COMA, DEFINITIONS AND DIFFERENTIAL DIAGNOSES

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Introduction

Coma is a medical emergency and as such, no discussion, even one essentially confined to definition and etiology, can occur without appreciating the complexity of the task facing the clinician. Whilst attending to the support of vital functions, the clinician will endeavor to answer the following questions:

1. What is the level of functional impairment (the extent of the lesion's effect)?
2. What is the rate of progression?
3. What is the likely pathological process?
4. Is there an immediately treatable cause?

Coma in children, particularly the infant, has added complexity introduced by the developmental stage of the child. Alterations in consciousness leading to coma are only noticeable when the behavioral responses vary from those a carer has come to expect. Where the repertoire of behaviors is limited to the simple reflex responses of the very young, which can remain almost unchanged until coma supervenes, the early recognition of alterations in conscious state requires both experience and frequent observation. It is not surprising, therefore, that conditions that in an adult may be recognized early, appear in the child to present late and often in an immediately life-threatening manner. Early recognition, prompt management, and intervention are infinitely preferable to resuscitating the moribund.

Regardless of age, coma can be produced only by processes that either depress cortical function bilaterally or directly impinge on the brainstem reticular activating formation. Broadly categorized, coma is caused either by a metabolic insult of sufficient severity to create diffuse, bilateral cortical dysfunction or by structural lesions directly affecting the reticular activating system. It is expected that the reader will be familiar with the definition of coma. Aspects of particular relevance in this article are those that help appreciate the nuances required to see the pediatric perspective.

Definition

Coma is a term used to describe the point in a process of evolving unconsciousness at which the sufferer is no longer capable of a purposeful response to the most vigorous of stimuli. It is, therefore, a behavioral description rather than a single clinical state and represents a state anywhere between the unimpaired to the completely unresponsive.

A variety of descriptions of the intermediate states of responsiveness are possible. "Lethargy" is often used to describe the state of diminished arousal that is responsive to a light stimulus such as voice. "Obtundation" is typically used to describe the poorly sustained but purposeful arousal elicited by mild stimulus (e.g., touch), whereas "stupor" requires vigorous (e.g., painful) stimuli for the elicitation of a purposeful response. Coma supervenes when it is no longer possible to elicit a purposeful response. This, of course, does not mean that no response is elicitable. A comatose patient may exhibit a range of reflex nonpurposeful responses of diminishing complexity culminating in a total absence of response to even the most intense of painful stimuli.

This complexity of stimulus/response is captured in the Glasgow Coma Scale (GCS). Though initially developed as a means to objectify the altered states of consciousness caused by head trauma, it can serve as a useful tool in nontraumatic coma. It also allows one to rephrase the definition of coma more specifically – coma requires the GCS to be <9. The obvious limitations of this scale for children have led to many attempts at modification, particularly of the verbal component (see Table 1).

It can be readily seen that for the purposes of defining coma (GCS <9), both the adult and pediatric versions are equivalent.

This, of course, does not mean that the scales are equally useful to detect the presence of coma.

Table 1 Glasgow Coma Scale (modified for pediatrics)

		All ages (best function)[a]		*Score*
Eye opening		Spontaneous		4
		To speech		3
		To pain		2
		None		1
	<1 year		>1 year	
Motor response	Spontaneous movement		Obeys commands	6
	Localizes pain		Localizes pain	5
	Flexion to pain		Withdraws to pain	4
	Abnormal flexion		Abnormal flexion	3
	Extension to pain		Abnormal extension	2
	Flaccid		Flaccid	1
	0–2 years	2–5 years	>5 years	
Verbal response	Smiles/coos	Appropriate words	Oriented and converses	5
	Consolable cry	Inappropriate words	Confused	4
	Inconsolable cry	Cries/screams	Incomprehensible sounds	3
	Grunts	Grunts	Grunts	2
	No response	No response	No response	1

[a]Whilst coma is defined as a GCS <9 condition, the changes in consciousness that precede it can also be defined by the GCS. Children <9 months do not localize pain.

Considerable debate has centered on the individual components of the GCS and their precision as predictors of severity/outcome as compared to the composite score. A nonlocalizing flexor response to a painful stimulus is sufficient to determine the presence of coma in children over the age on 1, the total score adding little. Reliance on the motor scale as the sole determinant of coma in those below the age of 1, and in particular those less than 9 months old (who at their best do not consistently localize pain) is clearly inappropriate. In such patients, the composite score is a better representation of their capacity.

Some investigators have commented that an inexperienced observer tends to score the same patient lower than a more experienced counterpart. This is even more likely to be the case in children. Finally, a number of common situations can make the scale inapplicable – endotracheal intubation precludes assessment of the verbal component, whereas facial swelling can prevent eye opening. There is no common agreement as to how to score such patients.

Despite the obvious utility of the GCS in describing coma, additional observations of signs such as pupillary response, brainstem reflexes, and respiratory pattern will be necessary for the assessment of likely cause.

Developmental Considerations

Coma is easy to define but hard to detect. This seemingly incongruous statement deserves explanation. The appearance of the comatose child can resemble the sleeping child. Newborns and infants up to 2 months of age can spend more than 90% of their day asleep. Whilst sleep, for the most part, can be readily differentiated from coma by the application of a stimulus and subsequent arousal, both the intensity of the stimulus needed to provoke arousal and the duration of that arousal can vary. A normal newborn, recently fed, may require a painful stimulus to wake and, when eventually woken, may fall asleep as soon as the stimulus ceases.

The limited reflex behaviors exhibited by the newborn are primarily those consequent to arousal, or the capacity for wakefulness. With maturation, the infant increasingly exhibits signs of awareness – visible actions consequent to sensations, emotions, or thoughts triggered by environmental stimuli. The observer must therefore have sufficient experience of the range of child behaviors across the developmental spectrum such that their expectations match the capacity of the child. Consistent purposeful responses to pain are readily observable to most (including lay) observers by 1 year of age. Whilst the experienced observer will be able to readily discern alterations of conscious state at all ages, it should be remembered that it is the parents who initiate most healthcare visits. A variable ability to detect subtle and early changes is to be expected.

Indeed, the readily observable consequence of this difficulty is on the perceived rate of onset of some of the common causes. In general, coma of acute onset suggests causes such as trauma, ingestion, seizure, or cerebrovascular event. Onset over hours to days

suggests infection, metabolic disorder, or mass lesions. The frequency of emergency presentations of conditions in the latter category points to the difficulty in the recognition of early alterations in conscious state.

Pediatric Vulnerability

The development of the central nervous system commences in the 3rd week of gestation and continues rapidly for at least the first 2 years of life. Progressive increase in cell number, myelinization, dendritic interconnections, and axonal growth leads to a rapid increase in brain weight. At birth, the brain is already 25% of its adult weight, whilst the child's total weight represents only 5% of adult weight. Not surprisingly, cerebral blood flow and oxygen consumption are proportionately greater in the infant than the adult. This equates to an increased vulnerability to the so-called metabolic causes of coma that interfere with substrate availability – hypoglycemia, hypoxia, shock, etc. The frequency of hypoxic–ischemic injury at postmortem in patients succumbing to coma is a clear reflection of this vulnerability. The capacity for wakefulness (arousal) is dependent on a region of interconnected neurons located ventral to the ventricular system and extending from the mid-brain to the lower pons. Known as the reticular activating system (RAS), it receives information from multiple sources and disseminates widely into the cortex. This region of the child's brain is susceptible to trauma consequent to the imbalance between the relative size of the head versus torso and the strength of the supporting musculature. Extremes of rotation or flexion/extension of the head and neck are readily generated, creating rotational/shear forces sufficient to disrupt the function of the RAS leading to abrupt onset of coma. It is believed the relatively fixed brainstem acts as a fulcrum around which the more mobile cortex can move during which it is subject to rotational/distraction injury. Magnetic resonance imaging (MRI) has confirmed that, in up to 60% of pediatric traumatic coma victims, brainstem injury is not only present but that its extent determines prognosis.

The pediatric brain, with its incomplete myelination, seems more susceptible to injury consequent to textural differences, variable rates of deformation in adjacent tissue creating shear stress, and the lack of protection offered by the thin calvarium. The corpus callosum appears particularly vulnerable. Somewhat paradoxically, the capacity for tolerating expanding lesions is enhanced by the incomplete fusion of sutures.

Apnea is a frequent accompaniment to pediatric brain injury, adding the risk of secondary injury from hypoxia/hypercarbia.

Finally, and perhaps most telling of all, is the physiological vulnerability to hypoxia of the young in any circumstance where airway patency and ventilatory adequacy are challenged. Airway obstruction and hypoventilation with the accompanying hypoxia and hypercarbia are all believed to potentiate the central nervous system (CNS) injury and are thought to be key triggers for acute brain swelling seen in some cases of trauma.

The prognosis for children suffering coma is commonly underestimated. Indeed it was once believed that for an equivalent injury children, with the plasticity of function believed to exist, had an improved outcome compared to that experienced by adults. It is now clear that the opposite is the case – indeed, the earlier the injury the more occult its consequence and the more delayed and extensive the effect.

Etiology

The differential diagnosis of coma is broad. The nonspecific nature of coma, with causes as diverse as septicemia and inborn errors of metabolism, requires a planned approach to diagnosis. Knowledge of the common categories of cause and their typical presentation as well as the age-specific frequency of individual diagnoses can help structure examination and investigation as well as ensure the rapid recognition of the treatable.

Typically, one of four pathophysiologic categories is suggested by the history and examination: (1) supratentorial expanding lesion; (2) infratentorial expanding lesion; (3) brainstem lesion; and (4) metabolic disorders. Whilst the overall incidence of pediatric coma is unknown, and is likely to be location and health system specific, some broad generalizations are possible.

In most of the published literature, children under the age of 1 year are not only the group most likely to suffer coma from any cause but also are those most likely to suffer from nontraumatic coma. The age group at greatest risk from traumatic coma is the 15–18-year-old group. That the likely cause varies with age is not surprising. Congenital causes are more likely in the first few months, infections more common in the young child, and trauma/intoxications in the older age groups.

Where only nontraumatic causes of coma are considered, infectious causes make up the clear majority of cases. This is in contrast to most adult series and requires the clinician to be particularly vigilant in

Table 2 Etiology of coma by age

< 1 year		*1–12 years*		*13–18 years*	
Cause[a]	*Frequency (%)*[b]	*Cause*[a]	*Frequency (%)*[b]	*Cause*[a]	*Frequency (%)*[b]
Infection[c]	50	Infection[c]	30	Trauma	40
Congenital	15	Seizures	20	Intoxication	25
Seizures	10	Trauma	20	Infection[c]	20
Metabolic	5	Metabolic/ingestion	10	Accidental[d]	5
Trauma	5	Accidental (nontrauma)[d]	10		

Source: Data from multiple sources, reflects the case for developed countries and is approximate only.
[a]Infrequent causes are omitted.
[b]Frequency in descending order by age; in up to 10% of cases the cause may remain unknown.
[c]Infection includes CNS-specific and systemic infections.
[d]Accidental: category includes causes such as asphyxiation, carbon monoxide poisoning, etc.

exploring the possibility of occult infection (Table 2). As indicated, the cause may remain undetermined. In many of these instances, the explanation is likely multifactorial. Hyperthermia, electrolyte abnormalities, hypovolemia, and acidosis coexist in many of the common infectious diseases of childhood, and whilst individually insufficient to explain the coma, in combination they can cause profound CNS depression.

The initial presentation of the child reflects not only this diverse etiology but also the difficulties inherent in the assessment of the under-1-year age group. Presentations in this age group are as likely to be nonspecific (e.g., vomiting, poor feeding, pallor) as they are to be CNS-specific (e.g., seizures, altered conscious state).

It is not uncommon for children to arrive at emergency medical services already intubated, having been given sedatives/relaxants. In these patients, examination can be limited to purely evaluation of the pupillary response. In such instances, management should reflect the broad possibilities. It should be particularly emphasized that it is important not to ignore any incongruities in the history or examination – what may have appeared to emergency personnel as a tonic seizure may be the early indication of central herniation. Mortality overall is in the range of 40–60%; it is cause-specific and time-weighted. The longer the duration of coma, regardless of cause, the worse the outcome. Coma consequent to complications of congenital abnormalities has a much higher mortality than intoxications for example. The low mortality from accidental ingestions, intoxications, and some metabolic causes supports the focus on supporting vital functions and treating the immediately treatable.

A number of "coma-like" conditions have been known to cause some initial confusion in diagnosis. The locked-in syndrome caused by a bilateral anterior pontine lesion is rare in pediatrics. Coma can, however, be mimicked by conditions causing severe neuromuscular weakness. Infantile botulism is one such condition where widespread weakness, often with early bulbar weakness, ophthalmoplegia, and ptosis, can cause the child to appear comatose. Psychogenic coma is unlikely.

The minimally conscious child is used to describe the chronic state where a child may exhibit arousal (clear sleep/wake cycles) but absent to nonpurposeful responses to stimuli.

Finally, in a brief discussion focused on definition and etiology, the clinical imperatives of this life-threatening complication have been given only passing mention. It is fitting to conclude that the challenge of the resuscitation, examination, investigation, and diagnosis and treatment of the comatose child is one that even the most experienced practitioner will find daunting.

See Also

Coma, Definitions and Differential Diagnoses: Adult

Further Reading

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Adult

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Definition of Coma

Coma can be defined as a state of unresponsiveness in which an individual is unaware of him/herself and the environment and cannot be aroused into a state of awareness or respond to the environment.

The Anatomical Substrate of Consciousness

Consciousness depends upon the interaction between the reticular formation within the brainstem and the cerebral hemispheres. The reticular formation is a complex polysynaptic pathway consisting of a network of small and large neurons that are present within the brainstem and extend to the diencephalon. All major sensory pathways have projections to the reticular formation where interaction takes place before there is projection to the cerebral cortex. The reticular activating system in particular is chiefly concerned with arousal and the maintenance of a wakeful state.

In principle, coma may be caused by dysfunction of the reticular activating system within the brainstem or diencephalon, notably the thalamus, or by bilateral problems affecting the cerebral hemispheres.

Pathophysiology

Lesions in the supratentorial compartment may cause a depression in the level of consciousness by displacing the brain laterally or caudally, with resultant dysfunction of the reticular formation. Infratentorial mass lesions initially compress adjacent structures, then with further expansion, downward herniation of the cerebellar tonsils and medulla through the foramen magnum occurs, as does upward herniation of the brainstem through the tentorial hiatus (Figure 1).

Within the supratentorial compartment, two types of brain herniation are recognized: first, subfalcine herniation, where the cingulate gyrus passes beneath the falx cerebri and, second, transtentorial herniation, where the parahippocampal gyrus and uncus pass through the tentorial hiatus.

An expanding lesion in one cerebral hemisphere will cause the cingulate gyrus on the medial surface of the hemisphere to herniate under the falx cerebri (Figure 2). This can cause displacement of the internal cerebral vein and the anterior cerebral artery and its branches. Although often clinically silent, such herniation can result in infarction in the territory of the anterior cerebral artery.

Transtentorial herniation takes place when an expanding lesion results in downward herniation of the parahippocampal gyrus and uncus through the tentorial incisura (Figure 3). Three structures of importance are compromised as a result:

1. the ipsilateral oculomotor nerve, resulting in pupillary dilatation due to the now unopposed sympathetic innervation of the pupil

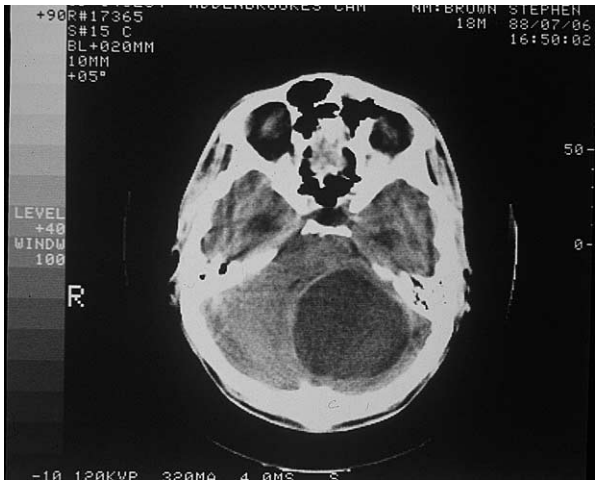

Figure 1 This computed tomography head scan shows a large cystic lesion in the right cerebellar hemisphere. It is producing a mass effect and is displacing the brainstem and cerebral aqueduct. The patient presented in a coma and at operation was found to have a hemangioblastoma, which was resected with good result.

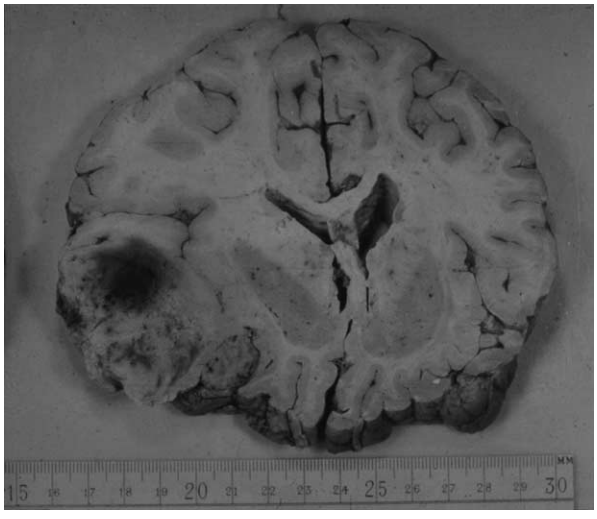

Figure 2 This coronal brain slice shows a glioblastoma multiforme in the left temporal lobe. Note the shift of midline structures and the herniation of the cingulated gyrus.

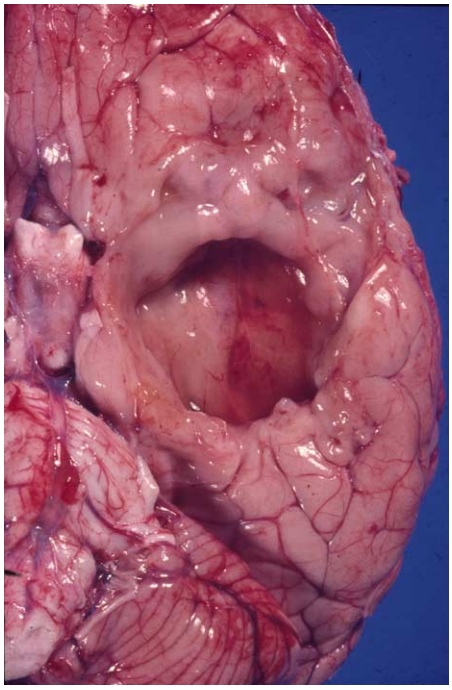

Figure 3 This patient had a cystic astrocytoma of the left temporal lobe and died. This autopsy photograph of the base of the brain shows the lesion and how the medial temporal structures have been displaced by transtentorial herniation.

2. the brainstem, resulting in a depression of the level of consciousness due to dysfunction of the reticular activating system. Long tract signs may also occur
3. the posterior cerebral vessels, leading in agonal cases to infarction of the medial aspect of the occipital lobe (calcarine infarction) (Figure 4).

It should be clearly appreciated that it is not just mass lesions such as tumors, abscesses, or hematomas that may result in coma, but other diffuse disturbances of the brain may also produce coma. These are classified into metabolic disturbances; toxic problems such as drug overdose and alcohol ingestion; inflammatory problems such as meningoencephalitis; epilepsy; and other causes such as subarachnoid hemorrhage or primary psychiatric illness. With metabolic disturbances, a general rule is that "all the failures" may produce coma, for example, renal failure, hepatic failure, cardiac failure, and so forth. Table 1 outlines the primary intracranial causes of coma while Table 2 looks at conditions of other etiologies that may result in coma.

Causes of Coma

It is important to have a systematic approach to managing patients in coma and a useful way to analyze the etiology of coma is described in Table 3.

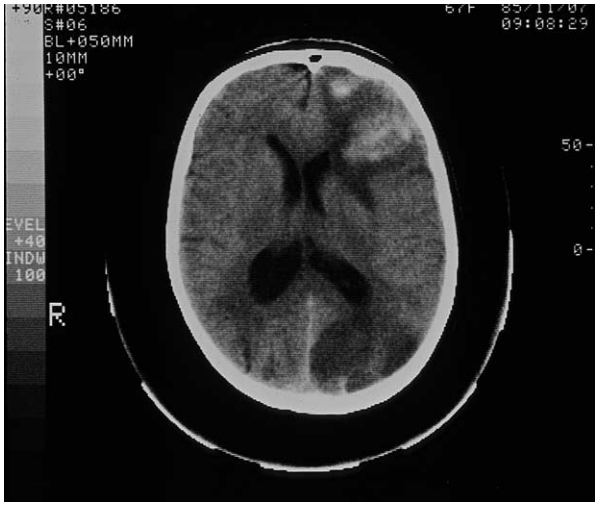

Figure 4 An area of low density is seen in the right occipital region. This represents infarction of the occipital lobe consequent upon transtentorial herniation. The confluent frontal contusion has resulted not only in transtentorial herniation but also in subfalcine herniation in this patient who fell downstairs.

Table 1 Primary intracranial causes of coma

- Head injury
- Intracranial tumor
- Meningitis
- Encephalitis
- Intracranial suppuration, e.g., abscess, subdural empyema
- Thrombosis of the venous sinuses of the dura mater
- Cerebral infarction

Table 2 Other conditions resulting in coma

- Toxic states, e.g., drugs, alcohol
- Generalized seizures
- Hypoxia
- Hyponatremia
- Hyperglycemia
- Hypoglycemia
- Hepatic failure
- Renal failure
- Disorders of calcium metabolism
- Thyroid problems, e.g., hypothyroidism
- Hypothermia
- Thiamine deficiency

Glasgow Coma Scale

This is the most universally accepted measure of the level of consciousness (Table 4). It is measured by assigning a patient a score relating to three parameters: first, the stimulus necessary to cause the patient to open his/her eyes; second, the best motor response in the limbs; and third, the best verbal response obtained from the patient. The maximum

Table 3 Etiology of coma

Coma with focal neurological signs or evidence of head injury
Supratentorial or infratentorial space-occupying lesion, e.g., tumor, hematoma, abscess
Hypoglycemic encephalopathy
Hepatic coma
Coma with meningism with no focal or lateralizing neurological signs
Meningoencephalitis
Subarachnoid hemorrhage
Coma without focal or lateralizing neurological signs or meningism
Hypoxic encephalopathy
Hyponatremia
Hypoglycemic encephalopathy and hyperglycemia
Liver failure
Renal failure
Hypocalcemia
Hypothermia
Hypothyroidism
Thiamine deficiency
Drug overdose
Generalized epilepsy, including postictal states
Nonneurological causes
Malingering
Hysteria
Catatonic schizophrenia

Table 4 Glasgow Coma Scale

	Score
Eye-opening	
Nil	1
To pain	2
To verbal stimuli	3
Spontaneously	4
Best verbal response	
No response	1
Incomprehensible sounds	2
Inappropriate words	3
Disoriented and converses	4
Oriented and converses	5
Best motor response	
No response	1
Extension	2
Abnormal flexion	3
Flexion	4
Localizes to pain	5
Obeys commands	6

score that can be obtained is 15 for a patient who is fully oriented with eyes open and obeying commands. The minimum score is 3 – it is impossible to score less than 3. It is important to record the score a patient obtains in each area of the Glasgow Coma Scale so that deterioration or improvement can be monitored.

Investigations

In patients in coma with evidence of head injury or who have focal neurological signs, a computed tomography (CT) or magnetic resonance imaging (MRI) scan of the brain will detect the presence of mass lesions in the supratentorial or infratentorial compartments. Blood glucose should be estimated and liver function tests performed. Lumbar puncture should not be performed in the presence of space-occupying lesions as this may precipitate coning.

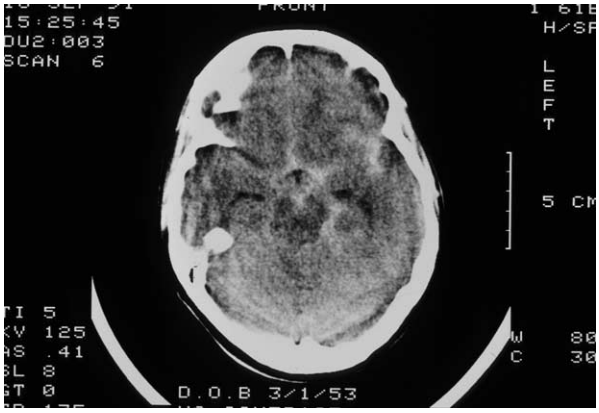

Figure 5 This patient was found collapsed with a Glasgow Coma Scale of 5. The computed tomography head scan shows widespread blood within the subarachnoid spaces. At autopsy, a saccular aneurysm of the anterior communicating artery was found to be the source of the hemorrhage.

In patients who are in coma and have evidence of meningism but in whom no focal or lateralizing neurological signs are present, a CT head scan should be performed. This will detect the presence of blood within the basal cisterns and subarachnoid spaces as well as other focal collections of blood or pus (Figure 5). Lumbar puncture is indicated in the investigation of these patients if there is no contraindication on CT scanning and in these circumstances, it is important to examine the cerebrospinal fluid for the presence of blood, microorganisms, protein, and inflammatory cells.

In patients who are in coma but who do not exhibit focal or lateralizing neurological signs or evidence of meningeal irritation, the following investigations should be performed:

- full blood count and erythrocyte sedimentation rate
- urea electrolytes and blood glucose
- liver function tests
- red blood cell transketolase
- plasma and urine osmolality
- serum calcium
- thyroid function tests
- arterial blood gases
- thiamine
- toxicology screen of urine and blood

- an electroencephalogram should be carried out, as this may show characteristic patterns in the hepatic and renal failure and may also detect nonconvulsive status epilepticus.

In patients without focal and lateralizing signs it may still be necessary to perform a CT or MRI scan and again, a lumbar puncture may be indicated if the other diagnostic tests fail to disclose the etiology of coma.

Management

A detailed management plan for all the various causes of coma is beyond the scope of this article and readers are referred to the Further reading section at the end of this article. However, certain principles of management should be clearly understood and these are outlined below:

- Resuscitation – ensure that the airway is clear, breathing is occurring, and that an adequate circulation is being maintained.
- Intravenous access should be adequate and may necessitate the insertion of central lines.
- At the same time as intravenous access is secured, blood should be withdrawn for various biochemical and hematological tests.
- As resuscitation is proceeding, clinical, laboratory, and radiological evaluation of the patient should be taking place and this is obviously aimed at detecting why the patient is in a coma.
- Certain etiological factors that result in coma may be treated specifically. For example, hypoglycemia can be managed by intravenous glucose infusion, opiate intoxication can be treated with naloxone and Wernicke's encephalopathy caused by thiamine deficiency may be treated with intravenous thiamine.
- The continuing care of patients in coma includes attention to pressure areas, monitoring of fluid and electrolyte balance, cardiac function and the maintenance of breathing, and control of epileptic seizures within or without the intensive care setting.

Prognosis

This is determined by a number of factors:

1. the etiology of the coma
2. the depth and duration of coma
3. clinical signs relating to brainstem reflexes.

Differential Diagnosis

A number of important conditions need to be distinguished clearly from conditions producing coma.

Brain Death

This refers to irreversible loss of function of the brain and brainstem irrespective of whether the heart is beating or not.

Etiology

1. head trauma
2. drug toxicity
3. central nervous system infections, including encephalitis and meningitis
4. cerebrovascular disease, including hemorrhagic and ischemic stroke
5. hypoxic–ischemic insults, including cardiac arrest, carbon monoxide poisoning, and status asthmaticus.

Diagnosis

This is clinical and assessment should be carried out by two doctors experienced in the performance of these tests on two separate occasions 12–24 h apart.

The essential diagnostic criteria are that:

- The patient is unconscious.
- The patient is apneic.
- The patient has absent brainstem function with the following features: (1) absent pupillary responses; (2) absent corneal reflexes; (3) absent oculovestibular reflexes, that is, doll's eyes, and caloric-induced eye movements are absent; (4) absent cough and gag reflexes; and (5) absent ventilatory reflexes, that is, there is no spontaneous respiration when the patient's respirator is disconnected and sufficient time elapses to allow arterial carbon dioxide tension to rise above the threshold required for the stimulation of respiration ($PaCO_2 > 8$ kPa).
- Stimulation of any region within the cranial nerves should not elicit a motor response.
- Patients should not be hypothermic or hypotensive. The patient should exhibit a flaccid tone and absent spontaneous or induced movement.

Although the diagnosis of brain death is essentially clinical, it is important to ensure that a remediable or reversible condition has been eliminated by appropriate imaging and other investigations, including blood tests and toxicology screens.

It is well recognized that in the presence of brain death the electroencephalogram shows electrical silence and that cerebral angiography shows no blood flow through the brain.

Persistent Vegetative State (PVS)

At one stage this condition was known as coma vigil. It is defined as a condition of wakefulness

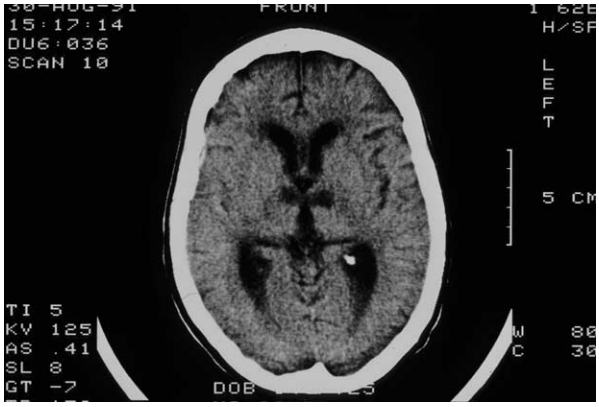

Figure 6 This computed tomography scan shows bilateral thalamic damage from carbon monoxide poisoning. The victim was found in a room that was heated by a defective gas fire. He remains in a persistent vegetative state.

Table 5 Principal causes of persistent vegetative state

- Head injury
- Cerebrovascular disease
- Hypoxia–ischemia, e.g., carbon monoxide poisoning, cardiac arrest
- Intracranial infection, e.g., meningitis, encephalitis
- Terminal stages of degenerative central nervous system disorders

without awareness. In the vegetative state the patient displays unawareness of him/herself and the environment and has a stable circulation, breathes spontaneously, and exhibits cycles of eye opening and eye closure with periods of sleep and wakefulness. For a vegetative state to be classified as persistent, it must continue for more than 4 weeks and it is therefore unlikely that it represents a phase in the recovery from coma.

When a vegetative state continues for more than 12 months after head injury or 6 months after other etiological causes of this condition, it is referred to as a permanent vegetative state.

In broad terms of neuropathology, PVS is attributable to severe and diffuse damage to the cerebral hemispheres in the presence of an intact brainstem. It should however be noted that damage to the rostral part of the brainstem in conjunction with damage to the cerebral hemispheres can also be consistent with the development of PVS. Diffuse axonal injury is a frequent cause of PVS, as is extensive laminar necrosis of the cerebral cortex consequent upon global cerebral ischemia or hypoxia. Thalamic necrosis of whatever etiology, for example, carbon monoxide poisoning, may also result in PVS (Figure 6). The principal causes of PVS are listed in Table 5.

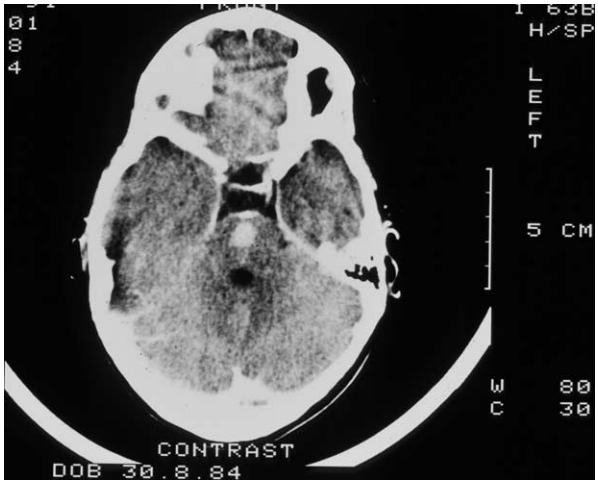

Figure 7 This computed tomography scan through the posterior cranial fossa shows a high-density area within the pons. This was due to a hypertensive hemorrhage and the patient was locked in.

Locked-In Syndrome

This is a particularly unpleasant and unfortunate condition in which patients are aware of themselves and their environment but are unable to respond due to loss of motor and speech function. The condition may be caused by supranuclear lesions affecting the corticospinal tracts, usually in the region of the ventral pons below the level of the oculomotor nerve nuclei (Figure 7). Alternatively the condition may be attributable to nuclear or infranuclear disease of motor nerves. Causes within the brainstem include head injury, tumor, demyelination, central pontine myelinolysis, and hemorrhage or infarction usually of hypertensive origin. Nuclear or infranuclear causes include Guillain–Barré syndrome or polyneuropathy.

Other Differential Diagnoses of Coma

These include narcolepsy, a condition characterized by excessive sleepiness, with abnormally regulated rapid eye movement sleep. The other important differential diagnosis of coma is syncope. This is essentially due to a variety of causes, all of which result in loss of postural tone and consciousness from an acute reduction in the blood flow to the brain. Disorders of cardiac rhythm, reduction in stroke volume, and reduced peripheral resistance of whatever etiology can result in syncope.

See Also

Carbon Monoxide Poisoning: Clinical Findings, Sequelae In Survivors; **Coma, Definitions and Differential Diagnoses:** Pediatric; **Head Trauma:** Pediatric and Adult, Clinical Aspects; Neuropathology

Further Reading

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