The approach to the patient who presents to the Emergency Department (ED) is altogether different than the approach to the patient who is presenting to the clinic or inpatient setting. A clinic patient is usually considered healthy unless pathology is proven, whereas an ED patient should be considered to have a life-threatening etiology for his or her complaint until proven otherwise. In the ED, history is often scarce or vague, no medical records are immediately available for review, and the patient is usually not known to the practitioner. Emergency physicians often are required to treat patients prior to arriving at specific diagnoses. We must first treat the patient, “rule-out” life-threatening causes for the patient’s symptoms, and then sort through the facts to arrive at a diagnosis.

This is all the more true in the patient who presents with a neurologic complaint. Subject to varying degrees of symptomatology, the patient is often unable to provide an accurate HPI, and the practitioner must rely on observations of family members, friends, or EMTs for data. The following is intended to provide a brief introduction and initial approach to the patient that presents to the ED with selected neurologic problems, i.e., patients with an altered level of consciousness, seizures, or stroke syndrome.

**APPROACH TO THE PATIENT WITH ALTERED MENTAL STATUS**

First and foremost, the standard approach to all patients presenting to the ED should be applied to the patient with altered mental status (AMS). Specifically, this refers to the “ABCs” – Airway management, assessment of Breathing and Circulation, and immobilization of the Cervical spine. Once the “ABCs” have been assessed and the patient has been stabilized, you can then step back and attempt to determine the etiology of the patient’s problem.

Definitions

We often hear patients being described as being lethargic or delirious, and an appropriate understanding of these terms is essential. Consciousness is defined as an awareness of oneself, one’s acts, and one’s surroundings.

Patients who appear asleep may actually just be sleeping. These patients are arousable to a normal level of awareness. This is to be compared with the patient who is **lethargic**, a state in which the patient may have global depressed awareness of self and environment, and may even appear wakeful. The **stuporous** patient is the patient that appears asleep, is arousable with noxious stimuli but not to a full state of awareness. **Coma** describes the state of unresponsiveness from which one cannot be aroused.

Confusional states present in a wide variety of ways. **Delirium** is an acute confusional state associated with psychomotor excitement, marked by impaired perception and memory, and often accompanied by hallucinations. Examples include alcohol withdrawal, toxic ingestions, metabolic abnormalities, and CNS infection. **Dementia** is characterized by chronic or gradual deterioration of mental function. Dementia is usually seen in elderly patients, and is exemplified by the patient with Alzheimer’s disease.

Pathophysiology

The main causes of AMS can be classified into diffuse or global causes, or causes that involve suppression of the reticular activating formation (RAF). It is important to remember that focal lesions in the cerebral cortices usually cause focal deficits rather than AMS. If a focal lesion does result in AMS, it should be a lesion that involves bilateral cerebral cortices. The diffuse or global causes of AMS are most commonly secondary to toxins such as alcohol or drugs, hypoxia, or hypoglycemia.

The reticular activation system consists of a group of fibers that traverse the brainstem en route to the thalamus. The RAF is responsible for maintaining the state of awareness. The function of the RAF can be suppressed by supratentorial or infratentorial direct pressure. A mass lesion in the supratentorial area results in the displacement of tissue that can compress the brainstem and RAF. An increase in pressure in the posterior fossa may displace the contents of the fossa (the cerebellum, the fourth ventricle, and the Aqueduct of Sylvius) upward through the tentorial notch or downward through the foramen magnum, thus compressing the RAF. Pontine hemorrhage or infarction may directly involve the RAF.
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General Approach to the Patient with AMS

As in all patients presenting to the ED, first assess and secure the airway, check breathing and circulation, and immobilize the cervical spine. If intubation is required, it should be followed by hyperventilation to a pCO2 of 25-30 mmHg. Lowering of the pCO2 is the most effective and rapid means of reducing intracranial pressure.

An intravenous line should be inserted and blood withdrawn for laboratory analysis. The patient should be placed on a cardiac monitor, and pulse oximetry measurement should be obtained. It is standard practice to then administer thiamine 100mg, glucose 25-50g, and naloxone 2-4mg IV. Thiamine is given to facilitate carbohydrate metabolism and prevent Wernicke-Korsakoff syndrome in nutritionally deficient patients. If rapid serum glucose testing can be done immediately, the administration of glucose can be guided by test results. Naloxone is an opiate and synthetic narcotic antagonist.

Once the patient had been stabilized, the history of the patient’s illness may be obtained. The patient may not be in a condition to give a reliable history, therefore, all available resources should be utilized. EMTs are a valuable source of information in the initial phase of management. Further history should be obtained (by phone if necessary) from family, friends, and/or witnesses of immediately preceding events. The patient’s medical record should be obtained for review.

A detailed physical exam may provide clues to the etiology of the patient’s symptoms. Examination of the skin may reveal needle tracks (IV drug overdose), cyanosis (hypoxia), or cellulitis (infection/sepsis). Breath odors, such as that of ethanol or acetone (diabetic ketoacidosis) may be helpful. (The prudent physician, however, must never assume that AMS is due to alcohol simply because the patient smells of alcohol). Cardiac examination may reveal an arrhythmia. Examination of the abdomen may show ascites or organomegaly (hepatic encephalopathy) or a pulsatile mass (abdominal aortic aneurysm). Evaluation of respiratory patterns may provide clues. Cheyne-Stokes respirations (a gradual increase in depth of respirations followed by a period of apnea) result when the cerebral cortex is no longer functioning. Hyperventilation may be due to a variety of causes, such as attempts to correct hypoxia or compensate for a metabolic acidosis.

The neurologic status of the patient should be initially assessed using the Glasgow Coma Scale. This will rapidly categorize the severity of injury as well as allow standardized objective assessment of the patient’s neurologic status (Table 1). Cranial nerve function should then be assessed. The eye function is the best measure of cranial nerve integrity. The size, shape, and reactivity of the pupils should be evaluated and funduscopic exam should be performed. Ocular movement can be evaluated by cold-caloric testing, and the corneal reflex should be assessed. Facial assymetry should also be noted, if present. Testing of cranial nerves VIII-XII is seldom helpful in those patients with coma (with the exception of the gag reflex). Physical findings of ataxic respirations, contralateral hemiparesis, and ipsilateral pupillary dilation are indicative of impending herniation of the temporal lobe (uncus) into the infratentorial space. If these findings are present, immediate neurosurgical consultation is indicated before proceeding with further work-up.

Table 1. Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Spontaneously</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>To verbal command</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Best Verbal Response</td>
<td>Oriented &amp; converses</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Disoriented and converses</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Best Motor Response</td>
<td>Obeys</td>
<td>6</td>
</tr>
<tr>
<td>To verbal command:</td>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td>To painful stimulus:</td>
<td>Flexion-withdrawal</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Abnormal extension</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>3-15</td>
<td></td>
</tr>
</tbody>
</table>
Neurologic Emergencies

Routine ancillary studies should include: CBC, electrolytes, BUN, creatinine, glucose, ABG, and toxic drug screening. Further laboratory evaluation should be guided by the history and physical findings (i.e., liver function tests, serum ammonia level, serum acetone).

CT scan is indicated in the patient with AMS and any of the following: 1) evidence of or suspected trauma, 2) suspected intracranial hemorrhage, 3) presence of an unexplained focal neurologic deficit, 4) papilledema, 5) suspected intracranial abscess, or 6) other causes of AMS have been ruled out or etiology remains unexplained. Cervical spine x-rays should also be obtained.

A lumbar puncture (LP) is indicated if meningitis is suspected. LP should be done immediately if no focal neurologic findings or evidence of increased intracranial pressure are found on physical exam. LP is also indicated if the CT scan is negative and either intracranial abscess or subarachnoid hemorrhage is suspected. Evidence of trauma and/or increased intracranial pressure are contraindications to LP.

Etiology of AMS

The differential diagnosis for causes of AMS and coma is extensive. Table 2 represents a limited but useful mnemonic aid for causes of coma, with the following additional points:

1) The most frequent cause of AMS in the ED is hypoglycemia. This is seen not only in diabetics, but also in patients with liver disease, pancreatic tumors, and chronic alcoholism.
2) Space-occupying lesion include not only neoplasms, but also intracranial hemorrhages (i.e., epidural or subdural hematoma) as well as focal infections and abscesses.
3) Patients with seizure disorder may present in a postictal state with AMS.
4) Psychogenic coma is a diagnosis of exclusion.
5) Endocrine/exocrine disorders include thyroid storm, as well as myxedema coma.

<table>
<thead>
<tr>
<th>Table 2. Causes of AMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A – alcohol, arrhythmia</td>
</tr>
<tr>
<td>E – endocrine/exocrine, electrolytes, encephalopathy</td>
</tr>
<tr>
<td>I – insulin</td>
</tr>
<tr>
<td>O – oxygen, opiates</td>
</tr>
<tr>
<td>U – uremia</td>
</tr>
<tr>
<td>T – trauma, temperature disorders</td>
</tr>
<tr>
<td>I – infection</td>
</tr>
<tr>
<td>P – psychiatric, porphyria, poisons</td>
</tr>
<tr>
<td>S – space-occupying lesions, stroke, subarachnoid hemorrhage</td>
</tr>
<tr>
<td>shock, seizures</td>
</tr>
</tbody>
</table>

Treatment

As with most entities in medicine, treatment of AMS is directed toward the underlying disorder. Specific management of the myriad of causes of AMS is beyond the scope of this discussion. The most critical portion of ED management is the initial stabilization of the patient. However, it is important to note the following “pearls”:

1) Immediate neurosurgical consultation is indicated if signs of uncal herniation are present. In addition to intubation and hyperventilation, mannitol 1-2 g/kg IV is indicated.
2) If infection is suspected, administration of antibiotics should not be delayed by the work-up.
3) Do not be misled by the odor of alcohol.
4) Immobilization of the cervical spine is imperative. All patients with AMS must be considered to have a cervical spine injury until proven otherwise.
5) Toxic drug screens only test for common drugs of abuse. Specific drug assays may be indicated.
6) Romazicon® (a benzodiazepine antagonist) should be considered if isolated benzodiazepine overdose is determined to be the etiology of AMS. However, Romazicon® is to be avoided in any patient with a history of seizure disorder or cyclic antidepressant overdose, as seizures and/or death can be precipitated by its administration.
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**APPROACH TO THE PATIENT WITH SEIZURES**

As with the patient presenting with altered mental status, the first step in management of the patient with seizures is assessment and stabilization of the “ABCs”. This includes immobilization of the cervical spine and compression of any obvious hemorrhage, as previously mentioned. After the “ABCs” have been assessed and the patient has been stabilized, you may then begin assessment and treatment of the patient’s seizure activity.

Definitions and Classification

A seizure is a paroxysmal event due to excessive neuronal discharge that leads to focal or generalized manifestations. *Status epilepticus* is defined as a seizure lasting >30 minutes, or intermittent seizure activity, also lasting >30 minutes, during which the patient does not regain consciousness.

Seizures can be divided into two classes: generalized, which are always associated with loss of consciousness, and focal, which may or may not be associated with impaired consciousness. Although this text is not intended to describe in detail the pathophysiology of each type of seizure, further classification of seizures is noted in Table 3. Generalized seizures are bilaterally symmetrical and without focal onset, and begin with an abrupt loss of consciousness. Auras may occur, but are not always a component of seizures. Partial seizures begin focally, but may progress to generalized seizures. Simple partial seizures, consciousness is not impaired, whereas, complex partial seizures are accompanied by impaired consciousness.

<table>
<thead>
<tr>
<th>Table 3. Seizure Classification</th>
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<tbody>
<tr>
<td><strong>Generalized</strong></td>
</tr>
<tr>
<td>a. Absence</td>
</tr>
<tr>
<td>i. Typical (petit mal)</td>
</tr>
<tr>
<td>ii. Atypical</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>b. Tonic and/or Clonic (grand mal)</td>
</tr>
<tr>
<td>c. Myoclonic/atonic</td>
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</tbody>
</table>

**HISTORY**

Seizure activity is not always due to a primary seizure disorder. Seizures may be occurring secondary to an infectious process (such as meningitis), a toxin, or a metabolic disorder. A cardiac arrhythmia may result in syncope, which may be mistaken for a seizure. Several sleep disorders, as well as movement disorders, may present as seizure activity. Psychiatric disorders including hyperventilation syndromes, fugue states, and pseudoseizures can be mistaken for a primary seizure disorder. Therefore, the precise history of events surrounding the seizure activity must be carefully elicited from witnesses.

If the patient had a seizure, he or she will have no true recall of the event. The patient may be able to tell you the immediately preceding activities, but will not be able to recall the actual event. Beware of the patient who “heard everyone talking” but couldn’t move or respond. Information that should be obtained from witnesses includes a description of the patient’s activity at the onset of symptoms, the progression of the patient’s activity during the episode, the duration of the activity, and the patient’s activity and mental state immediately following the event. A seizure typically has an abrupt onset and termination, and most only last 1-2 minutes. The pattern of activity may assist in the classification of seizures, or may altogether rule out a seizure as the etiology of the event. Movements during seizures are generally purposeless and inappropriate. With the exception of most petit mal or simple partial seizures, most seizure activity is followed by a period of confusion and lethargy, termed a “postictal state.”

The patient may have an established diagnosis of seizure disorder, but may have concomitant problems that lowered the seizure threshold and precipitated the current seizure activity. Therefore, it must be determined from witnesses if the patient has had other recent health problems, recent trauma, or if the patient has been compliant with the prescribed anticonvulsant regimen. A careful review of systems must be obtained to determine if the patient has had recent complaints indicative of concurrent infection, or if there has been other drug use. The patient’s usual pattern of seizure activity, as well as the frequency of
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occurrence, must be ascertained to determine if the current event deviates from the patient’s “baseline” disorder.

If the patient has no history of seizure disorder, it is imperative that a very detailed review of systems be obtained.

PHYSICAL EXAM

A thorough physical exam should be performed and directed toward discovery of any systemic illness that may have precipitated the event or any injury that may have been sustained during the event. An elevated temperature should prompt a search for a source of infection. Laceration of the tongue often occurs during a seizure. Once it has been determined that no injury has occurred involving the cervical spine, examine the neck for nuchal rigidity (i.e., evidence of meningitis). Examine the lungs for rales, as pneumonia may have precipitated the seizure activity, or may have occurred as a complication of the seizure (i.e., aspiration). The extremities should be examined for evidence of trauma (seizures are the most common cause for posterior dislocation of the shoulder). The neurological exam may be difficult to perform, as the patient may remain lethargic in a postictal state. Serial neurologic exams should be performed as the patient progresses through the postictal course.

ANCILLARY STUDIES

Specific ancillary studies that should be obtained depend on the clinical situation. For example, in a patient with a known seizure disorder who has just had a seizure that is typical for them, consider only a finger stick glucose determination and relevant serum anticonvulsant levels. However, in a patient with new-onset seizures, consider obtaining a CBC, electrolytes, BUN, creatinine, calcium and magnesium levels, liver function tests, urinalysis, toxic screen, alcohol level, and a pregnancy test.

Patients with any of the following should undergo neuroradiographic imaging (CT scan or MRI): a patient with a new-onset seizure, persistent global or focal deficits, recent history or evidence of head trauma, and/or a sudden change in the patient’s usual seizure pattern.

Further studies, such as an EEG, may be obtained in consultation with a neurologist.

TREATMENT

The treatment of seizures is based on protecting the patient from self-harm, as well as from complications that may arise from seizure activity. The mortality of status epilepticus has been reported as approximately 10%. Complications may include trauma, hypoxia, and circulatory collapse.

Treatment and assessment should begin concurrently, with assessment and stabilization of the “ABCs” including cervical spine immobilization. One of the most important things is to protect the patient from aspiration. Do not place anything in the patient’s mouth – a nasal airway may be used when necessary. Place the patient in the Trendelenburg position, with suction available at the bedside. Soft restraints may be necessary to prevent a fall or other injury. After initial assessment and stabilization has been performed, place the patient on a monitor, including pulse oximetry. Obtain IV access (withdraw blood for labs), and place the patient on oxygen. Obtain a finger stick glucose or administer 1 amp of dextrose. At this point, perform a thorough physical exam. If infection is suspected, administer antibiotics. If seizure activity persists, institute pharmacologic therapy (Table 4.).

DISPOSITION

Not all patients presenting with seizures must be admitted to the hospital. Those that require admission are those with status epilepticus and most patients with new-onset seizures. A patient with a known seizure disorder should be admitted if 3 or more seizures have occurred in a 24 hour period, or if the patient is significantly subtherapeutic on anticonvulsants and cannot be loaded in the ED (e.g., carbamazepine, valproic acid).

Patients with a history of seizure disorder who have had their typical seizures may be safely discharged home, provided their anticonvulsant levels are therapeutic, their mental status has returned to normal, they have no other condition mandating admission, and specific follow-up has been arranged. A neurologic consultant may choose not to admit a patient with a new-onset seizure. This is acceptable only if all of the following conditions are met: the physical exam is normal, CT scan is unremarkable, no other condition mandating admission is present, the home situation is adequate (including the availability of
close patient observation by family or friends), the patient is reliable, and specific follow-up has been arranged.

Table 4. Pharmacologic Control of Active Seizures

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lorazepam</td>
<td>0.1 mg/kg IV (&lt;2 mg/min; total dose 4-8 mg)</td>
<td></td>
</tr>
<tr>
<td>or</td>
<td></td>
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</tr>
<tr>
<td>Diazepam</td>
<td>0.15-0.25 mg/kg IV (&lt;5 mg/min; usual adult dose is 5 mg; maximum total adult dose is 20 mg)</td>
<td></td>
</tr>
<tr>
<td>then</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>15-20 mg/kg IV load (no faster than 50 mg/min)</td>
<td></td>
</tr>
</tbody>
</table>

If seizure activity persists:
- ET intubation (if not already performed)
- Phenobarbitol 20 mg/kg IV at 100 mg/min
- Consider lidocaine 100 mg IV bolus

If seizure activity persists:
- Consider barbiturate coma, general anesthesia, or diazepam drip. If this is done, continuous EEG monitoring is mandatory.
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APPROACH TO THE PATIENT WITH A STROKE SYNDROME

As in every patient who presents to the ED, first assess and secure the “ABCs”. Once this has been accomplished, you may proceed with further evaluation. In the patient presenting with a stroke syndrome, the goals of the ED physician are stabilization and support, exclusion of nonvascular causes of the patient’s symptoms, definition of the type and vascular territory of the TIA or stroke, expedition of appropriate consultation, minimization of secondary damage, and assistance with the prevention of subsequent similar events.

Definitions

A stroke, as defined by the World Health Organization (WHO), is the rapid development of clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting longer than 24 hours or leading to death, with no apparent cause other than that of vascular origin. A Transient Ischemic Attack (TIA) is defined as an acute episode of focal loss of cerebral function (including focal visual field loss) lasting < 24 hours, and is attributed to a temporarily inadequate blood supply. Therefore, a patient presenting to the ED with a new-onset (<24 hour) neurologic deficit cannot be classified into either category, and must be presumed to be experiencing an acute stroke.

Pathophysiology

Stroke is the leading cause of disability and the 3rd leading cause of death in the United States. Stroke causes brain damage via one of two mechanisms: 1) from ischemia due to vessel occlusion (primary atherosclerosis or an embolic event) which deprives neurons of oxygen and nutrients, or 2) from hemorrhage due to vessel rupture, which causes brain injury by direct cell trauma, mass effect, elevated intracranial pressure, and/or the release of detrimental biochemical substances. Approximately 80-85% of all strokes are ischemic in etiology, with the remaining 15-20% attributable to hemorrhage.

History

Risk factors for stroke are much the same as those for coronary artery disease, as atherosclerotic disease globally effects the vasculature. In fact, cardiac disease is a risk factor for stroke. This includes arrhythmias, such as atrial fibrillation, due to the potential for intracardiac thrombus formation. Other risk factors include: age (elderly), hypertension, diabetes, smoking, hypercholesterolemia, obesity, and oral contraceptives.

A detailed account of the patient’s symptoms should be obtained. This may, of necessity, be obtained from family or witnesses, since the patient may often be dysarthric or aphasic. The time of onset, the rapidity of onset, and the activity at onset should be determined. Gradual onset with waxing and waning neurologic symptoms suggests a thrombotic stroke, whereas sudden onset of symptoms suggests an embolic or hemorrhagic event. A history of recent TIAs or similar symptoms should be obtained. A TIA with a resultant deficit that is in the same distribution as the acute presenting deficit suggests thrombotic disease, whereas, TIAs with deficits in varying distributions (as compared with the current deficit) suggests an embolic event.

A history of headache or trauma should be obtained, if present. Headache occurs in the majority of patients with an intracranial hemorrhage, but occurs in only 10-20% of those with ischemic stroke.

In addition to the usual past medical, social, and family history, current medications and a complete review of systems should be obtained.

Physical Exam

A general physical exam should be preformed prior to initiating a detailed neurologic exam. Certain findings may need to be immediately addressed, while others may suggest the mechanism of the stroke.

Vital signs may reveal fever, which may suggest underlying infection that is “unmasking” the cerebral event. It is not uncommon for a patient with a stroke to have concurrent hypertension. The thickened smaller perforating vessels may require a higher pressure to remain patent.

Examination of the head and neck should include funduscopic exam, which may reveal papilledema (suggestive of mass effect) or preretinal hemorrhage (suggestive of subarachnoid hemorrhage). Pupil size, equality, and reactivity should be assessed. The temporal artery should be palpated for tenderness suggestive of arteritis. Nuchal rigidity may indicate meningitis or subarachnoid hemorrhage.
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Cardiac examination may reveal an arrhythmia such as atrial fibrillation, which may suggest a cardioembolic event. Other stigmata of cardioembolism should be sought out, such as Osler’s nodes, Janeway lesions, and splinter hemorrhages.

The goal of the neurologic evaluation is to localize the site of the lesion, while excluding other neurologic diseases. Mental status should be assessed, including level of consciousness, orientation, memory, speech, and understanding. Cranial nerves II-XII should be assessed, including visual field assessment. Muscle tone, strength, and fine motor control should be included in the motor exam. Pronator drift suggests cortical dysfunction. Deep tendon reflexes are initially hypoactive on the affected side; later they become hyperactive. Plantar responses (Babinski’s) should be performed. The sensory exam should include assessment of pain, temperature, light touch, and vibration sense. Cerebellar function testing should be performed, including balance, heel-to-shin, finger-to-nose, and, if the patient is safely ambulatory, gait.

Stroke Syndromes

The following are examples of classical patterns of stroke, categorized into ischemic and hemorrhagic syndromes.

Ischemic stroke syndromes

- **Dominant hemispheric infarction**: In all right-handed patients, and in up to 80% of left handed patients, the left hemisphere is the dominant hemisphere. Ischemia in this hemisphere results in contralateral weakness and numbness, contralateral visual field deficits, a gaze preference, dysarthria, and aphasia.
- **Nondominant hemispheric infarction**: Findings may be similar to those seen as a result of infarction in the dominant hemisphere; however, patients may be dysarthric, but typically are not aphasic. Patients often will neglect the contralateral extremities.
- **Middle cerebral artery infarcts**: Infarcts in the distribution of the middle cerebral artery results in a contralateral sensory deficit as well as motor weakness, with the face and the affected arm weaker than the leg. These patients may have aphasia if the dominant hemisphere is involved.
- **Anterior cerebral artery infarct**: An infarct in the distribution of the anterior cerebral artery results in deficits similar to those found in middle cerebral artery infarcts, except, in this case, leg weakness is greater than arm weakness.
- **Vertebrobasilar artery syndrome**: The posterior circulation supplies the brainstem, cerebellum, and visual cortex. Dizziness, vertigo, diplopia, dysphagia, cranial nerve palsies, and bilateral limb weakness may be associated with infarcts involving the posterior circulation. The finding of crossed neurologic deficits is the hallmark of a vertebrobasilar artery infarct (ipsilateral cranial nerve weakness with contralateral motor weakness).
- **Basilar artery occlusion**: Occlusion of the basilar artery results in severe quadriplegia, coma, or locked-in syndrome.
- **Lacunar infarct**: These infarcts result in pure motor or pure sensory deficits, and result from infarction from small penetrating arteries, and are primarily located in the pons and basal ganglia. Lacunar infarcts are commonly associated with chronic hypertension.
- **Arterial dissection**: Most often associated with severe trauma, arterial dissection can also result from minor events, such as a quick turn of the head. Dissection may occur in both the carotid and vertebral circulation. Patients may complain of sever headache or neck pain hours to days prior to onset of neurologic deficits.

Hemorrhagic stroke syndromes

- **Intracerebral hemorrhage**: This entity may be clinically indistinguishable from cerebral infarction. However, the presentation differs in that most patients are lethargic and may have hypertension. Headache, nausea, and vomiting often precede the onset of neurologic deficit.
- **Cerebellar hemorrhage**: Patients with cerebellar hemorrhage experience the sudden onset of dizziness, vomiting, marked truncal ataxia, and inability to ambulate.
- **Subarachnoid hemorrhage (SAH)**: These patients experience the sudden onset of a severe headache (“thunder-clap headache”) that is often occipital or nuchal in location. The headache is often accompanied by vomiting. Patients with SAH may have associated neurologic deficits secondary to the compression of brain tissue or cranial nerves by an enlarging aneurysm.
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Ancillary Studies

Every patient presenting with a stroke syndrome should receive the following ancillary studies: CBC, electrolytes, glucose (hypoglycemia can often mimic acute stroke), BUN, creatinine, PT, PTT, pregnancy (in females of child-bearing age), chest XR (to assess cardiac size, and rule out congestive heart failure as well as infiltrates), and EKG (4% of patients with stroke will have a life-threatening arrhythmia and 3% will have concurrent myocardial infarction). They should also have a noncontrast head CT. The primary goal of performing a head CT in these patients is to rule out hemorrhage and mass effect. Areas of acute ischemia are usually not seen on CT scan until 12-48 hours after onset of symptoms. CT scan has been reported to be 90-95% sensitive for visualizing SAH; if SAH is suspected and CT is negative, a lumbar puncture (LP) is mandatory to exclude the diagnosis. (SAH is discussed in further detail in the next section). Magnetic Resonance Imaging (MRI) is the preferred method of imaging in the evaluation of a brainstem stroke, subacute hematoma (age 10-20 days), demyelinating diseases, and arteriovenous malformations.

Additional studies should be considered on a case-by-case basis. Obtaining an erythrocyte sedimentation rate (ESR) is mandatory on any patient 50 years old or more with a headache or transient vision loss, to rule out temporal arteritis. LP is indicated if meningitis is suspected. Carotid artery doppler (duplex), echocardiogram, angiography, and magnetic resonance angiography (MRA) are all useful adjuncts to therapy and evaluation, and should be obtained in consultation with a neurologist.

Differential Diagnosis

The differential diagnosis of stroke includes the following:

- Hypotension (from cardiogenic, hypovolemic, or septic shock, or from an arrhythmia)
- Complications of migraine variants
- Labyrinthine disorders, including acoustic neuroma and vertigo
- Postictal (Todd’s) paralysis
- Infections, including meningitis, brain abscess, encephalitis, and neurosyphilis
- Toxins
- Metabolic disorders
- Subdural or epidural hematomas
- Demyelinating diseases, such as Multiple Sclerosis and optic neuritis

Treatment

The approach to the patient with a stroke syndrome should begin as with any patient who presents to the ED: assessment and stabilization of “ABCs”. Because of the significant risk of arrhythmia, the patient should be placed on a cardiac monitor. IV access should be established (blood should be withdrawn for laboratory analysis), and pulse oximetry should be obtained. Consider a finger stick glucose (for reasons mentioned above).

Do not attempt to lower the blood pressure in the hypertensive TIA or stroke patient, unless the systolic blood pressure is >220 mmHg, the diastolic blood pressure is >120 mmHg, or the mean arterial pressure is >140 mmHg on three repeated measurements made at 15 minute intervals, or if the patient is in danger of myocardial, aortic, or renal damage associated with elevated blood pressure. If blood pressure reading meet the aforementioned criteria, urgent consultation with a neurologist (or a neurosurgeon in the event of intracerebral hemorrhage) is mandatory prior to initiating therapy.

History and physical should be performed; ancillary tests should be obtained. Appropriate neurologic consultation should then be obtained.

A new treatment that is currently at the forefront is the use of thrombolytic agents in those patients with ischemic stroke. This is an instance in which the time of onset of symptoms is critical. If this therapy is to be instituted, it must be initiated within 3 hours of onset of symptoms, intracranial hemorrhage must be ruled out, and therapy should only be instituted in consultation with a neurologist.

Disposition

All decisions regarding the disposition of a patient with a stroke syndrome should be made in consultation with a neurologist. If a patient with a stroke is to be discharged home, the patient must be
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reliable, otherwise well without other conditions mandating admission, with a small completed stroke >48 hours old, and close follow-up has been arranged.

A patient with an isolated TIA may be safely discharged and referred for an outpatient work-up if he or she is reliable, otherwise well, and does not live alone; if symptoms have completely resolved and the patient now has a normal neurologic exam; carotid duplex has been performed and reveals <70% occlusion; evidence of a cardioembolic source has been ruled out, and there is no indication for anticoagulation with heparin; if antiplatelet therapy (aspirin or ticlodipine) has been initiated; and if close follow-up has been arranged.

All patients with new onset strokes should be admitted to the hospital. Patients with TIAs that do not meet the aforementioned criteria should be admitted.

BIBLIOGRAPHY


