Chapter 168: Altered Mental Status and Coma

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INTRODUCTION

Disorders of consciousness may be divided into processes that affect either *arousal* or *content of consciousness*, or a combination of both. Arousal behaviors include wakefulness and basic alerting. Anatomically, neurons responsible for these arousal functions reside in the reticular activating system, a collection of neurons scattered through the midbrain, pons, and medulla. The neuronal structures responsible for the content of consciousness reside in the cerebral cortex. Content of consciousness includes self-awareness, language, reasoning, spatial relationship integration, emotions, and the myriad complex integration processes that make us human. One simplistic model holds that dementia is failure of the content portions of consciousness with relatively preserved alerting functions. Delirium is arousal system dysfunction with the content of consciousness affected as well. Coma is failure of both arousal and content functions. Psychiatric disorders and altered mental states may share features such as hallucinations or delusion. Some distinctions between the different states are summarized in Table 168-1.

TABLE 168-1

Features of Delirium, Dementia, and Psychiatric Disorder

Characteristic	Delirium	Dementia	Psychiatric Disorder
Onset	Over days	Insidious	Sudden
Course over 24 h	Fluctuating	Stable	Stable
Consciousness	Reduced or hyperalert	Alert	Alert
Attention	Disordered	Normal	May be disordered
Cognition	Disordered	Impaired	May be impaired
Orientation	Impaired	Often impaired	May be impaired
Hallucinations	Visual and/or auditory	Often absent	Usually auditory
Delusions	Transient, poorly organized	Usually absent	Sustained
Movements	Asterixis, tremor may be present	Often absent	Absent

Mental status is the clinical state of emotional and intellectual functioning of the individual. The mental status evaluation may be divided into six areas (Table 168-2). Testing the mental status is done both formally and informally in patient evaluation by emergency physicians.[1] Assessment of higher mental or cognitive functions requires specific tests. Screening tests are described in the Diagnosis subsections under Delirium, Dementia, and Coma.

TABLE 168-2

Six Elements of Mental Status Evaluation

Appearance, behavior, and attitude
Is dress appropriate?
Is motor behavior at rest appropriate?
Is the speech pattern normal?
Disorders of thought
Are the thoughts logical and realistic?
Are false beliefs or delusions present?
Are suicidal or homicidal thoughts present?
Disorders of perception
Are hallucinations present?
Mood and affect
What is the prevailing mood?
Is the emotional content appropriate for the setting?
Insight and judgment
Does the patient understand the circumstances surrounding the visit?
Sensorium and intelligence
Is the level of consciousness normal?
Is cognition or intellectual functioning impaired?

DELIRIUM

INTRODUCTION

Delirium, acute confusional state, acute cognitive impairment, acute encephalopathy, altered mental status, and other synonyms all refer to a transient disorder with impairment of attention and cognition. The patient has difficulty focusing, shifting, or sustaining attention. Confusion may fluctuate (Table 168-1).

The incidence of delirium in ED populations is not clear. It is estimated that 10% to 25% of elderly hospitalized patients have delirium at the time of admission.[2,3] The literature suggests that up to one quarter of all ED patients aged 70 years or older have impaired mental status or delirium and contends that routine evaluation is not satisfactory to identify many of these patients.[4,5]

PATHOPHYSIOLOGY

Pathologic mechanisms producing delirium are complex and are thought to involve widespread neuronal or neurotransmitter dysfunction. There are four general causes[6]:

1. Primary intracranial disease
2. Systemic diseases secondarily affecting the CNS
3. Exogenous toxins
4. Drug withdrawal

CLINICAL FEATURES

Delirium or acute confusional state generally develops over days. Attention, perception, thinking, and memory are all altered. Alertness is reduced as manifested by difficulty maintaining attention and focusing concentration. The patient may appear quite awake, but attention is impaired. Activity levels may be either increased or decreased. The patient may fluctuate rapidly between hypoactive and hyperactive states. **Symptoms may be intermittent, and it is not unusual for different caregivers to witness completely different behaviors within a brief time span.** The sleep-wake cycles are often disrupted, with increased somnolence during the day and agitation at night, or "sundowning." Tremor, asterixis, tachycardia, sweating, hypertension, and emotional outbursts may be present. Hallucinations tend to be visual, although auditory hallucinations can also occur.[2,3,7]

DIAGNOSIS

Both historical and physical examination findings indicating delirium are necessary to confirm the diagnosis. Obtaining a history from caregivers, spouse, or other family members is the primary method for diagnosing delirium.[2,3,7] **The acute onset of attention deficits and cognitive abnormalities fluctuating in severity throughout the day and worsening at night is virtually diagnostic of delirium.** Examine medication history, including over-the-counter medications taken and prescribed medications, in detail. Check for drug interactions. Assess for an underlying process, such as pneumonia or urinary tract infection. Ancillary testing should include serum electrolyte levels, hepatic and renal studies, urinalysis, CBC, and a chest radiograph. Order a head CT if a mass lesion such as subdural hematoma is suspected; follow this by performing a lumbar puncture if meningitis or subarachnoid hemorrhage is considered.

One key tool for detecting delirium is the mental status examination and other cognitive screening instruments.[8,9,10,11,12] These tests are valuable in directing the physician to study aspects of attention and memory that might not otherwise be formally tested. Parts of the Mini-Mental State Examination are sometimes used in the ED.[8,9,10] The complete test takes 7 to 10 minutes to administer, but copyright restrictions limit reproduction and use.[11] Age, education, chronic cognitive impairment, and verbal abilities all may affect scores. The Mini-Mental State Examination does not detect mild impairment. The median positive and negative likelihood ratios for the test are 6.3 and 0.19, respectively.[11] Several other shorter evaluation tools have been proposed. [9,10,11,12] The **Quick Confusion Scale** has been tested in ED patients, yields scores that correlate well with those on the Mini-Mental State Examination ($r = 0.61$ in one study[10] and

0.783 in another[9]), and takes <3 minutes to administer. The patient does not need to read, write, or draw to complete the test[10] (Table 168-3).

TABLE 168-3

The Quick Confusion Scale

Item	Score (Number Correct)	¥ (Weight)	=	(Total)
What year is it now?	0 or 1 (Score 1 if correct, 0 if incorrect)	× 2	=	_____
What month is it?	0 or 1	× 2	=	_____
(Present memory phrase): Repeat this phrase after me and remember it: "JohnBrown, 42Market Street, New York."				
About what time is it? (Answer correct if within 1 h.)	0 or 1	× 2	=	_____
Count backward from 20 to 1.	0, 1, or 2	× 1	=	_____
Say the months in reverse.	0, 1, or 2	× 1	=	_____
Repeat the memory phrase (each underlined portion correct is worth 1 point).	0, 1, 2, 3, 4, or 5	× 1	=	_____
Final score is the sum of the totals.			=	_____
Notes:				
15 is the top score; a score <15 may indicate the need for additional assessment.				
Scores highest number in category indicates correct response; lower scoring indicates increased number of errors.				
Item 1. What year is it now?	Score 1 if answered correctly, 0 if incorrect.			
Item 2. What month is it?	Score 1 if answered correctly, 0 if incorrect.			

Item	Score (Number Correct)	¥ (Weight)	=	(Total)
Item 3. About what time is it?	Answer considered correct if within 1 h; score 1 if correct, 0 if incorrect.			
Item 4. Count backward from 20 to 1.	Score 2 if correctly performed; score 1 if one error, score 0 if two or more errors.			
Item 5. Say the months in reverse.	Score 2 if correctly performed; score 1 if one error, score 0 if two or more errors.			
Item 6. Repeat memory phrase: "JohnBrown, 42Market Street, New York."	Each underlined portion correctly recalled is worth 1 point in scoring; score 5 if correctly performed; each error drops score by 1.			
Final score is the sum of the weighted totals; items 1, 2, and 3 are multiplied by 2 and summed with the other item scores to yield the final score.				

Depression may resemble hypoactive delirium, with withdrawal, slowed speech, and poor results on cognitive testing present in both conditions. However, rapid fluctuation of symptoms is common in delirium but generally absent in depression. In addition, clouding of consciousness is absent in patients with depression. **Patients with depression are oriented and able to perform commands.**

An unusual cause of confusional state, but one that is suspected to be underrecognized, is **nonconvulsive status epilepticus**, or **complex partial status epilepticus**. This twilight state may persist for hours or even months. Suspicion and electroencephalography are required for recognition.[10] (See chapter 171, Seizures, for further discussion of nonconvulsive status epilepticus.)

TREATMENT

Direct treatment at the underlying cause. Common medical causes of delirium are listed in **Table 168-4**. Multiple causes may be present in a given patient.

TABLE 168-4

Important Medical Causes of Delirium

Infectious	Pneumonia Urinary tract infection Meningitis or encephalitis Sepsis
Metabolic/toxic	Hypoglycemia Alcohol ingestion Electrolyte abnormalities Hepatic encephalopathy Thyroid disorders Alcohol or drug withdrawal
Neurologic	Stroke or transient ischemic attack Seizure or postictal state Subarachnoid hemorrhage Intracranial hemorrhage CNS mass lesion Subdural hematoma
Cardiopulmonary	Congestive heart failure Myocardial infarction Pulmonary embolism Hypoxia or carbon dioxide narcosis
Drug related	Anticholinergic drugs Alcohol or drug withdrawal Sedatives-hypnotics Narcotic analgesics Selective serotonin or serotonin-norepinephrine reuptake inhibitors Polypharmacy

Environmental manipulations such as adequate lighting, psychosocial support, and mobilization may be helpful in enhancing the patient's ability to interpret the surroundings correctly.[2,6,7] Sedation may be needed. Haloperidol is a frequent initial choice at a dose of 5 to 10 milligrams PO, IM, or IV with reduced dosing of 1 to 2 milligrams in the elderly. Repeat at 20- to 30-minute intervals as needed. Benzodiazepines such as lorazepam, 0.5 to 2.0 milligrams PO, IM, or IV, may be used in combination with haloperidol in doses

of 1 to 2 milligrams, with the dose varying widely depending on the age and size of the patient and the degree of agitation. Chapter 287, Acute Agitation, provides further discussion on management of agitation.

DISPOSITION AND FOLLOW-UP

Admit the patient into the hospital for further treatment and additional diagnostic testing unless a readily reversible cause for the acute mental status change is discovered and treatment initiated. This decision is individualized with consideration of patient characteristics, the resources in the home or healthcare facility, and the patient's safety.

DEMENTIA

INTRODUCTION

Dementia implies a loss of mental capacity. Psychosocial level and cognitive abilities deteriorate, and behavioral problems develop. The largest categories of dementia are idiopathic dementia (Alzheimer's disease) and vascular dementia, which are essentially diagnoses of exclusion. However, other, more treaTable disorders may cause or simulate dementia.

The typical course of dementia is slow with insidious symptom onset. The abrupt onset of symptoms or rapidly progressive symptoms should prompt a search for other diagnoses. Presentation to the ED is usually precipitated by some sentinel event. Hallucinations, delusions, repetitive behaviors, and depression are all common.

PATHOPHYSIOLOGY

Most cases of dementia in the United States are due to Alzheimer's disease, a neurodegenerative disorder of unknown etiology. The pathophysiology is complex, with a reduction in neurons in the cerebral cortex, increased amyloid deposition, and the production of neurofibrillary tangles and plaques. Other neurodegenerative diseases have their own unique pathologies. Vascular dementia accounts for the next largest number of dementia cases. The pathology is that of cerebrovascular disease with multiple infarctions. A listing of different types of dementia is provided in Table 168-5.

TABLE 168-5

Classification of Dementia by Cause

Degenerative
Alzheimer's disease
Huntington's disease
Parkinson's disease
Vascular
Multiple infarcts
Hypoperfusion (cardiac arrest, profound hypotension, others)
Subdural hematoma
Subarachnoid hemorrhage
Infectious
Meningitis (sequelae of bacterial, fungal, or tubercular)
Neurosyphilis
Viral encephalitis (herpes, human immunodeficiency virus), Creutzfeldt-Jakob disease
Inflammatory
Systemic lupus erythematosus
Demyelinating disease, others
Neoplastic
Primary tumors and metastatic disease
Carcinomatous meningitis
Paraneoplastic syndromes
Traumatic
Traumatic brain injury
Subdural hematoma
Toxic
Alcohol
Medications (anticholinergics, polypharmacy)
Metabolic
Vitamin B_{12} or folate deficiency
Thyroid disease
Uremia, others
Psychiatric
Depression (pseudodementia)
Hydrocephalic
Normal-pressure hydrocephalus (communicating hydrocephalus)
Noncommunicating hydrocephalus

CLINICAL FEATURES

Impairment of memory, particularly recent memory, is gradual and progressive. Remote memories are often preserved. Impairment of memory and orientation with preservation of motor and speech abilities is said to be characteristic of the onset of dementia associated with Alzheimer's disease. Degenerative dementias are divided into early, middle, and late stages. Early in the disease, complaints of memory loss, naming problems, or forgetting of items is common. The middle stage shows progression of these problems plus loss of reading, decreased performance in social situations, and loss of direction. The late stage of the illness may include extreme disorientation, inability to perform self-care tasks, and personality change. Clinical features may include affective symptoms such as depression and anxiety, behavioral disorders, and speech difficulties.

Patients with vascular or multi-infarct dementia often show similar symptoms, but may have findings on physical examination of exaggerated or asymmetric deep tendon reflexes, gait abnormalities, or weakness of an extremity.

DIAGNOSIS

General physical examination does not determine the diagnosis of dementia but may be helpful in identifying associated causes.[13] The presence of focal neurologic signs may suggest vascular dementia or a mass lesion. Increased motor tone and other extrapyramidal signs such as rigidity or a movement disorder may suggest Parkinson's disease. Perform mental status testing as outlined earlier.

Laboratory assessment typically includes a CBC, comprehensive metabolic profile, urinalysis, thyroid function tests, serum vitamin B_{12} level, and serologic testing for syphilis (in patients at risk).[14,15] Other helpful laboratory tests may include erythrocyte sedimentation rate, serum folate level, human immunodeficiency virus testing, and chest radiography. Consider CT or MRI imaging in every patient at some point in the diagnostic evaluation. Perform lumbar puncture if the diagnosis is not readily apparent.

Diagnosis of probable vascular dementia requires signs of cerebrovascular disease. The relationship between stroke and cognitive decline must be temporally related, with dementia within 3 months of the stroke or abrupt deterioration in memory and other cognitive abilities. A fluctuating, stepped course suggests vascular dementia.[16]

The possibility of a concurrent medical condition suddenly causing cognitive functioning to deteriorate should be strongly considered and often is the thrust of investigation in the ED. Urinary tract infection, congestive heart failure, and hypothyroidism are just a few of the conditions that may cause a mildly demented but functioning individual to show rapid decline. The symptoms overlap with those of delirium as discussed earlier in the Clinical Features section under Delirium, and the two conditions may overlap.[3,7] Consider depression imitators, the so-called treaTable causes of dementia (included in **Table 168-5**), in the differential diagnosis.

Depression may coexist with dementia, and also consider depression-imitating dementia (pseudodementia). Arrange appropriate inpatient or outpatient follow-up for further behavioral health evaluation. If depression is severe, consider hospital admission.

TREATMENT

All types of dementia are treatable, at least to some degree, by environmental or psychosocial interventions. Antipsychotic drugs are used to manage psychotic and nonpsychotic behaviors, but adverse drug effects complicate treatment. Reserve antipsychotic drugs for patients with persistent psychotic features or those with extreme disruptive or dangerous behaviors.[3,17] Coordinate treatment with caregivers who are in a position to monitor the patient's behavior patterns over time.

Treatment of vascular dementia is limited to treatment of risk factors, including hypertension.

Normal-pressure hydrocephalus is suggested by the presence of excessively large ventricles on head CT and can prompt consideration of a trial of lumbar puncture with cerebrospinal fluid drainage or ventricular shunting. **Consider normal-pressure hydrocephalus if urinary incontinence and gait disturbance develop early in the disease process.**

DISPOSITION AND FOLLOW-UP

A new diagnosis of dementia may be entertained in the ED, but the depth of the required diagnostic evaluation usually exceeds the time available during the ED visit. A decision to admit or to arrange an outpatient diagnosis is the usual disposition after the major differential diagnostic possibilities have been eliminated. Direct attention toward the presence of delirium or a treaTable cause of dementia. Consider hospital admission if comorbid medical problems, a rapidly progressive or atypical clinical course, or an unsafe or uncertain home situation exists.

COMA

INTRODUCTION

Coma is a state of reduced alertness and responsiveness from which the patient cannot be aroused.[18,19] The Glasgow Coma Scale (**Table 168-6**; see also chapter 257, Head Trauma) is a widely used clinical scoring system for alterations in consciousness. Advantages are the simplicity of the scoring system and assessment of separate verbal, motor, and eye-opening functions. Disadvantages include lack of acknowledgment of hemiparesis or other focal motor signs and lack of testing of higher cognitive functions. Interrater variability has been noted in assessments using the **Glasgow Coma Scale**.[20] Another coma scale, the FOUR (*F*ull *O*utline of *U*n*R*esponsiveness) score, has been used in intensive care units and has the advantages of assessing simple brainstem functions and respiratory patterns, as well as eye and motor responses.[21] Causes of coma likely to be encountered in the ED are noted in **Table 168-7**.

TABLE 168-6

Glasgow Coma Scale

Component	Score	Adult	Child <5 y	Child >5 y
Motor	6 5 4 3 2 1	Follows commands Localizes pain Withdraws to pain Flexion Extension None	Normal spontaneous movements Localizes to supraocular pain (>9 mo) Withdraws from nail bed pressure Flexion to supraocular pain Extension to supraocular pain None	Follows commands
Verbal	5 4 3 2 1	Oriented Confused speech Inappropriate words Incomprehensible None	Age-appropriate speech/vocalizations Less than usual ability; irritable cry Cries to pain Moans to pain No response to pain	Oriented Confused Inappropriate words Incomprehensible
Eye opening	4 3 2 1	Spontaneous To command To pain None	Spontaneous To voice To pain None	

TABLE 168-7

Differential Diagnosis of Coma

Coma from causes affecting the brain diffusely

Encephalopathies

Hypoxic encephalopathy

Metabolic encephalopathy

Hypertensive encephalopathy

Hypoglycemia

Hyperosmolar state (e.g., hyperglycemia)

Electrolyte abnormalities (e.g., hypernatremia or hyponatremia, hypercalcemia)

Organ system failure

Hepatic encephalopathy

Uremia/renal failure

Endocrine (e.g., Addison's disease, hypothyroidism, etc.)

Hypoxia

Carbon dioxide narcosis

Toxins

Drug reactions (e.g., neuroleptic malignant syndrome)

Environmental causes—hypothermia, hyperthermia

Deficiency state—Wernicke's encephalopathy

Sepsis

Coma from primary CNS disease or trauma

Direct CNS trauma

Diffuse axonal injury

Subdural hematoma

Epidural hematoma

Vascular disease

Intraparenchymal hemorrhage (hemispheric, basal ganglia, brainstem, cerebellar)

Subarachnoid hemorrhage

Infarction

Hemispheric, brainstem

CNS infections

Neoplasms

Seizures

Nonconvulsive status epilepticus

Postictal state

PATHOPHYSIOLOGY

The pathophysiology of coma is complex. Coma can result from deficiency of substrates needed for neuronal function (as with hypoglycemia or hypoxia). With systemic causes, the brain is globally affected, and signs that localize dysfunction to a specific area of the brainstem or cortex are usually lacking. With primary CNS causes, coma may result from a brainstem disorder such as hemorrhage or from bilateral cortical dysfunction. Signs localizing to specific areas of CNS dysfunction such as hemiparesis or cranial nerve abnormalities may be present. Unilateral hemispheric disease, such as stroke, should not alone result in coma. The function of the brainstem and/or both hemispheres must be impaired for unresponsiveness to occur.

The herniation syndromes are models for alterations of consciousness, but their mechanisms are unknown. In **uncal herniation syndrome**, the medial temporal lobe shifts to compress the upper brainstem, which results in progressive drowsiness followed by unresponsiveness.[19,22] The ipsilateral pupil is sluggish, eventually becoming dilated and nonreactive as the third cranial nerve is compressed by the medial temporal lobe. Hemiparesis may develop ipsilateral to the mass from compression of the descending motor tracts in the opposite cerebral peduncle. **Central herniation syndrome** is characterized by progressive loss of consciousness, loss of brainstem reflexes, decorticate posturing, and irregular respiration.[19] Because midline shift without herniation, as demonstrated by neuroimaging, seems to correlate with a decreased level of consciousness, vascular compression due to local cerebral edema or local increased intracranial pressure (ICP) may be an underlying mechanism for these syndromes.

A diffuse increase in ICP can cause diffuse CNS dysfunction. Cerebral blood flow is constant at mean arterial pressures (MAPs) of 50 to 100 mm Hg due to the process of cerebral autoregulation. At MAPs outside this range, cerebral blood flow may be reduced, and diffuse ischemia may develop. **Cerebral perfusion pressure is equal to the MAP minus the ICP (cerebral perfusion pressure = MAP – ICP).** In extreme uncontrolled elevation of the ICP, cerebral perfusion pressure is diminished as the ICP approaches the MAP, which causes brain ischemia.

Particularly in unresponsive patients with a history of seizures, the possibility of ongoing nonconvulsive seizures must be considered. Subtle status epilepticus or ictal coma may represent transformed generalized convulsive status epilepticus. Electrical seizures may continue in the absence of clinical seizures.[23]

CLINICAL FEATURES

The clinical features of coma vary with both the depth of coma and the cause. For example, a patient in a coma with a hemispheric hemorrhage and midline shift may have decreased muscle tone on the side of the hemiparesis. The eyes may conjugately deviate toward the side of the hemorrhage. **With expansion of the**

hemorrhage and surrounding edema, increase in ICP, or brainstem compression, unresponsiveness may progress to a complete loss of motor tone and loss of the ocular findings as well.

A variety of abnormal breathing patterns may be seen in the comatose patient.[19] They offer little information in the acute setting. Pupillary findings, the results of other cranial nerve evaluation, hemiparesis, and response to stimulation are all part of the clinical picture that need assessment. These findings can assign the cause of the coma into a probable general category—diffuse CNS dysfunction (toxic-metabolic coma) or focal CNS dysfunction (structural coma). A further division of structural coma into hemispheric (supratentorial) or posterior fossa (infratentorial) coma is often possible at the bedside.

Toxic-Metabolic Coma

Many different toxic and metabolic conditions cause coma. The diffuse CNS dysfunction is reflected by the lack of focal physical examination findings that point to a specific region of brain dysfunction. For example, in toxic-metabolic coma, if the patient demonstrates either spontaneous movements or reflex posturing, the movements are symmetric without evidence of hemiparesis. Muscle stretch reflexes, if present, are symmetric. Pupillary response is generally preserved in toxic-metabolic coma. Typically the pupils are small but reactive. If extraocular movements are present, they are symmetric. If extraocular movements are absent, however, this sign is of no value in differentiating toxic-metabolic from structural coma. A noTable exception is severe sedative poisoning as from barbiturates; the pupils may be large, extraocular movements absent, muscles flaccid, and the patient apneic, which simulates the appearance of brain death.

Coma from Supratentorial Lesions

Coma caused by lesions of the hemispheres, or supratentorial masses, may present with progressive hemiparesis or asymmetric muscle tone and reflexes. The hemiparesis may be suspected with asymmetric responses to stimuli or asymmetric extensor or flexor postures. Uncal herniation syndrome, as described earlier in Pathophysiology, is an example of a supratentorial syndrome. Frequently, however, large acute supratentorial lesions are seen without the features consistent with temporal lobe herniation. Coma without lateralizing signs may result from decreased cerebral perfusion secondary to increased ICP. Reflex changes in blood pressure and heart rate may be observed with increased ICP or brainstem compression. Hypertension and bradycardia in a comatose patient may represent the Cushing reflex from increased ICP.

Coma from Infratentorial Lesions

Posterior fossa or infratentorial lesions comprise another structural coma syndrome. **An expanding lesion, such as cerebellar hemorrhage or infarction, may cause abrupt coma, abnormal extensor posturing, loss of pupillary reflexes, and loss of extraocular movements.** The anatomy of the posterior fossa leaves little room for accommodating an expanding mass. Early brainstem compression with loss of brainstem reflexes may develop rapidly. Another infratentorial cause of coma is pontine hemorrhage, which may present with the unique signs of pinpoint-sized pupils.

Pseudocoma

Pseudocoma or psychogenic coma is occasionally encountered and may present a perplexing clinical problem. Adequate history taking and observation of responses to stimulation reveal findings that differ from those in the syndromes described in the previous sections. Pupillary responses, extraocular movements, muscle tone, and reflexes are shown to be intact on careful examination. Tests of particular value include responses to manual eye opening (there should be little or no resistance in the truly unresponsive patient) and extraocular movements. Specifically, if avoidance of gaze is consistently seen with the patient always looking away from the examiner, or if nystagmus is demonstrated with caloric vestibular testing, this is strong evidence for nonphysiologic or feigned unresponsiveness.

DIAGNOSIS

In the approach to the comatose patient, perform stabilization, diagnosis, and treatment actions simultaneously. Examination, laboratory procedures, and neuroimaging allow differentiation between structural and metabolic causes of coma in almost all patients in the ED. History and physical examination findings allow that initial assignment in many patients, but liberal use of CT scanning is encouraged because exceptions to the tentative clinical diagnosis are frequent.

Address airway, breathing, and circulation immediately. Consider reversible causes of coma, such as hypoglycemia or opiate overdose. Access all available historical sources (EMS personnel, caregivers, family, witnesses, medical records, etc.) to aid in diagnosis.

The tempo of onset of the coma is of great diagnostic value. Abrupt coma suggests abrupt CNS failure with possible causes such as catastrophic stroke or seizures. A slowly progressive onset of coma may suggest a progressive CNS lesion such as tumor or subdural hematoma. Metabolic causes, such as hyperglycemia, may also develop over several days.

Address general examination and measurement of vital signs (including oxygen saturation and temperature) following stabilization and resuscitation. General examination may reveal signs of trauma or suggest other diagnostic possibilities for the unresponsiveness. For example, a toxidrome may be present that suggests diagnosis and treatment, such as the opiate syndrome with hypoventilation and small pupils.

Neurologic testing deviates from the standard examination. Fine tests of weakness, such as testing for pronator drift of the outstretched upper extremities, are not possible in the unresponsive patient. However, asymmetric findings on examination of cranial nerves through pupillary examination, assessment of corneal reflexes, and testing of oculovestibular reflexes may suggest focal CNS lesions. Abnormal extensor or flexor postures are nonspecific for localization or cause of coma but suggest profound CNS dysfunction. Asymmetric muscle tone or reflexes raise the suspicion of a focal lesion. **The goal of the physician is to rapidly determine if the CNS dysfunction is from diffuse impairment of the brain or if signs point to a focal (and perhaps surgically treatable) region of CNS dysfunction.**

CT is the neuroimaging procedure of choice. Acute hemorrhage is readily identified, as is midline shift and mass lesions. Consider lumbar puncture if CT scan findings are unremarkable and subarachnoid hemorrhage or CNS infection is suspected. Suspect basilar artery thrombosis in a comatose patient with "normal" results on head CT, in which the only finding may be a hyperdense basilar artery.[24] MRI or cerebral angiography is needed to make the diagnosis of basilar artery thrombosis.

SPECIAL CONSIDERATIONS IN COMA

If trauma is suspected, maintain stabilization of the cervical spine during assessment. If protection of the airway is in doubt or the coma state is likely prolonged, then protect the airway by intubating the patient. Rapid-sequence intubation techniques are discussed at length in chapter 29, Intubation and Mechanical Ventilation. **ingestions, infections, and child abuse in the appropriate clinical setting.**

Patients who have had generalized seizures and remain unresponsive may be in a continuing state of electrical seizures without corresponding motor movements. This is called **nonconvulsive status epilepticus** or **subtle status epilepticus** and can be described as electromechanical dissociation of the brain and body. **If the motor activity of the seizure stops and the patient does not awaken within 30 minutes, then consider nonconvulsive status epilepticus.** Obtain neurologic consultation and electroencephalography.[23]

TREATMENT

Treatment of coma involves identification of the cause of the brain failure and initiation of specific therapy directed at the underlying cause. Attend to airway, ventilation, and circulation. Evaluate and treat for readily reversible causes of coma, such as hypoglycemia and opioid toxicity.

Antidotes

Rapid point-of-care glucose determination can identify the need for dextrose. Although thiamine should be administered before glucose infusion in patients with a suspected history of alcohol abuse or malnutrition, thiamine is not necessary for all patients. Routine use of flumazenil in coma of unknown cause is not recommended.[25] Naloxone, the opiate antagonist, is useful in coma because typical signs of opiate overdose may be absent.

Increased Intracranial Pressure

If history, physical examination, or neuroimaging findings suggest increased ICP, specific steps can reduce or ameliorate any further rise in ICP. Any noxious stimulus, including "bucking" the ventilator, can increase ICP, so use paralytic and sedative agents. A general recommendation is to keep the head elevated about 30 degrees and at midline to aid in venous drainage. **Mannitol** (0.5 to 1.0 gram/kg IV) can decrease intravascular volume and brain water and may transiently reduce ICP. In cases of brain edema associated with tumor, dexamethasone, 10 milligrams IV, reduces edema over several hours. Hyperventilation with reduction of partial pressure of arterial carbon dioxide can reduce cerebral blood volume and transiently lower ICP.

Current recommendations are to avoid excessive hyperventilation (partial pressure of arterial carbon dioxide ≤35 mm Hg) during the first 24 hours after brain injury. Brief hyperventilation may be necessary for refractory intracranial hypertension. Data to recommend specific therapy are lacking, and preferences among individuals and institutions vary greatly, so communicate early with consultants and admitting physicians.

DISPOSITION AND FOLLOW-UP

Patients with readily reversible causes of coma, such as insulin-induced hypoglycemia, may be discharged if home care and follow-up care are adequate and a clear cause for the episode is suspected. Admit patients with persistent altered consciousness. Most institutions depend on emergency physicians to stabilize the patient's condition and correctly assign a tentative diagnosis so that the patient may be admitted to the proper specialty service. If the appropriate service is not available, then consider transfer to another hospital after patient stabilization.

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USEFUL WEB RESOURCES

Foundation for Education and Research in Neurological Emergencies—http://www.ferne.org

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