Assessing Patients With Altered Level of Consciousness

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Patients with alterations in level of consciousness are among the most difficult to assess, so knowledge of how to assess these patients is important for tracking trends and identifying changes. This article discusses methods used to assess patients admitted with an altered level of consciousness and describes the neurological assessment of and potential causes for altered level of consciousness. Identifying and understanding certain examination findings enable faster recognition and intervention for life-threatening neurological events, directly impacting outcomes for neurologically compromised individuals. (*Critical Care Nurse*. 2023;43[4]:58-65)

Patients with neurological conditions in any hospital unit can have a multitude of presentations, including altered level of consciousness (LOC) and cognitive, behavioral, vision, motor, and sensory changes.[1] For optimal outcomes, it is critical for the bedsxide nurse to understand assessment techniques and normal and abnormal assessment findings to enable quick diagnosis and intervention by the team. Neurological changes can be vague, can occur quickly, and may go unnoticed. These changes can be life-threatening and significantly impact quality of life. Interpreting assessment findings and correlating them to the neuroaxis can be challenging. Serial assessments are required to identify changes.[2] It takes knowledge, skill, and time to confidently perform a comprehensive neurological assessment of a patient with altered LOC.[3] Concerning examination findings may be difficult to identify in patients with altered LOC, potentially delaying the needed immediate interventions.[4] This article provides guidance for assessing patients with altered LOC.

A thorough neurological assessment includes vital signs, LOC, mental status, motor and sensory function, cranial nerve (CN) function, pupillary response, language and speech, reflexes, and cerebellar function.[5] At minimum, a neurological assessment should include vital signs, LOC, motor and sensory function, pupil size and reaction to light, and reflexes[6] (Table 1).

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Table 1 Assessment of patients with altered level of consciousness[2,4,7,8]

Assessment	What to look for	Assessment findings
Vital sign patterns	Bradycardia, tachycardia, hypertension, hypotension, temperature fluctuations, arrhythmias	
Level of consciousness	Same or different from the previous encounter?	
Eye opening	Spontaneous to voice, touch, pain?	
Body movement/posture	Spontaneous? Stimulation induced?	Normal, abnormal
Pupils	Equal size and shape? Reactive to light?	Brisk, sluggish, fixed, nonreactive
Gaze preference	Do the eyes follow an object as it is moved in horizontal, vertical, and diagonal patterns? Is there a gaze preference?	Normal, abnormal
Corneal reflex	Is there a blink response in both eyes?	Brisk, sluggish, none
Cough and gag reflexes	Does cough occur with stimulation? Gag present on both sides?	Present, weak, none
Doll's eye reflex	Do eyes move with coordinated movement opposite the direction the head is turned?	Present, absent
Deep tendon reflexes	Arms (biceps, triceps, brachioradialis), legs (patellar, Achilles), triple flexion	Absent, 0; hypoactive,1; normal, 2; hyperactive without clonus, 3; hyperactive with clonus, 4
Primitive reflexes	Babinski, grasp, snout, sucking, rooting	Present, absent
Breathing patterns	Hyperventilation, Cheyne-Stokes, apneustic, ataxic	

Vital Signs

Injury severity and comorbid conditions can cause a wide range of vital sign pattern changes. Neurons in the medulla of the brainstem regulate cyclic inspirations and expirations. The pons coordinates patterns, and the cerebral cortex provides voluntary control with each breath. Chemoreceptors adjust respiratory output according to blood gases.[7] High intracranial pressures compromise the medulla, causing an increase in systolic pressure and pulse pressure, bradycardia, and irregular respiratory patterns. Hypertension and bradycardia progressively worsen at variable rates while breathing becomes increasingly ataxic or agonal, although a change in breathing pattern may be masked by mechanical ventilation. These signs indicate loss of brain compliance, loss of brainstem function, or herniation.[4,6,9,10] Thus, monitoring vital signs, including rates and patterns, is an important component of the neurological examination.

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General Appearance

Before and while approaching the bedside, observe the patient's behavior and body position. Are the head and body in neutral alignment? Are the patient's eyes open? Is the patient looking around? Observe for spontaneous movement. Is the patient attempting to explore and understand the environment (for example, by intentionally pulling at restraints or tapping their body) or does the patient seem disconnected from reality (for example, by picking at the air)?[1]

Level of Consciousness

Consciousness is a state of knowledge and perception. Level of consciousness is the degree of arousal, and content of consciousness is the degree of awareness.[3] Evaluating both will optimize treatment and recovery

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Table 2 Altered levels and disorders of consciousness[1,2,4,7,11]

Term	Definition	Stimulus used	Assessment findings
Consciousness	Awake and aware of surroundings	Verbal	Awake, aware, and responsive to verbal and visual stimuli
Coma	Unconscious; a state of unarousable unresponsiveness	Verbal, tactile, pain	Unawake, unaware; closed eyes, immobile, no reflexive movements
Unresponsive wakefulness; formally known as vegetative state	Similar to coma	Verbal, tactile, pain	Unaware, some upper brainstem function, has sleep-wake cycles; may open and close eyes, spontaneously breathe, and have reflexive movements; appears awake. Could be transitory state between coma and recovery; could persist for extended time. Persistent vegetative state lasts >30 d, permanent vegetative state lasts >3 mo.
Obtunded	Reduction in alertness and environmental interest; decreased physiological responsiveness	Verbal, tactile, pain	Drifts off to sleep during assessment; slow in verbal, cognitive, and motor responses; responds appropriately to pain
Stupor	Aroused only with vigorous and repeated stimuli; when left undisturbed, immediately lapses back to the unresponsive state	Verbal, tactile, pain	Generally unresponsive, except for repeated, forceful stimulation; responds appropriately to pain
Delirium	Neurocognitive disorder; acute and fluctuating behavioral change resulting from consciousness and cognitive impairments		Hyperactive: restlessness, agitation Hypoactive: flat affect, apathy, lethargy, decreased responsiveness to the environment Mixed subtype: fluctuates between hyperactive and hypoactive
Dementia	Chronic disorder of executive function in at least 1 cognitive domain (memory, language, judgment, attention)		

Table 3 Tools used to assess level of consciousness[4,12-14]

Tool	Factors assessed	Scoring	Clinical significance
Glasgow Coma Scale	Eye opening Verbal responses Motor responses to spontaneous, verbal, tactile, and painful stimuli	15-Point scale 1 point for no response in each category; minimum score of 3	Score decrease of 2 or more without clear cause (eg, sedation) indicates neurological deterioration. Patients with lower scores need further assessment.
FOUR (Full Outline of UnResponsiveness) score	Eye Motor Brainstem reflex Respiratory response to stimuli	16-Point scale 4 components each with maximum score of 4	Lower scores indicate greater mortality risk (scores of 0-7, high risk; scores of 15-16, low risk).
Richmond Agitation-Sedation Scale	Sedation levels and behavior in patients who are and are not receiving mechanical ventilation Level of arousal Cognition Sustainability of sedation efforts	10-Point scale (–5) to (+4)	Positive scores indicate mild to severe symptoms. Negative scores indicate the patient is drowsy to unarousable. Score of 0 indicates the patient is calm and alert.

(Table 2).[1,4,7,10] Many validated assessment scores are pertinent to LOC (Table 3).

Multiple conditions can contribute to alterations in consciousness (Table 4). The many causes of changes in LOC include hypoxia, hypoglycemia, hypotension, hypoventilation, and medications such as sedatives.[1,4,7,16] A change in LOC is typically the first sign of a new neurological injury or deterioration. Therefore, observing the amount of stimulation (the nurse's presence, voice, or touch) needed to elicit a response is important.

Table 4 Disorders of consciousness: causes and presentation[4,7,15]

Cause of disorder of consciousness	Onset	Consciousness	Pupils	Motor	Etiology
Structural or inflammatory	Rapid	Impaired	Unilaterally nonreactive, may progress to bilaterally nonreactive	Motor dysfunction precedes coma; localized findings	Brainstem compression (direct or indirect), seizures, subarachnoid hemorrhage
Metabolic or systemic disorders	Progressive	Impaired	Preserved	Coma precedes motor dysfunction	Hypoxia, anoxia (eg, drowning, strangulation, arterial dissection, cardiac arrest, systemic hemorrhage) Ischemia (eg, embolic, thrombotic, DIC) Systemic disease (eg, cancer, diabetes) Toxin (eg, alcohol, poison, drug overdose) Meningitis (eg, vasculitis, encephalitis) Fluid and electrolyte imbalance Hypoglycemia Therapeutic coma (eg, barbiturate)
Psychiatric			Firmly closed eyes	Normal tone,	Psychogenic/functional (normal muscle tone, eyes firmly closed, EEG shows awake rhythm) Catatonic (appears unresponsive, but is conscious)
Coma of unknown cause	Rapid or progressive	Impaired	Impaired	Impaired	Potentially life-threatening; diagnosis of exclusion

Abbreviations: DIC, disseminated intravascular coagulation; EEG, electroencephalogram.

Mental Status Assessment

As a patient's condition deteriorates, changes in their mental state may not be fully appreciated. Disorientation to situation occurs first, followed by disorientation to time, place, and then person. Delirium is a common condition characterized by an acute change in cognitive function.

Nurses commonly screen patients for delirium with validated screening tools in conjunction with mental status assessment.[12-14,17-24] Although widely validated in most critically ill patient populations, tools like the Confusion Assessment Method for the Intensive Care Unit are not validated in neurologically compromised patients.[12-14,17-24]

Motor and Sensory Function

The patient's movement and the extent of stimulation needed to elicit a response determine the severity of cerebral impairment.[2] To assess motor and sensory function, first use a normal speaking voice, then progress to using a louder voice or a noise like hand clapping. If the patient does not respond, proceed to tactile stimulation by gently tapping or shaking the patient while calling the patient's name. Painful stimuli may elicit either a central or a peripheral response. A central noxious stimulus is applied by grasping the trapezius muscle of the shoulder, pressing on the orbital notch (unless the patient has facial trauma), or pinching the anteromedial section of the forearm near the axilla.[25] A central noxious stimulus should use intense pressure with limited duration to prevent tissue injury (for example, bruising or avulsion).[1,2,4,6,7] A response elicited by pressure on the nail bed reflects the spinal reflex arc, not cerebral function.[2,10]

In evaluating the extremities, comparing upper and lower extremity responses on the left and right sides is important. A normal response to painful stimuli would be purposeful, defensive, protective movements. Localization to painful stimuli indicates intact sensation and the presence of cortical functioning needed to push away the offending stimuli. Localization occurs when the patient's arm crosses the midline toward a central noxious stimulus applied to the opposite side of the body. Withdrawal responses do not cross the midline, reflecting an intact thalamus with cortical impairment. Nonpurposeful

Table 5 Motor responses in unconscious patients[2,4]

Purposeful	Localization: crossing midline of body in response to removing or stopping noxious stimulus Withdrawal: flexing response toward noxious stimulus, not able to connect with stimulus
Nonpurposeful	Muscle contraction Abnormal flexion Abnormal extension No response

Table 6 Injured cerebral structures and abnormal assessment findings[2,4]

Cerebral structures	Abnormal assessment findings
Cortex: hemispheric and/or thalamic injury	Sensory/motor deficits Decorticate posturing Possible respiratory/CV compromise
Midbrain	CN III: impaired pupillary response, impaired eye movement; roving pupils, nystagmus CN IV: impaired downward and inward gaze Sensory/motor deficits Decerebrate posturing
Pons	CN V: impaired facial sensation, corneal weakness CN VI: impaired lateral eye movement CN VII: facial movement, corneal weakness CN VIII: impaired hearing Sensory/motor deficits Possible respiratory/CV compromise
Medulla	CN IX: impaired gag reflex CN X: impaired cough reflex CN XI: impaired neck strength CN XII: impaired swallowing Respiratory/CV compromise

Abbreviations: CN, cranial nerve; CV, cardiovascular.

movements include withdrawal, muscle contractions, abnormal flexion (decorticate posturing), abnormal extension (decerebrate posturing), and no response (Table 5). Muscle contractions may be very slight and may not coordinate with other responses.

Decorticate and decerebrate posturing are pathological responses that can be spontaneous or result from tactile or painful stimulation. Decorticate posturing presents as the upper extremities rigidly flexed at the elbows and wrists with fisted hands while the legs are internally rotated and extended. Direct injury to or indirect downward pressure from large hemispheric lesions compressing the thalamus (relay station of sensory integration and motor response) yields decorticate posturing.

Decerebrate posturing presents as the upper extremities rigidly extended at the elbows and wrists with fisted hands rotated inward while the legs are extended. Decerebrate posturing reflects upper brainstem structural damage, severe metabolic disorders, or impairment in the midbrain that is severe and life-threatening.

Both decorticate and decerebrate posturing are clinically poor prognostic signs, with decerebrate posturing being more concerning. If they are new findings, these conditions mandate immediate intervention to prevent permanent, severe brainstem destruction or death.[1,4,6,7,9,10,25,26]

Variations in blood supply contribute to oscillating examination presentations. Sometimes patterns appear meaningful when they are not, complicating prognostication.[1,4,7,25] Flaccidity (no motor response, limpness) or intermittent flexor response without stimulation indicates injury to the medulla.

Cranial Nerves

Eyes

The neurological examination of the eyes represents several ocular nerves and is important to establish a baseline for detecting changes. Severe alterations in LOC inhibit the assessment of the optic nerve (CN II), needed for visual acuity. The oculomotor nerve (CN III), trochlear nerve (CN IV), and abducens nerve (CN VI) can be assessed by watching the patient's eyes follow an object as it is moved in horizontal, vertical, and diagonal patterns with or without command. If the patient cannot open the eyes, gently lift the patient's eyelids and observe the gaze position and eye movement. The presence of gaze preference provides valuable insight into the underlying neurological cause. Any resistance to eye opening is considered a voluntary response. In a patient in a comatose state, the lids close slowly when the eyebrows are raised and released; in states other than coma, the eyes close quickly.[6,16]

Impairment to the vestibulocochlear, or acoustic, nerve (CN VIII) produces nystagmus. Nystagmus is involuntary rotary, vertical, or horizontal eye movements that can occur spontaneously, with gaze, or with

head motion.[4,7,16,25] Eye patterns reflecting injury in various structural locations are shown in Table 6.

Decreases in corneal responses directly correlate to LOC deterioration.[25] The trigeminal nerve (CN V) and facial nerve (CN VII) create the corneal reflex and are assessed by placing a drop of sterile normal saline directly on the eye. If the normal saline drop does not elicit a response, touching the eyes gently with a cotton-tipped applicator, not gauze, is recommended to avoid corneal scratching.

The oculocephalic reflex (doll's eye reflex), present only in the nonconscious patient, reveals the functional status of the oculomotor nerve (CN III), abducens nerve (CN VI), and vestibulocochlear nerve (CN VIII).[10,26] Avoid testing this reflex if a cervical spinal injury is known or suspected. The oculocephalic reflex highlights passive movement or a fixed gaze and is tested by holding the patient's eyelids open and rapidly rotating the head to both sides. A desirable response is a coordinated eye movement opposite the direction the head is turned. An undesirable or absent response is a lack of eye movement so that the eyes align with the shoulder when the head is turned, indicating injury at or below the midbrain. Absence of response suggests a poor prognosis and regaining consciousness is unlikely.

The oculomotor nerve (CN III) constricts the pupil. Pupils are normally between 2 and 6 mm in diameter, with both pupils being the same size.[1,4,25] However, a small subset of patients will normally have anisocoria, or pupils slightly unequal in size (a difference of about 1 mm).[4] Pupils should be assessed with the same amount of ambient light each time. Begin by inspecting pupil size, shape, and symmetry. Pupillary response is assessed by using a pupillometer or shining a bright, narrow beam of light into the pupils. A pupillometer quantitatively measures the resting size of the pupil and reactivity to light. Although not widely available in all institutions, the pupillometer is the preferred method to assess pupils.[1,7] If a bright light with a narrow beam is used, the direct and consensual (opposite eye) pupillary responses are assessed. Wider beams (such as household flashlights) may capture a consensual rather than a direct pupillary response. Pupillary responses, eye positioning, and eye activity can provide significant insight. For example, when a patient with brain injury exhibits pupillary and brainstem signs, their condition can quickly deteriorate, and treatment interventions may become ineffective if delayed.

Face and Head

Assessment of the olfactory nerve (CN I) is typically omitted except when a basilar skull fracture is a possibility. The facial nerve (CN VII) is evaluated by assessing (at rest or with stimulation) the appearance of the patient's eyelids, cheeks, and mouth. Depressed nasolabial folds and a widened palpebral fissure indicate hemifacial weakness. If the patient is intubated, the response to a cotton-tipped applicator inside the nose will determine weakness.

The vestibulocochlear nerve (CN VIII) can be assessed by observing the patient's response to verbal stimuli. Speech and language cannot be fully assessed in patients who are intubated or obtunded. Left hemisphere or brainstem impairment compromises the patient's communication efforts (expressed and comprehended), interfering with the quality of the examination. For example, a patient may hear the sound of the command but be unable to understand and perform the given command because of an interruption in language processing. Do not provide the patient visual cues of the intended action because doing so interferes with assessment of the patient's ability to follow complex commands. Dysarthria is caused by neuromuscular weakness due to involvement of CNs V, VII, IX, X, and XII.[1,4,7,25]

The return of primitive reflexes after the first year of life indicates interruption between the corticospinal tract and motor cortex.

The hypoglossal nerve (CN XII) is assessed by having the patient extend the tongue and examining it for atrophy and weakness.[1,4,7,25] The glossopharyngeal nerve (CN IX) and vagus nerve (CN X) produce the gag and swallowing reflexes bilaterally and require separate assessment. Touching a Yankauer suction tip or tongue depressor to both sides of the posterior pharynx will elicit left and right responses even if the patient is undergoing mechanical ventilation.[4,8,25,26]

The spinal accessory nerve (CN XI) is assessed by observing the patient's ability to turn the head or raise the shoulders against the examiner's resistance.[24] Avoid

assessing head movement if cervical spinal fracture is possible and has not yet been ruled out.

Reflexes

Assessing the presence or absence of specific reflexes is valuable. The return of primitive reflexes after the first year of life indicates interruption between the corticospinal tract and motor cortex. The most commonly assessed primitive reflex, the Babinski reflex, is tested with heel-to-toe stimulation of the sole of the foot. When the Babinski reflex is present, the toes fan out in dorsiflexion. When the Babinski reflex is absent, the toes curl. The grasp reflex is assessed by placing something in the patient's hand; if this reflex is present, the patient's hand will grasp the object as if to hold it. The examiner can clarify whether this reflex is present by asking the patient to let go or attempt to identify the object by sensory integration. The snout (pouting or lip pursing when the closed lips are touched near the midline), rooting (turning the head and opening the mouth to follow and root when the corner of the mouth is stroked or touched), palmomental (twitching the chin when the palm is stroked), and glabellar (eye blinking when the forehead above the bridge of the nose is tapped) reflexes should also be assessed. These reflexes are abnormal, pathological responses.

By understanding cerebral processes, these minute changes can be quickly identified and interventions critical for brain preservation can be rapidly implemented.

Deep tendon reflexes in the arms (biceps, triceps, and brachioradialis tendons) and legs (patellar and Achilles tendons) are evaluated for involuntary responses to stimulation by partially stretched muscle groups when stimulated sensory fibers initiate impulses that progress to the spinal cord via the peripheral nerve. Triple flexion is a spinal reflex that occurs with stimulation of the flexor muscles in the legs, causing the entire limb to contract by flexing the hip, knee, and ankle simultaneously. This response reveals the integrity of transmission pathways between the sensory nerve endings of the tendons, spinal cord nerve root, and motor root. Clonus is a rapid, shaky, rhythmic, involuntary muscle contraction. Asymmetrical findings indicate a focal lesion, whereas symmetrical findings indicate generalized dysfunction.[4,7] Reflex findings are graded as absent (0), hypoactive (1), normal (2), hyperactive without clonus (3), or hyperactive with clonus (4).

Coordination

Motor control includes coordination, or the ability to execute smooth, accurate, controlled movements. A patient's motor control should be clinically assessed until the patient's LOC improves to the point at which the patient can engage in a full cerebellar assessment. Awkward, extraneous, uneven, or inaccurate movements characterize coordination impairment.[2,4,7]

Nursing Implications

Patients with altered LOC are vulnerable and are at a greater risk for a myriad of hospital-acquired complications such as infections, muscular atrophy, pressure injuries, deep vein thrombosis, and delirium. Numerous care bundles should be incorporated into routine care to prevent these complications. Additionally, this patient population may experience pain, anxiety, joint dislocation, stress fractures, contractures, tracheal ulceration or necrosis, anemia, paralytic ileus, electrolyte imbalances, and other issues that could potentially become long-term problems.[2,11] Variables that affect LOC assessment include underlying brain pathophysiology, the influence and effect of opioids and sedatives, preexisting conditions, pain, agitation, delirium, and the presence of more than 1 underlying neurological condition.[18,22,27]

Changes in status can be subtle, requiring astute monitoring. By understanding cerebral processes, these minute changes can be quickly identified and interventions critical for brain preservation can be rapidly implemented. Rapid implementation prevents long-term complications while maintaining compassion for all individuals involved. Each patient is unique and requires skill, creativity, patience, and a multidisciplinary effort to individualize care.

Nurses are at the forefront of care and therefore must comprehend the continuum of neurological disorders of consciousness. Many of the components of a neurological assessment are subjective, and assessments can change rapidly. Obtaining accurate education, skills, and neurological assessment competency helps nurses empower patients and families, thereby increasing their involvement in patient care. When a patient's family or caregiver feels informed, they are motivated to honor

the patient's wishes and make treatment decisions on the patient's behalf.

Conclusion

Confidence and competence in performing a neurological assessment are crucial to providing excellent care to neurologically impaired, vulnerable patients who are often unable to communicate, advocate for, or defend themselves. The condition of patients with altered LOC is tenuous and neurological changes are often subtle. Therefore, it is imperative to understand key components of an assessment and evaluate trends rather than isolated events. Nurses who understand the complexity of neurological assessments in patients with altered LOC can think critically and protect vulnerable patients. CCN

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See also

To learn more about neurological care in the critical care setting, read "Circadian Rhythmicity of Vital Signs at Intensive Care Unit Discharge and Outcome of Traumatic Brain Injury" by Boots et al in the *American Journal of Critical Care,* 2022;31(6):472-482. https://doi.org/10.4037/ajcc2022821. Available at **www.ajcconline.org**.

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