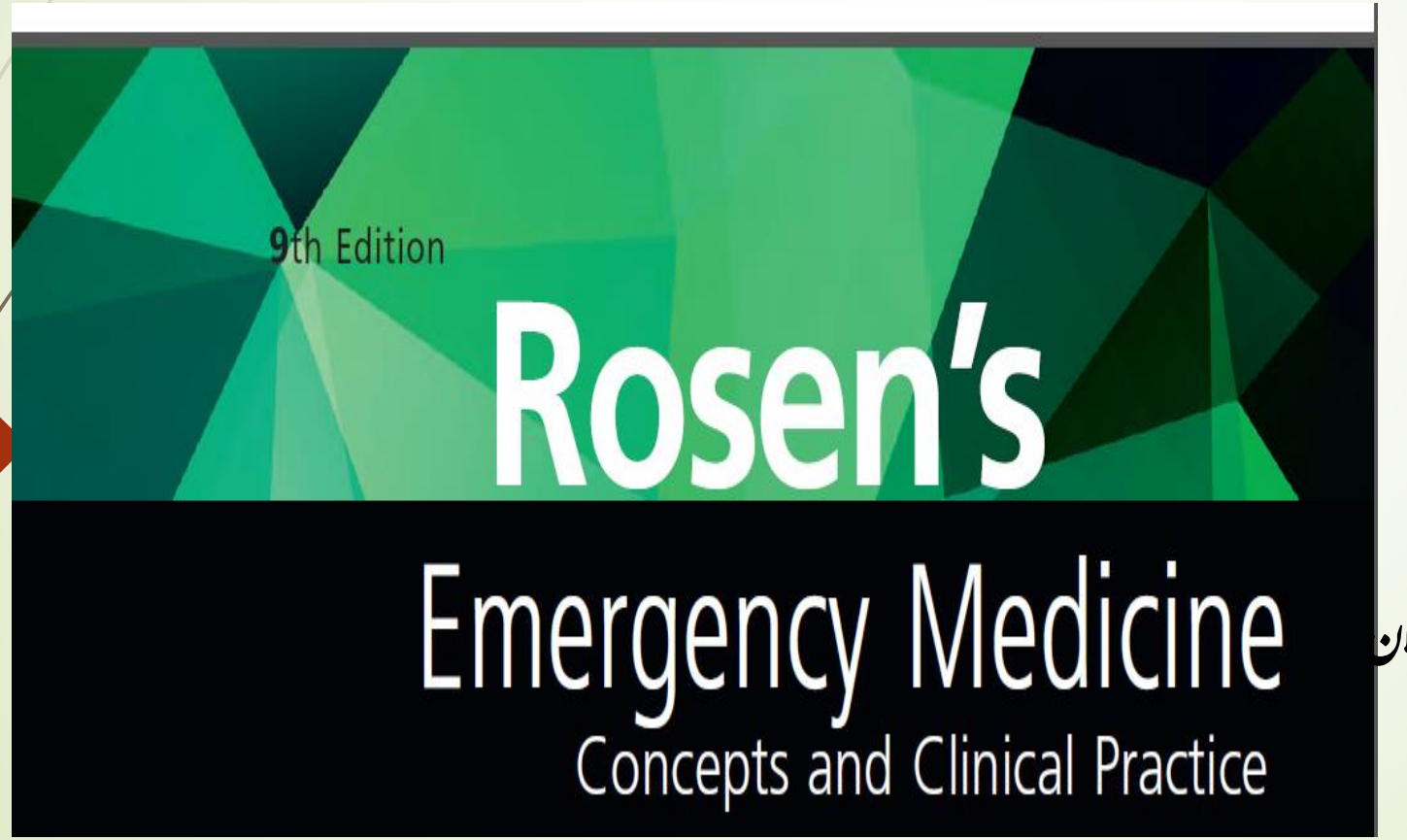

بسم الله الرحمن الرحيم

Stroke



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Background

Stroke is the fifth leading cause of death in the United States and a leading cause of long-term disability. It affects about 795,000 people per year. On average, someone has a stroke every 4 seconds, and someone dies of a stroke every 4 minutes . Stroke patients have an in-hospital mortality rate of 5% to 10% for ischemic

stroke and 40% to 60% for intracerebral hemorrhage (ICH). Only 10% of stroke survivors will recover completely, making stroke a leading cause of adult disability

any vascular injury that reduces cerebral blood flow (CBF) to a specific region of the brain, retina, or spinal cord, causing neurologic impairment. The onset of symptom may be **sudden or stuttering**, often with transient or permanent loss of neurologic function. 87% of all stroke are ischemic in origin, caused by the occlusion of a cerebral vessel. 13% are hemorrhagic strokes caused by the rupture of a blood vessel (ICH) or [SAH]). **Prior** treatment for stroke was not focused on reversal of damage and consisted of stabilization observation, and rehabilitation. **Current** acute interventionaltreatment regimens are designed to reverse or minimize brain damage..

Epidemiology

These may result from either in situ thrombosis or embolic obstruction from a more proximal source, usually the heart. Approximately one-third of all ischemic strokes are thrombotic in nature. These can be caused by either large- or small vessel occlusions. Common areas for large-vessel occlusions are cerebral vessel branch points, especially in the distribution of the internal carotid artery. Thrombosis usually results from clot formation in the area of an ulcerated atherosclerotic plaque that forms in the area of turbulent blood flow, such as a vessel bifurcation

A marked reduction in flow results when the stenosis occludes more than 90% of the blood vessel diameter. With further ulceration and thrombosis, platelets adhere to the region. A clot then either embolizes or occludes the artery. Lacunae, or small-vessel strokes, involve small terminal sections of the vasculature and more commonly occur in patients with diabetes and hypertension. A history of hypertension is present in 80% to 90% of patients who experience lacunar strokes. The subcortical areas of the cerebrum and brainstem often are involved. The infarcts range in size from a few millimeters to 2 cm and are seen most commonly in the basal ganglia, thalamus, pons, and internal capsule. They may be caused by small emboli or by a process termed *lipohyalinosis,* which occurs in patients with hypertensive cerebral vasculopathy.

One-fourth of all ischemic strokes are cardioembolic in nature. Embolization of a mural thrombus in patients with atrial fibrillationis the most common mechanism, fivefold increased risk for development of a stroke.

Noncardiac sources of emboli

diseased portions of extracranial arteries, resulting in an arteryto- artery embolus. One common example is amaurosis fugax, in which emboli from a proximal carotid artery plaque embolizes to the ophthalmic artery, causing transient monocular blindness. Although stroke risk increases with **age**, approximately 3% to 4% of all strokes occur in patients 15 to 45 years old, **Pregnancy**, the use of **oral contraceptives**, **antiphospholipid antibodies** (such as, lupus anticoagulant and anticardiolipin

protein S and C deficiencies, sickle cell anemia, and polycythemia all predispose patients to sludging or thrombosis, **Fibromuscular dysplasia** of the cerebral vasculature and **in rare** instances prolonged vasoconstriction from a **migraine syndrome** causes stroke. **Recreational drugs** such as cocaine, phenylpropanolamine, and amphetamines are potent vasoconstrictors that have been associated with both ischemic and hemorrhagic stroke. **Infectious processes**, particularly varicella and recently fungal meningitis, can induce vasculopathies that lead to stroke as well or can induce longer-term inflammatory processes that ultimately cause a clinical stroke.

Carotid and vertebral dissections

trauma ,sneezing. Dissections

stroke in the young

Carotid and vertebral

such as in fibromuscular dysplasia and connective tissue disorders

patient may report a minor preceding event, such as spinal manipulation, yoga, working overhead, coughing, or vomiting. Presenting manifestations may include headache, facial pain, visual changes, cranial nerve (CN) palsies, pain over the affected vessel, Horner’s syndrome, amaurosis fugax, SAH, or an ischemic stroke

headache frequently is unilateral and may occur days before onset of the other neurologic symptoms.

diagnosed by

ultrasonography, magnetic resonance angiography (MRA), and computed tomography angiography (CTA).

Medical therapy options

anticoagulation if SAH/intracranial dissection is not suspected. The existing data comparing antiplatelet treatment to anticoagulation is generally limited and antiplatelet treatment is generally simpler and safer. The use of tissue plasminogen activator (tPA) is considered as a safe and effective in extracranial carotid or vertebral dissection patients as in any other eligible patient.

Transient Ischemic Attack

A transient ischemic attack (TIA) was historically defined as a neurologic deficit with complete resolution within 24 hours; however, a portion of TIA cases have evidence of permanent brain ischemia on neuroimaging. Therefore, the American Heart Association (AHA) has adopted a tissue-based definition: A transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction. TIAs constitute an important warning sign for the future development of cerebral infarction. Approximately 10% of the patients who experience a TIA will experience a stroke within 3 months of the sentinel event, and one-half of these occur within the first 2 days.

Hemorrhagic Stroke

It carries a 30-day mortality rate of up to 50% with one-half of patients dying in the first 2 days. Among survivors, only one in five are living independently at 6 months. The two major underlying causes of ICH are **hypertensive vasculopathy** (caused by long-standing hypertension) and cerebral amyloid angiopathy (usually found in elders, which is the result of **amyloid deposition** in cerebral vessel walls). Hypertensive hemorrhage occur in the deep regions, including basal ganglia and thalamus. ICH caused by amyloid angiopathy tends to be lobar in nature and to occur more commonly in older adults.

Other factors leading to ICH include underlying vascular malformations (ie, arteriovenous malformations [AVMs] and aneurysms, drug intoxication [particularly sympathomimetics, such as cocaine], malignant hypertension, saccular aneurysms, blood dyscrasias, venous sinus thrombosis, hemorrhagic transformation of an ischemic stroke, moyamoya disease, and tumors).

BOX 91.1

Most Common Sites for Hypertensive Intracranial Hemorrhage

AFFECTED AREA (FREQUENCY)	COMMON CLINICAL PRESENTATION
Putamen (44%)	Contralateral motor/sensory loss
Thalamus (13%)	Limb pain, speech difficulty
Cerebellum (9%)	Uncoordinated movements of trunk and limbs
Pons (9%)	Numbness, weakness, ataxia, dizziness
Other cortical areas (25%)	Numbness, weakness, language disturbances

CLINICAL FEATURES

Ischemic Stroke

Ischemic strokes can be classified as anterior or posterior circulation strokes, depending on the vasculature involved. The presence of neurologic deficits is highly dependent on collateral flow. In addition to the vascular supply involved, ischemic strokes can be further described by the temporal presentation of their neurologic deficits.

“stroke in evolution” is one in which focal neurologic deficits worsen over the course of minutes or hours. Approximately 20% of anterior circulation strokes and 40% of posterior circulation strokes will show evidence of progression. Anterior circulation strokes may progress within the first 24 hours, whereas posterior strokes may progress for up to 3 days),

Occlusions in the anterior cerebral artery mainly affect frontal lobe function. The patient has altered mentation coupled with impaired judgment and insight, as well as the presence of primitive grasp and suck reflexes on physical examination. Bowel and bladder incontinence may be features of anterior cerebral artery stroke. Paralysis and hypesthesia of the lower limb opposite the side of the lesion are characteristic. Leg weakness is more pronounced than arm weakness in anterior cerebral distribution stroke. Apraxia or clumsiness in the patient’s gait also may be noted.

Marked motor and sensory disturbances are the hallmarks of occlusion of the middle cerebral artery. They occur on the side of the body contralateral to the side of the lesion and usually are worse in the arm and face than the leg. Such disturbances may involve only part of an extremity or the face but almost always are accompanied by numbness in the same region as that of the motor loss. Hemianopsia, or blindness in one-half of the visual field, occurs ipsilateral to the lesion. Agnosia, or the inability to recognize previously known subjects, is common, and aphasia may be present if the lesion occurs in the dominant hemisphere. Patients often have a gaze preference toward the affected hemispherebecause of disruption of the cortical lateral gaze centers.

Aphasia, a disorder of language in which the patient articulates clearly but uses language inappropriately or understands it poorly, also is common in dominant-hemisphere stroke. Aphasia may be expressive, receptive, or a combination of both. *Wernicke's aphasia* occurs when the patient is unable to process sensory input, such as speech, and thus fails to understand verbal communication (receptive aphasia). *Broca's aphasia* refers to the inability to communicate verbally in an effective way, even though understanding may be intact (expressive aphasia). Aphasia should be distinguished from **dysarthria**, which is a motor deficit of the mouth and speech muscles; the dysarthric patient articulates poorly but understands words and word choices. Aphasia is important to recognize because it usually localizes a lesion to the dominant (usually left) cerebral cortex in the middle cerebral artery distribution. *Aphasia* and *dysphasia* are terms that are used interchangeably but must be distinguished from *dysphagia,* which is difficulty in swallowing.

the vertebrobasilar system (ie, **posterior circulation strokes**) can cause the widest variety of symptoms and as a result may be the most difficult to diagnose. The symptoms reflect CN deficits, cerebellar involvement, and involvement of neurosensory tracts. The brainstem also contains the reticular activating system, which is responsible for mediating consciousness, and the emesis centers. Unlike those with anterior circulation strokes, patients with posterior circulation stroke can have loss of consciousness and frequently have nausea and vomiting. vision and thought processing are impaired. Visual agnosia, the inability to recognize seen objects, may be a feature, as may alexia, the inability to understand the written word. A third nerve palsy may occur, and the patient may experience homonymous hemianopsia. One of the more curious facets of this syndrome

is that the patient may be unaware of any visual problem (visual neglect). Vertigo, syncope, diplopia, visual field defects,

weakness, paralysis, dysarthria, dysphagia, spasticity, ataxia, or nystagmus , crossed deficits, such as motor deficits on one side of the body and sensory loss on the other. In anterior circulation strokes, by contrast, abnormalities are always limited to one side of the body.

A focused neurologic examination should assess level of consciousness, speech, CN function, motor and sensory function, and cerebellar function. Level of consciousness and fluency of speech can be rapidly assessed in a dialogue with the patient to determine the presence of dysarthria or aphasia. The head should be evaluated for signs of trauma. Pupillary size and reactivity and extraocular movements provide important information about brainstem function, particularly CN III through CN VI; an abnormal third nerve function may be the first sign of tentorial herniation. Gaze preference suggests brainstem or cortical involvement. Central facial nerve weakness from a stroke should be distinguished from the peripheral causes of CN VII weakness. With a peripheral lesion, the patient is unable to wrinkle the forehead. Assessment of facial sensation, eyebrow elevation and squinting, smiling symmetry, gross auditory acuity, gag reflex, shoulder elevation, sternocleidomastoid strength, and tongue protrusion complete the CN evaluation.

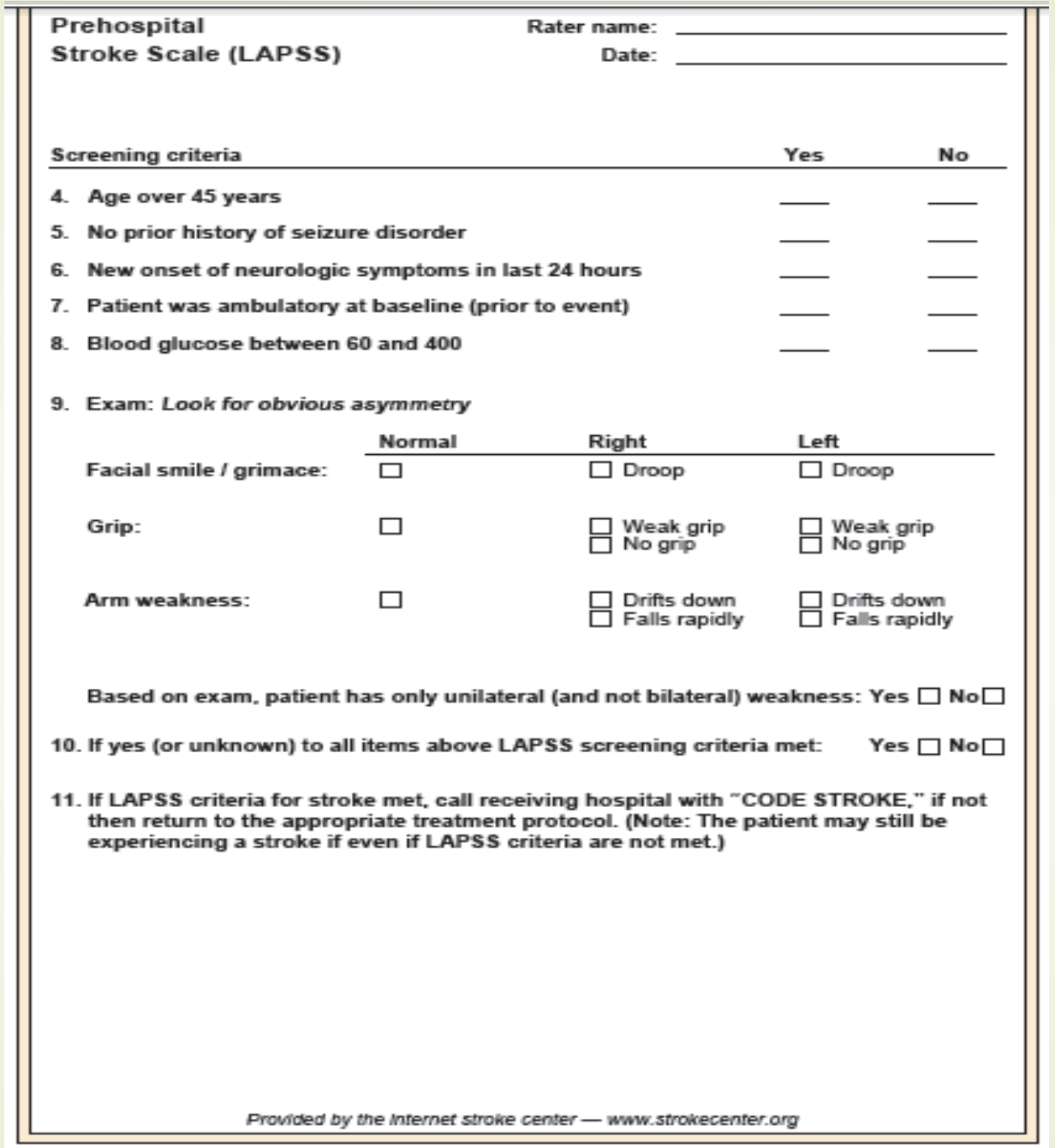
Motor and sensory testing is performed next. Muscle tone can be assessed by moving a relaxed limb. Proximal and distal muscle group strength is assessed against resistance. Pronator drift of the arm is a sensitive sign of motor weakness and can be tested simultaneously by having the patient sit with eyes closed and arms outstretched, with palms toward the ceiling, for 10 seconds. Asymmetrical sensation to pain and light touch may be subtle and difficult to detect. Double simultaneous extinction evaluation tests for sensory neglect and can be easily performed by simultaneously touching the right and left limbs. The patient may feelboth the right and left sides being touched individually but may not discern touch on one side when both are touched simultaneously. Similarly, the ability to discern a number gently scratched on a forearm, graphesthesia, is another easily tested cortical parietal lobe function. These tests can help differentiate a pure motor deficit of a lacunar stroke from a sensorimotor middle cerebral artery deficit.

Cerebellar testing and the assessment of reflexes and gait complete the examination. Finger-to-nose and heel-to-shin evaluations are important tests of cerebellar functions. Asymmetry of the deep tendon reflexes or unilateral Babinski's sign may be an early finding of corticospinal tract dysfunction. Gait testing is commonly omitted yet is an informative part of the neurologic examination when it can be safely performed. Observing routine ambulation and heel-to-toe walking can assess for subtle ataxia, weakness, or focal cerebellar lesions.

Several prehospital stroke scales have been created to assist emergency medical service (EMS) personnel with the rapid assessment of potential stroke patients. Many of these prehospital stroke scales have been prospectively validated for their accuracy in stroke detection. Two of the more commonly used scales include the Cincinnati Prehospital Stroke Scale (Fig. 91.1) and Los Angeles Prehospital Stroke Screen (Fig. 91.2).

Cincinnati Prehospital Stroke Scale	
Facial Droop	
Normal:	Both sides of face move equally
Abnormal:	One side of face does not move at all
Arm Drift	
Normal:	Both arms move equally or not at all
Abnormal:	One arm drifts compared to the other
Speech	
Normal:	Patient uses correct words with no slurring
Abnormal:	Slurred or inappropriate words or mute

Fig. 91.1. Cincinnati Prehospital Stroke Scale. (Adapted from Kothari RU, Pancioli A, Liu T, et al: Cincinnati Prehospital Stroke Scale: reproducibility and validity. Ann Emerg Med 33[4]:373-378, 1999.)



Prehospital Stroke Scale (LAPSS)

Rater name: _____

Date: _____

Screening criteria	Yes	No
4. Age over 45 years	____	____
5. No prior history of seizure disorder	____	____
6. New onset of neurologic symptoms in last 24 hours	____	____
7. Patient was ambulatory at baseline (prior to event)	____	____
8. Blood glucose between 60 and 400	____	____

9. Exam: *Look for obvious asymmetry*

	Normal	Right	Left
Facial smile / grimace:	☐	☐ Droop	☐ Droop
Grip:	☐	☐ Weak grip ☐ No grip	☐ Weak grip ☐ No grip
Arm weakness:	☐	☐ Drifts down ☐ Falls rapidly	☐ Drifts down ☐ Falls rapidly

Based on exam, patient has only unilateral (and not bilateral) weakness: Yes ☐ No ☐

10. If yes (or unknown) to all items above LAPSS screening criteria met: Yes ☐ No ☐

11. If LAPSS criteria for stroke met, call receiving hospital with "CODE STROKE," if not then return to the appropriate treatment protocol. (Note: The patient may still be experiencing a stroke if even if LAPSS criteria are not met.)

Provided by the Internet stroke center — www.strokecenter.org

Fig. 91.2. Los Angeles Prehospital Stroke Screen. (Adapted from Kidwell CS, Starkman S, Eckstein M, et al: Identifying stroke in the field: prospective validation of the Los Angeles Prehospital Stroke Screen [LAPSS]. Stroke 31:71-76, 2000.)

The National Institutes of Health Stroke Scale (NIHSS) is a useful and rapid tool for quantifying neurologic deficit in patients with stroke and can be used in determining treatment optionsn(Table 91.1). NIHSS scores have been shown to be reproducible and valid and to correlate well with the amount of infarcted tissue on computed tomography (CT) scan. The baseline NIHSS score can identify patients who are appropriate candidates for fibrinolytic therapy, as well as those at increased risk for hemorrhage, although it is possible for patients to have disabling strokes with an NIHSS of zero (severe truncal ataxia). In addition, it has been used as a prognostic tool to predict outcome and is currently being used by some stroke centers to stratify patients for entry into treatment trials.

Hemorrhagic Stroke

The classic presentation of ICH is the sudden onset of headache, vomiting, severely elevated BP, and focal neurologic deficits that progress over minutes. Similar to ischemic stroke, ICH is often associated with a motor and sensory deficit contralateral to the brain lesion. Almost 40% of patients will demonstrate significant growth in hemorrhage volume within the first few hours.

Although headache, vomiting, and coma are common, many patients do not have these findings, and the clinical presentation can be identical to that of patients with ischemic stroke; the two cannot be reliably differentiated in the absence of neuroimaging Ongoing assessment of airway and mental status is of paramount importance in patients with ICH because precipitous deterioration is always a possibility. Emergency airway management requires careful judgment: On the one hand, airway control can prevent aspiration, hypoxia, and hypercarbia; on the other, sedation and paralysis can make it difficult to follow the neurologic examination, which can help monitor for hemorrhage expansion, elevated intracranial pressure (ICP), seizure activity, and brainstem herniation.

As with ischemic stroke, a careful neurologic examination is important in localizing the region and extent of injury. Baseline NIHSS and Glasgow Coma Scale scores can be used to assess stroke severity, although the Glasgow Coma Score (GCS) may be more practical to follow for neurologic deterioration (Table 91.2).

In addition, serial examinations can detect early changes that may suggest ongoing bleeding during the acute phase. The ICH score can predict mortality (Table 91.3).

Poor prognostic indicators for patients with ICH include a decreased level of consciousness on arrival, intraventricular hemorrhage, and large ICH volume, all of which can be assessed in the emergency department (ED) (Fig. 91.3).

TABLE 91.1

National Institutes of Health Stroke Scale Scoring Form

ITEM	SCORING DEFINITIONS	SCORE
1a. Level of consciousness (LOC)	0 = Alert and responsive 1 = Arousable to minor stimulation 2 = Arousable only to painful stimulation 3 = Reflex responses or unarousable	
1b. LOC-related questions: Ask patient's age and month. Must be exact.	0 = Both correct 1 = One correct (or dysarthria, intubated, foreign language) 2 = Neither correct	
1c. Commands: Open and close eyes, grip and release nonparetic hand. (Other one-step commands or mimic also acceptable.)	0 = Both correct (acceptable if impaired by weakness) 1 = One correct 2 = Neither correct	
2. Best gaze: Horizontal EOM by voluntary or doll's eye maneuver.	0 = Normal 1 = Partial gaze palsy; abnormal gaze in one or both eyes 2 = Forced eye deviation or total paresis that cannot be overcome by doll's eye maneuver	
3. Visual field: Use visual threat if necessary. If monocular, score field of good eye.	0 = No visual loss 1 = Partial hemianopsia, quadrantanopia, extinction 2 = Complete hemianopsia 3 = Bilateral hemianopsia or blindness	
4. Facial palsy: If patient is stuporous, check symmetry of grimace to pain.	0 = Normal 1 = Minor paralysis, flat NLF, asymmetrical smile 2 = Partial paralysis (lower face = UMN lesion) 3 = Complete paralysis (upper and lower face)	
5. Motor arm: Arms outstretched 90 degrees (sitting) or 45 degrees (supine) for 10 seconds. Encourage best effort. Indicate paretic limb in score box.	0 = No drift for 10 seconds 1 = Drift but does not hit bed 2 = Some antigravity effort, but cannot sustain 3 = No antigravity effort, but even minimal movement counts 4 = No movement at all X = Unable to assess owing to amputation, fusion, fracture, and so on	L or R

	X = Unable to assess owing to amputation, fusion, fracture, and so on	
6. Motor leg: Raise leg to 30 degrees (from supine) for 5 seconds. Indicate paretic limb in score box.	0 = No drift for 5 seconds 1 = Drift but does not hit bed 2 = Some antigravity effort, but cannot sustain 3 = No antigravity effort, but even minimal movement counts 4 = No movement at all X = Unable to assess owing to amputation, fusion, fracture, and so on	L or R
7. Limb ataxia: Check finger-nose-finger, heel-shin position sense; and score only if out of proportion to paralysis.	0 = No ataxia (or aphasic, hemiplegic) 1 = Ataxia in upper or lower extremity 2 = Ataxia in upper *and* lower extremity X = Unable to assess owing to amputation, fusion, fracture, and so on	L or R
8. Sensory: Use safety pin. Check grimace or withdrawal if patient is stuporous. Score only stroke-related losses.	0 = Normal 1 = Mild-moderate unilateral loss but patient aware of touch (or aphasic, confused) 2 = Total loss, patient unaware of touch; coma, bilateral loss	
9. Best language: Describe cookie jar picture, name objects, read sentences. May use repeating, writing, stereognosis.	0 = Normal 1 = Mild-moderate aphasia (speech difficult to understand but partly comprehensible) 2 = Severe aphasia (almost no information exchanged) 3 = Mute, global aphasia, coma; no one-step commands	
10. Dysarthria: Read list of words.	0 = Normal 1 = Mild-moderate; slurred but intelligible 2 = Severe; unintelligible or mute X = Intubation or mechanical barrier	
11. Extinction or neglect: Simultaneously touch patient on both hands, show fingers in both visual fields, ask about deficit, left hand.	0 = Normal, none detected (visual loss alone) 1 = Neglects or extinguishes to double simultaneous stimulation in any modality (visual, auditory, sensation, spatial, body parts) 2 = Profound neglect in more than one modality	

Android Free App: https://play.google.com/store/apps/details?id=com.myprograms.nihss
Apple Free App: https://itunes.apple.com/us/app/nih-stroke-scale-from-statcoder/id408788598?mt=8
Online NIHSS Calculator: www.mdcalc.com/nih-stroke-scale-score-nihss/
EOM, Extraocular movement; *L*, left; *LOC*, level of consciousness; *NLF*, nasolabial fold; *R*, right; *UMN*, upper motor neuron.
Modified from Massachusetts General Hospital Stroke Service. NIH stroke scale materials. Scoring form. Available at www2.massgeneral.org/stopstroke/pdfs/scoring_form.pdf.

DIFFERENTIAL DIAGNOSIS

Ischemic Stroke

Extra-axial collections of blood secondary to trauma can mimic stroke. An epidural or subdural hematoma can cause an altered mental status, focal neurologic signs, and rapid progression to coma. Elders, who represent the age group at highest risk for stroke, can be victims of recurrent falls that lead to chronic subdural hematomas. Carotid dissection may occur after neck trauma or sudden hyperextension and may be associated with focal neurologic signs and symptoms, as with an aortic dissection extends into the carotid arteries. Other structural lesions that may cause focal neurologic sign include brain tumors and abscesses. Air embolism should be suspected in the setting of marked atmospheric pressure changes, such as in scuba diving or during medical procedures or injuries that may allow air into the vascular system. Seizures, altered mental status, and focal neurologic findings also may be manifestations of air embolism.

Metabolic abnormalities also can mimic stroke syndromes.

Hypoglycemia often is responsible for an altered mental status and is a well-known cause of sustained focal neurologic symptoms that can persist for several days.Wernicke's encephalopathy causes ophthalmoplegia, ataxia, and confusion that can be mistaken for signs of cerebellar infarction Migraine may present with focal neurologic findings, with or without headache.

seizure followed by Todd's postictal paralysis may mimic stroke. Bell's palsy, labyrinthitis, vestibular neuronitis, peripheral nerve palsy, and demyelinating diseases may all mimic stroke. Ménière's disease may be difficult to distinguish from a posterior circulation stroke or TIA. Dizziness, vertigo, hearing loss, and tinnitus in Ménière's disease are common, whereas difficulties with vision or speech or other focal symptoms are uncommon

giant cell arteritis is a disease of older adults. It may cause severe headache, visual disturbances, and, rarely, aphasia and hemiparesis. Other symptoms include intermittent fever, malaise, jaw claudication, morning stiffness, and myalgias.

The diagnosis should be suspected in patients with a very high erythrocyte sedimentation rate (ESR) and is confirmed by temporal artery biopsy.

Collagen vascular diseases such as polyarteritis nodosa, lupus, and other types of vasculitis may cause stroke syndromes.

Cerebral venous sinus thrombosis (CVST) is another cause of focal neurologic symptoms that most commonly affects the superior sagittal sinus and lateral sinuses The diagnosis of CVST can be difficult because of the nonspecific nature of symptoms, as well as the variable time frame of symptom onset (from hours to a few weeks).

Patients may have generalized headaches, nausea, vomiting, paresis, visual disturbances, depressed

level of consciousness, seizures, or even symptoms generally ascribed to psychiatric disorders (such as, depression).

Depending on the location of the thrombus, physical examination of the patient may reveal papilledema, proptosis, or palsies of CNs III, IV, and VI, as well as other focal neurologic signs and symptoms.

Risk factors for CVST include trauma, infectious processes, hypercoagulable states, low-flow states, compression of the venous sinus, dehydration, various drugs (such as, androgens, “ecstasy,” and oral contraceptives), and pregnancy or the postpartum state

Hemorrhagic Stroke

The differential diagnosis for ICH is similar to that for ischemic stroke; considerations include migraine, seizure, tumor, abscess, hypertensive encephalopathy, and trauma. Once ICH is appreciated on neuroimaging, it can be difficult to determine the underlying cause. Primary ICH typically manifests as a parenchymal hematoma with new onset neurologic symptoms. Patients with hemorrhagic transformation of an ischemic stroke may have recurrence or worsening of previously established deficits. Patients with known underlying cancer, or perihematomal edema out of proportion to the hemorrhage, should be considered for hemorrhage into a metastasis or primary tumor. Finally, patients with known underlying venous thromboembolic risk factors may have underlying CVST.

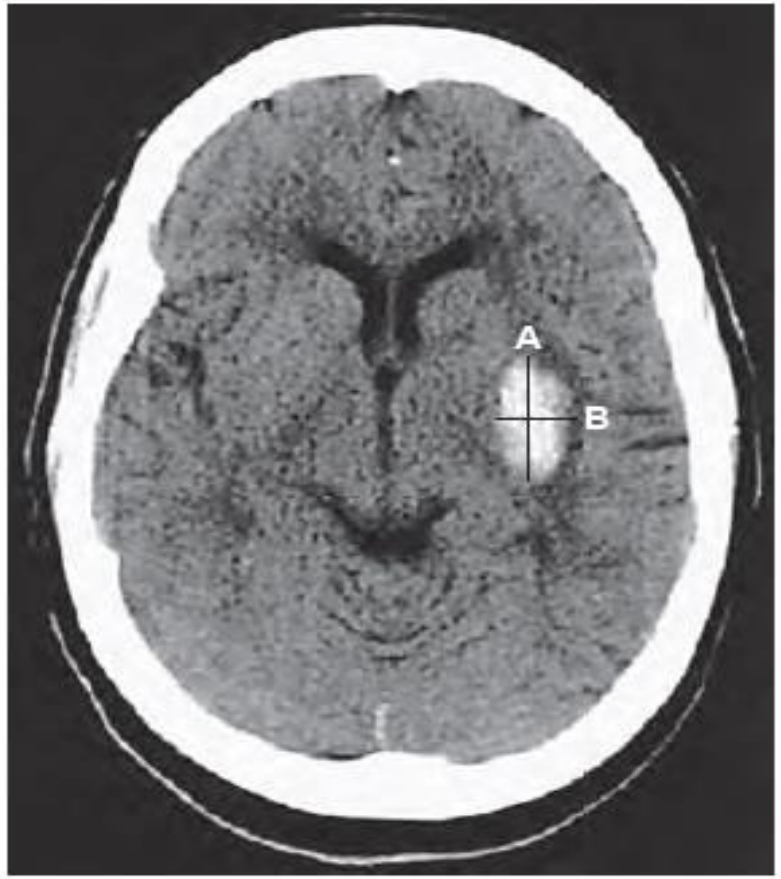


Fig. 91.3. The computed tomography (CT) slice with the largest area of hemorrhage is identified. The largest diameter of the hemorrhage on this slice is measured in centimeters *(line A)*. The largest diameter 90 degrees to A on the same slice is measured *(line B)*. C is the approximate number of 10-mm slices on which the intracerebral hemorrhage (ICH) was seen. (Many centers use 5-mm slices, in which case an adjustment can be made by dividing by 2.) The volume of the hemorrhage = A × B × C ÷ 2 (ABC/2).

TABLE 91.2

Glasgow Coma Scale Score*

EYE OPENING (E)	VERBAL RESPONSE (V)	MOTOR RESPONSE (M)
4 = Spontaneous	5 = Normal conversation	6 = Normal
3 = To voice	4 = Disoriented conversation	5 = Localizes to pain
2 = To pain	3 = Words, but not coherent	4 = Withdraws to pain
1 = None	2 = No words; only sounds	3 = Decorticate posture
	1 = None	2 = Decerebrate posture
		1 = None

*Total score = E + V + M

Shoestring Graphics: Glasgow coma score.
Available at www.ssgfx.com/CP2020/medtech/glossary/glasgow.htm.

TABLE 91.3

Intracerebral Hemorrhage Score Predicting Mortality After Acute Intracerebral Hemorrhage

FEATURE	POINTS
GLASGOW COMA SCALE SCORE	
3 to 4	2
5 to 12	1
13 to 15	0
INTRACEREBRAL HEMORRHAGE VOLUME	
>30 mL	1
≤30 mL	0
INTRAVENTRICULAR HEMORRHAGE (INTRAVENTRICULAR BLOOD)	
Present	1
Absent	0
INTRACEREBRAL HEMORRHAGE LOCATION	
Infratentorial	1
Supratentorial	0
AGE	
≥80 years	1
<80 years	0
30-DAY MORTALITIES FOR TOTAL INTRACEREBRAL HEMORRHAGE SCORES	
0 = 0%	
1 = 13%	
2 = 26%	
3 = 72%	
4 = 97%	
5 = 100%	
6 = Estimated to be 100%; no patients in the study fell into this category	

Adapted from Hemphill JC, et al: The ICH Score. Stroke 32:891-897, 2001.

DIAGNOSTIC TESTING

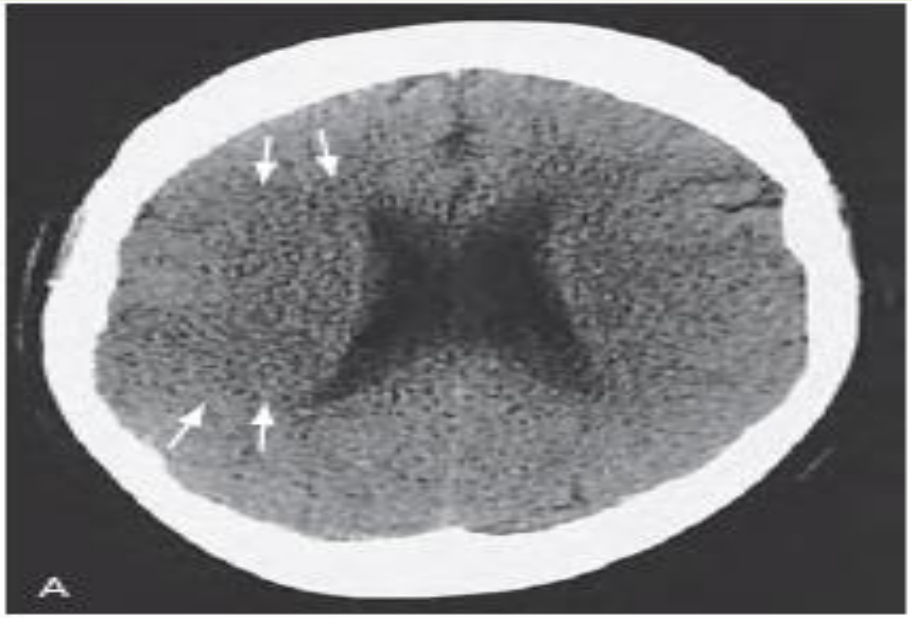
Ischemic Stroke

Although clinical data can help establish the diagnosis, cause, and location of the stroke, confirmatory diagnostic tests are often required to establish the final cause or to eliminate other causes for the deficits. The immediate evaluation includes **cranial imaging, an electrocardiogram (ECG) and hematologic testing**,

particularly blood glucose determination.

An emergent non contrast cranial CT is the standard initial imaging technique for evaluating a patient with a potential stroke. It can quickly differentiate an ischemic stroke from ICH and other mass lesions. This information is crucial to the subsequent therapeutic decisions that will be rapidly made. A CT scan can identify almost all parenchymal hemorrhages larger than 1 cm in diameter and it has a high sensitivity for the detection of SAH.

of ischemic strokes, gross signs of infarction will not appear on routine CT scans for at least 6 to 12 hours, depending on the size of the infarct. However, subtle, early ischemic changes have been noted in up to 67% of non contrast CT scans within the first3 hours. These early ischemic changes include the hyperdense artery sign (acute thrombus in a vessel), sulcal effacement, loss of the insular ribbon, loss of gray-white interface, mass effect, and acute hypodensity (Fig. 91.4). In addition, CTA can be used to identify the presence of intravascular thrombosis, vasculature dissection, or stenosis. In cases in which arterial dissection is suspected, a imaging with MRA or CTA is indicated



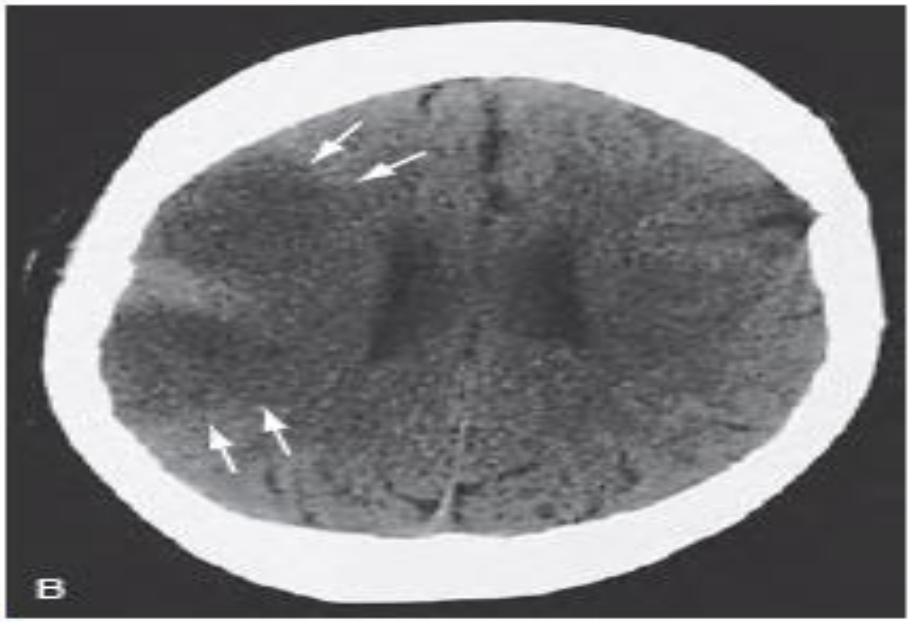
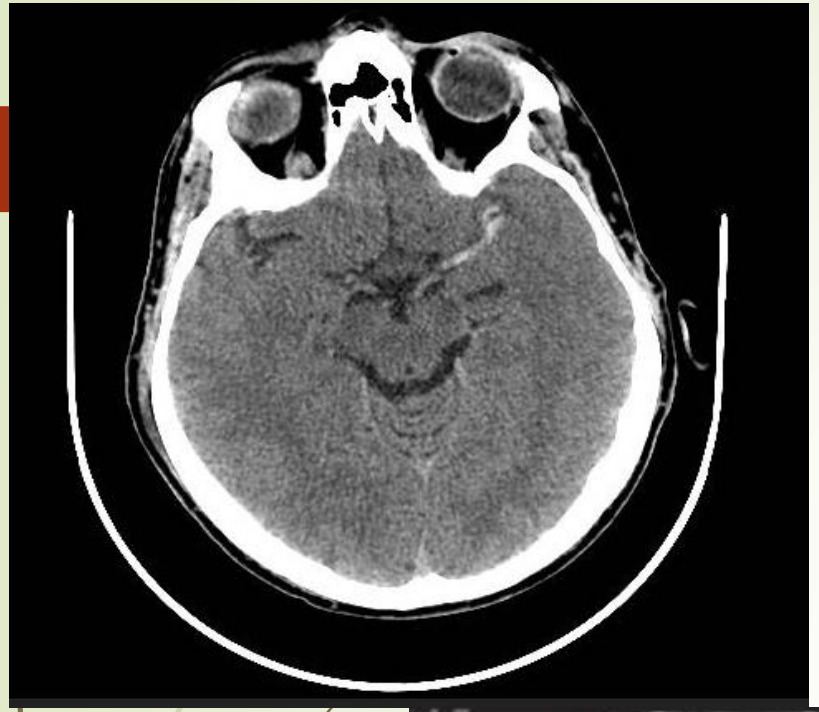
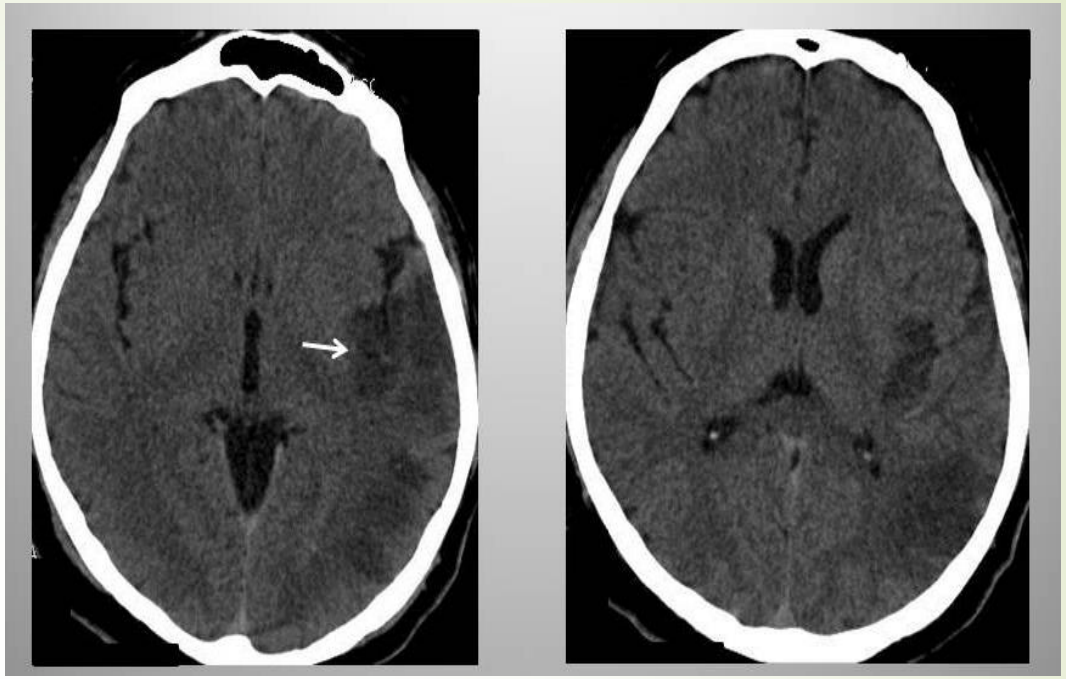
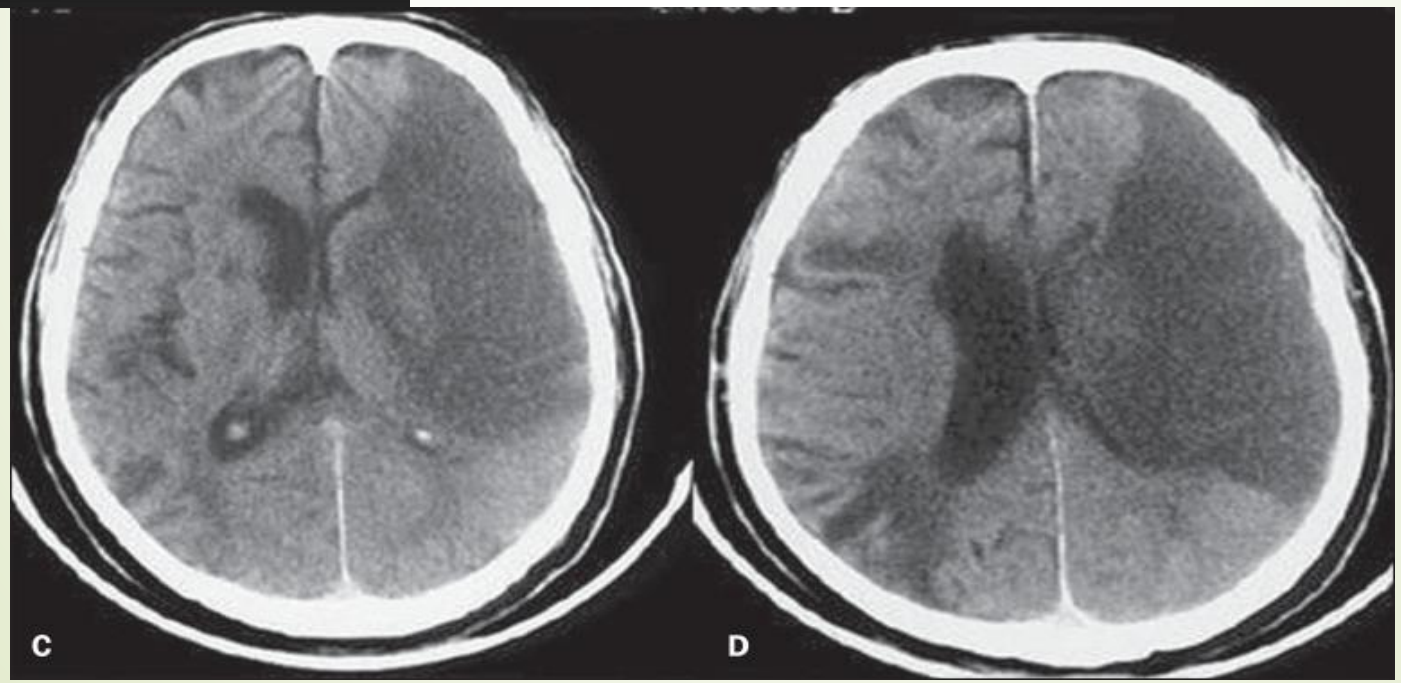


Fig. 91.4. **A,** Computed tomography (CT) scan taken 2 hours and 50 minutes after a large right middle cerebral artery occlusion. There are subtle, ultra-early ischemic changes, including loss of the gray-white interface *(arrows)* and subtle evidence of sulcal effacement. **B,** CT scan of same patient approximately 8 hours after symptom onset shows acute hypodensity *(arrows)* and more prominent sulcal effacement.



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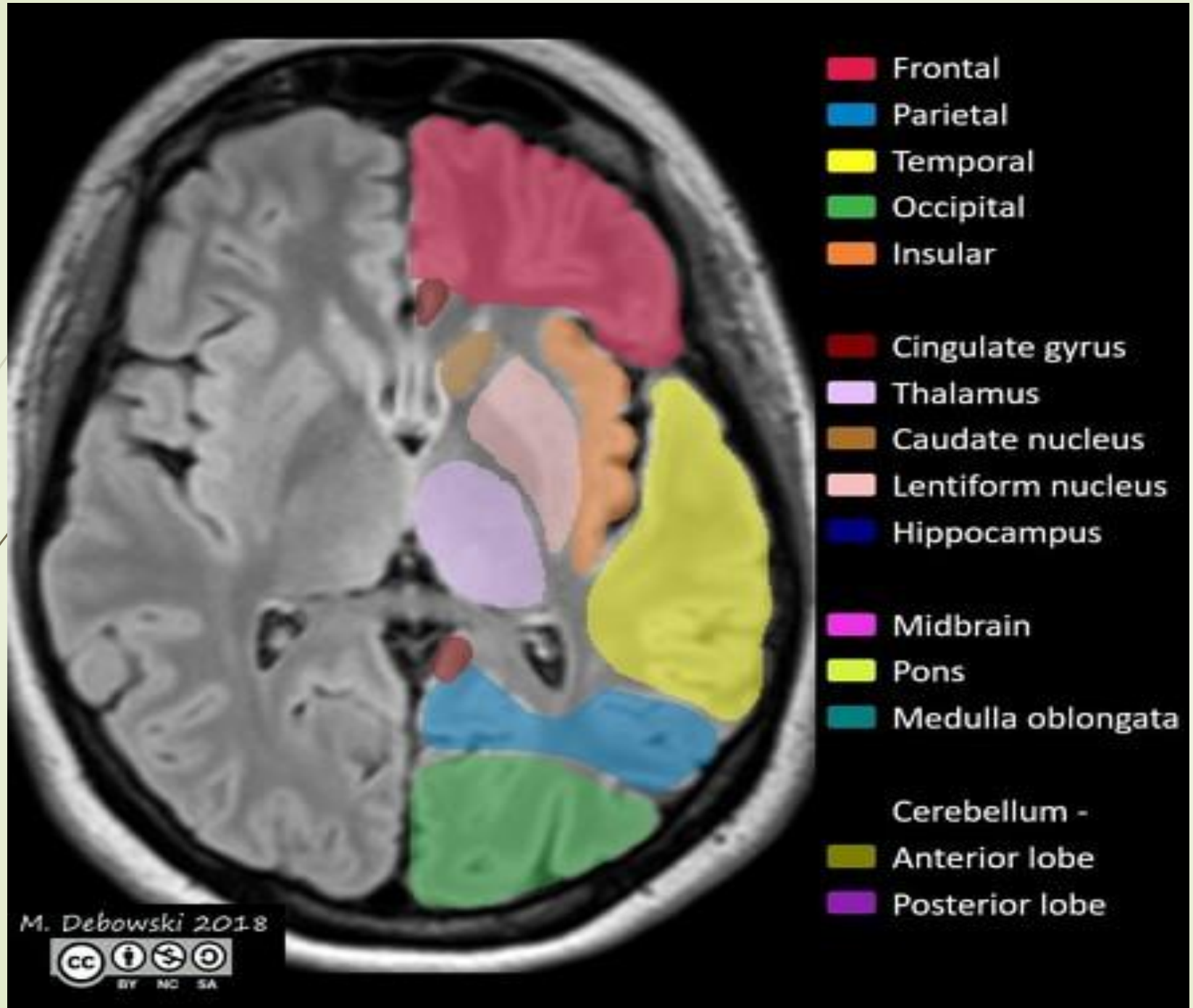
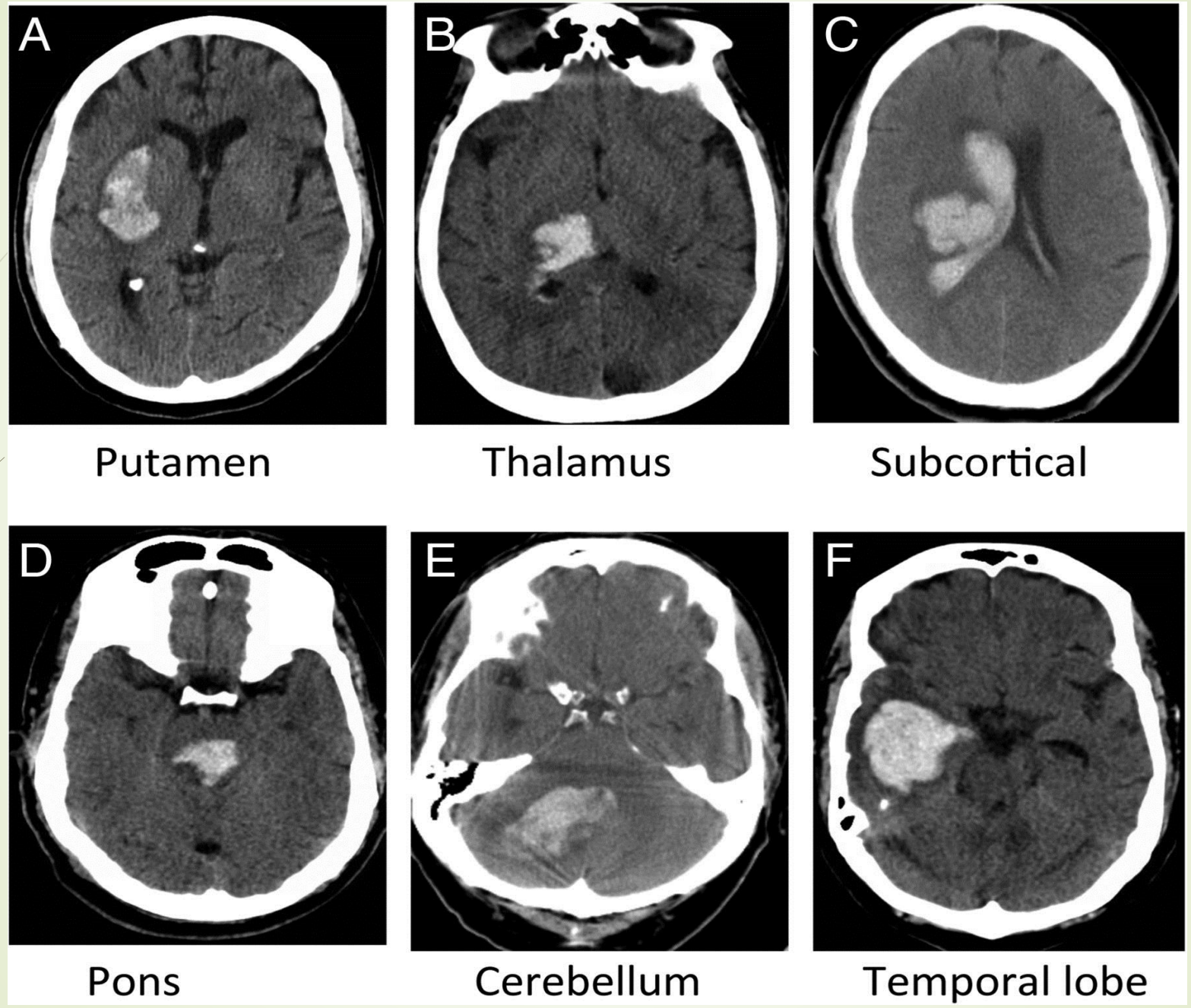

Frontal
Parietal
Temporal
Occipital
Insular
Cingulate gyrus
Thalamus
Caudate nucleus
Lentiform nucleus
Hippocampus
Midbrain
Pons
Medulla oblongata
Cerebellum -
Anterior lobe
Posterior lobe
M. Debowski 2018
BY NC SA


A
B
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Putamen
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Subcortical
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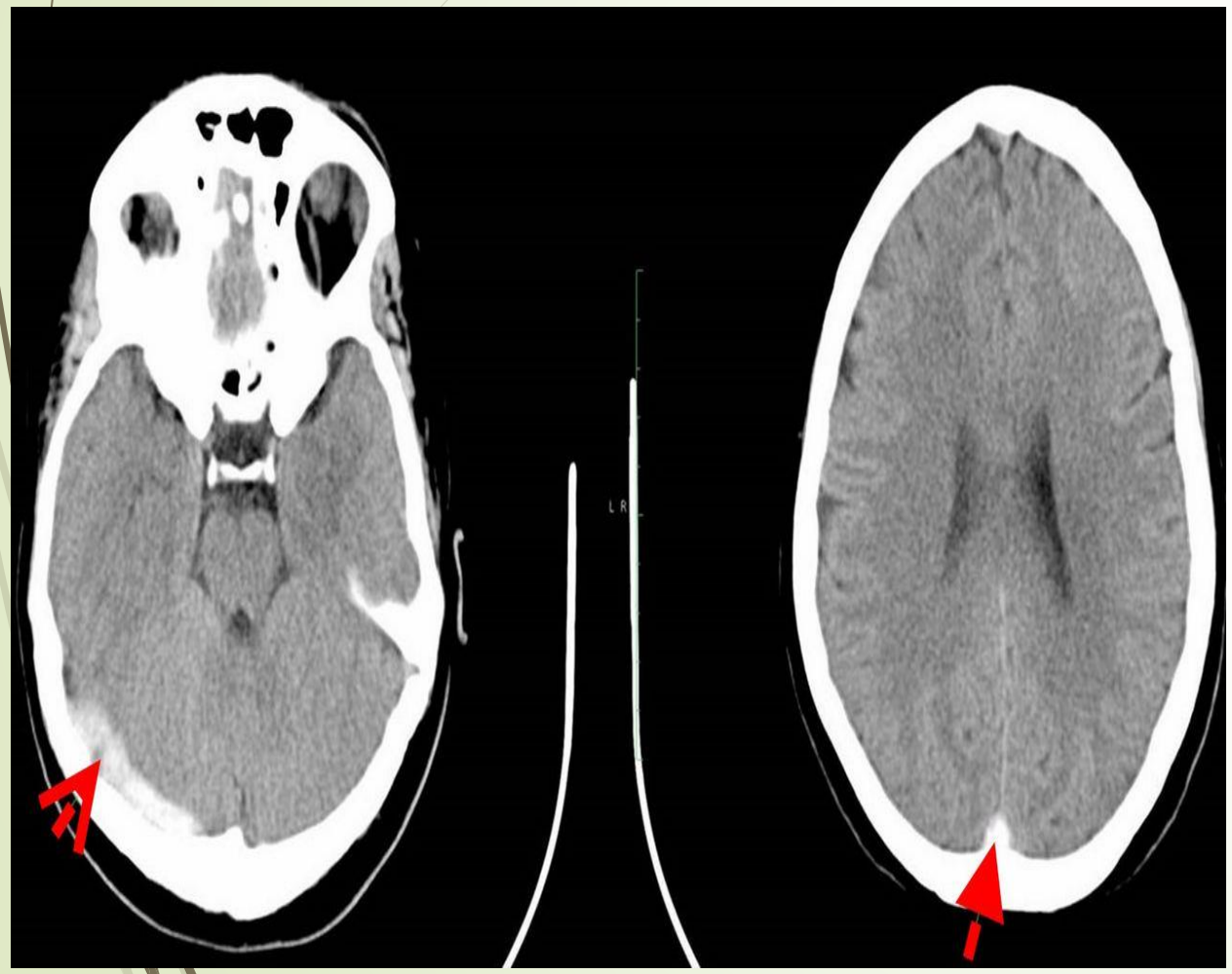
Pons
Cerebellum
Temporal lobe



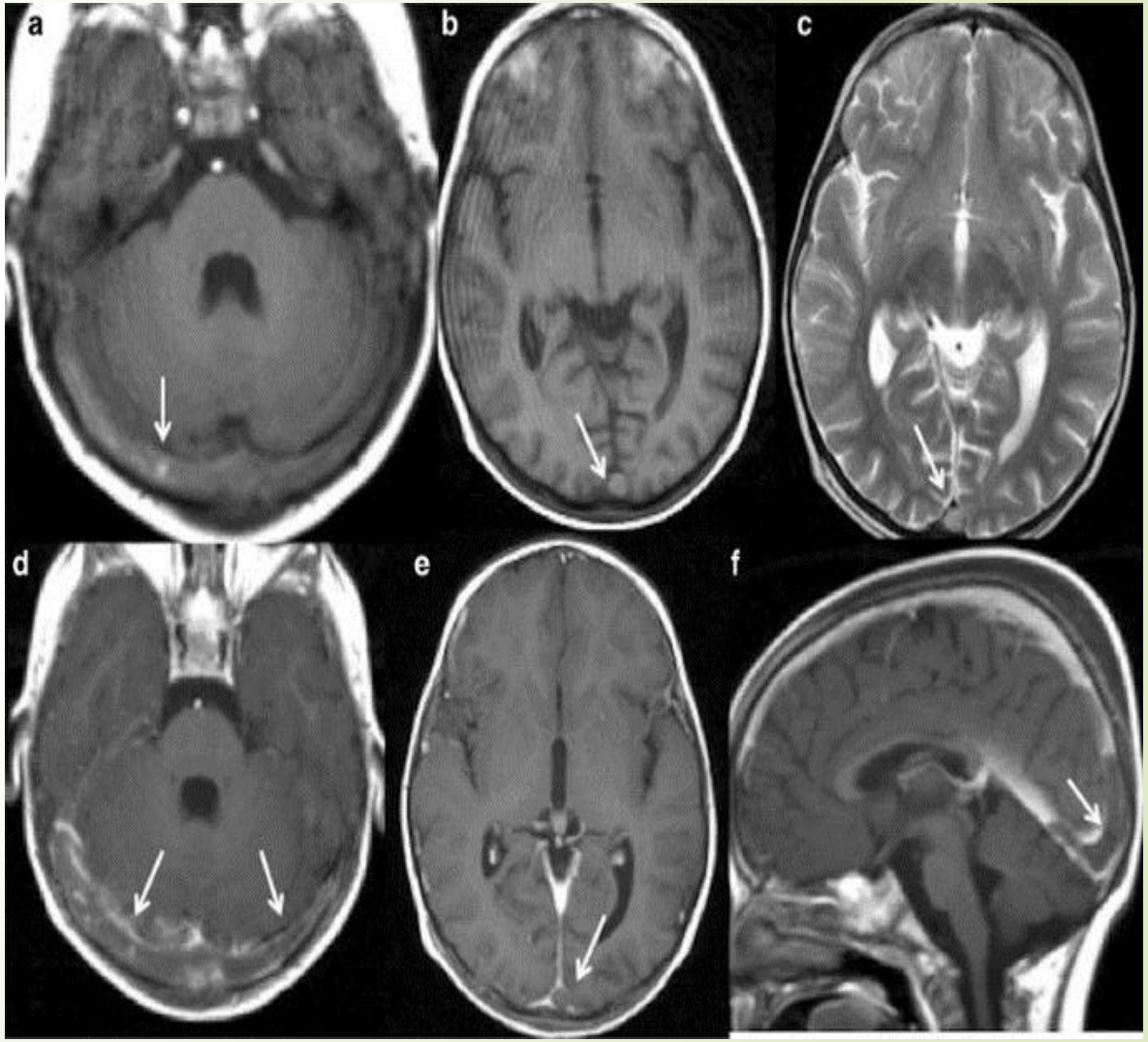

a
b
c
d
e
f

TABLE 91.4

National Institute of Neurological Disorders and Stroke Recommended Stroke Evaluation Targets for Potential Thrombolytic Candidates

MANAGEMENT COMPONENT	TARGET TIME FRAME
Door to doctor	10 minutes
Door to CT completion	25 minutes
Door to CT scan reading	45 minutes
Door to treatment	60 minutes
Access to neurologic expertise*	15 minutes
Access to neurosurgical expertise*	2 hours

*By phone or in person.
CT, computed tomography.

The clinical importance of early ischemic CT findings with regard to fibrinolytic therapy within 3 hours of symptom onset is questionable, because the ability of treating physicians to reproducibly identify these findings is poor and their clinical significance is questionable. Only acute hypodensity and mass effect have been shown to be associated with an increased risk of ICH after fibrinolysis (over that in treated patients without these findings). However, these findings do not exclude patients from fibrinolytic therapy, which is associated with an improved neurologic outcome. Patients with a hyperdense artery sign and acute hypodensity of one-third of the middle cerebral artery distribution tend to have a poorer prognosis; however, their outcomes are still better with tPA treatment than without such treatment.

MRI can visualize ischemic infarcts earlier and identify acute posterior circulation strokes more accurately than CT, and it may be as effective as CT in identifying ICH.10 However, availability, difficulty in accessing critically ill patients, and scan time limit the use of MRI in acute stroke. Advances in MRA technology have allowed a noninvasive method of demonstrating large-vessel occlusions of the anterior and posterior circulation, although small intracranial vascular occlusions may not be readily apparent. With the improvements in MRI and MRA speed and resolution, some stroke centers are replacing CT protocols with limited “stroke protocol” MRI or MRA as the initial imaging modality of choice. The choice of initial cranial imaging modality is highly dependent on the speed with which these scans can be performed and interpreted at each individual center.

Diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) are MRI techniques that take minutes to perform and may allow differentiation between reversible and irreversible neuronal injury. Other potential imaging modalities include CTA and perfusion scans. In CTA, a CT scan is enhanced by an intravenous (IV) contrast agent to better define the vasculature of the brain. Areas of vascular stenosis and occlusion can be visualized with this technique. This information can then be used by interventionalists to determine whether a lesion is amenable to endovascular thrombectomy. Also requiring IV contrast, perfusion CT scans can reveal perfusion deficits within different regions of the brain

In addition, CTA and perfusion CT can differentiate reversible from irreversible ischemic insults.

An **ECG** is indicated in all patients with acute ischemic stroke, because atrial fibrillation and acute myocardial infarction are associated with up to 60% of all cardioembolic strokes.

The hematologic evaluation includes :

CBC, platelet ,PT,PTT,INR, cardiac enzymes, and serum glucose

Other ancillary diagnostic tests to consider include an echocardiogram,carotid duplex scan, and angiogram.

Finally, conventional angiography can demonstrate stenosis or occlusion of both large and small blood vessels of the head and neck. It can detect subtle abnormalities, such as with dissection, that may not be demonstrated with noninvasive imaging techniques.

unresponsive on presentation, their ability to communicate may be altered by dysphasia.

After an ischemic stroke, patients usually can maintain their airway unless the brainstem is affected or significant cerebral edema is compressing the opposite hemisphere. Patients with intact protective airway reflexes should receive

a monitor and IV line should be established.

Routine oxygen supplementation of normoxic stroke patients should be avoided.

Overhydration should be avoided to prevent cerebral edema.

Out-ofhospital personnel should attempt to rapidly ascertain the patient's blood sugar; if this is not possible, glucose should be given when hypoglycemia is strongly suspected with an understanding that hyperglycemia may be neurotoxic.

Electrocardiographic monitoring is recommended to identify life-threatening arrhythmias and atrial fibrillation.

The circumstances surrounding the stroke as well as concomitant medical conditions should be ascertained. A key part of the initial information on stroke patients is the prehospital providers' documentation of the exact time the patient was last seen to be neurologically normal and the level of neurologic functioning. This is especially important because reversible defects may completely resolve by the time the patient has arrived at the hospital. The level of consciousness, gross focal motor deficits, difficulty with speech, clumsiness, facial asymmetry, and any other focal deficits should be noted. Prehospital stroke scales assist in identifying patients who have had a stroke and who are potential candidates for fibrinolytic therapy. Early recognition, notification, and transport by EMS are associated with delivery of fibrinolytic treatment and improved patient outcomes.

In the ED setting, the vital signs should be reassessed on an ongoing basis because patients may rapidly deteriorate even with subacute stroke. Some stroke patients are found at home 1 or 2 days after the event has occurred and may have concomitant illnesses, such as aspiration pneumonia, dehydration, hypothermia, rhabdomyolysis, or myocardial ischemia. Fever necessitates an evaluation to identify sources of infection, followed by prompt institution of treatment. Even minor degrees of hyperthermia have been associated with increased neurologic injury. Oral medications (and food) should be withheld until some form of swallowing assessment has been performed, given the risk of aspiration in patients with an acute stroke.

Blood Pressure Management

The management of BP in patients with acute ischemic stroke and TIA is controversial because of limited data. Current guidelines for the management of hypertension in patients with acute ischemic stroke recommend that antihypertensive treatment be reserved for those with markedly elevated BPs, unless fibrinolytic therapy is planned or specific medical indications are present. These medical indications include acute myocardial infarction, aortic dissection, hypertensive encephalopathy, and severe left ventricular heart failure. Oral or parenteral agents are withheld unless the **patient's systolic pressure is greater than 220 mm Hg, diastolic pressure is greater than 120 mm Hg, or mean arterial pressure (MAP) is greater than 130 mm Hg** (Box 91.2). If parenteral agents are used, labetalol 10 to 20 mg IV push, or calcium channel blocker (eg, nicardipine starting at 5 mg/hour IV), is favored because of ease of titration and limited effect on cerebral blood vessels. Sublingual nifedipine or sublingual nitroglycerin are not recommended, either agent can produce a precipitous drop in BPbecause

.

If fibrinolytic therapy is planned, stringent control of BP is indicated to reduce the potential for intracranial hemorrhage after the thrombolytic is administered (see Box 91.2). Thrombolytic therapy is not recommended for patients whose systolic pressure is consistently higher than 185 mm Hg or whose diastolic pressure is 110 mm Hg at the time of treatment. Simple measures can be used to try lowering BP below this level. Recommended approaches include the use of IV labetalol 10 to 20 mg or continuous nicardipine.Normally normotensive stroke patients with low BP or normally hypertensive stroke patients with low or even low-normal BP are given a fluid bolus to try to increase cerebral perfusion. This is especially important in patients in a dehydrated state. If initial fluid challenge is ineffective, the patient may require vasopressor therapy(eg, with dopamine) to gradually increase MAP and improve cerebral perfusion.

The current consensus regarding management of ICH is to provide antihypertensive treatment with parenteral agents for systolic pressures higher than 160 to 180 mm Hg or MAP higher than 130 mm Hg. Recommended agents include labetalol, esmolol, nicardipine, clevidipine, and hydralazine.

BOX 91.2

Emergency Antihypertensive Therapy for Acute Ischemic Stroke

INDICATION THAT PATIENT IS ELIGIBLE FOR TREATMENT WITH INTRAVENOUS RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR OR OTHER ACUTE REPERFUSION INTERVENTION

Blood Pressure Level

Systolic >185 mm Hg or diastolic >110 mm Hg

Labetalol 10 to 20 mg IV over 1 to 2 minutes; may repeat 1 time

or

Nicardipine infusion, 5 mg/hr; titrate up by 2.5 mg/hr at 5- to 15-minute intervals, maximum dose 15 mg/hr; when desired BP attained, reduce to 3 mg/hr

Other agents (hydralazine, enalaprilat, and so on) may be considered when appropriate.

If BP does not decline and remains >185/110 mm Hg, do not administer rtPA.

MANAGEMENT OF BLOOD PRESSURE DURING AND AFTER TREATMENT WITH RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR OR OTHER ACUTE REPERFUSION INTERVENTION

Monitor BP every 15 minutes during treatment and then for another 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours.

Blood Pressure Level

Systolic 180 to 230 mm Hg or diastolic 105 to 120 mm Hg

Labetalol 10 mg IV over 1 to 2 minutes; may repeat every 10 to 20 minutes; maximum dose of 300 mg

or

Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min

Systolic >230 mm Hg or diastolic 121 to 140 mm Hg

Labetalol 10 mg IV over 1 to 2 minutes; may repeat every 10 to 20 minutes; maximum dose of 300 mg

or

Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min

or

Nicardipine infusion, 5 mg/hr; titrate up to desired effect by increasing 2.5 mg/hr every 5 minutes to maximum of 15 mg/hr

If BP not controlled, consider sodium nitroprusside.

BP, Blood pressure; *IV,* intravenous; *rtPA,* recombinant tissue plasminogen activator.

Adapted from Jauch EC, Saver JL, Adams HP Jr, et al: Guidelines for the early management of adults with ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 44(3):870-947,

Thrombolytic Therapy

The recommended dose for **rtPA is 0.9 mg/kg IV to a maximum of 90 mg (10% of the dose given as a bolus followed by an infusion lasting 60 minutes).** Although the initial recommended time window for IV rtPA administration was 3 hours because the patient was last known to be at their neurologic baseline, a subsequent study has demonstrated the usefulness of IV rtPA at 3 to 4.5 hours in a carefully selected subgroup of acute ischemic stroke patients (Table 91.5 and Box 91.3).

The more recent CHANCE trial found aspirin and clopidogrel for 90 days significantly reduced recurrent stroke (from 11.7% to 8.2%) in Aspirin should not be given for the first 24 hours to patients who have received a fibrinolytic agent and not until a swallowing study has been performed.

BOX 91.3

Fibrinolytic Therapy for Acute Ischemic Stroke in the 3- to 4.5-Hour Time Window Inclusion and Exclusion Criteria

INCLUSION CRITERIA

Diagnosis of ischemic stroke causing measurable neurological deficit
Onset of symptoms within 3 to 4.5 hours before beginning treatment

RELATIVE EXCLUSION CRITERIA

Older than 80 years old
Severe stroke (NIHSS > 25)
Taking an oral anticoagulant regardless of INR
History of both diabetes and prior ischemic stroke

INR, International normalized ratio; *IV*, intravenous; *NIHSS*, National Institutes of Health Stroke Scale; *rtPA*, recombinant tissue plasminogen activator.

TABLE 91.5

Comparison of AHA/ASA Acute Stroke Management Guidelines and Previous and New FDA Prescribing Information for Alteplase (Activase) Treatment in Acute Ischemic Stroke

Inclusion criteria for fibrinolytic therapy

- Diagnosis of ischemic stroke causing measurable neurological deficit
- Onset of symptoms less than 3 hours before beginning treatment

CRITERION	AHA/ASA ACUTE STROKE MANAGEMENT GUIDELINE 2013[a]	OLD ALTEPLASE (ACTIVASE) PI (UPDATED 2009)	NEW ALTEPLASE (ACTIVASE) PI (FEBRUARY 2015)
Prior stroke	Exclusion: prior stroke within 3 mo	Contraindication: recent (within 3 mo) previous stroke	Removed entirely
Seizure at onset	Relative exclusion: seizure at onset with postictal neurological impairments	Contraindication: seizure at the onset of stroke	Removed entirely
Bleeding diathesis/ OACs	Exclusion: Platelet count <100 000/mm^3 Heparin received within 48 h, resulting in abnormally elevated aPTT Current use of anticoagulant with INR >1.7 or PT >15 s Current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevated sensitive laboratory tests	Contraindication: known bleeding diathesis including but not limited to: Current use of OACs (eg, warfarin sodium), an INR >l.7, or a PT >15 s Administration of heparin within 48 h preceding the onset of stroke with an elevated aPTT at presentation Platelet count <100 000/mm^3 Warning for all indications: patients currently taking OACs	Bleeding diathesis remains a contraindication, but all laboratory values and specific examples removed

ICH	Exclusion: history of previous ICH	Contraindication: history of ICH	Contraindication removed Warning added for recent ICH
BP	Exclusion: Elevated BP (systolic >85 mm Hg or diastolic >10 mm Hg)	Contraindication: uncontrolled hypertension at the time of treatment (eg, >185 mm Hg systolic or >110 mm Hg diastolic)	Contraindication: current severe uncontrolled hypertension remains, specific BP values removed Warning for BP >175/110 mm Hg remains for all alteplase (Activase) indications
Blood glucose	Exclusion: blood glucose <50 mg/dL	Warning: because of the increased risk for misdiagnosis of acute ischemic stroke, special diligence is required in making this diagnosis in patients whose blood glucose values are ≈50 or >400 mg/dL	Removed entirely
Severe stroke	Not listed	Warning: patients with severe neurological deficit (NIHSS score >22) at presentation; there is an increased risk of ICH in these patients	Removed entirely
Mild stroke	Relative exclusion: only minor or rapidly improving stroke symptoms (clearing spontaneously)	Warning: safety and efficacy in patients with minor neurological deficit or with rapidly improving symptoms have not been evaluated; therefore, treatment of patients with minor neurological deficit or with rapidly improving symptoms is not recommended	Removed entirely
Neuroimaging findings	Exclusion: CT demonstrates multilobar infarction (hypodensity >1/3 cerebral hemisphere)	Warning: Major early infarct sign (substantial edema, mass effect, or midline shift on CT)	Removed entirely
SAH	Exclusion: symptoms suggest SAH	Contraindication: Suspicion of SAH on pretreatment evaluation	Contraindication: subarachnoid hemorrhage

Comparison of AHA/ASA Acute Stroke Management Guidelines and Previous and New FDA Prescribing Information for Alteplase (Activase) Treatment in Acute Ischemic Stroke—cont'd

CRITERION	AHA/ASA ACUTE STROKE MANAGEMENT GUIDELINE 2013[a]	OLD ALTEPLASE (ACTIVASE) PI (UPDATED 2009)	NEW ALTEPLASE (ACTIVASE) PI (FEBRUARY 2015)
Use in specific populations			
Pregnancy	Relative exclusion	Warning: pregnancy Category C	No change
Nursing mothers	Not listed	Not mentioned	Unknown risk
Children	Inclusion: ≥18 y of age	Indicated for adults	Pediatric use not established
Elderly	Not listed	Warning for all indications: advanced age (eg, >75 y) may increase risks	Warning added: age >77 y was 1 of several interrelated baseline characteristics associated with an increased risk of ICH; efficacy results suggest a reduced but still favorable clinical outcome
Gastrointestinal or genitourinary bleeding	Warning: gastrointestinal or genitourinary bleeding within the past 21 d	Warning: gastrointestinal or genitourinary bleeding within the past 21 d	Warning: gastrointestinal or genitourinary bleeding

[a]From Jauch EC, Saver JL, Adams HP Jr, et al; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 44:870–947, 2013.

AHA/ASA, American Heart Association/American Stroke Association; *aPTT*, activated partial thromboplastin time; *BP*, blood pressure; *CT*, computed tomography; *FDA*, US Food and Drug Administration; *ICH*, intracerebral hemorrhage; *INR*, international normalized ratio; *NIHSS*, National Institute of Health Stroke Scale; *OAC*, oral anticoagulant; *PI*, prescribing information; *PT*, prothrombin time; *SAH*, subarachnoid hemorrhage.

Endovascular Rescue Therapy

studies have conclusively demonstrated that patients with severe strokes and evidence of proximal large vessel occlusions have significantly better functional outcomes when treated with the new generation devices. trials emphasize the need for better regionalization of stroke care to reduce the time to definitive reperfusion. Unlike acute ischemic stroke, non-acute cases of intracranial occlusions are best treated with medical therapy and not permanent intracranial stents.

Intra-arterial thrombolysis currently does not have a defined role in the treatment of acute ischemic stroke.

Anticoagulation

current AHA guidelines recommend against the routine use of heparinoids in this population. However, heparin is sometimes considered by vascular neurologists in select patients at high risk for stroke progression, including patients with crescendo TIAs or TIA from a cardioembolic source (eg, atrial fibrillation, patients with a highgrade carotid artery stenosis, patients with posterior circulation TIA, and patients with evolving strokes). Heparin or LMWH is often instituted to treat carotid and vertebral artery dissection, unless a contraindication such as intracranial extension is present. If a dissection is diagnosed and the patient has no symptoms of ischemia, treatment with antiplatelet therapy alone may be an option. Heparin therapy should not be initiated in patients with suspected endocarditis or in any patient until a CT scan has ruled out intracranial bleeding.

Many patients are coagulopathic at the time of their ICH; and for patients on anticoagulants, emergency reversal will theoretically minimize the risk of further bleeding, although this is not backed up by clinical trial evidence. For patients on warfarin, reversal is achieved using IV vitamin K (10 mg IV or subcutaneously), supplemented with either fresh frozen plasma (FFP) (2 to 4 units) or prothrombin complex concentrate (PCC) (25 to 50 units/kg depending on INR—dosing varies for other PCCs by formulation). Of the new generation oral anticoagulants, at this time only dabigatran has a specific antidote (idarucizumab)

Fourfactor PCC is likely to be the most quickly available reversal agent for apixaban and rivaroxaban; hemodialysis will rapidly eliminate dabigatran from the circulation but is not generally practical for patients with serious bleeding.

For patients with clinical or radiographic evidence of **elevated ICP**, therapies aimed at lowering ICP should be considered. First, neurosurgical consultation is obtained to evaluate the benefits of an external ventricular drain (EVD) placement or hematoma evacuation. Hyperventilation can be a temporizing measure pending more definitive treatment. Mannitol moves fluid from the intracranial compartment, thereby reducing cerebral edema. Hypertonic saline (3% or 23.4%) can be used as an alternative to mannitol or in combination. Other experimental modalities include barbiturate coma and hypothermia.

AEDs

should be reserved for patients with known or suspected seizure, including nonconvulsive seizures

DISPOSITION

Ischemic Stroke and Transient Ischemic Attacks

Most patients can be managed on a general medical or telemetry unit, although there is evidence suggesting a benefit from admission to a stroke-specific unit. Patients with large acute hemispheric strokes (associated with increased risk of herniation) or with significant posterior circulation-related changes and those treated with a fibrinolytic agent should be monitored in a step-down or ICU for at least 24 hours. In many centers, patients require admission to receive a prompt evaluation for TIA. However, some centers have developed ED

observation unit protocols or rapid outpatient TIA clinics to ensure an expedited evaluation.

TABLE 91.6

ABCD2 Score for Assessing Stroke Risk in Patients With a Transient Ischemic Attack

RISK FACTOR	POINTS
Age >60 years old	1
Initial BP >140/90 mm Hg	1
Unilateral weakness	2
SPEECH IMPAIRMENT	
Without weakness	1
Symptoms 10 to 59 minutes	1
Symptoms ≥60 minutes	2
History of diabetes	1
RESULT	
0 to 3 = Low risk (1% risk of stroke in 48 hours)	
4 to 5 = Moderate risk (4.1% risk of stroke in 48 hours)	
≥6 = High risk (8% risk of stroke in 48 hours)	

BP, Blood pressure.


THANKS FOR YOUR ATTENTION