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REVIEW ARTICLE

Dazed and confused: the neurological examination of the unconscious patient

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Abstract

The neurological examination of unconscious patients is critical in emergency settings, where up to 5% of consultations are due to impaired consciousness. Despite the challenges posed by the lack of patient cooperation, various maneuvers can help establish the cause and localization of neurological issues. This paper presents an approach to the neurological examination in unconscious patients, emphasizing its importance in differential diagnosis and treatment planning.

Keywords: *Clinical exam. Emergencies. Unconsciousness.*

Aturdido y confundido: el examen neurológico en el paciente inconsciente

Resumen

La exploración neurológica de pacientes inconscientes es crítica en entornos de emergencia, donde hasta el 5% de las consultas se deben a alteraciones de la conciencia. A pesar de los desafíos que plantea la falta de cooperación del paciente, varias maniobras pueden ayudar a establecer la causa y localización de los problemas neurológicos. Este artículo presenta un enfoque para la exploración neurológica en pacientes inconscientes, destacando su importancia en el diagnóstico diferencial y la planificación del tratamiento.

Palabras clave: *Examen clínico. Urgencias. Inconsciencia.*

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Introduction

The neurological examination, like any physical examination, aims to prove or disprove the differential diagnosis derived from the patient's history[1]. As a versatile tool, it can be adapted to various scenarios and needs, including the assessment of an unconscious patient.

The classic structured neurological examination largely depends on the patient's cooperation and interaction. While some components (e.g., gait assessment) cannot be performed on an unresponsive or non-cooperative patient, there are still numerous maneuvers that can be conducted to establish the cause and localization of the patient's problem. This is crucial, as up to 5% of all emergency department consultations are due to impaired consciousness. The differential diagnosis in such cases is broad, including seizures, cerebrovascular disease, infections, and intoxications, among others. This underscores the importance of performing an accurate and methodical neurological examination[2,3].

Here, we present our approach to the neurological examination of the unconscious patient focused on adult population. Pediatric examination will not be discussed here (Fig. 1).

History and general physical examination

Several neurological textbooks highlight the importance of history taking and the general physical examination in the inpatient neurological setting[4,5].

For the acute coma scenario, the most important aspects of the history to consider are the onset, past medical history, current list of medications, and context (what was the patient doing?). With these four questions, a clever physician can start formulating their working hypothesis.

The minimum general physical examination involves inspection and vital signs. Since in most hospitals, the first physician to evaluate patients is rarely a neurologist, most patients will be stable, monitored, and will have basic bloodwork by the time a neurological evaluation is required. Abnormalities in respiration are important and can help localize lesions (Fig. 2). The inspection often reveals clues about comorbidities that patients may have, and signs suggesting skull fractures (Battle's sign or raccoon's eyes sign)[4].

Level of consciousness

Consciousness, simply put, involves three main axes: wakefulness, awareness (the ability to interact with the environment), and the ability to produce movement[6]. Impairment in any of these can cause acute or chronic impairment of consciousness, as described in tables 1 and 2[5,7,8].

In chronic cases, it may be difficult to differentiate between a minimally conscious state and a vegetative state (now often referred to as unresponsive wakefulness syndrome). In such cases, the Coma Recovery Scale-Revised is one of the most sensitive tools used to detect disorders of consciousness[9,10].

Cranial nerves

The full cranial nerves examination requires the patient's cooperation, which could lead to the assumption that in unconscious patients, this assessment cannot be done. However, there are useful maneuvers that are even included in brain death criteria and aid prognosis in post-cardiac arrest patients[11,12].

As with the cooperative patient, a systematic approach is often helpful and prevents overlooking important steps.

For the II cranial nerve, fundoscopy can be performed, some authors emphasize that technical difficulties, the time-consuming nature of the examination, and an overall low sensitivity for detecting papilledema in undilated pupils by non-ophthalmologists may render this test less useful in the acute coma setting; nevertheless, it should always be checked[13]. The pupillary light reflex involves the II and III cranial nerves, and its absence can be considered a reliable predictor of poor functional outcome in post-cardiac arrest patients[12].

The blink to threat (also known as the menace reflex) is frequently used in clinical practice. In response to a sudden lateral movement directed toward the eyes, a person momentarily closes their eyelids. There is little information about the sensitivity and specificity of this maneuver. Some authors propose the following rule of thumb: in daily clinical practice, the presence of the menace reflex excludes hemianopia, but its absence is of little localizing value and does not necessarily imply a visual field deficit[14].

The examination of eye movements is one of the most valuable tools for diagnosing neurological disease. Detailed evaluation can help physicians localize lesions and assist in establishing the prognosis for unresponsive patients (Tables 3 and 4).

The rest of the cranial nerves' examination involves evaluating brainstem reflexes (e.g., corneal reflex,

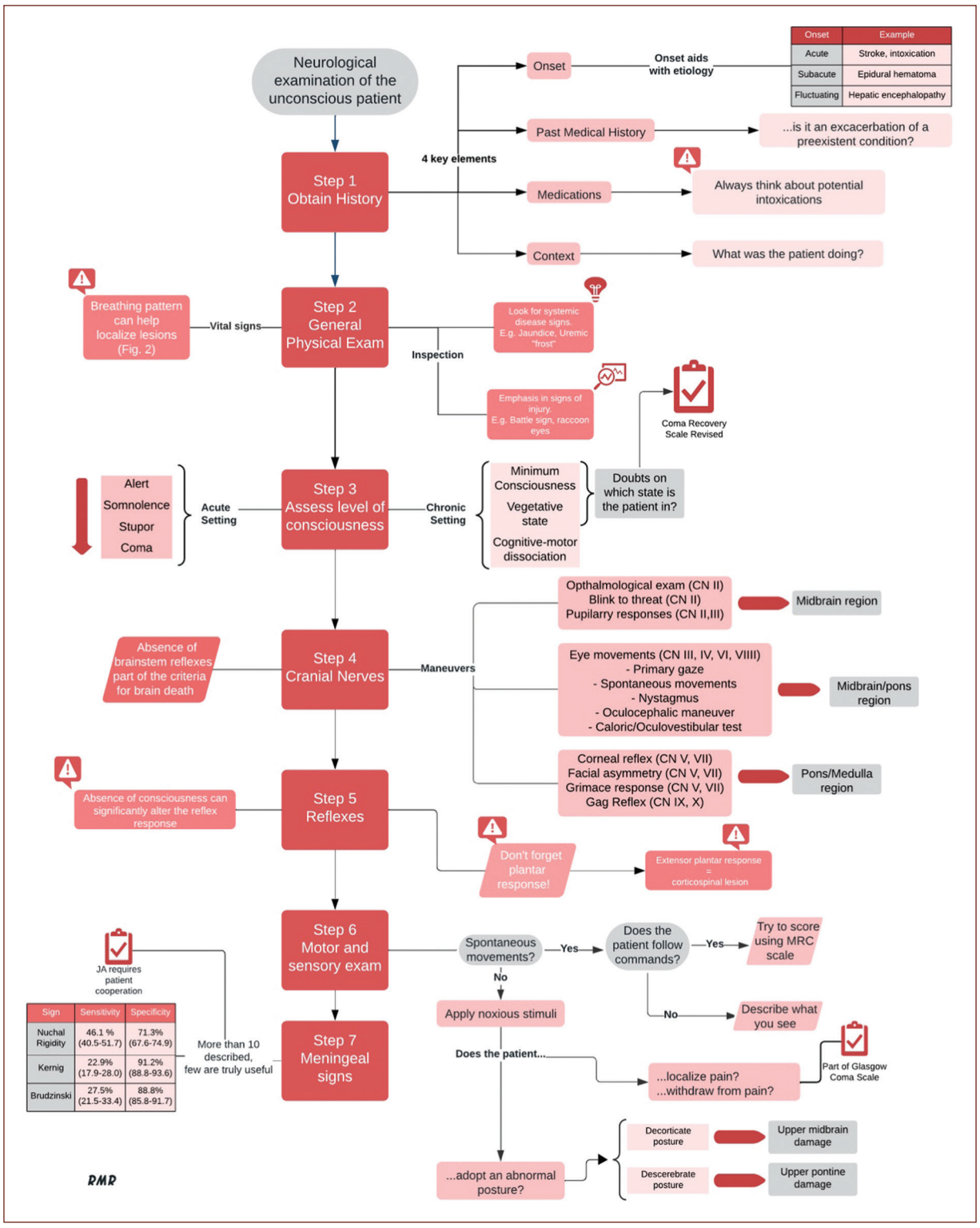

Figure 1. The neurological examination of the unconscious patient.

facial symmetry at rest, grimace response, and gag reflex), which are essential. Their lack of response can also aid in localizing lesions[11,13]. Dude to the need for patient's cooperation, the lower cranial nerve's examination is usually limited to the gag reflex.

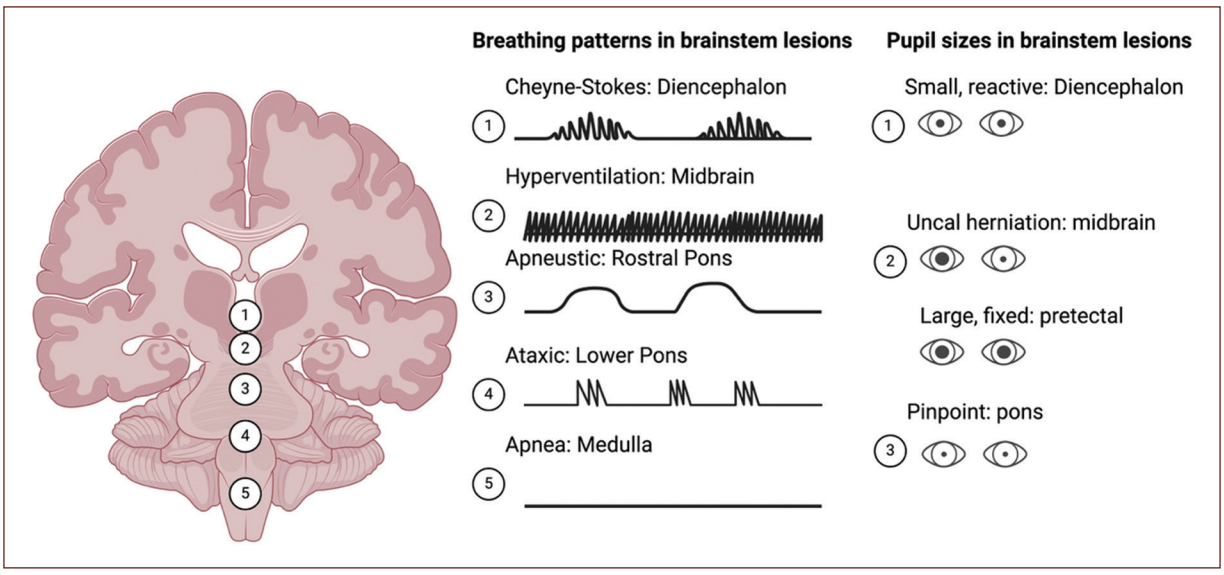

Figure 2. Breathing patterns in brainstem lesions *(adapted from Posner et al., 2019)*[5].

Table 1. Acute unconscious states definitions

Conscious state	Definition
Alert	The subject is awake. Full awareness of self and one's relationship with the environment is required
Somnolence	A condition characterized by diminished alertness where the individual tends to nod off when not actively engaged
Stupor	A *deep-sleep* like state wherein the person can only be aroused with intense and sustained stimulation
Coma	The individual lies with eyes shut and despite of the vigorous stimulation (*e.g., painful stimuli*), the individual cannot be aroused

Adapted from Posner et al., 2019[5]

Table 2. Chronic unconscious states definitions

Conscious state	Definition
Minimum consciousness	The subject shows sporadic or minimal but clear evidence of self or environmental awareness which is consistently demonstrated (e.g., purposeful behavior)
Vegetative state	There is absence of behavioral evidence of awareness of the self and the relationship with the environment. There remains the stimulus-induced arousal and normal sleep-wake cycles
Cognitive-motor dissociation	Also known as covert consciousness. Detection of intentional brain activity through MRI or EEG, but no response to the examiner and no detectable command following behaviors

Adapted from Posner et al., 2019[5]

Reflexes

Reflexes in unconscious patients may not be as critical as in cooperative patients, but their absence in acute settings (< 24 h) may indicate a spinal cord injury. Over time, the natural course is for these reflexes to become exaggerated (hyperreflexia), which is commonly seen in the progression of spinal cord injuries[15].

Monosynaptic reflexes, also known as stretch reflexes, involve a single synapse between the afferent (sensory) and efferent (motor) neurons. The most recognized one is the patellar or knee-jerk reflex. This reflex examination involves striking the patellar tendon with a reflex hammer. A typical response is the contraction of the quadriceps muscle, causing extension of the leg. Absence or reduction of this reflex may suggest a lesion at the L2-L4 level of the spinal cord. Other important localizing reflexes include the bicipital (C5-C6), brachioradialis (C6), tricipital (C7), and ankle-jerk (S1) reflexes[16]. Cranial to caudal inspection order is always recommended.

Polysynaptic reflexes, on the other hand, involve multiple synapses and interneurons, providing a more complex response. The nociceptive flexion reflex, commonly examined in unconscious patients, is believed to be a polysynaptic reflex aimed at facilitating the withdrawal of

Table 3. Eye movement maneuvers in non-responsive patients

Eye movement	Description
Primary gaze	Primary gaze refers to the resting position of the eyes, looking forward without specific eye movements. Dysconjugate eyes indicate lesions affecting individual nuclei located in the brainstem
Spontaneous eye movements	Spontaneous eye movements are the natural, effortless eye movements that occur without conscious effort
Nystagmus	Nystagmus is an involuntary, rhythmic eye movement that can be horizontal, vertical, or rotatory. It can be a sign of neurological or vestibular conditions
Oculocephalic maneuver	The oculocephalic maneuver, also known as the doll's head test, is used to assess vestibular function. It involves turning the patient's head while observing eye movements. Abnormal horizontal doll's head eye movements are indicative of lesions affecting oculomotor nerves (III), abducens nerves (VI), and pons. Abnormal vertical doll's head movements are indicative of lesions affecting oculomotor nerves (III), trochlear nerves (IV), and midbrain
Caloric tests	Caloric tests are used to assess vestibular function by stimulating the inner ear with cold or warm water. The patient's eye movements are observed in response to temperature changes in the inner ear

the biceps femoris muscle in response to noxious stimulation[17]. This reflex involves a multilevel area of the spinal cord and the integrity of multiple sensory and motor pathways[18]. Other helpful localizing polysynaptic reflexes include the abdominal cutaneous (T9-T11), cremasteric (L1-L2), bulbocavernosus (S2-S4), and anal wink (S2-S4) reflexes[19-21].

The Babinski sign, another crucial examination, is assessed by stimulating the sole of the foot and observing toe movements. An extensor response is considered abnormal and indicates a corticospinal lesion[22]. Several alternate methods for eliciting an extensor response exist, with the Chaddock and Oppenheimer signs being the most useful variations of the Babinski sign with a sensitivity of 50% but with a specificity of 99%[4,23].

An exaggeration of reflex responses, including abnormal posturing (decerebrate or decorticate), can indicate severe brainstem dysfunction, as will be discussed in the motor examination[5].

Motor and sensory examination

Examining unconscious patients poses unique challenges, as these patients are unable to provide feedback or follow commands. Therefore, the sensory and motor examination techniques employed must be tailored to these circumstances. Motor examination starts with inspection to determine spontaneous or reflex movements[5]. If a patient follows verbal commands, the Medical Research Council score is recommended for motor evaluation. If the patient has spontaneous movements but does not respond to verbal commands, only a description of abnormal movements is recommended.

Tone examination can be done, but we should always remember that in a physiologic state, the tone is diminished during sleep states; however, passive movement can reveal chronic tone changes such as spasticity[24].

In unconscious patients or those without spontaneous movements, motor response to vigorous stimuli should be elicited. These stimuli include pressing the orbital roof, performing a sternal rub, or stimulating the periungual region. Decerebrate or decorticate posturing helps infer the level of brainstem or cerebral dysfunction[5]. Decorticate posturing is characterized by flexion of the elbows and wrists with extension of the legs and feet, while decerebrate posturing is characterized by rigidity and sustained contraction of the extensor muscles of all four extremities. Decorticate posturing is produced by extensive lesions from the forebrain to the rostral midbrain. In contrast, decerebrate posturing is associated with upper pontine lesions, indicating the release of vestibulospinal reflexes. Decerebrate posturing is usually more severe than decorticate posturing, as patients are less likely to recover. Abnormal posturing is associated with a worse prognosis, with only 37% of decorticate patients surviving following a head injury and only 10% of patients with decerebrate posturing[25].

Some patients can have abnormal postures in response to sensory stimuli such as the "Lazarus sign" where in response to neck flexion the upper limbs move and can adopt a dystonic-like posture[26].

Sensory examination in unconscious patients is more intricate because it heavily relies on patient feedback, which is not feasible in this scenario. The Glasgow coma scale (GCS) is commonly used, focusing on eye-opening, verbal, and motor responses to stimuli, providing indirect inferences about sensory function. Although GCS does not specifically measure sensory input, abnormal responses usually correlate with severe sensory impairment[27]. Since the clinical evaluation

Table 4. Eye movement findings in lesions at specific levels of injury

Eye movement	Midbrain lesion	Pons lesion	Medulla lesion
Primary gaze	Impaired horizontal and vertical gaze control due to disruption of the midbrain's superior colliculus and cranial nerve nuclei. Convergence may also be affected	Impaired horizontal gaze control due to damage to the abducens nucleus. Vertical gaze may remain intact. Convergence may be impaired	Severely impaired horizontal and vertical gaze control due to extensive involvement of cranial nerve nuclei and pathways. Convergence is often lost
Spontaneous eye movements	Generally preserved as the midbrain lesions tend to spare the vestibular and brainstem reticular formation. Spontaneous nystagmus may occur	May exhibit spontaneous nystagmus due to disruption of vestibular pathways, but some preservation of spontaneous eye movements is possible	Often severely impaired due to extensive brainstem involvement. Spontaneous nystagmus is common, and spontaneous eye movements may be limited
Nystagmus	Depending on the location of the lesion within the midbrain, various forms of nystagmus can occur, including vertical, rotatory, or gaze-evoked nystagmus	Horizontal nystagmus, often with a specific pattern based on the affected pontine structures, such as horizontal gaze palsy with pontine lesions (HGPP)	Horizontal nystagmus, typically horizontal gaze palsy with pontine lesions (HGPP), but may also involve other patterns
Oculocephalic maneuver	May show impaired or absent oculocephalic reflex (doll's head maneuver) due to disruption of the superior colliculus and oculomotor pathways	Oculocephalic reflex may still produce some eye movement, but it can be altered or diminished	Oculocephalic reflex is often absent or severely impaired, reflecting extensive brainstem damage
Caloric tests	Cold caloric tests may reveal reduced or absent nystagmus due to impaired vestibulo-ocular reflex (VOR). Warm caloric tests may produce minimal response	Cold caloric tests may yield reduced nystagmus or directional changes due to damage to vestibular nuclei. Warm caloric tests may show diminished response	Both cold and warm caloric tests typically result in minimal to no nystagmus, indicating severe vestibular dysfunction

of the sensory system is limited in this scenario, evoked potential studies are useful in evaluating the functionality of sensory pathways. In this procedure, sensory stimuli are delivered, and the subsequent brain electrical responses are recorded[28].

Meningeal signs

Central nervous system infections are life-threatening conditions that can present as disturbances in consciousness in the emergency department[3]. Clinical diagnosis is one of the most difficult topics in neurology, and due to this, several signs have been described to aid in its diagnosis[29]. Most of these signs lack enough sensitivity and specificity to confidently rule in or out meningitis. Inconsistency between studies, uncertain microbiology (sometimes confounded by pretreatment with antibiotics), and interobserver variability are some reasons why a high suspicion of a central nervous system infection is needed when interpreting these signs[30].

The most useful signs are Jolt Accentuation, nuchal rigidity, and Kernig and Brudzinski's signs. Their sensitivity is low, but they are highly specific, which makes them very practical when present[30,31].

Other reflexes

Primitive reflexes (those that are present in early life, but are suppressed by development), also so-called frontal release reflexes (e.g., palmomental reflex and palmar grasp) are rarely useful since they are found in healthy people and rarely help in determining the localization and etiology of the problem. However, a very exaggerated release reflex does help pointing to a brain disease[32].

Conclusion: order even in chaos

The purpose of our approach is to avoid biases and promote a systematic evaluation of patients in the acute setting at the emergency room. Nowadays, there are clinical scales (e.g., FOUR scale) designed to standardize the evaluation of patients and facilitate communication between specialists. These scales are useful but were designed for particular settings, such as intubated patients, and not as a first evaluation in the emergency department[33].

Since impaired consciousness is a frequent reason for consultation in neurology and has several differential

diagnoses, clinical examination remains crucial in the diagnostic and therapeutic process.

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Ethical disclosures

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Confidentiality of data. The authors declare that no patient data appear in this article. Furthermore, they have acknowledged and followed the recommendations as per the SAGER guidelines depending on the type and nature of the study.

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