Chapter 10 – Common Neurologic Emergencies

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This chapter will address common neurologic emergencies: coma, stroke, and status epilepticus. These are time-sensitive situations in which effective therapy may lessen or completely reverse a potentially catastrophic insult to the central nervous system.

Coma may be anticipated as an integral component of the terminal event of all fatal human illnesses. However, coma, which represents failure of the brain’s alerting system, may be completely reversible. The earlier the process inciting the comatose state is treated, the greater the likelihood of a more rapid complete recovery. The differential diagnoses of the causes of coma are numerous and varied. While proceeding towards the correct diagnosis with a directed history, physical examination, and prioritized laboratory investigations, therapies that may be antidotes to the cause of the coma are administered. A systematic approach is described which ensures reaching the definitive diagnosis promptly.

Ischemic stroke, the most common form of stroke, has one therapeutic intervention, which increases the likelihood of a complete or near-complete recovery without increasing morbidity or mortality. The thrombolytic agent, tissue plasminogen activator (tPA), must be administered within 3 hours of stroke onset; the sooner after stroke onset tPA is given, the greater its therapeutic efficacy. However, tPA administration is associated with a definite risk. Thus, the selection of eligible patients requires an extremely rapid response system and strict adherence to inclusion and exclusion criteria. A user-friendly checklist is provided.

Convulsive Status Epilepticus (continuous generalized tonic-clonic seizure activity) needs to be aborted in less than 60 minutes which, depending on the etiology of the seizures, improves the chances of a good outcome. A step-by-step annotated timeline is described below.

Coma

The comatose patient manifests a depressed level of consciousness. To be conscious, the patient has to be both awake and aware. Patients with severe anoxic cortical damage, with brainstem sparing, exhibit wakefulness and sleep but are not aware and are, therefore, unconscious. This is the hallmark of a
vegetative state. Once awareness is impaired, the level of consciousness is described as depressed or altered. Terms like lethargy, obtundation, stupor, and coma are used to indicate varying degrees of depression of normal physiologic alertness. The term ‘coma’ means a deep depression in the state of ‘altered level of consciousness.’ Coma is characterized by the patient’s arousal response to verbal or painful stimuli. The degree of decrease in level of consciousness correlates with the severity of the disease process and dictates urgency of response and type of treatment. The altered level of consciousness may indicate a primary effect on the brain or it may be a sign of other serious illness such as septic shock.

Alertness or arousal depends on an intact reticular formation or reticular activating system (RAS) running through the brainstem from which it then projects to the thalami and on to both cortical cerebral hemispheres. Thus, alteration in the level of consciousness, including coma, can be functionally localized to two areas. There are only two types of coma: brainstem or bilateral hemisphere. One or both areas can incur a chemical or structural insult resulting in an altered level of consciousness. A unilateral lesion does not itself cause an alteration in the level of consciousness, unless by mass effect it also causes significant distortion of the brainstem, which affects the functioning of the RAS in the brainstem.

For example, if there are signs of brainstem coma, such as certain abnormal eye movements or a dilated pupil due to pressure on the III cranial nerve adjacent to the brainstem, then the patient may be herniating and the situation represents a possible neurosurgical emergency. CT scanning may be needed immediately while measures to reduce increased intracranial pressure are being instituted. If these brainstem signs are absent, then the coma is probably due to nonstructural effects on the RAS or bilateral hemisphere disease suggesting a totally different course of action for diagnosis and treatment.

Case Report: Part 1

A 50-year-old man is found unconscious in a downtown park. 911 is called and the patient is transported to the emergency department. The paramedics report that the patient is comatose with a Glasgow Coma Scale of 7 (no eye opening = 1; unintelligible sounds = 2; nonspecific withdrawal movements = 4). His breathing was noisy, but improved with a jaw thrust maneuver. He has been placed on a backboard with spine precautions. His C-spine has been immobilized. Vital signs are 140/90, 90 pulse, 24 respiratory rate, pulse oximetry 94 percent. Oxygen at 6L/min by nasal cannula has been applied. An IV line is initiated. Serum glucose level is 80. Cardiac monitor shows sinus rhythm. There was no response to intravenous naloxone. There is a nearly empty bottle of Thunderbird wine in his jacket along with a full bottle of (phenytoin) Dilantin® capsules dated two weeks earlier. The patient smells of alcohol and his pants are urine stained. There are numerous healed scars about the head with encrusted sutures along his right parieto-occipital scalp. His right pupil is 6 mm and does not appear to react, while the left is 3 mm reactive. According to his buddies, he had been drinking more than usual recently to alleviate a headache. He has been lying on the ground for several hours.

Although the patient is a known alcoholic and appears to be in an alcoholic stupor or perhaps in a postictal state, the history and physical examination point to a more urgent situation. The pupil asymmetry (presumably not due
to a previous insult) suggests that coma is due to brainstem compromise with herniation. Because acute or chronic intracranial hematoma is the likely cause, the trauma team is notified.

A careful history, along with timely and appropriate interventions is necessary in the management of patients presenting with an altered level of consciousness. Examination, differential diagnosis, and treatment options are discussed below.

**History**

Obtain history from witnesses, friends, and family keeping in mind that certain interventions (see below) must be performed while gathering information. Is there any history of trauma, seizures, diabetes, allergies or other medical problem? How long has the patient been unconscious? Were there any bottles or medication containers at the scene? Is the patient taking prescribed medications? Is there anything special about the environment in which the patient was found? Indoors or outdoors? Any unusual odors? Any others in the vicinity in a similar state? Is the patient wearing a Medical Alert bracelet?

Examine clothes pockets for identification, suicide notes, or drug bottles. If possible, initiate an immediate search through any previous medical records. They will help to confirm information gathered from witnesses, family and friends, and may add vitally important data. For example, it would be unfortunate to load a patient with phenytoin who has a previous history of a Stevens-Johnson allergic reaction to the medication.

**Interventions**

*Always assume there is a C-spine fracture.* The unconscious patient may have suffered a head injury and simultaneous cervical spine injury as the initial event or during the fall when becoming unconscious. *Ensure a patent airway maintaining C-spine precautions.*

If respirations are inadequate, a jaw-thrust or chin-lift maneuver may assist respirations initially. A nasopharyngeal airway or an oropharyngeal airway may keep the airway patent prior to intubation. The comatose patient cannot protect the airway and needs to be intubated. This is obviously the case for the patient with an absent gag reflex. The patient may vomit at any time resulting in an increase in morbidity and mortality associated with aspiration pneumonia. The comatose patient may have a seizure at any time, further complicating the situation.

- Oxygen? Initially high-flow oxygen by nasal cannula or mask. Note response.
- Repeat and monitor vital signs including temperature. If the patient is hyperthermic or hypothermic, institute appropriate management.

Intravenous line—Normal Saline at a rate to maintain euvoelia is used initially if the blood pressure is normal. If blood pressure is low due to hypovolemia, then appropriate fluid replacement should be instituted. Hypertension may be secondary to elevated intracranial pressure, which should be treated before aggressive use of anti-hypertensive medications is undertaken. Bloods are sent for electrolyte and other analyses.

ECG monitor—The rhythm should be observed throughout and treated as needed. Obtain an electrocardiogram.

Thiamine—Wernicke’s encephalopathy may present as coma; the treatment is
thiamine. Giving glucose to a patient who appears malnourished and thiamine depleted, as occurs in alcoholics, may precipitate Wernicke’s encephalopathy. In such patients, administer 100mg of thiamine by intravenous injection before giving glucose. Note response.

Glucose—If the patient is hypoglycemic, administer 50 cc D50W intravenously. In children give 2 cc/kg of D25W. If the patient is not hypoglycemic, giving a glucose load to a patient with a stroke or other brain injury may aggravate the brain damage. Note Naloxone—Give 2 mg intravenously. Be prepared for the patient who is a narcotic overdose individual to awaken in response to the naloxone and to become combative and resist further medical evaluation. Note response.

Flumazenil—If pure benzodiazepine overdose is definite, administer 0.2 mg/min up to a maximum of 1 mg IV. If the patient ingested other drugs, flumazenil may induce seizures. Note response.

- Review responses to glucose, thiamine, naloxone, flumazenil and oxygen.
- With the airway protected, an orogastric tube for gastric lavage is inserted and activated charcoal is instilled when there is a possibility of a toxic ingestion.
- A Foley catheter is inserted for obtaining urine for laboratory tests and monitoring urine output.

Observe for status epilepticus. A rhythmical twitching of some of the digits of either hand or a rhythmical small amplitude horizontal jerking of the eyes may be the only clue that the patient is in status epilepticus. If the patient is having seizures, treat accordingly (see Chapter 7: Episodic Disorders).

If meningitis is suspected, perform a lumbar puncture. If there are signs of increased intracranial pressure or focality on examination, the LP may be temporarily withheld pending results of a CT scan. Appropriate antibiotics should be initiated prior to LP if there is going to be any delay in obtaining the CT scan in patients suspected of having bacterial meningitis.

If a unilaterally dilated pupil (sluggish or unresponsive to light) is present, suggesting uncal cerebral herniation, the patient is hyperventilated to a pCO2 of about 35 mm Hg and given mannitol IV at 1 gram/kg as a temporizing measure for the increased intracranial pressure. Obtain a head CT scan while neurosurgery consultation is requested emergently.

**Physical Examination**

What is the patient’s level of consciousness? What are the size of the pupils and their response to light? Is there evidence of trauma?

1. Confirm the comatose state. Voice, touch or noxious stimuli (pressure to sternum or to nail bed of middle finger of each hand, and to the supraorbital nerve) should be used to arouse the patient. Observe and record response.

2. Use the **Glasgow Coma Scale** to assess the degree of coma prior to intubation and the use of paralytic or sedative agents. Compare the Glasgow Coma Scale scores.
### Table 10-1: Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
<th>Eye Opening</th>
</tr>
</thead>
<tbody>
<tr>
<td>To verbal command</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Best Motor Response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws to pain</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Best Verbal Response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Oriented converses</td>
<td>5</td>
</tr>
<tr>
<td>Disoriented</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
</tr>
</tbody>
</table>

The worst score obtainable is not 0, but 3. Note that score for the motor response is based on the best response so that a hemiplegia on one side with a normal contralateral side receives a motor score of 6. A patient with quadriplegia from a spinal cord injury receives a motor score of 1. The Glasgow Coma Scale provides a useful standard for comparison to help determine if there is deterioration, improvement, or no change in the patient’s level of consciousness.

### Components of the Physical Examination

#### Vital Signs

**Blood pressure:** Presence of hypotension requires immediate management. Hypertension with diastolic pressure of at least 140 may indicate hypertensive encephalopathy. Consider eclampsia at a lower blood pressure in the pregnant patient. Cushing’s triad of hypertension, bradycardia, and bradypnea is seen in acute marked increased intracranial pressure. Cerebral perfusion pressure (CPP = mean arterial pressure minus intracranial pressure) should be maintained at a minimum of 60 mm Hg.

**Respirations:** Observe the pattern. Hyperventilation may indicate metabolic acidosis. Cheyne-Stokes,(crescendo-decrescendo followed by apnea), ataxic (irregular rate and depth), or apneustic (pause at inspiration) breathing may be present indicating different CNS levels of dysfunction. **Temperature:** Fever may be associated with CNS or systemic infection. Fever may also be a symptom of CNS hemorrhage or status epilepticus. Consider heat stroke, environmental causes, as well as thyroid disease in hyperv/hypothermia.

#### Head

**Skull palpation:** Look for hemotympanum. Battle sign (post-auricular ecchymosis) and raccoon eyes are signs of basal skull fracture, which take hours to develop.

**Inspect mouth:** Note any toxic/metabolic odors on breath. Tongue laceration...
may indicate a seizure disorder.

**Neck**

*Look for meningismus:* When the C-spine has been cleared, examine for nuchal rigidity, Brudzinski, or Kernig sign. If present, a lumbar puncture should be performed looking for meningitis or subarachnoid hemorrhage. A CT scan should be obtained prior to LP in the presence of increased intracranial pressure, papilledema, a history of head trauma, or suspicion of a CNS lesion causing focal lateralizing neurologic signs such as hemiparesis. Begin antibiotic therapy for meningitis and re-evaluate after CT scan. In the comatose patient meningismus (nuchal rigidity) may disappear.

**Eyes (see special section on Pupils and Extraocular Movements below)**

*Pupil size, symmetry, reactivity:* After ABCs (airway, breathing and circulation) and spine immobilization, the pupils are checked. Tiny pinpoint pupils are commonly caused by opiates. Pontine lesions may also cause pinpoint pupils, but are associated with other brainstem cranial nerve signs. Cholinesterase inhibitor, organophosphate insecticide poisoning and clonidine overdose also cause miotic pupils. Notice eyelid fluttering or the presence of tone with voluntary eyelid control in psychogenic coma.

*Extraocular movements:* Instruct all patients who appear comatose to open their eyes and look up. These voluntary movements may be all that patients with locked-in syndrome due to brainstem ischemic or hemorrhagic stroke may be capable of performing. These patients are sometimes erroneously diagnosed as comatose, but are fully awake and may only be able to communicate with eye blinking or eye motion. After opening the eyelids, the resting position of the eyes is noted. *Conjugate tonically deviated eyes usually indicate a large ipsilateral hemisphere lesion. Nystagmoid unidirectional jerks may be the only sign of ongoing seizure activity.*

*Funduscopic exam:* Subhyaloid hemorrhages indicate subarachnoid bleeding. Papilledema takes 12–24 hours to develop after acute increase in intracranial pressure.

**Motor System**

Look for spontaneous movements and response to noxious stimuli as well as asymmetrical movements, which may indicate a hemiparesis. Finger twitching may be only residual of ongoing seizure activity. Observe for decorticate and decerebrate posturing.

**Skin**

Look for petechia, ecchymoses, presence or absence of sweating, skin changes, or needle marks.

**Lungs, Cardiac, Abdomen**

Auscultate and palpate looking for systemic illnesses and secondary effects of CNS insults, e.g., neurogenic pulmonary edema.

**Extremities**

Observe position of limbs; an out-turned leg may be due to hemiparesis or a hip fracture.
Special Section: Pupils and Extraocular Movements

The brainstem is small and compact; it imparts the pupillary light reflex, houses the extraocular muscle nuclei and their connections, and the reticular activating system (RAS).

The size of the pupils is maintained by a balance between the parasympathetic and sympathetic autonomic nervous systems. Normal pupils are equal in size within one millimeter and equally change in response to light or dark. The light stimulus to either eye travels via the optic nerve, chiasm and tract to the midbrain Edinger-Westphal nucleus from which the information travels along the parasympathetic fibers running on the outside of the III (oculomotor) cranial nerve to the pupil. The III nerve also innervates the levator palpebrae muscle and the medial rectus, inferior rectus, superior rectus, and inferior oblique extraocular muscles. Anything pressing on the III nerve causes ipsilateral pupillary dilatation in addition to ptosis and eye movement disorder. This is why the unilateral dilated unreactive pupil is such a valuable sign of cerebral herniation of the uncus of the temporal lobe over the edge of the tentorium where the III nerve is running alongside on the way from the brainstem to the globe.

The sympathetic fibers also affect pupillary size. The sympathetic fibers innervate the tarsal muscles in the upper and lower eyelids and lesions can then cause the appearance of ptosis by narrowing the palpebral fissure. Horner’s syndrome, which is a sign of sympathetic fiber interruption, consists of the triad of ipsilateral ptosis, meiosis, and anhydrosis. Pupil asymmetry may also be the result of direct eye trauma, surgery, Adie’s tonic pupil, or accidental or intentional instillation of a mydriatic anticholinergic drug like scopolamine or cholinergic substances like pilocarpine.

Normally, both eyes move conjugately to maintain binocular fixation of objects. This control is mediated through the medial longitudinal fasciculus (MLF) in the brainstem, which connects the ipsilateral III nerve nucleus with the contralateral VI (abducens) nerve nucleus. The MLF ensures that movement of the ipsilateral medial rectus muscle is yoked to the contralateral lateral rectus muscle. This neurologic observation provides the basis for the vestibular-induced oculocephalic (doll’s eyes) and caloric testing of the eye movements. The cervical spine is protected in the comatose patient and doll’s eyes maneuvers can be performed when there is no suspicion of cervical spine injury. Ice water calorics provide a much more powerful and reliable stimulus than doll’s eyes maneuvers for testing the eye movement system. The eyes may be dysconjugate in sleep or with a depressed level of consciousness, but in response to an alerting stimulus or to caloric testing they become conjugate if there is no pathology of the extraocular movement system.

Case Report: Part 2

On arrival at the emergency department, the rescue squad’s findings were confirmed. The patient was comatose with a nonreactive 6 mm right pupil. In response to noxious stimuli, his left side moved much less than the right. Immediately, the C-spine was examined radiologically and the patient was intubated, slightly hyperventilated, and placed on a ventilator with 100 percent oxygen. Mannitol 100 grams was given intravenously and a CT of the head obtained. The neurosurgeon was at the bedside and the operating room staff
were notified of possible emergent surgery. Preoperative laboratory and X-ray studies were performed (see Diagnostic Adjuncts below).

**Differential Diagnosis**

The problem of patients presenting in coma is common. It may appear to be an overwhelming task to properly evaluate and manage these patients since just about everything in a textbook of medicine can result in decreased level of consciousness and coma. Regardless of the cause, the approach to these patients requires rapid assessment while instituting therapeutic and diagnostic measures, which identify and correct or amend any processes, which might lead to progressive and irreversible damage. The proper evaluation and management of these patients relies on the history, physical examination, and ancillary tests. An organized approach will nearly always give the correct diagnosis and provide the proper management sequence.

The preliminary differential diagnosis addresses the question, "Is the coma due to a primary central nervous system disease or the consequence of a systemic illness?"

This differentiation is primarily based on the presence or absence of focality (localization of a specific anatomical deficit) on the neurological examination. Focal findings strongly suggest the presence of a specific lesion. Systemic problems, such as a lack of nutrients (glucose, oxygen) or metabolic problems (sodium, calcium) or an accumulation of toxins (carbon dioxide, carbon monoxide, alcohol) cause diffuse central nervous system dysfunction. Occasionally hyponatremia, hepatic coma, or nonketotic hyperosmolar coma can present with focal neurologic signs. When there is focality on examination, a structural lesion is sought.

The first 10 items in the differential diagnosis list below are the most life- and function-threatening. Measures for therapy are instituted while emergent diagnostic techniques are undertaken to identify the cause of coma.

1. Shock or Hypertensive encephalopathy: decreased cardiac output, myocardial infarction, congestive heart failure, and pulmonary embolus
2. CO2 Narcosis or Hypoxia: pulmonary disease, hypoventilation
3. Hyperthermia or Hypothermia
4. Hypoglycemia (insulin overdose)
5. Wernicke's encephalopathy (thiamine deficiency)
6. Exogenous toxins (e.g., opiates, carbon monoxide, cyanide, barbiturates, benzodiazepines, antidepressants, antihistamines, atropine, organic phosphates, bromides, anticholinergics, ethanol, methanol, ethylene glycol, hallucinogens, ammonium chloride, heavy metals, over-the-counter drugs including salicylates)
7. Stroke (ischemic)
8. Intracranial hemorrhage (with or without trauma): subarachnoid hemorrhage, intracerebral hematoma, epidural hematoma, subdural hematoma
9. Meningitis: bacterial, syphilis, fungal, carcinomatous Encephalitis: Herpes simplex
10. Reye's syndrome (pediatric)
11. Trauma: diffuse axonal injury to the brain without significant intracranial hemorrhage
12. Tumor: CNS meningioma, glioma, and remote effects (e.g., lung cancer)
15. CNS Infections: progressive multifocal leukoencephalopathy, Creutzfeldt-Jakob disease
16. Seizures: status epilepticus, including non-convulsive; prolonged postictal state
17. Blood: anemia, sickle cell disease
18. Vascular: systemic lupus erythematosus (SLE)
19. Metabolic: hypercalcemia, uremic encephalopathy, hepatic encephalopathy, hyperosmolar state, thyrotoxicosis or myxedema coma, Cushing’s disease, pituitary apoplexy, porphyria
20. Psychiatric: especially depression; hysterical conversion reaction
21. Other: migraine, basilar (especially in children) intussusception in children

The popular mnemonic **TIPPS** on the **VOWELS** lists the frequent causes of coma.

- A alcohol
- E epilepsy
- I insulin
- O opiates
- U urea(metabolic)
- T trauma
- I infection
- P poisoning
- P psychogenic
- S shock, stroke

**Diagnostic Adjuncts**

The sequence for ordering of the laboratory tests depends on the history and physical examination. If there is a history of head trauma, the important laboratory test is the CT scan. A patient with a head injury may have had a precipitating event that caused a fall that resulted in the head injury. A diabetic patient may have been hypoglycemic; an elderly patient may have had a myocardial infarction or an arrhythmia; and a single car motor vehicle accident may have been a suicide attempt after taking a toxic ingestion.

**Laboratory Evaluation**

- CBC, differential, platelets
• Serum glucose, electrolytes, calcium, magnesium, phosphorus
• Alcohol level
• Renal function tests
• Hepatic function tests
• Arterial blood gases; carboxyhemoglobin level
• Urine for urinalysis and toxicology studies, myoglobin and porphobilinogen
• EKG
• CT/MRI scan
• Thyroid function studies
• Lumbar puncture for CSF—cells, protein, sugar, India ink prep, fungal cultures, and extra tubes as needed
• EEG (immediately if suspect status epilepticus)

Case Report: Part 3
The CT scan showed a large right parieto-occipital chronic subdural hematoma, which probably explains the patient’s recent headaches. There was fresh blood within the chronic subdural. This recent bleeding probably resulted in the patient’s acute decompensation. The patient was taken immediately to the operating room to have the subdural hematoma evacuated.

Special Conditions

Coma in Children
Head injuries in children differ in several ways from those in adults. Children in traumatic coma usually do not have a surgically correctable lesion, though one should always be sought. A relatively minor injury can have an apparent initial recovery, followed by several hours of decreased level of consciousness with waxing and waning signs, then complete recovery (post-traumatic stupor and delayed non-hemorrhagic encephalopathy).

Seizures
The postictal state in children can occasionally be prolonged and last two to three days.

Reye’s syndrome
This post viral illness tends to be associated with salicylate ingestion and presents with decreased level of consciousness and elevated ammonia levels. Due to a decrease in use of aspirin for fever in children, it is now less common.

Coma in the elderly
Ischemic stroke is more common in the elderly. Basilar artery thrombosis impairs brainstem perfusion and can cause coma at onset. Large hemisphere ischemic strokes may develop massive cerebral edema and result in compression of the
brainstem over days from onset. Cerebellar hemisphere strokes (ischemic or hemorrhagic) can result in coma over hours to days.

- Chronic subdural hematomas present much more commonly in the elderly and about half the time a history of head trauma, which may be very minor, is obtained.
- Hypothyroidism must always be considered in the elderly.
- The elderly patient can be very sensitive to medications including sedative hypnotic medications.
- The elderly may accidentally, or intentionally, take an overdose of drugs.
- The postictal state following seizures may be prolonged in the elderly patient.

**Narcotics**

Naloxone will reverse the coma, respiratory depression and meiosis of opiates. The presence of pinpoint pupils, skin track marks, and a history of intravenous drug abuse all point to opiate overdose. Even in the absence of meiosis, check for accidental overdose. A child may have gotten into the parent’s medication or an elderly person may have taken too much acetaminophen with codeine. The narcotic overdose patient will awaken in response to naloxone, and possibly become combative and resist further medical evaluation. Remember that the naloxone duration of action is one hour and the opiate taken may have a much longer half-life (methadone or propoxyphene). Patients may, consequently, lapse back into coma. Do not forget the need for evaluation of possible complications, which occurred during the comatose state (e.g. aspiration which could lead to pneumonia).

**Alcohol**

Patients who are in coma due to alcohol intoxication need to be observed and monitored until they fully recover their normal state. The problem of alcoholism should be addressed prior to discharge, preferably by a social worker.

**Insulin**

Patients presenting in coma due to a hypoglycemic reaction secondary to an insulin overdose, who have returned to their normal mental state, do not require hospitalization. However, their diabetes management must be re-evaluated so they can avoid a recurrence. Patients that have taken long-acting oral hypoglycemic agents may relapse and need to be observed.

**Psychogenic**

Psychogenic coma does occur, but is uncommon. Usually it resolves with patience and support. Psychogenic seizures, which can be difficult to diagnose by even the most astute neurologists, are probably the most common form of hysterical coma. Clues to the diagnosis are the presence of eyelid fluttering, Bell’s phenomenon (elevation of the globes when eyelid opening is resisted by the patient), and the absence of a postictal phase after generalized seizures. Spontaneous crossing of the legs is not a reliable sign of pseudocoma. The neurological exam is otherwise normal.

In hysterical coma, ice water caloric tests give normal ipsilateral deviation with fast phase nystagmus in the opposite direction. If you suspect hysterical coma,
calorics should be used only as a last resort, and preferably with a neurologist present. Often times opening the eyelids, noting the normal tone, and bringing your face or a mirror close up to the patient’s eyes, results in the patient’s looking around or away, confirming that they are indeed awake. Bizarre behavior mimicking psychiatric illness can be seen in individuals under the influence of drugs and alcohol, and those who are postictal, hypoglycemic, or have suffered a head injury or a subarachnoid hemorrhage. In any patient presenting in coma, if the diagnosis is unclear or the patient is not responding as expected, obtain prompt neurologic expert consultation.

**Referral**

Transfer should be considered when the patient is stable enough to be transferred and definitive care exists which your facility does not offer. Referral or consultation should be considered if there is uncertainty regarding the diagnosis or management, if the clinician does not have privileges to provide the type of care needed (e.g., surgery), the clinician is ethically opposed to providing care (e.g., hospice care, or the opposite, definitive care at the patient’s/family’s request when it would be futile). Referral or consultation can also be considered when there is a conflict (e.g., personality conflict or the patient is a close friend or family member) and the clinician feels they cannot be objective. Consultation as a second opinion may be wise when the diagnosis is unexpected (e.g., young person with severe injury), serious, the prognosis is grim or the patient is not getting better. It should also be considered if the patient requests a consultation. The first sign that the patient desires a consultation may be provided by the family (i.e., the patient does not want to challenge or doubt the doctor/patient relationship).

Specific examples of consultation for neurologic emergencies include: all neurologic emergencies requiring surgery, status epilepticus requiring general anesthesia, and for rehabilitation for neurological emergencies with sequelae if the clinician is not comfortable. Consider transfer when there is inadequate equipment (e.g., no dialysis, CT or EEG) or facilities (e.g., limited ICU) in your hospital. There is some evidence that specialized stroke and closed-head injury units have better outcomes than routine ICU’s. If a specialized unit is not available, special staff training for managing victims of stroke or closed-head injury may be beneficial (e.g., monitoring mean arterial pressure, post-thrombolytic monitoring, etc.)

**Psychosocial Impact**

Surviving a neurological emergency can be very stressful for the patient and/or family members. This is especially true if the etiology is recurrent. Although a discussion of stress is beyond the scope of this book, long-term stress can cause physiologic and psychological signs and symptoms, for individuals or families, resulting in family disruption, depression, etc. It can also be associated with medical illnesses, and if coping skills are not developed, with a poor prognosis. Individuals and families should be monitored closely and counseled or treated as necessary. However, long-term stress is not always harmful. At least part of the stress can have positive results if the individual or family is motivated to obtain future care in a timely manner. Long-term stress may also motivate planning for emergencies, even resulting in patients moving to another town or location to be near adequate facilities. Planning can also include such decisions as who will make medical and financial decisions in the event that the outcome from a
future emergency is not good.

Survivors of neurologic emergencies with risk of recurrence go through many of the same stages of any patient receiving bad news: denial, anger, bargaining, possibly depression and eventually resolution. Active listening by the clinician may be an important management tool. Often the clinician will observe these stages if they take the time to listen to the patient or family.

Otherwise, the psychological impact of a neurological emergency is dependent upon the cause, especially coma, and whether it is an acute situation, a treatable/preventable situation (e.g., seizures) or potentially a chronic, recurrent and/or disabling situation. The impact from status epilepticus has already been discussed in the chapter on Episodic Disorders (Seizures). The impact from stroke has been partially discussed in the chapter on Weakness.

If coma (and for that matter, stroke or seizure) is secondary to a medical condition and the condition is entirely correctable, the patient and family members may only need reassurance. The patient and family should be prepared for the same psychological impact they could expect from any acute, serious illness. They should also be observed for sequelae related to an intensive care admission (e.g., ICU delirium, post-traumatic stress syndrome, etc.) and a brief recovery.

If the cause of coma is trauma (e.g., closed head injury), or any other cause requiring a prolonged recovery and rehabilitation, patients are at risk for long-term sequelae, a loss of self-dependence, self-confidence, and self-esteem. Support groups as listed below are often helpful.

If we do a good job as clinicians, the psychological impact from an ischemic stroke should be identical to that of a patient newly diagnosed with coronary artery disease following an event. The management is often very similar. In fact, the most common cause for death following an ischemic stroke is a myocardial infarction. After the patient/family absorbs this information, they should also be informed that current prognosis has never been better, especially due to new medications and therapies. Adherence to medical regimens will be very important. The patient and family should be observed for any evidence of the psychological defense known as denial, especially regarding necessary lifestyle changes to prevent recurrences.

The psychological impact on the family for any of these emergencies that result in a disability increases the risk of “caregiver burnout,” especially if there is one primary caregiver. Depending on severity of the disability, issues of placement, finances, scheduling/coordinating care, or even activities of daily living may all be dependent upon the caregiver. Caregiver burnout has been well described in the literature, including risk for anxiety, depression, and other major medical illnesses. With severely disabled patients, many geriatricians suggest that the caregiver be considered the primary patient rather than the disabled patient because the caregiver may be at greater health risk. Support groups and community resources as listed below are very valuable when attempting to avoid or manage caregiver burnout.

**Community Resources**

For patients with status epilepticus or weakness following a stroke, community resources are listed in the chapters on Episodic Disorders (seizures) and Weakness. For additional clinician or patient questions, for patient or family education, recent advances in treatment, or a listing of support groups for stroke
or coma, several resources are listed below. It is important to recommend these resources to patients or family members with serious or chronic illnesses. Support groups are often vital for recognizing and preventing caregiver burnout.

**References**


**Case Report: Part 3**

*After evacuation of the subdural hematoma, the patient had an uneventful course and recovered completely.*

**Ischemic Stroke**

Stroke is a major health problem and the third leading cause of death in the United States. Unfortunately symptoms are not as readily recognized by the general public as are those for myocardial infarction. A major health initiative is currently underway to educate the public about the symptoms of stroke. Readers are referred to the AAFP patient education web site *familydoctor.org,* “Stroke: Warning Signs and Tips on Prevention.” Use of the term “brain attack” has been promulgated to achieve public recognition equivalent to “heart attack”. This initiative is of paramount importance because of the recent development in treating stroke with tissue plasminogen activator (t-PA). If administered in a timely and appropriate fashion, tPA increases the likelihood of a complete or near-complete recovery.

At this time the reader should review the clinical symptoms associated with stroke as outlined in the Episodic Disorders chapter. Familiarity with these symptoms assures that patients are appropriately selected for t-PA therapy.

**Case Report**

You have just completed your morning rounds at the hospital, and are informed that a long-time patient has notified your office that her husband (a 65-year-old man who has always been in excellent health and on no medications) is en route to the Emergency Department (ED). You go directly to the ED where the paramedics arrive at 8 a.m. They report that the patient’s wife called 911 when she noticed upon awakening at 7:30 a.m. her husband was having difficulty speaking, his face was crooked and his right arm was limp at his side. They had established an intravenous line, obtained a blood glucose measurement...
of 120 mg/dL, and placed the patient on low-flow oxygen by nasal cannulae. Blood pressure in the field was 200/100 mm Hg, and it was the same in the ED, heart rate was 82 and regular, with a respiratory rate of 18. The patient’s temperature was recorded along with the initial vital signs in the ED as 37°C. You examine the patient and the key findings are neurologic. There is no speech output but he seems to follow some simple commands (close your eyes, lift up your arm). There is right facial asymmetry, a right field cut to threat, a flaccid right arm with no withdrawal to a painful stimulus to the nail bed digits of the right hand, and spontaneous weak movement of the right leg compared to the left. You join the Emergency Physician in providing care.

Question 1
Your initial management includes:
1. Sublingual nifedipine
2. Chewable aspirin
3. Obtain an emergency head computerized tomography (CT) scan
4. All of the above

Question 2
1. Ordering tissue plasminogen activator (t-PA) at 0.9 mg/kg estimated weight
2. Ordering a heparin infusion at 10 units/kg/hour
3. Performing a lumbar puncture before either 1 or 2
4. None of the above

None of the above. You may order and even prepare t-PA but do not administer it since the time of stroke onset is not known in a patient who awakens with stroke symptoms. The ischemic stroke may have occurred just before awaking or any time since known to be last neurologically intact (e.g., before going to sleep). In the United States, the manufacturer has established a no-charge pharmacy resupply program for any t-PA, which is opened but not used. Although heparin is often used as therapy for acute ischemic stroke, heparin has not been proven to be beneficial and may increase risk of intracerebral hemorrhage in large hemispheric stroke, which this patient manifests. Lumbar puncture is indicated to help diagnose subarachnoid hemorrhage or meningitis. Neither is suggested in this patient and would be a contraindication for immediate thrombolysis or anticoagulation if those became treatment options.

Minutes later the patient’s wife arrives. She states that she and her husband had arisen early that morning, awakening in time to catch a beautiful sunrise. At that time her husband was perfectly normal. They returned to bed at 6:30 a.m., and when they awoke at 7:30 a.m., he was noted to have the neurologic deficit. She knows he’s having a stroke and is asking that you do something. You re-evaluate the patient. He’s neurologically the same. The radiologist states the CT scan shows no hemorrhage and there are no early infarct signs. The nurse provides you with the blood test results from clinical laboratory. A complete blood count including platelets, glucose, electrolytes, renal function tests, pro-time and partial thromboplastin time are all within normal limits.
Question 3

Your management now includes:

1. Ordering t-PA at 0.9 mg/kg, 10 percent initial bolus, and the remainder over an hour because the patient has a greater likelihood of functionally independent recovery with minimal or no disability than without t-PA therapy, despite a 6 percent increase in the likelihood of a symptomatic intracerebral hemorrhage.

2. Ordering tenecteplase as a single weight-based IV bolus dose since no IV infusion is needed.

3. Withholding thrombolytic therapy for the acute ischemic stroke since the patient cannot give informed consent.

4. Withholding thrombolytic therapy for acute ischemic stroke until the patient’s private physician arrives since it is now only just over two hours from stroke onset and the patient may still be suffering only a transient ischemic attack.

The best choice is 1. The dosage of t-PA in acute stroke is lower than in acute myocardial infarction with a maximum dose of 90 mg. Although tenecteplase (TNK) has been shown to be beneficial for thrombolysis in acute myocardial infarction, TNK has not been shown to improve outcome in acute ischemic stroke, though such trials are being conducted. Patient understanding of risks and benefits of any therapy physicians provide is important. However, when patients are unable to participate in the decision-making, physicians are responsible for providing the best possible medical care. In this case, the patient’s spouse can provide any needed informed consent. However, if a legally authorized representative is unavailable, therapy should not be withheld. The rationale for treatment should be documented in the medical record, as well as discussions with appropriate individuals. As for concern that the patient’s symptoms may still only be the manifestations of a transient ischemic attack, in the NINDS t-PA stroke trials report, only 2 percent of patients in the placebo group had normal National Institutes of Health Stroke Scale (NIHSS) scores at 24 hours. As in this report, patients who show rapid improvement prior to receiving medication are excluded since they may be experiencing a TIA. However, if a significant deficit persists, these improving patients may benefit from IV t-PA in reducing the likelihood of a poor outcome, best reviewed with physicians experienced with the use of t-PA for stroke.

Your management is based on your understanding of the NINDS t-PA acute stroke trial published in the New England Journal of Medicine December 14, 1995, the subsequent Food and Drug Administration approval in the US in June 1996, and the FDA equivalent in Canada in February 1999. Attention to inclusion and exclusion criteria as organized below is important to replicate the results of the NINDS trial.

Checklist for t-PA for Acute Ischemic Stroke

Inclusion Criteria

1. Ischemic stroke with a defined onset of less than three hours from time t-PA is to be started. Ascertain last time patient known to be awake and deficit-free.
2. Measurable deficit on NIH Stroke Scale. Neurologic deficit minimal weakness, isolated ataxia, isolated sensory deficit, or isolated dysarthria.

3. CT scan shows no evidence of intracranial hemorrhage. If early signs of new major hemisphere infarct are present (e.g., edema, mass effect, sulcal effacement), reassess time of onset. The presence of these CT findings is associated with an increased risk of hemorrhage.

Exclusion Criteria History:
1. Stroke or serious head trauma within past three months.
2. Major surgery or serious trauma within past 14 days.
3. History of intracranial hemorrhage, AVM, or aneurysm.
4. GI or urinary tract hemorrhage within previous 21 days.
5. Arterial puncture at a noncompressible site OR lumbar puncture within previous seven days.

Clinical:
1. Rapidly improving neurologic signs or minor symptoms.
2. Systolic blood pressure > 185 mm Hg OR Diastolic blood pressure > 110 mm Hg OR aggressive (IV) treatment required to reduce patient’s blood pressure to specified limits.
3. Seizure at onset.
4. Symptoms suggestive of subarachnoid hemorrhage.
5. Recent myocardial infarction (post-MI) pericarditis.

Laboratory:
1. Patient taking anticoagulants AND prothrombin time (PT) greater than 15 seconds (International Normalized Ratio [INR] 1.7).
2. Patient has received heparin within 48 hours preceding stroke onset AND has an elevated partial-thromboplastin time (PTT).
3. Platelet count below 100,000 per mm3.
4. Glucose concentration below 50 mg/dl (2.7 mmol/liter) OR above 400 mg/dl (22.2 mmol/liter)
5. Patient of childbearing age who has a positive pregnancy test

Discuss the risks and benefits of thrombolytic therapy with the patient and family (if possible) and document the discussion in the medical record

Prior To Administering t-PA:
Review checklist to confirm inclusion and exclusion criteria. Confirm patient is not showing spontaneous improvement.

Treatment and Patient Management:
1. t-PA 0.9 mg/kg total or maximum 90 mg.
2. Administer 10 percent of t-PA dose as a bolus.
3. Administer remaining 90 percent of t-PA as a constant infusion for one hour.
4. **DO NOT** give anticoagulants for 24 hours from start of t-PA administration.
5. **DO NOT** give antiplatelet agents for 24 hours from start of t-PA administration.
6. Admit to Intensive Care Area OR Acute Stroke Unit.
7. Maintain systolic blood pressure **UNDER** 180 diastolic blood pressure **UNDER** 105
8. Restrict central venous line placement OR arterial puncture for 24 hours.
9. **DO NOT** insert indwelling bladder catheter for >30 minutes after t-PA administration.
10. **AVOID** insertion of nasogastric tube for 24 hours after t-PA administration.

**Blood Pressure Management:**

1. Monitor BP for 24 hours after starting t-PA infusion every 15 minutes for 2 hours; every 30 minutes for 6 hours; then hourly for next 16 hours.
2. If systolic BP 180–230 OR diastolic BP 105–120, **THEN** repeat in 5–10 minutes.
   If elevated on both readings:
   **ADMINISTER** Labetalol 10 mg IV over 1–2 minutes.
   **MONITOR** every 15 minutes.
   **REPEAT** 10 mg or 20 mg every 10–20 minutes as needed up to 150 mg.
   **AVOID** hypotension.
3. If systolic BP > 230 OR diastolic BP 121–140, **THEN** use labetalol as above, repeating every 10 minutes.
   **If response inadequate, use IV nitroprusside.**
4. If diastolic BP > 140, **THEN** use IV nitroprusside (0.5–10 mcg/kg/minute).
   Monitor closely, avoid hypotension. **USE WITH CAUTION!**

**If a sudden major rise in BP occurs, consider intracerebral hemorrhage, stopping t-PA infusion, and obtaining emergency CT scan.**

**Status Epilepticus**

The reader is advised to review the material on seizures in the Episodic Disorders chapter.

Definition: Patient does not recover to a normal alert state between two or more tonic-clonic seizures or duration of seizures greater than 20 minutes. Although most epileptic seizures are self-limited, some go on for prolonged periods, whereas others recur so rapidly that the condition is referred to as status epilepticus. The most serious form of this disorder is generalized convulsive status epilepticus, in which convulsive seizures are repeated without return of consciousness in between.
Goals of Treatment

1. Terminate seizure activity as soon as possible, preferably within 30 minutes of onset.
2. Prevent recurrence of seizures.
3. Ensure adequate cardiorespiratory function and brain oxygenation by establishment and maintenance of an adequate airway and support of blood pressure.
4. Correct any precipitating factors (e.g., hypoglycemia, hyponatremia, hypocalcemia, or fever).
5. Prevent or correct any systemic complications, especially hyperpyrexia, which may exacerbate neuronal damage caused by the continuous seizure activity.
6. Evaluate and treat possible causes of the episode of status epilepticus.

Treatment of Generalized Status Epilepticus

Immediate Action:

Obtain vital signs including temperature: If hypertensive, consider hypertensive encephalopathy. If febrile, use appropriate antipyretic measures vigorously.

Maintain airway orally or nasally. Monitor respirations. Cardiac monitor and BP monitor. Draw glucose, lytes, BUN, Ca, Mg, P, CBC with differential, creatinine and CK.

Accu-Chek-Treat hypoglycemia with 50 cc D5OW. Pediatrics 1 cc/kg D25W. Thiamine 100 mg IV to prevent possible precipitation of Wernicke-Korsakoff syndrome in malnourished patients (eg, alcohol and other drug abuse patients). If IV unavailable, consider glucagon 2 mg IM to treat hypoglycemia.

Obtain antiepileptic drug levels and arterial blood gas levels, if indicated. It is not necessary to treat status-induced metabolic acidosis if there is a good airway and seizures stop. If acidosis persists, consider other causes.

Save blood for toxicology screen. Consider theophylline, tricyclic antidepressants or other overdose. Consider amphetamine or cocaine use.

Obtain urine. In addition to urine toxicology screen when appropriate, check for myoglobinuria.

Obtain allergy history, particularly to phenytoin

At 5 minutes:

IV NS to maintain euvolemia. If an IV cannot be obtained by a peripheral line, consider intraosseous infusion, cutdown or central line placement. Consider endotracheal, rectal, IM or gastric administration of needed medication. Recognize your own limitations and obtain consultation for drug route, dosage, concentration, precautions, and complications.

Lorazepam no faster than 2 mg/min IV up to 0.1 mg/kg. Give in increments of 2 mg. Repeat increments no more often than every two minutes. Diazepam 2 mg/min up to 20 mg in 5 mg increments may be used, but lorazepam may be more effective for immediate control of status epilepticus. For pediatrics
diazepam may be used up to 0.25 mg/kg in four divided doses and it may be equally effective as lorazepam in children. These benzodiazepines may be administered rectally. Do not exceed dosage of benzodiazepines if already given in prehospital care. Stop benzodiazepine when clinical seizures stop. Clinical seizures may have subtle features such as nystagmoid jerking of eyes, small rhythmic finger movements, or twitching of the corner of the mouth. Observe for respiratory depression. Have flumazenil available. Place call for neurology consult.

At 15 minutes:
Proceed to load phenytoin at 20 mg/kg in NS at 50 mg/min by infusion pump with close monitoring. If BP drops, cease phenytoin infusion, wait for BP to return, then resume infusion at 25 mg/min and continue monitoring. Phosphenytoin may be infused at 100 mg phenytoin equivalents/min with similar precautions. If seizures stop and reoccur, resume benzodiazepine until maximum dose is reached. Arrange for admission to ICU.

Arrange for emergency EEG, if overt convulsive activity has stopped but patient is not improving in level of consciousness. Even when seizures are clinically no longer apparent, patient may be in electrographic status.

If status persists, intubate patient if not previously necessitated to maintain airway. Use short-acting neuromuscular agents so that clinical response can be assessed when paralytic drugs wear off.

At 30 minutes:
Repeat glucose Accu-Chek and temperature. Review laboratory results.
If still in status, additional phenytoin at 5mg/kg until cessation. Repeat again if necessary.

Obtain phenytoin level 30–60 minutes after completion of infusion.

At 60 minutes:
Arrange for general anesthesia with sodium pentothal or consider other antiepileptic anesthetic drugs. When status has been stopped, evaluate and treat patient for the precipitating cause. Head CT scan or MRI scan are performed to delineate structural brain lesions such as brain tumor or subarachnoid hemorrhage. Lumbar puncture should be performed if meningitis, encephalitis, or subarachnoid hemorrhages are suspected.