillary dilatation and vertical nystagmus with upward gaze.
  - *Parinaud syndrome* has a conjugate upward vertical gaze palsy resulting from a pineal tumor that compresses the midbrain or, less commonly, a tumor or infarct of the midbrain pretectum. It is characterized by the following:
    - Impaired upward gaze
    - *Collier sign:* Lid retraction
    - *Setting-sun sign:* Downward gaze preference
    - Dilated pupils that respond poorly to light but respond to accommodation
- *Downward gaze palsy:* Impaired voluntary downward gaze along with conjugate vertical gaze impairment along with preservation of reflex vertical movements is usually due to progressive supranuclear palsy.

Fig. 3: The eyes deviate to the same side of the brain lesion. A computed tomography (CT) scan of the brain showed that the infarct is on the right side and eyes are deviated toward the right side.

Pupils

Always compare the two sides.
- Shape
- Size

- Reaction:
  - *Neuromuscular blockers do not cause changes in pupil size or reactivity.*
  - *Toxic-metabolic causes of coma can change pupillary size but reactions are maintained.*

Shape: Differences between the two sides can occur as a result of ocular surgery, trauma to the eyes, or due to inflammation (iritis), which causes adhesions between the pupil and anterior capsule of the lens.

Size: Anisocoria (unequal pupil sizes) can occur as a result of traumatic injury, increased intracranial pressure (ICP), nerve palsies, medication, or poisoning. The pupil is innervated by sympathetic fibers (via cervical sympathetic chain), which dilate it (mydriasis), and parasympathetic fibers (via the third cranial nerve), which constrict it (miosis). The size depends on the balance between these opposing forces. Mydriasis occurs due to sympathetic stimulation or parasympathetic blockage and miosis due to parasympathetic stimulation or sympathetic blockage **(Fig. 4)**.

Causes of *miosis* in the ICU: Pontine lesions, Horner syndrome, organophosphorus poisoning, and opioid administration

Causes of *mydriasis* in the ICU: Oculomotor lesions (brain stem injury, uncal herniation, and posterior communicating artery aneurysm), adrenergic stimulation as a result of medications (adrenaline or high-dose dopamine and nebulized salbutamol getting into the eyes), poisoning (amphetamine, parasympathetic block due to atropine, and tricyclic antidepressant overdose), or brain death.

Ambient light and anisocoria: The defective pupil's reaction to light is always less brisk than the normal pupil's reaction. If the pupillary asymmetry increases in bright ambient light, the larger pupil usually demonstrates a defect of constriction (has a mydriasis: parasympathetic dysfunction), whereas an increase of anisocoria in dim ambient light usually indicates a deficiency of dilation of the smaller pupil (has a miosis: sympathetic dysfunction). Thus, *mild mydriasis is best recognized in a well-lit environment* **(Fig. 3)**. A bright environment tends to constrict both pupils (direct and consensual reflexes), but the normal pupil will constrict more, accentuating the asymmetry. *Mild miosis is conversely picked up in a dim environment.*

Fig. 4: Size of pupils and pupillary response to light. Note the left pupil is dilated.

TABLE 2: Pupil size, response to light in relation to the stage of the oculomotor nerve.

	Side of compression	***Opposite side***
Stage 1	Miosis	Normal
Stage 2	Mydriasis, impaired light reflex	Miosis
Stage 3	Mydriasis	Mydriasis

The nerve fibers of the oculomotor nerve pass through the interpeduncular fossa before passing between the posterior cerebral artery and the superior cerebellar artery to reach the cavernous sinus. During this course, the oculomotor nerve lies lateral to the posterior communicating artery. It then passes through the cavernous sinus and proceeds through the supraorbital fissure to reach the orbit of the eye. The *somatic (voluntary)* nerve fibers are bundled *deep inside* the nerve, whereas the *autonomic fibers* to the pupil are *superficial* on the outside of the nerve. Knowing the spatial layout of these fibers will help one understand the various forms of presentation in third nerve palsies.

In a patient who has increased ICP as the brain is pushed down, the third nerve is compressed leading to *Hutchinson's pupillary changes* **(Table 2)**.

As the parasympathetic fibers are superficially located, a compressive pathological process affects pupillary size before ocular movements are affected. Compression of the oculomotor nerve can also occur due to the following:

- An aneurysm (in posterior cerebral, superior cerebellar, posterior communicating)
- Intracranial space-occupying lesion causing uncal herniation

In contrast, in microvascular vascular lesions (diabetes mellitus, hypertension, and vasculitis), the pupillomotor fibers are relatively spared from the ischemic injury. This is because the core of the extra-axial segment of the oculomotor

nerve is infarcted while the superficial fibers are spared. The earliest manifestation may be only ptosis, which can then progress to involve other extra-ocular muscles innervated by the oculomotor nerve.

Hippus*:* Spontaneous changes in the pupillary size may be observed and are termed *hippus*. It can occur during recovery from a third nerve palsy or more rarely, it may be a sign of seizure activity in a patient who is paralyzed with neuromuscular blocking agents, and therefore the motor manifestation of the seizure activity is blocked.

Reaction: This may be due to light or accommodation.

Light reflex: Initially, shine the light from the lateral side (avoid shining from anterior to prevent triggering the accommodation response; if the patient is conscious and focuses on the light, the accommodation response will be activated). If no response is seen, the light can be shone directly. Both pupils need to be observed for any reaction. The response of the one into which the light is shown is the *direct response* and the reaction of the other pupil is the *consensual response*. Next, move the torch in an arc from pupil to pupil. If the eye has any optic nerve dysfunction, it will show a paradoxical dilatation when the torch is moved from the normal to the affected eye. This is the *afferent pupillary defect* (Marcus Gunn pupillary sign; **Table 3**). This occurs because the afferent impulse from the affected eye is reduced, so much so that when the light is shifted from the normal to the abnormal eyes, a paradoxical dilatation is noted. The pupillary dilatation *after a consensual constriction* (when the light is first shone in the normal eye and then moved to the abnormal side) dominates over the weak stimulus when light is shown in the affected eye (due to optic nerve disease).

The accommodation response: This is done by requesting the patient to look afar and then to a finger at about 30 cm midway between the eyes. The accommodation response involves constriction of both pupils **(Table 4)**. It involves pathways from the visual association cortex to the third nerve nucleus. In conjunction with the light reflex, it can give useful information.

It cannot be done in unconscious patients.

TABLE 3: Interpretation of pupillary light reflex.

Interpretation of pupillary light reflexes			
		Direct light reflex (eye into which light is shone)	
		Absent	*Present*
Consensual light reflex (opposite eye)	*Absent*	Ipsilateral optic nerve lesion	Contralateral oculomotor nerve lesion
	Present	Ipsilateral oculomotor lesion	Normal

TABLE 4: Interpretation of accommodation response of pupillary light reflex.

Interpretation of pupillary reflexes			
		Pupillary light reflex	
		Absent	*Present*
Accommodation response	*Absent*	Bilateral optic nerve lesion, Parinaud syndrome (some)	Occipital cortical blindness, midbrain lesion (rare)
	Present	Ciliary ganglion lesion, Holmes-Adie, Argyll-Robertson, Parinaud syndrome (most)	Normal

Trigeminal Nerve: V

The trigeminal nerve has three testable components as follows:

1. Motor
2. Sensory
3. Reflexes:
   i. Corneal
   ii. Jaw jerk

The motor component supplies the jaw muscles: Closing (masseter, temporalis) and opening (pterygoids). A lesion on one side causes the jaw to deviate to the ipsilateral side (side of the lesion) upon opening the mouth.

The sensory component carries the sensation of touch, pain, temperature, and proprioception from the face. Only touch and pain are tested.

Testing reflexes related to the V nerve are more useful in the ICU than testing the other components.

Corneal Reflex

The afferent is via the V sensory and efferent via the facial nerve (VII).

Stimulus: Remove any contact lens. Ask the patient to look away (so that the examiner's action is not visible) and

TABLE 5: Interpretation of corneal reflex.

Interpretation of corneal reflexes			
		Direct corneal reflex	
		Absent	*Present*
Consensual corneal reflex	*Absent*	Ipsilateral trigeminal nerve lesion	Contralateral facial nerve lesion
	Present	Ipsilateral facial nerve lesion	Normal

touch the lateral aspect of the cornea with a wisp of cotton. Touch the cornea not the conjunctiva. Touch only the outer part of the cornea so that the response is not in response to a visual stimulus (menace response) and also to prevent any possibility of a corneal abrasion in the pathway of light reaching the lens.

Response: Closure of both eyelids. The *direct corneal reflex* refers to closure of the eyelid on the ipsilateral side and the *consensual corneal reflex* refers to closure of the eyelid on the contralateral side.

The response can be interpreted as shown in **Table 5**.

Jaw Jerk (Masseter Reflex)

The patient's mouth is left open slightly. The examiner's thumb is placed on the lower jaw in the mental region above the chin and the other four fingers over the masseter muscle. A tap on the examiner's thumb with a tendon hammer normally elicits either no response or a slight contraction of masseters (palpable by the fingers on the muscle) or resulting in mouth closure (observation). Its significance is only when it is exaggerated—indicating a bilateral upper motor neuron lesion above trigeminal motor nucleus (above pons). This is commonly seen in pseudobulbar palsy.

Facial Nerve: VII

The facial nerve has the following components for testing:

1. Motor: *Facial movements*—wrinkling forehead, closure of eyes, and lip and cheek movements (blowing up cheeks, smiling, and showing teeth)
2. Sensory: *Taste* from the anterior two-thirds of tongue
3. Reflexes:
   a. Corneal reflex
   b. Glabellar tap

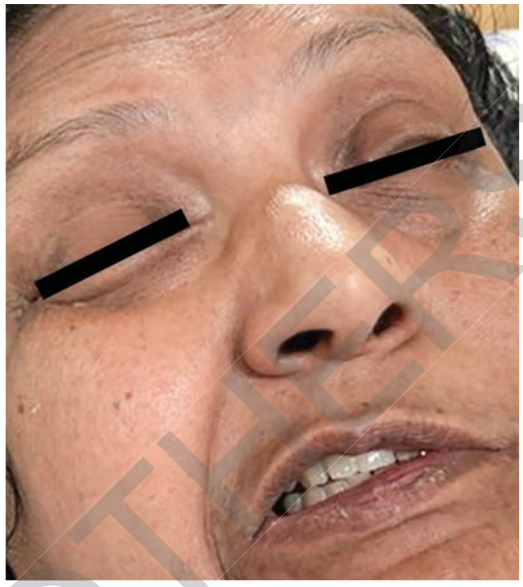


Fig. 5: The deviation of angle of mouth to the right side and incomplete closure of left eye with preserved wrinkling of forehead on both sides indicates upper motor neuron (UMN) facial nerve involvement of left side.

Motor

The motor component innervates the forehead (wrinkling) orbicularis oculi (closure of eyes) and the muscles of facial expression. First, observe facial asymmetry. Mild asymmetry can be normal. In a conscious patient, testing involves asking the patient to wrinkle their forehead, shut their eyes tightly, blow up cheeks, smile, and show teeth with teeth in apposition **(Fig. 5)**. In an unconscious patient, supraorbital pressure can reveal the weakness of facial muscles.

The facial nerve nucleus supplies the ipsilateral facial muscles via the LMN.

The UMN supply from the cortex to the facial nerve nucleus has a unique pattern: the upper part of the facial nerve nucleus is supplied from both cerebral cortices whereas the lower part of the facial nerve nucleus receives only UMNs from the contralateral cortex. This transition from bilateral control to unilateral control is gradual so that in some individuals, the top third of the nucleus (controlling wrinkling of the forehead) has bilateral supply whereas in others it is the top half (controlling forehead wrinkling and eye closure) that has the bilateral control. It is important to keep this anatomy in mind because in a UMN facial nerve lesion, only the lower facial movements

(contralateral to the UMN lesion) are affected (the upper part can be controlled through the ipsilateral cortex), whereas in an LMN facial lesion, one half of the face (ipsilateral, both upper and lower part) is affected.

It is also important to note that in a facial palsy due to a cortical lesion, emotional facial movements (crying or smiling) may be spared as they originate in other areas.

Testing the sensory component of taste is not useful in the ICU setting.

Reflexes

Corneal reflex has already been described in detail in the section on the examination of cranial nerves under trigeminal nerve.

Glabellar tap: The *glabellar reflex*, is also known as the "*glabellar tap sign.*" It is elicited by repetitive tapping of the glabella. The afferent signal is transmitted by the trigeminal nerve to the brainstem and the efferent signals go via the facial nerve to the orbicularis oculi muscle, causing blinking. The normal response to the first several taps is by blinking but if tapping is done persistently, it would normally lead to suppression of blinking. If the blinking continues with persistent tapping, it is a frontal lobe release sign, known as *Myerson's sign*. It can be present in those who have Parkinson disease, frontal lobe disease, and pseudobulbar palsy.

Vestibuloauditory: VIII

The functions of hearing and balance are subserved by the VIII cranial nerve.

A detailed testing of the auditory component is not useful in the ICU. However, bilateral deafness makes it difficult to waken a patient by calling her/him and can obviously make other testing difficult.

Vestibular function testing is useful in an unconscious patient (see the section on the examination of cranial nerves under ocular Nerves under oculocephalic reflex).

Glossopharyngeal-Vagus: IX, X

These are usually tested together.

IX: Transmits touch sensation from the nasopharynx and pharynx and taste from the posterior third of the tongue.

X: Sensory fibers from the pharynx and larynx; motor fibers to the pharynx and larynx **(Fig. 6)**.

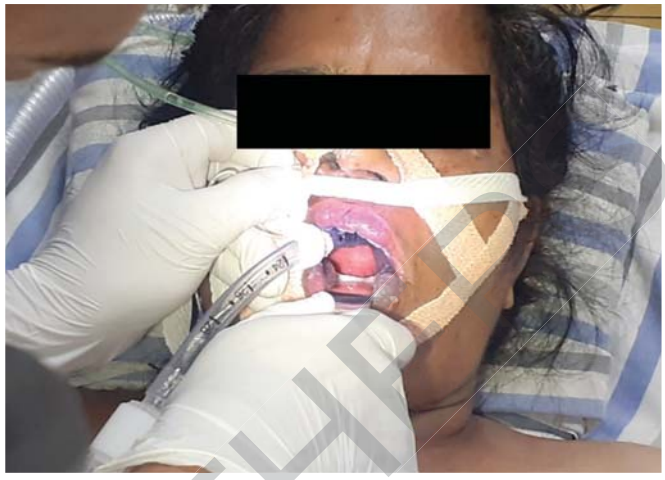


Fig. 6: Examination of a patient for gag reflex and uvula movement. First, explain the procedure to the patient. Keep the patient in a comfortable position, preferably upright. Tell the patient to open the mouth, and gently introduce the tongue depressor to depress the tongue enough to visualize the posterior pharynx and uvula. If the patient is cooperative, tell the patient to say "ah", and look for uvula movement. A swab stick can be used to gently touch the posterior pharynx to check the gag reflex.

It is of significance in two situations:

1. Absence of gag reflex makes nasal regurgitation and pulmonary aspiration likely
2. Bilateral laryngeal abductor muscle paralysis can result in life-threatening stridor. This can occur post-extubation and will need re-intubation followed by tracheostomy.

Accessory: XI

This is a motor nerve and its testing is not useful in the ICU setting. It has central and spinal components—the central component provides fibers to the vagus and the spinal component (C1 to C5) innervates the trapezius and sternocleidomastoid muscles of the ipsilateral side.

Hypoglossal: XII

Testing this nerve is not useful in the ICU. It is interesting to note that it is tested by requesting the patient to protrude the tongue—the tongue will deviate to the weak side because the intact genioglossus pulls the tongue forward contralaterally without any resistance from the weak side (which, if active, would have kept the protrusion in the midline; **Fig. 7**).

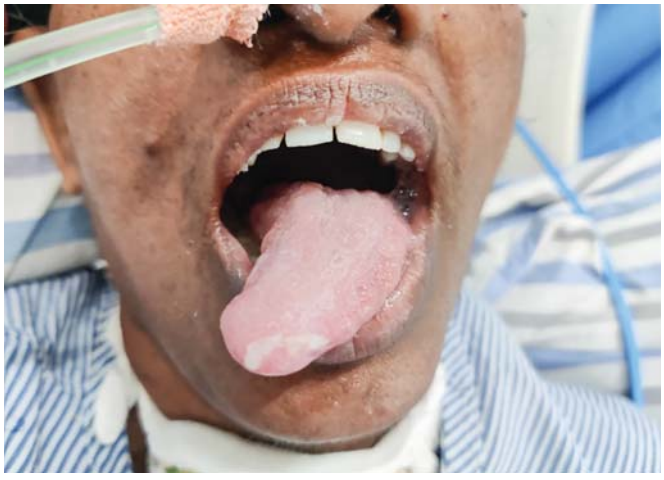


Fig. 7: Assessment of XII cranial nerve. The tip of the tongue has deviated toward the right side (weak side) due to the action of an intact nerve on the left side.

BEDSIDE EVALUATION WITH DEVICES

The traditional clinical skills of inspection, palpation, percussion, and auscultation (with the historical modification by orthopedicians of adding movement and measurement with deletion of percussion and auscultation) can be augmented by using bedside devices.

Imaging the brain has deepened our understanding of the pathophysiology of clinical signs. In a critically ill patient, this includes angiogram, CT scan, magnetic resonance imaging (MRI), isotope imaging, etc., all of which usually need patient transfer to the appropriate location.

However, there are bedside devices that can complement clinical examination, including the following:

1. Ophthalmoscopy
2. Ultrasound
3. Electroencephalogram (EEG)
4. Nerve conduction and electromyography (EMG)
5. Bedside CT and MRI

This discussion will focus on 1 and 2, which can be done by an intensivist.

Ophthalmoscopic Evaluation

The ophthalmoscope can be used to detect local eye disorders and systemic problems that can be reflected in the eye. In the context of a neurological evaluation, it can also be used to magnify and observe minimal pupillary reactions and mild nystagmus.

TABLE 6: Differences between papilledema and papillitis.

Papilledema	***Papillitis***
Usually bilateral	Usually unilateral
Visual acuity preserved, enlarged blind spot	Early loss of visual acuity
No ocular pain but headache may be present	Pain on eye movement
Spontaneous venous pulsations absent	Spontaneous venous pulsations present
Splinter hemorrhages may be present	No splinter hemorrhages
Normal pupillary reaction	Marcus Gunn pupils

This discussion will focus on two acute/subacute conditions:

1. Increased ICP
2. Bleeds in the eye

Increased Intracranial Pressure

Spontaneous retinal venous pulsations (seen using an ophthalmoscope) are present with normal ICP. However, it may not be visible in about 20% of normal individuals, so its absence does not necessarily indicate a high ICP. They may be elicited by exerting mild eyeball pressure in a person in whom it is not seen, but this could give a false positive sign that ICP is normal and hence should not be done.

Papilledema implies a swollen optic disc due to raised ICP. If it is due to a raised ICP, it is usually bilateral. Blurring and loss of a distinct disc margin are two of the earliest signs. Splinter hemorrhages around the disc can also be associated with papilledema.

Many conditions can imitate papilledema (pseudopapilledema). These include hypermetropia (no true disc protrusion), medullated nerve fibers (usually unilateral), and colloid bodies of the disc.

Nonpapilledematous optic disc swelling (papillitis) may be due to infection, inflammation, or demyelination (optic neuritis, multiple sclerosis, central retinal vein or artery occlusion, diabetic papillopathy, malignant hypertension or due to toxins).

The differences shown in **Table 6** may be useful to note.

Bleeds in the Eye

Splinter and flame-shaped: Blood follows the path of least resistance, which is along and between the nerve fibers

in the retina and not across them. In the inner nerve fiber layer, the axes of nerves are perpendicular to the light being shone—hence hemorrhage in this layer is feathery. Splinter-shaped hemorrhages are bleeds in the inner nerve layer. The individual splinter is a line of blood pooling between nerve fibers perpendicular to the light from the ophthalmoscope. A larger collection will have a flame-shaped or feathery edge—a flame-shaped hemorrhage is a fusion of splinter hemorrhages.

Subhyaloid hemorrhage (preretinal): Subhyaloid hemorrhages can be seen most often after a rupture of an intracranial aneurysms or sometimes after head injury. The shape of preretinal hemorrhages vary depending on patient position—round top when seen with the patient supine (as in an unconscious patient) or flat at the top (if patient is erect). They always obscure the retinal vessels.

Ultrasound

Optic Nerve Sheath Ultrasound

Optic nerve sheath ultrasound (ONSUS) is a bedside procedure that can be used to evaluate for increased ICP.

The optic nerve is an extension of the brain. The intra-orbital component is encapsulated by dura, arachnoid, and pia matter. The meningeal cover of the optic nerve is a continuation of the dural and subarachnoid space. This allows the optic nerve sheath to transmit cerebral spinal fluid pressure and its changes. The bulbous portion of the optic nerve, approximately 3 mm posterior to the globe, appears to be the most distensible and sensitive to changes in ICP.

Optic nerve sheath diameter (ONSD) is a reliable marker of intracranial hypertension. ONSD changes with ICP in real time. Monitoring it is useful in ICUs (where invasive ICP monitoring is not routinely available) in order to recognize early intracranial hypertension and for recognizing possible brain death.

The probe used is one with the highest available frequencies (12–14 MHz linear array transducer) because of the relatively shallow depth, small volume of the region of interest (ROI), and very low attenuation in ocular media. Higher frequencies offer better resolution and smaller, easier-to-handle probes with a small contact surface.

The upper limit of the normal ONSD includes:

- 4 mm in infants
- 5 mm in children and adults
- ONSD shows linear increment till 7.5 mm, after which it plateaus off.

Systematic reviews and meta-analyses have shown that ONSD of >5.70 mm has a concurrent ICP value >20 mm Hg.

Transcranial Doppler

Intracranial neurovascular examination is done by using a transcranial Doppler (TCD) study. TCD is a painless, noninvasive, radiation free, and easily repeatable bedside test. It measures the rate and direction of blood flow inside intracranial vessels (arteries in the circle of Willis). The ultrasonic beam that is emitted from the TCD probe crosses the skull at the target location and is reflected from the erythrocytes, which flow at different speeds in the cerebral vessels. The reflected signals are recorded. It provides an indirect assessment of the cerebral vessels' diameter. An ultrasound probe of low frequency is used (2 MHz) because soft tissues and bone attenuation are low at this range compared with higher frequencies.

It provides information regarding cerebral blood flow velocity (CBFV). The basic principle of TCD ultrasonography is based on the fact that the CBFV in a cerebral artery is inversely proportional to the diameter of that artery.

Three *acoustic windows* to evaluate the cerebral arteries are available:

1. Transtemporal window (between the eye and ear pinna) to assess the middle cerebral artery
2. Transorbital window to assess the ophthalmic artery
3. Transforaminal window (across the foramen magnum) to evaluate the vertebral arteries

The use of TCD helps in the following:

- Early detection of cerebral vasospasm following subarachnoid hemorrhage (SAH)
- Detection of ischemic stroke and monitoring of efficacy of treatment in patients with hyperacute stroke
- Early diagnosis of hyperperfusion syndrome after carotid endarterectomy
- Monitoring of arteriovenous malformation (AVM) after definitive treatment
- Confirmation of brain death

Its limitations are as follows:

- High operator dependency
- Measurements of velocity may be affected by multiple factors such as age, gender, hematocrit value, differences in the partial pressure of CO_2 in blood, and thickness of the skull bone

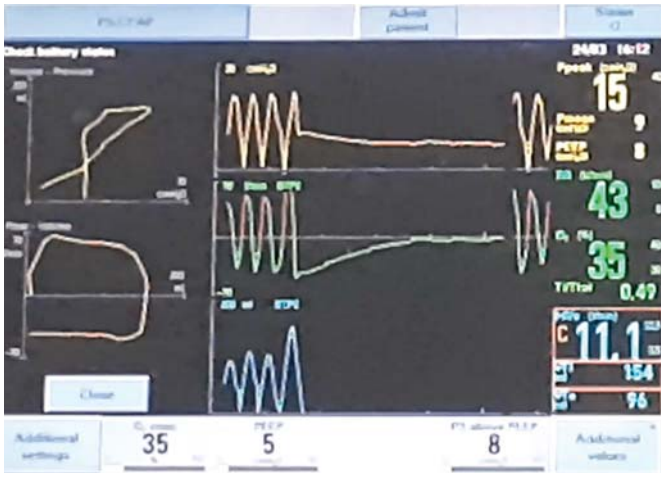


Fig. 8: Biot breathing: It is characterized by regular deep respirations interspersed with periods of apnea. This is the ventilator graphics from a patient who had a pontine infarct.

- Measurements are also limited to the large basal arteries and it can only provide an index of global and not local cerebral blood flow velocity
- Superficial AVM and those located in the parietal, occipital, and cerebellar regions are difficult to detect.

SUMMARY

The examination of the central nervous system in a critically ill patient comprises of examination of the brain, cranial nerves, peripheral nerves and muscles systematically. Examination of the brain and cranial nerves is included in this section and the rest in the next section. The examination is done within the constraints relevant to a critically ill patient with the goal of assessing the location and extent of neurological dysfunction.

The brain is evaluated in terms of the level and content of consciousness. The twelve cranial nerves are then examined systematically.

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CASE SCENARIO 2

A 20-year-old girl from a village was brought to the accident and emergency department of a tertiary care center with a history of being unarousable after going to sleep on the verandah of her house in the village. There was no history of ingestion of any poison.

The patient was deeply cyanosed. She was promptly intubated and put on mechanical ventilation. On detailed examination, she had a Glasgow Coma Score of 2T/10 with total ophthalmoplegia, dilated pupils, and absent pupillary, vestibulo-ocular, and corneal reflexes. There were no facial movements or limb movements in response to pain. Deep tendon reflexes were not elicitable. Abdominal and plantar responses were absent.

The heart rate was 100 beats/min, blood pressure 100/60 mm Hg, and respiratory rate was <10 breaths/min with an SpO_2 of 70%. Temperature was 36.6°C. Cardiorespiratory examination was normal. Abdominal examination showed possible fang marks on the right lateral abdomen with no local signs of inflammation.

The patient had been completely paralyzed on arrival and had thus been apneic for an indeterminate period of time. Therefore, hypoxic encephalopathy leading to brain death was suspected. However, in view of her age, potential reversibility and presence of fang marks, it was decided to treat her for 72 hours and reassess.

Thinking Through

This patient was unconscious and hypoxic on arrival.

The first question is whether the patient needs cardiopulmonary resuscitation (CPR).

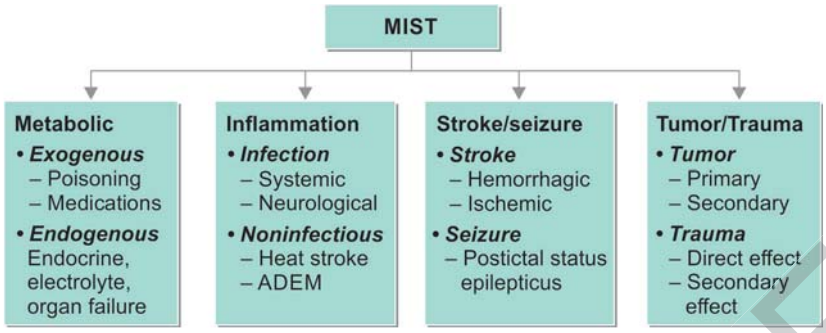
However, there is a pulse and blood pressure but respiratory effort was inadequate, and because diagnosis and treatment must proceed in parallel, she is intubated.

She had areflexic quadriparesis with nonreacting pupils.

The next question is: What is the cause of neuromuscular paralysis and pupillary unresponsiveness?

A rapid mnemonic to remember the causes of coma in terms of pathophysiology is MIST as depicted in **Flowchart 1**.

Flowchart 1: Mnemonic to remember the causes of coma in terms of pathophysiology.



(ADEM: *acute disseminated encephalomyelitis)*

Rapid testing for glucose is easily done using finger-prick sample.

The last two boxes (stroke/seizure, tumor trauma) are usually made out from a combination of history and localizing signs.

In the present scenario (lack of positive history and no localizing signs), the first two boxes are more likely as a cause for the coma.

In view of the acuity of onset, the *first box is* most likely and the highest priority would be poisoning as endogenous metabolic causes are usually slower (except for hypoglycemia).

In addition, the history in a person who has taken poison is notoriously unreliable so poisoning is still a possibility.

The one point against poison ingestion is the unresponsive pupils.

The additional finding of possible fang marks makes the diagnosis of envenomation likely. However, krait bites are notoriously difficult to detect due to lack of local signs.

In view of the potential reversibility of envenomation, she is given further treatment.

Subsequent Events

She was shifted to the ICU. Supportive therapy was initiated. In view of possible snake bite, she was given polyvalent antivenin but with no immediate change in neuromuscular status. Hematological and biochemical values were normal. A baseline cold caloric stimulation test was performed and was negative. CT brain was normal and an MRI subsequently showed no gross abnormality. Patient remained in a comatose state for the next 2 days and showed no change in neurological status. On day 3 of admission, the patient started to cough on endotracheal suctioning (this had earlier been absent). Further examination revealed slight pupillary constriction to bright light. Over the next 48 hours, the patient had a gradual improvement in ophthalmoplegia and muscle power of the extremities. Higher mental functions appeared to be intact. Mechanical ventilation was continued for another 5 days due to persistent respiratory weakness. After a week, the patient was successfully weaned off mechanical ventilation.

Clinical Examination of Critically Ill Patients

Salient Features

- A unique book that focuses on a systematic way of taking history and clinical examination of critically ill patients
- A case scenario in each chapter adds value to the chapters and provides opportunities for the readers to connect to real-life scenarios
- Predefined subsections help the reader grasp the basic principles of clinical examination, from focused examination to its application
- Well-experienced authors who are teachers, educators, and examiners in the field of critical care medicine
- The chapters are written out on the real-life experiences of the authors as the literature hardly exists
- The book has the potential to serve as a guide for all trainees in the field of critical care and to all practicing critical care physicians.

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