Revisión Bibliográfica

Neurocirurgical Assessment of Coma - Review

Coma en Neurocirugía - Revisión

Nunes N. [1], Nunes N. [2], Pereira C. [3], Moura C. [1], Alves G. [1], França F. [1], Alves G. [1], Rocha D. [4]. Delourence G. [5], Araujo L. [2]

RESUMEN:

El coma metabólico, es el más frecuente y su principal representante son las intoxicación exógenas. 1-2% de las consultas de pronto atendimiento son de paciente con cambio del nivel de consciencia. Describir la anatomía y fisiopatología del paciente en coma, evolución clínica del paciente comatoso y direccionar por una tabla y flujograma, los diagnósticos diferenciales posibles y tratamiento etiológico. Realizado una revisión de los bancos de dados: LILACS, Medline, PubMed, con las palabras claves: Escala de coma de Glasgow, coma post traumático da cabeza, coma, muerte cerebral. Fueron encontrados 39 trabajos para esta revisión, en 1981 hasta 2017. Tres estructuras básicas son las responsables por esa manutención de la vigilia y conocimiento: entre ellas: Sistema Reticular Activador Ascendiente (SRAA), los hemisferios cerebrales y lo sistema límbico. En el examen clínico evaluamos: 1) Conocimiento que es la validación más elemental de las funciones mentales superiores, también examinar cuanto el nivel y contenido del conocimiento. 2) El estándar respiratorio que reflete comprometimento neurológico son el Cheyne-Stokes y biot, este son un indicador indirecto de la validación. 3) Validación pupilar es por su forma, tamaño y reactividad pupilar y la 4) validación motora no se consiste en un bueno parámetro de validación de lo nivel y contenido del conocimiento. La importancia de la anamnesis y examen físico bien hechos, como la correcta definición de la etiología, lo adecuado tratamiento y inversión de lo coma en tiempo experto, sin dejar secuelas neurológicas.

Palabras claves: Trastorno de la conciencia, rehabilitación, coma, tratamiento del coma, neurocirugía.

1. Medical Student - Faculdade Atenas de Medicina; Paracatu, Minas Gerais, Brazil
2. Department of Neurosurgery - Hospital Santa Casa; Ribeirão Preto, São Paulo, Brazil
3. Department of Neurosurgery of FBHC and Neurosurgery Service, Aracaju, Sergipe, Brazil
4. Department of Cardiology and Internal Medicine - Faculdade Atenas; Paracatu, Minas Gerias, Brazil
5. Medical Student - FEMA - Fundação Educacional do Município de Assis; Assis, São Paulo

ABSTRACT:

The metabolic coma is the most prevalent and the exogenous toxic represent 1-2% in emergencies are by patients with level of consciousness dismissed. Describe the anatomic and pathophysiology of comatose patient, make the clinical exam and propose a flowchart and table directing the different possible diagnoses, and its etiologic treatment. A review of the data base LILACS, Medline and PubMed, with the keywords: Glasgow coma scale, coma post-head injury, coma and brain death. 39 reviews were selected, for this article, period of 1981-2017**.** Three basic structures are responsible for maintaining awake and consciousness: the ascending reticular activating system (ARAS), the cerebral hemisphere and the limbic system. In clinical exam it is important to evaluate: 1) The consciousness, that is the most elementary mental superior function, examining it by the level and content of consciousness. 2) The respiratory pattern that reflects indirectly on the neurologic damage. Two are important: Biot and Cheyne-Stokes. 3) Pupil exam by its form, size and reactivity. 4) The motor evaluation is not a good parameter to examine the level and content of consciousness. The importance of a well done physical exam and anamnesis, and the correct definition of the etiology of coma are the main concerns to the treatment and the potential reversible coma, on time, without leaving any neurologic damage.

Key Words: Disorder of consciousness, rehabilitation, coma, coma management, neurosurgery

INTRODUCCION

Coma is a state of unconsciousness characterized by a lack of arousal and awareness. The defining clinical feature is the complete loss of spontaneous or stimulus induced arousal. There is no eye opening, and EEG (electroencephalogram) testing reveals the absence of sleep-wake cycles. Structural lesions usually involve diffuse cortical or white matter damage, or a brainstem lesion. Those who survive this stage will begin to awaken and transition to a Vegetative State (VS), unresponsive wakefulness state (UWS) or Minimally Conscious State (MCS) within 2 to 4 weeks.[1]

Comatose patient management is divided into two major priorities. First, the cause should be elucidated, so that attempts can be made to reverse it. Second, these extremely vulnerable patients should be protected in every possible way. This includes appropriate airway management, adequate

oxygenation, and possibly mechanical ventilation. Other abnormal vital signs should also be corrected, such as blood pressure and temperature. Each of these simple tasks is essential in the initial hours of care. In patients brought to the emergency department, coma can be due to traumatic brain injury, cerebral or cerebellar hemorrhage, acute basilar artery embolus, anoxic-ischemic brain injury after cardiopulmonary resuscitation, and drug overdose. Once the cause of coma is established, management should proceed quickly. This is most pertinent in patients with an expanding mass lesion causing shift of the thalamus or brainstem, acute obstructive hydrocephalus, and central nervous system (CNS) infection he capacity of attention and vigil are the basics of our consciousness, of the environment and of ourselves. [2]

The capacity of attention and vigil are the basics of our consciousness, of the environment and of ourselves. Clinical Changes in consciousness and the coma state are clinical specters of the change in attention and vigil. Coma is defined, as a clinical syndrome characterized by a reduction of consciousness with many possible pathology causes. [2]

Some situations in which the level of attention is not committed, just the content, may confuse the diagnosis. These situations are: vegetative state, Acute confusional state, minimally aware states, akinetic mutism, abulia, captivity syndrome and psychogenic withdrawal. [1,2]

Coma is studied as part of a disease, a clinical syndrome, and not as a clinical sign. Because of that, its real prevalence is a challenge. The metabolic coma is the most prevalent and its mail representative is the exogenous intoxication. Yet, the structural one is represented mainly by Intracranial hemorrhages and subdural hematomas. In interned patients, 22% have delirium and 1-2% of queries in Emergency are by patients with changes in the level of consciousness. [1,2]

The objective of this paper is to describe the anatomy and the physiopathology of the comatose patient, from clinical evaluation of the comatose patient as well as to show a table and an algorithm with many differential diagnosis and their respective treatment.

METHOD

Review in data base was carried out: LILACS, Medline, PubMed, by the keywords: Glasgow Coma Scale, Head post traumatic coma, coma, brain death. 39 reviews were found between 1981 and 2017. Reviews about this subject were included and analytic articles were excluded.

DISCUSSION

Three basic structures are responsible for the maintenance of vigil and consciousness: (1) Ascending reticular activating system (ARAS), (2) the cerebral hemispheres and the (3) limbic system. The ARAS is responsible for the cortical activation and regulation of sleep cycle and vigil. The cortex, when

activated by ARAS, sends impulses to the cortex, and initiates the psychic phenomena command. This is the principal vigil formation mechanism, but when it is changed, it is showed by a coma, or by the changes in the level of consciousness, as evaluated by Glasgow Coma Scale (GCS). The GCS was originally developed to standardize the evaluation in cranioencerebral traumas, however it can be extrapolated by other clinical conditions to quantify the level of consciousness. [3,4,5]

ETIOLOGY

The physiopathology of the structural coma, which is less common, is by compression of structures, among which is the prototype of the consciousness state, the ARAS. There is the supratentorial ones, among which are: tumor, abscess, traumatic brain injury, ischemic stroke, hemorrhagic stroke, hydrocephalus and the infratentorial, represented by tumors, pontine mielynosis, occlusion the basilar artery. The diagnosis method is carried out mostly by a cranial tomography (CT) of the skull, and when this is not sufficient, angio CT, angio MR or arteriography are necessary.[4,5]

The most prevalent endocrine metabolite is a diffuse cerebral damage, represented by hypoglycemia, hyperglycemia, hyponatremia, hypernatremia, hypercalcemia, uremia, Hepatic encephalopathy, narcosis, hypothyroidism, Addison disease and hypopanhipotuitarism, after drugs, hypothermia, Inhalation of toxic gases (carbon monoxide) and Psychiatric causes (Table 1). [5]

differential diagnosis	anamnesis and clinic	exams and diagnosis	conduct
Intracranial hypertension/ hydrocephaly	reflex of cushing, papilledema, history of trauma, tumor and infection	CT - cranial tomography	operating, DVE, PIC, clinic according to evolution
AVCi	scale of Cincinnati, factors of risk for cardiovascular disease	cranial tomography (24-48 hrs) or NMR with diffusion(DWI) and dispersion (earlier phase)	If < 4,5 hrs and no contraindication: thrombosis. After, outpatient monitoring for secundary prevention.
AVCh	neck stiffness, the worsted headache of life, focal deficit, signal of intracranial hypertension	cranial tomography, angio CT, angio NMR,arteriography	surgical or clinic, fisher, hunt hess, deviation of midline line and localization of hip.
Infeccion (meningitis,sepsis, brain abscess)	fever, neck stiffness, seizure, HIV +	if the cranial tomography is normal, Liquor, serology and epidemiology	clinic or surgical according to the medical response or volume of abscess
Status epilepticus/ State of hardly epileptic not convulsive	some times the only presentation is decrease in the level of awareness	higher level of suspicion, EEG(electroencephalography),CT	clinic (sedation) or surgical, according to the pattern of EEG or CT
Metabolic	suspicion of specific case according to the clinic	sodium, potassium, magnesium, calcium, glucose, urea, creatinine,transaminase, hypoxia, hypocorticism, hyper/ Hypothyroidism, bilirubin, acidosis, toxicological, cardiac enzymes, coagulagram,heavy metals,carbon monoxide	treat the cause or replace the electrolytes
hypoglycemia/wernicke	every time check the glucose in the states of comatose	check dextro	replace the glucose and thiamine, alcoholism
tumor	focal signals, family history and epidemiology, intracranial hypertension	cranial tomography,NMR, biopsy, stereotaxic	chemotherapy, radiotherapy, surgery
subdural hematoma	elderly, alcoholic,trauma, use of anticoagulant, cortical atrophy. Think in different causes of dementia	CT- signal of crescent moon	surgical and clinical in some cases (use of statin)
extradural hematoma	trauma	TC with biconvex radiopaque image	hemostasis of medium meningeal arterial. Drain the hematoma
medication	opioid,benzodiazepinic, barbiturates,tricyclic, neuroleptico, aspirin, selective serotonin inhibitors, anticonvulsant drugs, acetaminophen	suspicion and clinical history	treat whit antagonist - flumazenil, naloxone, plasma, Acetylcysteine
others differentials	syndrome of heminegligencia , delirium, deep depression, dementia,acute psychosis, psychiatric disorder, persistent vegetative state(is one vigil whithout perception of the enviroment), Diffuse axonal injury (DAI)	exclusion of others causes	treat according to the cause

Table 1: Different diagnoses of patients in state of coma that could be treated or reverted

CLINICAL PRESENTATION

The physical exam of this patient had to be instructive for the validation of three aspects: Confirmation of mental alteration, state information that helped to localize neural dysfunction and that helped to elucidate the case of the disturb. [6]

The exam had to be standard and based on the examination of: 1- conscience, 2- respiratory breath, 3- exam of the eyes (pupil and external factor in ocular motricity), 4- motor response. [6,7]

1. *In the consciousness exam:* Superior mental function evaluation. Evaluate the level in the substance of consciousness. The level of conscious reflect upon the reduction or no feedback of awake reaction. Go through the spectrum of contusion, delirium, lethargy, stupor and coma. Another way of evaluating is through the quantity thought in the ECG (Glasgow Scale of Coma). Although the scale of coma of Jouvet is more complete than the Glasgow, it is more complex and could entail mistakes in their application. The substance of the conscience is referent to the appropriate patient relationship with the external environment, and elucidates many complex

mental functions like attention, memory, language and others. The vegetative state is one example of lack of consciousness, while awake. And because of that, there had to be attention in the evaluation of disturbs in conscience, because of the aphasia and the syndrome of the confinement, that characterizes motor disturbs with damage in the quality of consciousness, but not in the level. Pay attention to this type of evaluation in order to not entail in wrong diagnoses on the level of conscious. [7,8,9]

2. *The breath:* Is one integrated act for nerve influence of almost all the encephalon. Thus, breathing is one indirect evaluation of their functions, in spite of the low specifies. It could be influenced by the neural gene and metabolic. The epileptics breath inhibition, in the pos ictal period, in which the patient had moments of auto-limited apnea. The central neurogenic breath in which PaO_2 is necessarily normal or higher, occurs in mesencephalic lesions, cerebellar hemispheres and bulb. The apnea breath had long inspired spasm followed with total apnea in the brain bridge lesions, hypoglycemia, lack of cerebral oxygen, meningitis. The Cheyne-Stokes breaths, where periods of hyperpnea are altered with hypopnea, occur with diffuse involvement of the brain, increased intracranial pressure or cardiopulmonary impairment. These breaths increase in depth and volume to a peak and decline until there is a period of apnea. Associated respiratory rate changes are respiratory ataxia in which the respiratory pattern is irregular, with superficial and deep erratic movements. This respiration exists through dysfunction of the medullary respiratory centers and may indicate impending agonizing breaths and apnea. The principal cases are: metabolic disturb, bilateral cerebral infarction, hypertensive encephalopathy, imminent transtentorial herniation, cardiac insufficiency and shock. Central neurogenic hyperventilation is characterized by prolonged, rapid and regular hyperpnea. It is associated with diseases that affect the paramedian reticular formation in the mesencefalo inferior and in the superior bridge. Apnea, a rare form of respiratory rhythm dysfunction, develops with a prolonged respiratory phase, occurs in lesions on the bridge rostrally to the trigeminal nerve motor nucleus or in the cervicomedullary compression. Systemic diseases also cause changes in the respiratory pattern, such as diabetic

ketoacidosis or severe myxedema [8,9]

3. The pupils were controlled by the nucleus of brainstem (sympathetic and parasympathetic), when they are intact and realize the act of dilatation and constriction respectively. Because of these alterations, it is frequent in comatose patients. The form of evaluation is through form, size and reactive pupil. It is important to observe that the evaluation observes the ocular motor that reflects the integrity of this area that involves the consciousness. The unilateral alterations, in most cases, are from structural cases and symmetric from metabolic cases. Some of these reflexes could be searched thought the evaluate of the integrity of brainstem like the reflex of eyelid, corneal reflex, oculo-cephalic reflex, oculo-vestibular reflex. These are the most used in the protocols of encephalic dead. The alteration that had to be evaluated in the urgent care is the anisocoric with the photomotor reflex typic of aneurism of posterior communicant artery, for the relationship with the pair cranial III. [9] The main coma pupil changes are: (1) Oculomotor paralysis: Intracranial compression of one of the nerves of the third cranial nerve, contusion of the ocular glob. (2) Mydriasis: Anxiety, delirium, pain, seizure, botulism, atropine, hypermagnesemia, sympathomimetic drugs. Horner's syndrome: Traumatic carotid dissection, brachial plexus injury, internal jugular vein catheterization. (3) Myositis: Opiates, metabolic encephalopathies, pontine lesions, hypercapnia. (4)Medium-fixed pupils typical of encephalic death.[10]

4. The motor answer is separate from the areas that regulate the mental state, therefore, the evaluation does not even have relation with the depth of the coma, in other words, they are disproportionate. Not based in have a good baseline of evaluate in the level and conscience substance. The posture of decerebration and decortication refers to a lesion in the mainstream and could become one form of evaluation oriented for to coma. The movements like asterix suggest uremic encephalopathy, hypoglycemia. The evaluation of the signal in Babinski is of the extension of 1 year old before the reflection, if it is not normal, reflect the lesions of the pyramidal structure. [11]

Cerebral herniations

Inside the skull, which is a rigid sphere, are present the brain, blood and

cerebrospinal fluid (CSF). The volume formed by them is constant. When there is a mass or lesion that forms an increase volume within the cranial cavity, the outflow of liquor initially occurs in order to maintain this volume constant. It is worth remembering that in this case a pressure gradient occurs between the two hemispheres and between the posterior fossa and the portion above the cerebellum tent. As the lesion expands, more cerebrospinal fluid outflow and in some cases decreased blood volume. When there are alterations of the intercompartmental volume inside the skull there is a decoupling of encephalic tissue to the lower pressure compartment causing the hernias. [12]

The most important hernias are central transtentorial hernia and lateral hernia. The central hernia is seen in cases where the diencephalon is pushed down the cerebellum tent. Initially the patient evolved with sleepiness and Cheyne-Stokes breathing. From then on bilateral myosis occurs, but photoreagents, sometimes paratonia and also motor response in flexion (decortication). The next step of herniation is called mesencephalic. Here the pupils are medium fixed, with abduction deficit of the eyes and extension posture (decerebration). When the bridge compromises the eye movements are abolished, the respiratory rhythm becomes irregular. When the bulb is compressed the pupils are medium-fixed, with the patient in apnea and absence of motor reaction. The diencephalic phase is important, because at this time it is still possible to reverse the process.[13]

Lateral herniation occurs when the medial portion of the temporal lobe, that is, the uncus hints between the free edge of the tentorium of the cerebellum and the midbrain. In this case the lesions are in the temporal lobe or are extra-axial. Initially, there is compression of the opposite cerebral peduncle to the side injured side. From this point, the evolution is the same as that of the central.[14]

Minimally Conscious State (MCS)

The MCS is characterized by a severe impairment of consciousness, with evidence of wakefulness and partial preservation of awareness. Unlike the VS, there are discernible, purposeful behaviors that can be differentiated from reflexive behavior. The hallmark of MCS is inconsistent but reproducible, command following. The preservation of cortico thalamic connections might explain why patients in MCS retain the capacity for cognitive processing. The patient may exhibit visual pursuit, emotional responses, and gestures to appropriate environmental stimuli, but are unable to functionally communicate their thoughts or feelings.[15]

Acute Confusional State

Once emerged from the MCS, patients continue to experience a transient period of disorientation and agitation. The full array of symptoms associated with the acute confusional state can also include irritability, distractibility, anterograde amnesia,

restlessness, emotional lability, impaired perception, attentional abnormalities, and a disrupted sleep-wake cycle. A key pattern to this state is the day-to-day fluctuation of behavioral responses. The return of behavioral consistency despite situational stresses may indicate a resolution of this period. **16,17**

The FOUR score

To address the shortcomings of the GCS, the Full Outline of UnResponsiveness (FOUR) score was developed (**Table 2)**. To simplify scoring, the FOUR score includes four components – eyes, motor, brainstem, and respiratory – that are each graded on a scale from 0 to 4, producing a maximum combined total score of 16. With an equal to higher interrater reliability than the GCS, as well as validation in multiple patient populations, the FOUR score has proven to be an important initial tool in the evaluation of comatose patients. Perhaps because of its greater emphasis on brainstem reflexes and respiratory patterns, the FOUR score has also been shown to have greater predictive value in terms of eventual progression towards more severe injury, especially in patients with low GCS scores, or the relatively ubiquitous GCS score of 3 T, which is commonly reported by paramedics following intubation in the field after sedatives and paralytics have been given. Brainstem damage and failure to maintain adequate ventilation are reflections of injury severity. The FOUR score does not contain a verbal component, and can be measured with equal accuracy in intubated and non-intubated ICU patients. The FOUR score can be used to evaluate for clinical progression with serial examinations in patients with intracranial mass lesions. **16,17,18,19**

Table 2: FOUR score scale

Eye response
4 eyelids open or opened, tracking, or blinking to command 3 eyelids open but not tracking
2 eyelids closed but open to loud voice
1 eyelids closed but open to pain
0 eyelids remain closed with pain
Motor response
4 thumbs-up, fist, or peace sign
3 localizing to pain
2 flexion response to pain
1 extension response to pain
0 no response to pain or generalized myoclonus status
Brainstem reflexes
4 pupil and corneal reflexes present
3 one pupil wide and fixed
2 pupil or corneal reflexes absent
1 pupil and corneal reflexes absent
0 absent pupil, corneal, and cough reflex
Respiration
4 not intubated, regular breathing pattern
3 not intubated, Cheyne–Stokes breathing pattern 2 not intubated, irregular breathing
1 breathes above ventilator rate
0 breathes at ventilator rate or apnea

Delirium

It is an acute state and floating of the deficit of attention, disorganized thinking, alteration in the state of awake. The drowsiness could be present in alteration with the nocturnal

agitation. The fast identification of delirium is fundamental, because it could pronounce the neural dysfunction more seriously, that could be early treated and reverted. It occurs in 10 up 50% of hospitalized patients. Some factors of risk are advanced age, sensorial dysfunction, drugs, dangerous basic condition, dehydration, immobility, dementia. The CAM-ICU (confusion assessment method in an intensive care until) is used for diagnoses in critical patients. [20,21,22,23,24]

Vegetative State(VS)/Unresponsive Wakefulness State

The VS is thought of as an unconscious, dissociative state of wakefulness without awareness. The patient's eyes open spontaneously, and EEG testing reveals the presence of sleep-wake cycles. Patients may arouse by provocation or external estimulation, but they show no signs of conscious perception or deliberate action. Interestingly, these patients may perform stereotyped gestural movements such as yawning, chewing, crying, smiling, or moaning, but these are unrelated to context. The presence of wakefulness suggests preserved brainstem functioning, but the lack of awareness suggests an underlying cortical dysfunction. Likewise, functional neuroimaging has shown sensory estimulation will activate primary cortical areas, but not the higher order cortical areas thought necessary for awareness. With proper medical care, a patient in a VS can survive for many years. [25,26,27,28]

Management

Adequate management of eye, mouth, and skin at compression sites requires frequent change of linens. Every day, infections may present, skin may break down, and fluid shifts may cause rapid imbalance of homeostasis. Drugs (particularly antibiotics) have potentia adverse effects. [27,28]

Adequate management of eye, mouth, and skin at compression sites requires frequent change of linens, patches, and protective pads. Splinting of extremities may be needed to avoid contractures. Inability to close eyelids completely after trauma and, in particular, nocturnal lagophthalmos are risk factors for conjunctivitis and corneal erosion. Polyethylene moisture chambers are required to prevent early epithelial breakdown. Filamentary keratopathy is a common dry-eye syndrome in patients in prolonged coma. Prolonged eyelid contact with the cornea and reduced blinking impair lacrimal fluid turnover and may be contributing factors.[29,30]

Tracheostomy reduces pulmonary complications and provides easier access for pulmonary secretion. Tracheostomy will reduce length of stay, but it should generally be postponed until approximately 2 weeks in patients the most common healthcare-related infections are pneumonia, urinary tract infections, or infections of indwelling venous catheters. Potentially difficult to eradicate microorganisms include *Enterococcus faecalis* or *faecium, Staphylococcus aureus*, *Klebsiella pneumoniae, Acinetobacter baumannii,*

Pseudomonas aeruginosa, and *Enterobacter cloacae* . Clostridium difficile infections are also on the rise, particularly in patients with long hospital stays. The dilemma faced by treating physicians is that delayed initiation of antibiotics increases mortality, yet combination therapies to broaden the spectrum may lead to antibiotic resistance. Antibiotic therapy is changing as a result of infectious disease consultation. [31]

Fever in comatose patients is mostly caused by infections. Lingering infections have to be excluded before attributing fever to the brain injury. However, Paroxysmal sympathetic hyperactivity (PSH) syndrome is a commonly seen in " unexplained" fever of comatose patients. PSH or dysautonomic storming all too frequently remains unrecognized and untreated. These spells are most common in young patients with diffuse axonal traumatic brain injury, but can occur with any major brain injury. Episodes of PSH can begin during the acute phase, often in comatose patients, and continue into the rehabilitation phase. Patients become tachycardic, hypertensive (with widened pulse pressure), tachypneic, febrile, diaphoretic, and often develop markedly increased tone, which may result in dystonic posturing. [32]

Pupillary dilatation, piloerection, and skin flushing can also be seen. The manifestations of PSH respond best to bolus doses of morphine sulfate (2– 8 mg intravenously). This favorable response is not related to the analgesic effect of opiates, but rather to modulation of central pathways that are responsible for the autonomic dysfunction. The response to morphine is rapid and quite reliable in aborting spells of PSH. Other effective medications for the treatment of PSH include non cardioselective beta-blockers (such as propranolol), clonidine, and dexmedetomidine (central alpha 2-receptor agonists), bromocriptine (a dopamine D2-receptor agonist), baclofen (GABA B receptor agonist), benzodiazepines (GABA A receptor agonist), and gabapentin (which binds GABA receptors and voltage-gated calcium channels in the dorsal horn of the spinal cord). In our experience, beta-blockers and clonidine are useful in controlling the tachycardia and hypertension, but less so for the dystonia. Baclofen and benzodiazepines (especially diazepam) do cause muscle relaxation, but may not improve the other hypersympathetic features, who can potentially be liberated from the ventilator if they show early signs of substantial neurologic improvement. [33,34]

Gradually, after the patient is weaned off the ventilator, the tracheostomy can be closed, including in patients with prolonged unconsciousness. Pulmonary care involves frequent culturing of sputum when secretions change in color and texture and immediate antibiotic coverage to treat pneumonia and sepsis. Pleural effusions are frequent as a manifestation of anasarca and large pleural collections may need to be

drained. Gastrointestinal problems vary from gastroparesis to paralytic ileus, resulting in distension of the colon and increased risk of perforation. Daily bowel care may include motility agents. [35]

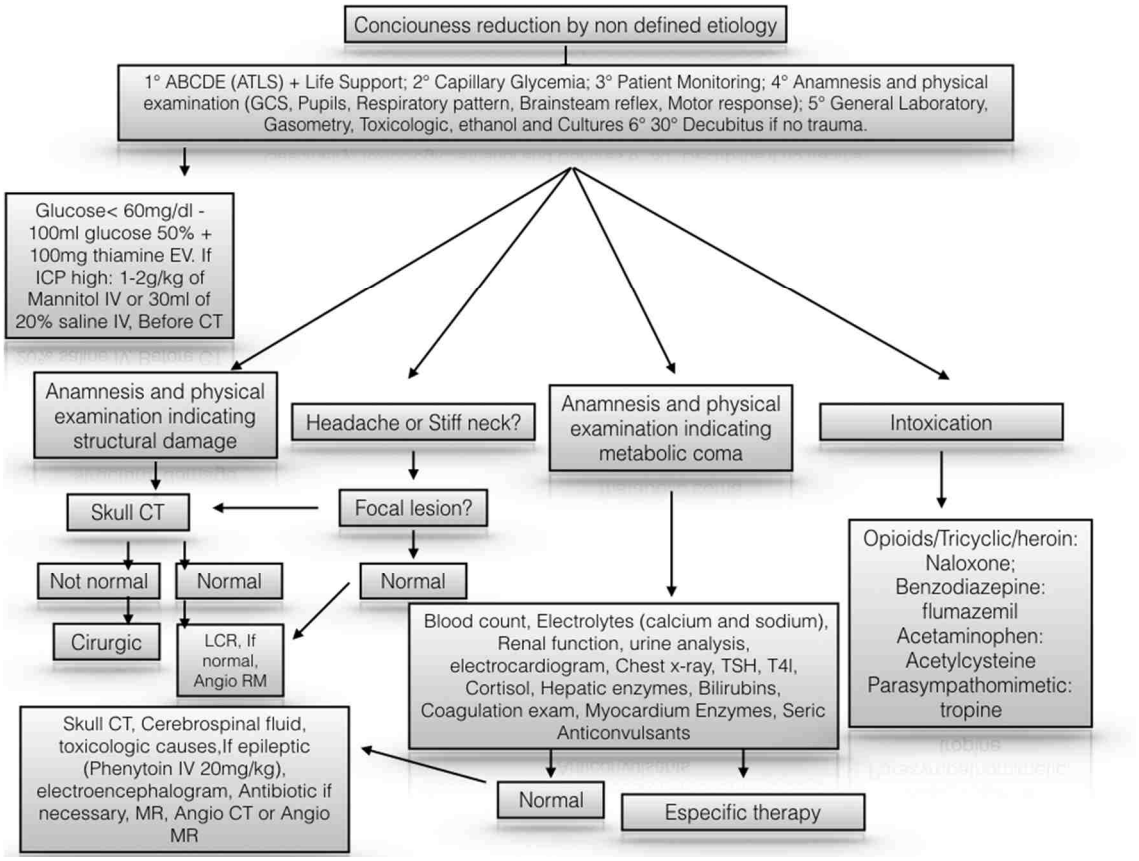
Continuous volume replacement is needed for longtime care. The adequate intravascular status is determined by satisfactory organ perfusion (urinary output, capillary refill, cold or warm extremities, blood lactate, and mixed venous oxygen saturation). Tissue edema may form over time, possibly as a result of overzealous, percutaneous gastrostomy (PEG) and Fluid administration (e.g., failure to adjust intravenous fluid rate while advancing enteral nutrition, failure to concentrate medications). Volume depletion is less common in the longterm but may occur, especially when extravascular compartment is expanded by sepsis. Hypotonic crystalloids, such as lactated Ringer' s or half normal saline, should be avoided in traumatic brain injury. Albumin (5%) is a good volume expander, and may have a role in sepsis resuscitation, but the safety in acute brain injury is unclear and may be deleterious in traumatic brain injury. In patients who have developed oliguria and a rise in BUN (BUN/creatinine ratio > 20), dehydration is very likely and should result in discontinuation of all diuretics and administration of normal saline. Nutrition is eventually provided through a PEG , which is very safe. Complications include wound infection, leakage, peritonitis, self-extubation or hemorrhage in the first weeks of placement, only in 2%. The risk of gastrointestinal hemorrhage may be increased. Compared with nasogastric tubes, gastroesophageal reflux is lower in patients with a PEG. [36,37]

A bowel care regimen should be initiated. Bowel incontinence is often present, and the task is to keep the skin clean and dry. Diarrhea may have many causes, but can be attributed to certain nutritional formulas and resolve with reducing fiber content. Antibiotics, as well as *Escherichia coli* or Clostridium difficile infections, are other possible causes of diarrhea. Failure to pass stool, or marble-like stools, should be treated with rectal enema or manual removal. Glycerol suppository can be helpful, but senna (10 mL) and lactulose (20 mL) are common maintenance therapies. Comatose patients are at risk of adynamic ileus. Metoclopramide (10 mg IV) or erythromycin (500 mg orally) can be very effective to resolve the bowel distension. Nosocomial urinary tract infections will likely occur in comatose patients with long-term indwelling catheters. [38]

Daily Check List in care of the comatose patient [39]

- *Lungs :* Mechanical ventilation settings, Weaning option, Tracheostomy care, Chest X-ray for infiltrates
- *Heart:* Cardiac arrhythmias, Electrocardiogram changes(i.e., QT prolongation), Inotropes/vasopressors/beta blockade

- *Gastrointestinal:* Oropharyngeal hygiene, Nutrition and choice of formula, Targets glucose/insulin drips, Bowel motility assistance, Bladder , Indwelling catheter, Urine analysis
- *Skin:* Decubitus, Conjunctiva/eye care
- *Prophylaxis:* Unfractionated heparin, Surveillance ultrasound of venous system, Gastrointestinal prophylaxis, Fever control
- *Access:* Peripheral catheter, Peripherally inserted central catheter, Subclavian, Peripheral intravenous
- *Medication:* Medication reconciliation, Antibiotic stop dates, Drug–drug interaction, Sedation/analgesia needs



Algorithm: This Algorithm's purpose is a faster and objective evaluation, bringing the immediate answer about the necessities or not of investigation and conduct to be taken

CONCLUSION

In most of the cases, 40% of coma is associated with brain lesions that could have a favorable rehabilitation. In contrast, in the post parade coma, 89% will die or will have some sequel. The metabolically etiologic coma presents one prognostic better than the structural origins.

In conclusion, there is a great importance in the well-done anamneses

and physical exam, as well as the correct definition of etiology, for the appropriate treatment or reversion of the coma in an adequate time, without neurological sequel. This chapter suggests a flowchart that could diagnose and treat the more prevailing causes, in a way as to minimize the neurological loss of patients that present a decrease in awake and in consciousness.

REFERENCIAS:

1. Feske SK. Coma and confusional states: emergency diagnosis and management. Neurol Clin 16:237-256,1998.
2. Zeman A: Consciousness. Brain 2001; 124:v1263–1289
3. Steriade M. Arousal: revisiting the reticular activating system. Science. 1996;272:225.
4. Young GB, Pigott SE: Neurobiological basis of consciousness. Arch Neurol 1999; 56: 153–157
5. Benseñor IM, Atta JA, Matins MA. Semiologia clínica. Caramelli P. Distúrbios da consciência. 1° ed. São Paulo: Sarvier, 2002, pg-446-450.
6. Sturmann K. The neurologic examination. Emerg Med Clin North Am. 1997;15:491-506
7. Radvany J. Ferraz AC. Estados Confusionais no paciente Grave. In: Knobel E. - Condutas no Paciente Grave. 2° ed. São Paulo, Editora Atheneu, 1998. p.617-629
8. North JB, Jennet B. Abnormal breathing patterns associated with acute brain injury. Arch Neurol. 1974;32:338
9. Dandan IS. Altered Consciousness. Top Emerg Med. 2004; 11:242-253
10. Masdeu JC. The Localization of Lesions Causing Coma. In: Brazis PW, Masdeu JC, Biller J. Localization in Clinical Neurology. 2° edition. Little, Brown and Company, Boston. 1990.p.457-485.
11. Bruno M-A, Vanhaudenhuyse A, Thibaut A, et al. From unresponsive wakefulness to minimally conscious PLUS and functional locked-in syndromes: recent advances in our understanding of disorders of consciousness. J Neurol 2011; 258(7):1373–84.
12. Sherer M, Nakase-Thompson R, Yablon SA, et al. Multidimensional assessment of acute confusion after traumatic brain injury. Arch Phys Med Rehabil 2005; 86(5): 896–904.
13. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. Lancet 1974; 2:81–84
14. Ely EW, Inouye SK, Bernard GR et al. Delirium in mechanically ventilated patients: validity and reliability of the confusion assessment method for the intensive care unit (CAM-ICU). Jama, 2001; 286: 2703-2710.
15. Robert D, Stevens, MD. Anish Bhardwaj, MD, FCCM Approach to the comatose patient Crit Care Med 2006 Vol. 34, No. 1
16. Levy DE, Bates D, Caronna JJ, Carlidge NE, et. al. Prognosis in nontraumatic coma. Ann Intern Med. 1981, 94 293-301
17. Young GB, Wang JT, Connolly JF: Prognostic determination in anoxic-ischemic and traumatic encephalopathies. J Clin Neurophysiol 2004; 21:379–390
18. Wijdicks EFM, Varelas PN, Gronseth GS et al. (2010). American Academy of N. Evidence-based guideline update: determining brain death in adults: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 74: 1911–1918.
19. Wijdicks EFM (2010). The bare essentials: coma. Pract Neurol 10: 51–60.
20. Moore SA, Wijdicks EFM (2013). The acutely comatose patient: clinical approach and diagnosis. Semin Neurol 33: 110–120.
21. Stavale M. Bases da Terapia Intensiva Neurológica: Fisiopatologia e Princípios terapêuticos, 2edição, Editora Santos
22. Greer DM,Young J, Scripko PD. Clinical Examination for Outcome Prediction in Nontraumatic Coma. Crit Care Med, 40:1150-1156, 2012

23. Forsberg S, Hojer J. Metabolic vs Structural Coma In The ED: An Observational Study. American Journal of Emergency Medicine, 30:1986-1990, 2012
24. Cavanna AE, Shah S, Eddy CM, et al. Consciousness: a neurological perspective. Behav Neurol 2011;24(1):107–16.
25. Giacino JT, Fins JJ, Laureys S, et al. Disorders of consciousness after acquired brain injury: the state of the science. Nat Rev Neurol 2014;10(2):99–114.
26. Edlow JA, Rabinstein A, Traub SJ et al. (2014). Diagnosis of reversible causes of coma. Lancet 384: 2064–2076.
27. Eapen BC, Georgekutty J, Subbarao B. Disorders of Consciousness.Phys Med Rehabil Clin N Am - (2017). http://dx.doi.org/10.1016/j.pmr.2016.12.003
28. Wijdicks EFM (2001). The diagnosis of brain death. N Engl J Med 344: 1215–1221.
29. Wijdicks EFM. The bare essentials: coma. Pract Neurol 2010; 10: 51–60.
30. Wijdicks EFM, Varelas PN, Gronseth GS et al. American Academy of N. Evidence-based guideline update: determining brain death in adults: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2010; 74: 1911–1918.
31. Laureys S, Owen AM, Schiff ND. Brain function in coma, vegetative state, and related disorders. Lancet Neurol 2004;3(9):537–46.
32. Wijdicks EFM (2016). Why you may need a neurologist to see a comatose patient in the ICU Crit Care 2016; 20: 193.
33. Wijdicks EFM (2017). Brain Death 3ed Oxford University Press.
34. Lavrijsen J, vaqn Rens G, van den Bosch H. Filamentary keratopathy as a chronic problem in the long-term care of patients in a vegetative state. Cornea 2005;24: 620–622.
35. Giacino JT, Ashwal S, Childs N, et al. The minimally conscious state: definition and diagnostic criteria. Neurology 2002;58(3):349–53..
36. Mijalski C, Lovett A, Mahajan R et al. Cerebral fat embolism: a case of rapid-onset coma. Stroke 2015; 46: 251–253.
37. Moore SA, Wijdicks EFM. The acutely comatose patient: clinical approach and diagnosis. Semin Neurol 2013; 33: 110–120.
38. Salottolo K, Carrick M, Levy AS et al. Aggressive operative neurosurgical management in patients with extra-axial mass lesion and Glasgow Coma Scale of 3 is associated with survival benefit: a propensity matched analysis. Injury 2016; 47: 70–76.
39. Stevens RD, Cadena RS, Pineda J. Emergency neurological life support: approach to the patient with coma. Neurocrit Care 2015; 23: 69–75.

Correspondencia:

Neiffer Nunes Rabelo
Email: neifferr@gmail.com

Recibido : 13/06/17

Aprobado : 09/07/17

Conflicto de intereses : Los autores declaran no presentar conflicto de intereses