



FOUNDATIONS  
Emergency Medicine

# Foundations Frameworks

## Approach to Stroke Management

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Here is a step-wise approach to managing patients with a suspected stroke (those with altered mental status and/or focal neurologic deficits):

### 1. Initial Stabilization

- a. ABCs
  - i. Evaluate the patient's ability to protect their airway and consider intubation. Recognize that ischemic strokes usually do not require intubation unless they have a large ischemic territory or a stroke affecting centers responsible for maintaining mental status (i.e. the brainstem)
  - ii. Hypertension can be an early physiologic response and a sign of stroke; if the patient is hypotensive, you must investigate the cause
  - iii. Beware of Cushing response (indicative of imminent herniation): irregular respirations, hypertension, bradycardia
    1. Emergently lower intracranial pressure (ICP) with hypertonic saline/mannitol and surgical decompression
- b. Evaluate pupils; consider ordering early mannitol (1 g/kg over 15 min) or hypertonic saline (3% 250 mL bolus) for suspected increased ICP
- c. Check a glucose and rule-out hypoglycemia

### 2. CT Brain

- a. Obtain an emergent CT brain to determine if the neurologic deficit is from hemorrhagic vs. (suspected) ischemic stroke. If there is concern for large vessel occlusion (LVO) stroke, obtain a CT angiogram.
- b. Hemorrhagic stroke
  - i. Hypertensive
    1. Patients usually have chronic HTN, vascular disease
    2. Blood seen in parenchyma, usual locations: Basal ganglia, thalamus, pons, cerebellum
  - ii. SAH
    1. Spontaneous: aneurysmal, arteriovenous malformations (blood typically in the basal cisterns)
      - a. 12% initial mortality for aneurysmal SAH. Patients can be severely obtunded, have associated cardiogenic shock/arrhythmias, neurogenic pulmonary edema. Provide supportive care as indicated.
  - iii. "Convexal": blood is high on convexities, outside of brain (seen in amyloid angiopathy, reversible cerebral vasoconstriction, or hypertensive encephalopathy)
  - iv. Hemorrhagic transformation: blood seen in areas outside of the deep structures (e.g., a hemorrhagic stroke seen in an MCA territory)
- c. Ischemic Stroke
  - i. Large vs. small vessel ischemic stroke: suspicion based on neurologic exam
  - ii. Large vessel = Cortical (large territory)
    1. Anterior Cerebral Artery (ACA): strength/sensation of leg
    2. Middle Cerebral Artery (MCA): strength/sensation of arm and face, aphasia if left-sided, neglect if right-sided
    3. Posterior Cerebral Artery (PCA): contralateral visual field deficit
    4. Basilar artery: motor and sensory deficit + cranial nerve findings, possible AMS

5. Superior Cerebellar Artery (SCA), Posterior Inferior Cerebellar Artery (PICA), Vertebral Arteries: ataxia, dysarthria, vertigo, cerebellar findings
6. Diagnostic pearls: (think large vessel IF)
  - a. Decreased consciousness
  - b. Motor AND sensory involvement
  - c. Higher level “thinking” processes affected
  - d. Aphasia, right sided neglect (“take your left hand and point at the ceiling”), eye deviation (frontal eye fields affected)
7. Complications: cerebral edema leading to herniation and death
- iii. Small vessel = Lacunar syndrome (damage to the white matter pathways [a.k.a. the wiring], not the higher level “thinking” processes)
  1. No change in consciousness
  2. No aphasia or neglect
  3. Motor OR sensory involvement, not both

### 3. Treatment Approach

- a. All strokes:
  - i. ABC stabilization
  - ii. Check glucose
  - iii. Head of bed up (if concerned for aspiration or ICP), NPO, bedrails up to prevent fall
  - iv. CBC, chemistry, coags, troponin, EKG
- b. Hemorrhagic:
  - i. BP control:
    1. Uncontrolled hypertension can worsen amount of hemorrhage, but hypotension will worsen ischemia to surrounding areas experiencing mass effect
    2. Aim for a systolic blood pressure of 140-180 using a nicardipine drip (CCB also reduces risk of cerebral vasospasm causing secondary ischemic stroke)
  - ii. Anticoagulation reversal – check platelets, coags, take a good medication reconciliation
    1. Anti-platelet medications- consider reversal with platelet transfusion, ddAVP (new data showing *potential harm* in platelet transfusion for spontaneous ICH)
    2. Warfarin- reverse with vitamin K, Fresh Frozen Plasma (FFP), or Prothrombin Complex Concentrate (PCC)
    3. Dabigatran- reversal agent (idarucizumab, i.e. Praxbind) or dialysis
    4. Anti-Xa NOACs- specific reversal agents are under investigation, consider PCC
  - iii. Treat all seizures with benzodiazepines. Consider seizure prophylaxis with levetiracetam (indicated for those who have had a seizure)
  - iv. Increased ICP
    1. Signs of increased ICP:
      - a. Worsening mental status, CN 6 palsy, fixed & dilated pupil, decorticate/decerebrate posturing
      - b. EKG with QT prolongation and diffuse, deep T wave inversions (cerebral T waves)
      - c. US – dilated optic nerve (> 5 mm)
      - d. CT showing midline shift, effacement of sulci
    2. Treatment:
      - a. Surgical decompression
      - b. Elevate head of bed
      - c. Optimize venous drainage - remove cervical collar, don't place internal jugular central lines
      - d. Hyperventilate to a pCO<sub>2</sub> of 35-40 – only a temporizing measure to be performed if patient begins to actively herniate
      - e. Osmotic agents
        - i. Hypertonic (3%) saline 250 mL bolus
        - ii. Mannitol (1 gram/kg over 15 minutes)

iii. 23% Hypertonic saline “bullet”

c. Ischemic Strokes

i. Treatment options:

1. tPA: patient presenting within 4.5 hours of symptom onset, no contraindications (SBP > 180, elevated INR on anticoagulation, previous ICH, recent surgery, > 3hr in pt > 80 yo, recent spinal injection/LP)
2. Clot retrieval: emerging data shows benefit of clot retrieval in patients with LVO and viable brain tissue on CT perfusion/MR up to 24 hours post onset of symptoms

d. Disposition: neuro ICU, +/- neurosurgical or neuro-interventionalist consult

**4. Consider Stroke Mimics**

a. Check a blood sugar

b. Evaluate for the following stroke mimics:

- i. Hypoglycemia
- ii. Seizure (e.g. postictal Todd's paralysis)
- iii. Metabolic encephalopathy
- iv. Illicit drug use or EtOH intoxication
- v. Complex migraines
- vi. Peripheral nerve compression
- vii. Peripheral causes of vertigo
- viii. PRES or hypertensive encephalopathy
- ix. Conversion disorder
- x. Recrudescence (re-emergence of symptoms of an old stroke from a toxicologic, metabolic, infectious, or cardiac cause)

**References:**

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