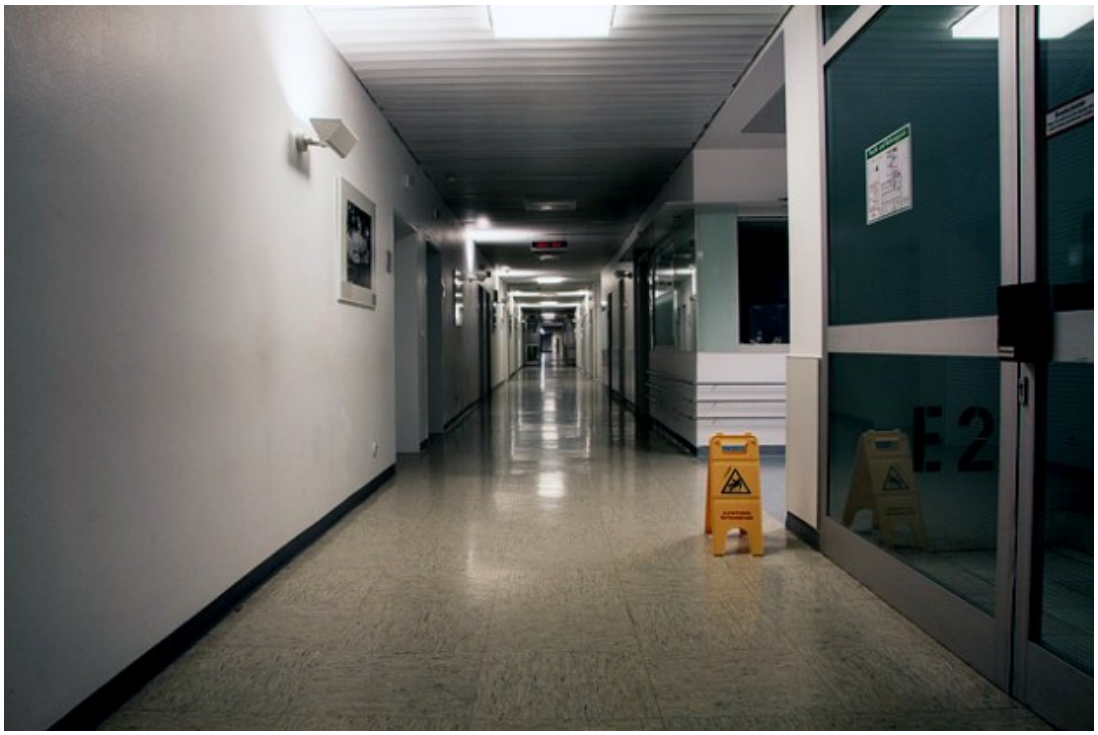
Coma — Definition and Types

See online here

Coma is a life-threatening process that often requires immediate recognition, appropriate diagnosis of the cause, and proper initial management. The causes of coma can be divided into structural versus causes of diffuse neuronal dysfunction. Causes of diffuse neuronal dysfunction can be further classified into toxic and metabolic. The definitive treatment of patients with coma needs to be disease-specific.



Beyond the Consciousness

Different areas of the nervous system jointly control the degree of consciousness

Alertness/Vigilance: ARAS region (activating the reticular ascending system in the formatio reticularis)

Awareness: Thalamus in the diencephalon

Self-revelation: Default mode network (DMN, with island involvement, singular cortex, and prefrontal cortex)

Self-location/self-perception: Seat in the parietal lobe

If the nerve cells of these centers do not communicate, or if the connecting nerve pathways are damaged, there is misregulation of the consciousness.

Falling Into Coma

According to the ancient Greeks, a coma is nothing more than ‘deep sleep’. The medical practitioner recognizes the coma to include the closed eyes, the impossibility of arousing the patient, and the fact that he does not react to pain. Coma can have a pathological cause or can be artificially initiated. **Any form of coma is life-threatening.** Also, there is no uniform concept for this syndrome.

The causes of ‘real’ coma in the context of morbid disorders are manifold. All impairments of the vital functions are possible. If the causes are not obvious, one should think of injuries or diseases of the brain and consider poisoning or metabolic disorders.

Causes: Any Cause of Diffuse Neuronal Dysfunction		
Structural	**Toxic**	**Metabolic**
• Stroke • Brain tumor • Intracranial hemorrhage • Intracerebral hemorrhage • Hydrocephalus	• Intoxication • Anesthetics • Dissociative agents • Carbon monoxide poisoning • Toxic alcohols • Antidepressants • Antiepileptics • Asphyxia • Serotonin syndrome • Neuroleptic malignant syndrome • Clonidine	• Respiratory failure • Dysthermia • Dysglycemia • Infection including meningitis and encephalitis • Hypothyroidism • Thiamine deficiency • Non-convulsive status epilepticus • Electrolyte disturbances

Coma mimics can present with a picture that is similar to coma however the patient will not be in a ‘real’ coma. They include:

- Locked-in syndrome
- Neuromuscular paralysis
- Akinetic mutism
- Psychogenic unresponsiveness

Although the search for causes is important, one should 1st and foremost take the deep unconsciousness seriously. Coma threatens the body due to the failure of important protective reflexes and the lack of regulation of the vital functions. The 1st step to take when one encounters a coma patient is, therefore—always—to **secure and keep the airways patent as well as to control the circulation**.

Estimating the Depth and Stages of the Coma

Consciousness is not a light switch, which can be turned on and off. Rather, it is like a dimmer, over which the brightness is infinitely adjustable. Before people fall into a coma, the **phases of somnolence, marked drowsiness, and the sopor usually precedes the condition**. In the latter, the patients are only aroused by very strong stimuli.

To be able to assess how deep a patient has fallen into a coma, tests are available for pain reactions. **The pressure on the fingernail is a common method and can suffice for a coma assessment:**

Grade 1: immediate and directed response

Grade 2: delayed but directed response

Grade 3: no targeted reaction

Grade 4: no reaction

The Glasgow coma scale

The use of the **Glasgow coma scale**, which was already developed in the 1970s, continues to be established in the clinical world.

It is easy to calculate that the point sums must lie between 3 and 15. A number below 8 points indicates a severe brain disorder that makes intubation necessary.

Points	Best Eye-Opening	Best Verbal	Best Motor	
6	—	—	Obeys command	**Cognition ↑ Level of Consciousness**
5	—	Oriented	Localizes pain	
4	Spontaneous	Confused	Withdraws to pain	
3	To speech	Inappropriate	Decorticate posturing	
2	To pain	Incomprehensible	Decerebrate posturing	
1	None	None	None	

Types of Coma

Artificial coma

An artificial coma is quite different. Rather, it corresponds to **long anesthesia, in which a patient is placed to protect himself**. The artificial coma involves 2 components – sedation by narcotic agents and analgesia by painkillers. If both components are used, 1 speaks of **analgosedation**.

After severe brain damage, the artificial coma should prevent the neuronal activity and the intracranial pressure from increasing, and the nerve cells from dying. The blood pressure is lowered. The metabolism is lowered. Also, anxiety and emotional stress situations can be interrupted, which would severely impair the success of the treatment.

The duration of an artificial coma should be kept as short as possible since it can lead to side effects:

- Cardiac and respiratory disorders
- Gastrointestinal disorders
- Drug dependence
- Disorders of the immune system
- The danger of infection, e.g., lung infection
- Coagulation disorders, e.g., thrombosis

Nevertheless, the duration of an artificial coma does not tell us about the state of mind after awakening. It is, therefore, conceivable that a long time is surmounted in an artificial coma without neurological impairments.

Examination of the Comatose Patient

- Say/yell the patient’s name (verbal stimulation)
- Shake/tap patient (tactile stimulation)
- Press on patient’s nail beds (peripheral painful stimulation)
- Sternal rub/nasal swab (central painful stimulation)

If these tests fail to reveal a response, your patient is possibly in a coma. The brainstem exam is done to then determine if brain death is present.

Brainstem reflexes

Pupillary light reflex

- Afferent: CN II
- Efferent: CN III (parasympathetic)

Oculocephalic reflex (doll's eyes)/cold

- Afferent: CN VIII
- Efferent: CN III, IV, VI

Corneal reflex

- Afferent: CN V (ophthalmic division)
- Efferent: CN VII

Gag reflex

- Afferent: CN IX
- Efferent: CN X

Cold caloric reflexes

- 50 mL of ice water injected into the ear
- Cold is an inhibitory stimulus to the vestibular nerve.
- The vestibular nerve projects to the contralateral CN VI nucleus.
- The normal response is a slow deviation of eyes toward the side of cold water, then fast return to midline (nystagmus).
- In a coma, if CN III/IV/VI/VIII pathways are intact, the eyes tonically deviate toward the cold stimulus with no fast phase.
- **Remember: the primitive brain looks to stimulus, the higher brain brings eyes to the midline.**

By now, it might become clear to you that the Glasgow coma scale is somewhat limited in assessing a comatose patient. It does not take into account the brainstem reflexes and respiration abnormalities which can give important clues about the severity and even cause of coma. The full outline of the unresponsiveness (FOUR) score was developed to address these limitations.

Points	Eye response	Motor response	Brainstem reflexes	Respiration
4	Open spontaneously or blink to command	Can make a thumbs up, peace, or fist 'responds to the command'	Pupil and corneal reflexes present	Not intubated, regular pattern
3	Open spontaneously but does not track your fingers 'does not respond to the command'	Localizes to pain	1 pupil wide and fixed	Not intubated, Cheyne-Stokes pattern
2	Open to loud voices	Flexion response to pain	Pupil or corneal reflexes absent	Not intubated, irregular breathing

1	Open to pain	Extension response to pain	Pupil and corneal reflexes absent	Breathes above the ventilator rate
0	Closed with pain	No response to pain	Absent pupil, corneal and cough reflexes	Breathes at ventilator rate/apnea

Time of Awakening

After severe brain damage, the patient may awaken from the coma, but his cerebrum cannot be activated or can no longer be activated. Vegetative control is essentially functional. But for the environment, the **consciousness processes seem to be reduced to a minimum**. This ‘waking coma’ can last for months or a whole lifetime. The so-called **apallic syndrome** describes this condition, in which the brain stem, the interbrain, and the spinal cord maintain the life of a person, while the higher mental processes are no longer present.

In his book ‘Time of Awakening’, the neurologist Oliver Sacks describes how he was able to wake up patients for a short time from the waking coma. After a period of apparently incomplete apathy, the patients were beginning to feel joy and ideas. This is hardly imaginable with a complete failure of the cerebrum. The real apallic syndrome is, therefore, difficult to diagnose in practice. No one can know whether the cerebrum of these patients has actually failed—or whether it simply does not communicate with the outside world.

References

Glasgow Coma Scale

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Notes