

CHAPTER 9

Stupor And Coma

The patient with reduced level of consciousness presents complicated clinical problem that requires prompt yet careful evaluation and management. Pathophysiologically, stupor and coma are most commonly produced when there is significant damage or dysfunction in ascending activating system of brain stem. The ascending activating system has its origin in reticular formation of pons and midbrain, with possible contributions from other nuclei such as the locus ceruleus and median raphe. Ascending axons project to thalamus, limbic system, and to widespread areas of the cerebral cortex.

Clinically, there are three main mechanisms by which this system is affected and consciousness compromised: one, by diffuse disruption of neuronal metabolism such as seen in drug overdose or metabolic disease (e.g., uremia, hypoglycemia); two, by direct damage to the activating system in the brain stem, e.g., occlusion of basilar artery, brain stem hemorrhage; three, unilateral or bilateral hemispheric lesion with lateral displacement due to mass effect [with diencephalon and brain stem compression] (e.g., intracerebral hematoma, neoplasm, abscess, traumatic contusion or hemisphere. A small unilateral hemispheric or subcortical lesion, e.g., lacunar infarct is not likely to cause impaired consciousness and these patients are alert.

The focus of this chapter is on the neurologic rather than medical aspects of the comatose patient. The reader, requiring a more extensive discussion of the medical evaluation and management of coma, is referred to the excellent monograph by Plum and Posner.

EVALUATION

Because of the urgent nature of the problem, the initial evaluation phase is carried out in conjunction with management of acute emergency. The initial phase of evaluation consists of brief history. If accompanying relative or friend is available, examiner should ask what happened. If this person does not know, as is often the case, physician should inquire if patient is diabetic, hypertensive, drug or alcohol abuser, or epileptic or has any known medical diseases.

After historical information is assimilated, attention is immediately turned to the patient. The physician should briefly examine the patient for evidence of trauma or meningeal irritation (hemorrhage or meningitis), establish an airway, and obtain vital signs. A large-caliber intravenous line is placed, and blood is drawn for emergency blood cell count and chemistries (sugar, blood urea nitrogen, alcohol, liver profile, and electrolytes, including calcium and magnesium). Next, 100 mg of thiamine (for Wernicke's syndrome) is administered intravenously, followed by 50 ml of 50% glucose solution (for hypoglycemic reactions). This can be lifesaving and will not worsen the condition in diabetic ketoacidosis. If patient does not promptly revive, the physician should insert urinary catheter, check urine for sugar and acetone, and save sample for toxicology screening (drugs often prove to be the explanation for coma, and exact identification of substance is very helpful in management). Consideration of

administration of naloxone if opiate overdose is suspected or flumazenil (0.2 mgm IV and repeat this injection to maximum of 3 mgm) if benzodiazepine overdose is suspected. Be careful as flumazenil may cause seizures and respiratory depression may not be reversed.

At this point, more extensive history can be obtained, and full physical and neurologic examination can be performed. From this examination the physician is usually able to:

- establish if the coma is caused by neurologic lesion or metabolic/toxic disorder (a very important factor in both management and prognosis.);
- determine depth of coma and presence or absence of brain stem reflexes (these correlate well with the prognosis); and
- establish specific cause. A hastily ordered CT scan may add to the diagnostic confusion, and an orderly step-wise evaluation is more useful.

Observation

The first step is to observe the patient on the stretcher. Examiner should look for spontaneous movements and assess if they are purposeful or reflex. In addition, patient is assessed for seizure activity, myoclonic jerks (common in metabolic encephalopathies), asterixis (irregular flapping movements of the hands, feet, and tongue), tremor, diffuse twitching (seen in hypoglycemia and hyponatremia), and unilateral twitching and fasciculations. Also note any asymmetries of motor activity, which would suggest focal lesion. Check breath odor--fruity, sweet suggests diabetic ketoacidosis; fishy, suggests hepatic encephalopathy; dirty smelly restroom suggests renal encephalopathy; garlicky suggests medication effect such as organophosphates. Hyperthermia suggests infectious-inflammatory disease or subarachnoid hemorrhage; hypothermia suggests environmental exposure, systemic illness, or drug intoxication. Examination of skin may provide important clues of systemic illness. Periorbital blood (raccoon eyes) or retroauricular ecchymosis (Battle sign) indicate traumatic origin. Assess for signs of meningeal irritation but these may be absent in comatose patients with meningitis or SAH.

Respiration

Changes in respiratory patterns are common in comatose patients and often have specific significance for the level of coma or structure involved.

Hypoventilation

Shallow regular respiration suggests an overdose of sedative medication, e.g., barbiturates, benzodiazepines, alcohol, or opiates.

Hyperventilation

High-amplitude rapid respiration can be seen in many conditions, including metabolic acidosis, hypoxia from pulmonary disease, and occasionally midbrain damage (central neurogenic hyperventilation).

Cheyne-Stokes

Cheyne-Stokes respiration is a pattern in which periods of hyperventilation are followed by

periods of apnea. The transition between the two is gradual with a crescendo/decrecendo pattern. This is commonly seen in metabolic disease and bilateral hemisphere disease and rarely with upper brain stem damage. Although definitely abnormal, it does not carry the ominous prognostic implications of the other abnormal patterns.

Apneustic

Apneustic breathing is a rare pattern in which the patient holds his or her breath for a few seconds on inspiration (inspiratory cramp), then exhales. It is seen in patients with lesions of the tegmentum of the lower pons and indicates serious brain stem dysfunction.

Cluster

Another uncommon pattern that has the same anatomic and prognostic implication as apneustic breathing is cluster breathing. The pattern is characterized by a cluster of three or four breaths followed by short periods of apnea.

Ataxic

Ataxic breathing is a very irregular pattern that signals dysfunction of the medullary respiratory centers. When this pattern is seen, it is wise to place the patient on assisted respiration.

Preapneic

There are several very ominous respiratory signs, such as gasping, expiratory push, and fish-mouthing (opening the mouth during inspiration) that are commonly seen before total apnea.

Level of Consciousness

Assessment of the level of arousability is important for two reasons: to determine the seriousness of the patient's condition and to establish a reliable parameter by which to follow the patient's progress. A number of standard terms are used to describe levels of consciousness (Box 9-1). By definition, comatose patients have closed eyes. Patients in minimally conscious and vegetative state patients have open eyes and spontaneous eyelid blinking.

BOX 9-1

- *Alert:* awake and normally responsive to environmental input
- *Lethargic or somnolent:* *sleepy and somewhat inefficient mentally but usually oriented; tends to drift off to sleep if not stimulated.*
- *Obtunded:* *Aroused with loud voice or gentle shaking but when roused tends to be confusional.*
- *Stupor or semicoma:* *requires vigorous stimulation to arouse.*
- *Coma:* either no response to painful stimulation (deep coma) or reflex movements only with painful stimulation (light coma).

Because of imprecision in these definitions, several objective systematic evaluation schemes have been devised. Basic principle of these evaluations is that both the stimulus necessary to

rouse the patient and level of response are graded, thus giving more objective description of the patient's responsiveness. The clinician should begin by calling patient's name; if this is ineffective, stimulus is increased in intensity. Examiner should next shout and gently shake patient and call his or her name. Finally, painful stimulus (firm pressure on sternum or supraorbital ridge, pinching the skin, tightly grasping the Achilles tendon) can be applied. Stimulus required for arousal is recorded, along with full description of motor response, level of vocalization, and ocular response. The following are organized in ascending order of severity:

TABLE 9-1 Patient Response to Arousal Stimulus		
Level of Vocalization	Ocular Response	Motor Response
Normal Conversation	Open with eye contact with examiner	Obeys commands
Confused discourse	Open but no contact	Purposeful movements
Incoherent mumblin	Open with dysconjugate gaze	Restless movements
Groaning	No eye opening	Decorticate posturing
Grunting		Decerebrate posturing
No response		Flaccid quadriplegia

By using this scheme, level of arousal can be tested serially and has advantage that different examiners can evaluate the patient and accurately assess progress or deterioration. One complicating factor that must always be considered in following patients with decreased level of consciousness is underlying effects of the sleep-wake cycle. This cycle is often disturbed in brain-damaged patients; therefore, fluctuations in arousability may not always represent deterioration in patient's condition but may merely be form of sleep.

Motor Response

When observing motor response, examiner should carefully note asymmetries of movement and tone. Absence of movement on one side usually indicates structural lesion on opposite side of brain or brain stem and will quickly turn examiner's attention toward primarily neurologic evaluation. Eversion of the foot suggests hemiparesis on that side. The quality of movements is also important because different types of movements are seen at different levels of coma. Certain movement patterns can also localize damage within the brain stem. Qualitative levels of motor response include those listed in Box 9-2. Abnormal flexor responses (decorticate posturing) indicate bilateral hemispheric and diencephalic level involvement and abnormal extensor response (decerebrate posturing) indicate brain stem lesions; however, these are nonlocalizing and have never been confirmed in humans but rather in experimental animal models.

Brain Stem Reflexes

Adequacy of brain stem functioning is critical factor in overall assessment of comatose patient. Loss of brain stem reflexes is usually sign of poor prognosis unless loss is transient. It signifies either major structural damage in brain stem or metabolic or toxic disturbances of marked degree.

Box 9-2

1. *Obey commands.* This is highest level of motor response and indicates that higher cortical language centers as well as the motor system are functioning.
2. *Purposeful movements.* This term is usually employed when stuporous patient localizes and attempts to fend off painful stimulus. Such movements indicate a fairly high degree of sensorimotor integration.
3. *Restless movements.* These are random movements in response to stimulus. They are not accurate in their localization of the stimulus or effective in attempts to prevent it. Such movements do involve abduction of limbs and would thus be considered as purposeful in rudimentary sense.
4. *Decorticate posturing.* This is a type of reflex posturing in which the arms are adducted and flexed and the legs extended. Decorticate posturing indicates a hemispheric lesion; when bilateral, it often represents the first stage in central herniation.
5. *Decerebrate posturing.* In this case, the patient extends, adducts, and internally rotates the arms and extends legs. Teeth are clenched, although on occasion there is reflex protrusion of the tongue. This posturing can be seen spontaneously or can be in response to stimulation. When present, this is a more ominous sign than decorticate posturing and usually indicates bilateral pyramidal tract dysfunction at the midbrain or upper pontine level. Decerebrate posturing can be seen in a metabolic coma, so it does not necessarily indicate damage to the brain stem.
6. *Mixed posturing.* On occasion, patients with pontine lesions will demonstrate mixed picture with extended arms and flexed legs.
7. *Flaccid quadriplegia.* When the brain stem is completely nonfunctional, tone is completely lost.

Pupils

Pupillary size, symmetry, and reactivity to light are all important. Pupillary reflex is integrated in midbrain through optic nerve, Edinger-Westphal nucleus, and oculomotor nerve. There is influence from sympathetic system, which begins in hypothalamus and descends through brain stem to synapse in superior cervical ganglia and ascends to orbit via nerve plexus along carotid artery. Pupillary size is determined by relative influences of parasympathetic and sympathetic systems (Box 9-3). Pupillary reaction to flashlight may be difficult to discern if pupillary size is small; therefore, magnifying glass may be required to evaluate difficult to assess, questionable pupillary responses.

Horner's Syndrome (Miosis, Ptosis, and Unilateral Anhidrosis)

With small reactive pupil on one side and loss of sweating on same side of face and body, it is likely that patient has either unilateral (ipsilateral) hypothalamic damage from early herniation or ipsilateral brain stem destructive lesion.

Box 9-3

1. *Very widely dilated (8 mm) nonreactive, particularly if unilateral:* Strongly suggests that cycloplegics have been used.
2. *Midrange, equal, and reactive:* This pattern in comatose patients usually indicates metabolic or toxic cause.
3. *Small and reactive:* Can be seen in early stage of central herniation.
4. *Midrange (5-6 mm), possibly irregular and unequal, and unreactive:* Midbrain damage is very likely; this can also be seen in glutethimide (Doriden) toxicity.
5. *One pupil dilated and fixed:* There is damage to one oculomotor nerve, which in comatose patients is most commonly caused by herniation of temporal lobe and pressure either to nerve in subarachnoid space or to the nucleus and nerve within midbrain.
6. *Bilateral pinpoint and reactive:* Pontine hemorrhage (sympathetic damage in which there is unopposed parasympathetic tone) or narcotic overdose (constricted pupils can be dilated with narcotic antagonists, e.g., naloxone).
7. *Bilateral dilated (6-7 mm) and fixed:* this can be seen with brain stem death or atropine toxicity.

Eye Position and Movement

The examiner opens the patient's eyes and observes primary position of eyes and any spontaneous movement. Open eyes and spontaneous eye opening is not consistent with comatose state.

- *Roving dysconjugate gaze:* In most comatose patients, eyes spontaneously move in random fashion. Patients do not fix on objects in the environment, and eye position is often dysconjugate. With this eye movement, brain stem is functioning.
- *Forced lateral gaze:* Frontal eye fields (area 8) exert tonic effect on horizontal eye movement. With focal seizures, gaze will be forced away from active hemisphere. With destructive lesion, such as cerebral infarct, tonic activity from intact hemisphere usually forces gaze toward damaged hemisphere. Forced horizontal gaze [in the opposite direction] can occur in brain stem lesions. [To remember this more easily--seizure activity is interesting and patient gaze is directed toward seizing limb; whereas, hemiplegia is depressing and eyes look away from hemiplegic limbs.]
- *Forced downgaze:* This is seen usually with thalamic hemorrhage, mass lesions in pineal region, or in some metabolic comas, particularly hepatic.
- *Nystagmus:* These rapid jerking eye movements can be seen with seizure, phenytoin toxicity, and pontine damage in which vestibular nuclei are damaged.
- *Ocular bobbing:* This pattern--rapid downgaze followed by slow upgaze in a patient who is incapable of horizontal eye movement--is seen in low brain stem lesions.

- *Bilateral paralysis of abduction:* Bilateral abducens palsy is commonly caused by increased intracranial pressure from either cerebral hemisphere or posterior fossa masses.
- *Total oculomotor paralysis in one eye:* This is caused by damage to third cranial nerve, from herniation or from burst internal carotid aneurysm. The eyelid droops, pupil is dilated and non-reactive and only movement is in lateral direction.

Oculocephalic Reflex (Doll's Head Phenomenon)

Oculocephalic reflex is tested by placing patient's head at 30 degrees above horizontal and quickly flexing head or rotating it to either side. (This should not be done in patients who are suspected of having sustained neck trauma.) In comatose patient with normal brain stem function, head rotation stimulates semicircular canals and also proprioceptive fibers in cervical spine. These stimuli enter brain stem and travel up medial longitudinal fasciculus to activate third and sixth nuclei, thus initiating lateral eye movement. Before head is rotated, eyes will be staring directly up at ceiling; with rotation of head to right eyes will continue to look at ceiling even though head is now to the right. The reflex system in brain stem has stimulated both left sixth nerve to rotate left eye left and right third nerve to make right eye also look left. If head is rapidly flexed, eyes will open and look up (positive doll's head phenomenon). When brain stem damage has occurred and reflex arc is impaired, eyes continue to stare ahead in same plane of head, and no ocular rotation occurs (negative doll's head phenomenon). This represents the testing of the oculocephalic reflex in a comatose patient. If this reflex is attempted in an awake patient, the eyes move toward the side of the head rotation.

Oculovestibular Reflex (Caloric Stimulation)

Oculovestibular reflex is similar to oculocephalic reflex, except that the stimulus to vestibular system is via cold or warm water on eardrum and not head rotation. This test should be performed if the doll's head response is negative because negative response can result if stimulus (head turning) was not adequate to stimulate the brain stem. Examiner must check to see that external ear canal is clean and that tympanic membrane is intact; then ice water is introduced into ear canal with small catheter. Ten milliliters is usually sufficient, but as much as 50 ml should be used before deciding that reflex is absent. In patient with functioning brain stem, eyes will slowly turn toward stimulated side. In patients taking barbiturates, tricyclic antidepressants, aminoglycoside antibiotics, and other vestibulotoxic drugs, this reflex can be absent secondary to vestibular dysfunction alone and not brain stem dysfunction. This is the response in a comatose patient. In an awake patient who is much more sensitive to the effect of cold water, 0.1 ml is irrigated. The normal response is slow horizontal deviation towards the irrigated ear and then horizontal nystagmus towards the opposite side. In comatose patients, nystagmus does not occur.

Corneal Reflex

Corneal reflex is tested by holding patient's eye open and then touching corneas. If facial paralysis prevents eye closure, examiner can observe for change in eye position. There is a normal forced upgaze when the patient attempts to close the eyes (Bell's phenomenon). Presence of corneal reflex indicates brain stem function.

Gag Reflex

The gag reflex is a medullary reflex that should only be tested if patient has been intubated because it can cause reflex vomiting, aspiration, and increased intracranial pressure.

Funduscopy

During funduscopy, the clinician should search principally for papilledema or subhyaloid hemorrhages; the latter is a classic sign of subarachnoid hemorrhage.

Muscle Stretch and Pathologic Reflexes

Asymmetries and pathologic reflexes such as Babinski signs or clonus should be sought. The Babinski sign can be positive and clonus can be present (but is usually bilateral) in metabolic disease, so this is not a sine qua non of structural neurologic disease. The presence of clonus and a Babinski sign indicates need for neuroimaging studies to exclude focal brain disease or injury in setting of severe enough metabolic encephalopathy to impair consciousness. For example, hepatic encephalopathy can cause these signs, but neuroimaging studies must be done to exclude associated intracranial hemorrhage caused by impaired coagulation mechanism or brain abscess caused by impaired immune response. Although there are rare exceptions, uni- or bilateral Babinski signs indicate structural brain disease.

Response to Pain

The patient's response to pain has basically been tested during assessment of the level of consciousness, but it is also important to look for asymmetries. For example, thalamic hemorrhage elicits a markedly decreased response to pain on side opposite the lesion.

DIFFERENTIAL DIAGNOSIS

Proper management of coma requires exact diagnosis. Because there is a wide variety of causes, it is useful to be familiar with relative frequency of conditions most commonly known to cause coma. In one series of 500 cases of nontraumatic coma, the general classifications were reported (Box 9-4).

Findings on neurologic examination alone permits examiner to place patient into one of these general diagnostic categories. Box 9-4 outlines the five general diagnostic categories of the coma patient.

BOX 9-4	
Metabolic disorders	35%
Exogenous toxins (drugs primarily)	30%
Supratentorial lesions	20%
Intracerebral hemorrhage	(9%)
Subdural hematoma	(6%)
Infarct	(2%)
Tumor	(1.5%)
Abscess	(1%)
Other	(0.5%)
Subtentorial lesions	13.5%
Brain stem infarct	(8%)
Tumor	(2.5%)
Pontine hemorrhage	(2%)
Cerebellar hemorrhage	(1%)
Psychogenic unresponsiveness	1.5%

BOX 9-5
<ol style="list-style-type: none">1. Metabolic or toxic coma.<ol style="list-style-type: none">a. No lateralizing neurologic findingsb. Pupils equal, mid range, and reactive to lightc. Eyes roving and dysconjugate, can be forced downgazed. Oculocephalic and oculovestibular reflexes intacte. Tremors, multifocal myoclonic jerks, asterixis2. Coma caused by structural brain lesions. An expanding hemispheric lesion causes shifts (herniations) of brain tissue from one brain region of higher pressure to one of lower pressure. The cerebral hemispheric mass can exert a downward or lateral force through the tentorial notch thus damaging the brain stem.<ol style="list-style-type: none">a. Frontal, parietal, or midline mass (central herniation).<ul style="list-style-type: none">• Stage 1 (diencephalic): hemiparesis with decorticate posturing• Stage 2 (midbrain/upper pons): Pupils mid range and fixed, respiration rapid,

- decerebrate posturing, oculocephalic reflexes absent
- Stage 3 (low pons): pupils fixed, extremities flaccid with possible leg flexion, and low, rapid respiration
 - Stage 4 (medullary): pupils fixed, breathing irregular or apneic, flaccid quadriplegia
- b. Temporal mass (uncal herniation): uncus or medial temporal lobe is displaced into tentorial notch to compress and distort midbrain
- Stage 1: subtle decrease in level of consciousness, pupillary dilation (usually ipsilateral to lesion) caused by ipsilateral oculomotor nerve and midbrain effect.
 - Stage 2: complete third nerve palsy, rapid decrease in level of consciousness, hemiparesis (caused by pressure on cerebral peduncle). Hemiparesis is usually opposite the lesion, but occasionally midbrain is pressed against opposite tentorial edge and ipsilateral hemiparesis develops (Kernohan's notch phenomenon). Posterior cerebral artery extends around brain stem; this artery can be compressed against tentorium by uncus to cause occipital lobe infarction and homonymous hemianopsia.
3. Subtentorial lesions. There are several main lesions in this group, but most are characterized by rapid onset of coma with evidence of damage to the brain stem. Vascular lesions cause more rapid onset and nonvascular mass lesions can cause a slower onset of symptoms. Neurologic deficit can be caused by upward (ascending) transtentorial herniation by subtentorial lesion, or by cerebellar tonsillar herniation with sudden death because of pressure on and damage to respiratory centers in lower medulla.
- a. Basilar artery occlusion: Acute loss of consciousness, bilateral pyramidal tract findings, evidence of primary brain stem dysfunction (variable cranial nerve and gaze abnormalities)
 - b. Pontine hemorrhage: Acute loss of consciousness, irregular respiration, tetraplegia often with decerebrate posturing, frequently very small but reactive pupils, abnormalities in ocular rotation, abnormal oculocephalic, and caloric testing
 - c. Mass lesion (including neoplasms, abscesses, and subdural hematomas): Gradual decrease in level of consciousness, vomiting, hyperventilation, loss of upgaze or lateral gaze; oculocephalic reflexes preserved
 - d. Cerebellar hemorrhage with secondary brain stem compression: Headache, ataxia, small pupils, nausea and vomiting, vertigo, dysarthria, sixth and often seventh nerve palsies, stiff neck, nystagmus, and bilateral Babinski signs. These signs progress rapidly as consciousness is being lost, so early recognition (before consciousness is impaired) is critical if surgical evacuation is to be successful.
4. Psychogenic unresponsiveness (hysterical coma). This exists, but should only be seriously considered in diagnostic process after organic disease is quite certainly ruled out. Such patients basically do not appear ill and show no abnormal physical or neurologic findings except their unresponsiveness. They may not respond to pain, and oculocephalic responses can be difficult to interpret, but caloric testing will elicit prompt forced gaze with nystagmus to the opposite side. The gaze shows fixation, and dysconjugate roving eye movements are not seen. After completely negative physical, neurologic, and laboratory investigation, it is usually safe to observe the patient.
5. Other states resembling coma.

- a. **Akinetic mutism:** The patient is lethargic and can appear alert but not responsive; can have spontaneous eye movements and visual pursuit but shows no signs of mental awareness even when stimulated; is capable of producing speech but is mute; and has no voluntary motor movement but is not paralyzed. The patient's eyes can be open, can blink in response to threat and can even visually track objects. Patient is inattentive and mute (never spontaneously speaking). Other akinetic muter patients appear wakeful with their eyes open and some eye contact can be made with examiners and family (coma vigil). Electroencephalogram shows marked generalized slow wave pattern. This condition can be caused by bilateral basal frontal lobe or midbrain lesions (damaging midbrain reticular formation).
- b. **Chronic vegetative state:** This term is used to describe patients who have survived severe head injury or diffuse hypoxic-ischemic brain injury. They have widespread brain lesions in cortical and subcortical regions, but brain stem is usually spared. Patient lies in bed with eyes open, can occasionally utter sounds or moans, but does not speak or respond to commands. The patient may blink in response to threatening stimulus or can have searching eye movements and may briefly fixate on the examiner or family member, but there is no real eye contact. Reflex movements (decerebration, decortication, myoclonic jerks) can occur. Depending on the specific damaged brain regions, patient can show bilateral limb weakness, spasticity, Babinski signs, and arousal response but no awareness of environment. Respiratory and circulatory function is normal. Electroencephalogram can show alpha rhythm (not well sustained) sometimes with normal sleep pattern; this contrasts with slowing of isoelectric pattern seen in other comatose patients.
- c. **Locked-in syndrome:** In this condition, patient is totally immobile with inability to move limbs and is not able to speak or swallow or show facial expression; the eyes, however, are spared. Patient responds to questions by opening and closing the eyes or moving the eyes horizontally or vertically. There is no impairment of awareness or consciousness. The electroencephalogram is normal. It can be caused by a lesion of the basis pontis (involving corticospinal and corticobulbar fibers) with sparing of ascending reticular activating fibers. This condition can occur in basilar artery occlusion, central pontine myelinolysis, or motor neuropathy involving peripheral and cranial nerves.
- d. **Catatonia:** The patient appears akinetic and mute. The neurologic examination shows normal respiration, normal pupillary response and extraocular motility, and intact motor function with no abnormal reflexes. The limbs may remain in the position they are placed (waxy flexibility) and appear rigid. The electroencephalogram is normal. This condition can be seen in manic depressive illness and schizophrenia, but many cases have an organic cause such as basal ganglion disease, encephalitis, drug reaction, and various other metabolic and structural lesions (Philbrick and Rummans, 1994).

MANAGEMENT

The management of the comatose patient has three basic stages. The first stage consists of nonspecific emergency procedures necessary to maintain vital functions. The second stage is specific medical or neurologic treatment of cause of coma. Unexplained causes of coma should be assessed with EEG for the possibility of nonconvulsive status epilepticus especially if there is ocular deviation, myoclonic jerks or rhythmic eye blinking. Specific medical treatment of toxic and metabolic disease is discussed in standard medical texts. The care of neurologic disease such as tumors, strokes, and infections is discussed in other chapters of this volume. The third stage involves specific preventive measures that are necessary in immobile patients:

- General skin care to prevent decubitus ulcers
- Antiembolic stockings and heparin in small doses to appropriate patients to prevent thrombophlebitis and pulmonary embolism
- Ointment in the eyes and patching to prevent corneal ulceration or abrasion
- Sponge padding of elbows to prevent pressure neuropathy of the ulnar nerves
- Nutrition--avoid catabolic state and give appropriate caloric levels utilizing enteral feeding
- Gastric stress ulcer prophylaxis utilizing H-2 blockers

PROGNOSIS

Knowing the expected long-term outcome in the comatose patient is useful both in discussing patient's condition with family and in planning patient's future care. An exact prognosis cannot be given for each patient, but there are some general observations and statistics available that clinician can use as a guideline. In a study of 500 nontrauma patients who were comatose for at least six hours, Plum and Posner reported that only 15% made a satisfactory recovery. Seventy-six percent had died within 1 month, and 85% had died within 1 year. In general, comatose patients from primary neurologic diseases will have a much worse prognosis than patients in metabolic coma. For example, a good recovery is expected in 25% of the patients in metabolic coma but in less than 5% of patients with coma secondary to subarachnoid hemorrhage. In metabolic disease, if coma is long or brain stem function is abnormal, prognosis worsens dramatically.

In patients with toxic overdose, prognosis is good and the death rate can be as low as 5% but the survival rate decreases with prolonged coma, concurrent medical illness, complications, or advanced age.

The case of coma after cardiac arrest deserves special mention. The best overall parameter for judging prognosis is length of time from arrest to initial return of consciousness. If time is short prognosis is good. If the patient is not awake in 10 to 12 hours, there is only a 40% chance of functional recovery. Quality of movement is also a good indicator. If patient is comatose 1 hour after cardiac arrest and has decorticate or decerebrate posturing, prognosis is much better. If patient remains comatose for 6 hours and does not initiate purposeful withdrawal, possibility of good recovery drops to below 5%. At 24 hours, if no purposeful movement has returned, most

patients will die or survive in vegetative state. Another poor prognostic sign is a loss of oculocephalic reflexes after circulation has been reestablished and failure to regain pupillary or brain stem reflexes by 48 hours.

BRAIN DEATH

Death can be declared either when heart function ceases or when the brain is nonfunctional and irreversibly damaged. Although it is usually the neurologist or neurosurgeon who is called on to make a legal pronouncement of brain death, it is important for all physicians to know the basic criteria required. Brain death can be declared when a patient has known structural brain disease (e.g., trauma, hemorrhage, and infarction) and over a period of 6 hours (some prefer to wait 24 hours) demonstrates no hemispheric or brain stem function. Pupils should be fixed to light, caloric testing with 50 ml of ice water should be negative, and the patient should be apneic for (5 to 10) minutes immediately after 10 minutes of ventilation with 100% oxygen. It is crucial to differentiate coma-brain death from "locked-in syndrome (de-efferented state) in which there is blinking and spontaneous vertical eye movements which occur in response to questions asked of the patient. These patients may communicate by code systems as they cannot communicate by usual means. If the coma is due to toxin, hypothermia, or metabolic cause, the prognosis is less certain than with structural lesions, and brain death should be diagnosed with great caution. Deep tendon reflexes and spinal withdrawal reflexes can be present in brain-dead patients. An electroencephalogram is not necessary, but if it is obtained, it should not show any brain activity exceeding 2 μ V. If above criteria are met, patient can be considered clinically dead. The legal status of the concept of brain death and irreversible coma is dependent upon individual hospital guidelines. It is important to be certain that all brain stem reflexes are absent and the course of the neurological dysfunction should be known and be irreversible. The diagnosis of brain death is important for organ donation. Always perform toxicology to exclude drug overdose even if neuroimaging studies show structural lesion.

SUMMARY

Comas are usually produced by either toxic (medication or drugs) or metabolic problems or by structural lesions. The clinical examination differs depending upon the cause of the coma. A careful neurologic examination can usually establish the diagnosis and lead to appropriate management.

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Evaluation

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