Chapter 101 – Stroke

Episode Overview:

1. List 10 conditions on the differential diagnosis for ischemic stroke
2. List 5 etiologies of ischemic stroke & list 3 specific causes of hemorrhagic stroke
3. Describe the expected findings in the following stroke syndromes:
   a. Anterior Cerebral Artery
   b. Middle Cerebral Artery
   c. Vertebrobasilar
4. List four common sites for hypertensive intracranial hemorrhage and six symptoms associated with hypertensive ICH
5. Define Transient Ischemic Attack
6. What is the risk of stroke after TIA?
7. How do you risk stratify a patient presenting with a TIA?
8. Describe the management of high-risk TIA and low-risk TIA; how does the presence of carotid stenosis change management?
9. What are the NINDS recommended stroke evaluation target times for potential thrombolytic candidates?
   a. Describe pre-hospital management goals for ischemic stroke
   b. List 3 immediate diagnostic steps to be performed in the ER for stroke
10. When should you consider thrombolysis in patients presenting with stroke? Outline the management for a patient with ischemic stroke who is not eligible to receive thrombolysis
11. Describe the main patient groups, target levels, and medications for emergency antihypertensive therapy for acute ischemic stroke (stroke patient with and without potential for reperfusion therapy)
12. What are the inclusion criteria for fibrinolytic therapy in stroke?
13. What are the AHA exclusion criteria for fibrinolytic therapy in stroke?
   a. How do they compare with the NEW alteplase guidelines (2015)?
14. What are the inclusion and relative exclusion criteria for acute ischemic stroke in the 3-4.5 hour time window?
15. What is the dosing strategy for rt-PA?
16. Describe BP targets in hemorrhagic stroke and indications for surgical intervention
17. List three potential complications of ischemic stroke
18. What are poor prognostic factors for ICH? What is a short formula for calculating ICH volume?

Wisecracks:

1. List 8 risk factors for stroke in young patients
2. Differentiate between Wernicke’s and Broca’s aphasia
3. List 5 early CT findings consistent with ischemic stroke
4. What were the exclusion criteria in ECASS III?
5. What did the NINDS trial show?
6. List inclusion criteria and exclusion criteria for treatment of rtPA in ischemic stroke
Rosen's in Perspective

Yes, it's here. The episode we all feared….

Let’s jump into this content. You know, we do heavy topics on CRACKCast, here’s another heavy weight…

“Stroke can be defined as 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 symptoms may be sudden or stuttering, often with transient or permanent loss of neurologic function.”

The brain needs oxygen and glucose and when a stroke occurs that fine balance is immediately altered. If there is a small amount of blood flow to an area of the brain we get “electrical silence” of the tissues, but if the flow is totally cut off beyond the “point of no return” membrane failure occurs with complete cell death. This process occurs with ischemia, or in intracranial hemorrhage (ICH) where the mass effect causes damage along with the subsequent cascade of inflammation and edema.

Remember:
- 87% of all strokes are ischemic - occluded cerebral vessel
- 13% are hemorrhagic - bleeding into the parenchyma of the brain = ICH (not SAH)

The big picture of current therapies are:
- Prevention of secondary neurologic injury by:
  - Blood Pressure management
  - Anticoagulation
  - Thrombolytic therapy
  - Catheter based interventions
  - Surgery

1) **List 10 conditions on the differential diagnosis for ischemic stroke.**

- **Structural**
  - acute/chronic subdural or epidural hematoma
  - Brain tumour
  - Brain abscess
- **Vascular**
  - Air gas embolism
  - Aortic dissection
  - Carotid / cervical artery dissection
  - Migraine
  - Giant cell arteritis
  - Polyarteritis nodosa
  - Lupus / vasculitis
  - Cerebral venous sinus thrombosis
- **Metabolic**
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www.canadiem.org/crackcast

- Hypoglycemia
- Wernicke’s encephalopathy (ophthalmoplegia, ataxia, confusion)
- Post-seizure induced Todd’s paralysis

- Infectious
  - Bell’s palsy
  - Labyrinthitis
  - Vestibular neuronitis

- Demyelination or Peripheral Neuropathy
  - Peripheral nerve palsy
  - Demyelinating disease
  - Meniere’s disease

This list is similar to the differential diagnosis of hemorrhagic stroke, but hemorrhagic stroke includes:

- Hypertensive encephalopathy
- Posterior Reversible Encephalopathy Syndrome

2) List 5 etiologies of ischemic stroke & list 3 specific causes of hemorrhagic stroke

- **Ischemic**
  - In situ thrombosis in ⅓ of these strokes
    - Large vessels
      - Occur at cerebral vessel branch points - e.g. internal carotid artery
      - and caused by ulcerated atherosclerotic plaque → platelet plugs
    - Small vessels
      - Lacunar or small vessel strokes at the small terminal vessel end points [diabetes, hypertension!]
  - Embolic obstruction in ⅓ of all strokes
    - Cardiac - atrial fibrillation; septic emboli from infective endocarditis.
    - Noncardiac
      - Extracranial proximal carotid plaque (amaurosis fugax)
      - AGE

- **Hemorrhagic:**
  - Hypertensive vasculopathy
  - Cerebral amyloid angiopathy (age related amyloid deposition in the cerebral vessel walls)
  - Ateriovenous malformations
  - Aneurysms
  - Drug related - cocaine
  - Malignant hypertension
  - Blood dyscrasias
  - Hemorrhagic transformation of ischemic stroke/tumour
3) Describe the expected findings in the following stroke syndromes:

Blood is supplied to the brain by the anterior and posterior circulations. The anterior circulation originates from the carotid system and perfuses 80% of the brain, including the optic nerve, retina, and fronto-parietal and anterior-temporal lobes.

The first branch off the internal carotid artery is the ophthalmic artery, which supplies the optic nerve and retina. As a result, the sudden onset of painless monocular blindness (amaurosis fugax) identifies the stroke as involving the anterior circulation (specifically the ipsilateral carotid artery) at or below the level of the ophthalmic artery. The internal carotid arteries terminate by branching into the anterior and middle cerebral arteries at the circle of Willis.

Although the posterior circulation is smaller and usually supplies only 20% of the brain, it supplies the brainstem (which is critical for normal consciousness, movement, and sensation), cerebellum, thalamus, auditory and vestibular centers of the ear, medial temporal lobe, and visual occipital cortex. The posterior circulation is derived from the two vertebral arteries that ascend through the transverse processes of the cervical vertebrae. The vertebral arteries enter the cranium through the foramen magnum and supply the cerebellum by the posterior inferior cerebellar arteries. They join to form the basilar artery, which branches to form the posterior cerebral arteries.
a) **Anterior Cerebral Artery**

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.

b) **Middle Cerebral Artery**

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.

c) **Vertebrobasilar**

Pathology in 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 cranial nerve 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.

The posterior cerebral artery supplies portions of the parietal and occipital lobes, so vision and thought processing are impaired. 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 may be associated with vertebrobasilar artery insufficiency.

Posterior circulation strokes also demonstrate 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 brief hot stroke neurological exam:
- GCS / LOC
- Speech (tell me what happened?) assessment
- Inspect for signs of trauma
- Cranial nerve assessment
- Sensory and motor x 4 limbs
- Cerebellar function
- Glucose

**4) List four common sites for hypertensive intracranial hemorrhage and six symptoms associated with hypertensive ICH.**
Most common sites for hypertensive intracranial hemorrhage - Box 91.1

- AFFECTED AREA (FREQUENCY)
  - Putamen (44%)
  - Thalamus (13%)
  - Cerebellum (9%)
  - Pons (9%)
  - Other cortical areas (25%)

- COMMON CLINICAL PRESENTATION
  - Contralateral motor/sensory loss
  - Limb pain, speech difficulty
  - Uncoordinated movements of trunk and limbs
  - Numbness, weakness, ataxia, dizziness
  - Numbness, weakness, language disturbances

5) Define 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."

NB: No time window is given!

6) What is the risk of stroke after TIA?

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.

7) How do you risk stratify a patient presenting with TIA?

A simple but suboptimal assessment called the ABCD² score (ie. ABCD squared, for Age, Blood pressure, Clinical features, Duration of symptoms, and Diabetes) was designed to identify patients at high risk of ischemic stroke in the first seven days after TIA.

It’s frowned upon because it was designed for the primary care setting and doesn’t do a very good job in predicting who is high or low risk in real life. Check out this 2015 systematic review: https://www.ncbi.nlm.nih.gov/pubmed?term=26136519

Anyway, it seems to be all we have to work with in the ER….
8) Describe the management of high-risk TIA and low-risk TIA; how does the presence of carotid stenosis change management?

Patients with new-onset TIAs should receive an expedited evaluation and treatment owing to the substantial short-term risk of stroke and other adverse events. Emergency neuroimaging, vascular imaging (such as with a carotid doppler study, MRA, or CTA), electrocardiography, and basic blood tests should be performed. A medically or surgically treatable cause for TIAs (eg. high-grade carotid stenosis, mural thrombus) should be sought, which would require in-hospital treatment such as anticoagulation, stenting, or carotid endarterectomy.

Check out: https://www.uptodate.com/contents/image?imageKey=NEURO%2F107065&topicKey=NEURO%2F1123&rank=1~150&source=see_link&search=tia

As per the Canadian Best practise guidelines here we should be starting patients with TIAs in the ED on antiplatelet therapy (as long as they have no contraindications). The NNT for this is 77. ASA alone if not currently on antiplatelet. Add second antiplatelet agent (eg. plavix plus ASA) if already on single agent and had a TIA.

The more recent CHANCE trial found that dual antiplatelet therapy with aspirin and clopidogrel for 90 days significantly reduced recurrent stroke (from 11.7% to 8.2%) in patients initially presenting with high-risk TIA or mild stroke.
(NIHSS below 4). 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 - Rosen's 9th Ed.

Currently viable strategies include blood pressure reduction, statins, antiplatelet therapy, and lifestyle modification, including smoking cessation.

9) What are the NINDS recommended stroke evaluation target times for potential thrombolytic candidates?

• Describe pre-hospital management goals for ischemic stroke
  o Identify stroke
  o Early hospital notification
  o Rapid transport
  o Ensure CNS oxygenation and perfusion (sp02>95%)

• List 3 immediate diagnostic steps to be performed in the ER for stroke
  o Blood glucose test (+ PTT, INR, CBC)
  o ECG
    ▪ To exclude atrial fibrillation or acute MI
  o Cranial imaging (CT or CT/CTA)

<table>
<thead>
<tr>
<th>MANAGEMENT COMPONENT</th>
<th>TARGET TIME FRAME</th>
</tr>
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<tbody>
<tr>
<td>Door to doctor</td>
<td>10 minutes</td>
</tr>
<tr>
<td>Door to CT completion</td>
<td>25 minutes</td>
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<tr>
<td>Door to CT scan reading</td>
<td>45 minutes</td>
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<tr>
<td>Door to treatment</td>
<td>60 minutes</td>
</tr>
<tr>
<td>Access to neurologic expertise*</td>
<td>15 minutes</td>
</tr>
<tr>
<td>Access to neurosurgical expertise*</td>
<td>2 hours</td>
</tr>
</tbody>
</table>

10) When should you consider thrombolysis in patients presenting with stroke?

• Thrombolysis Indications:
  o Clinical diagnosis of ischemic stroke causing measurable neurologic deficit
  o Onset of symptoms <4.5 hours before beginning treatment; if the exact time of stroke onset is not known, it is defined as the last time the patient was known to be normal
  o Age greater than or equal to 18 years
Outline the management for a pt with ischemic stroke who is not eligible to receive thrombolysis.

- Prevent secondary neurologic injury
  - Optimal fluid and electrolyte balance
  - Avoid hypo- or hyperglycemia
  - Prevent fever
- Start an antiplatelet (or add clopidogrel on to ASA) within 48 hrs
- Consider starting LMWH in hospital after consultation with neurology
- Start aggressive statin therapy
- Treat high blood pressure only if the hypertension is extreme (systolic blood pressure >220 mmHg or diastolic blood pressure >120 mmHg)
- Prevent aspiration, early mobilization and physiotherapy and multidisciplinary stroke unit care

11) Describe the main patient groups, target levels and medications for emergency antihypertensive therapy for acute ischemic stroke (stroke patient with and without potential for reperfusion therapy)

This is controversial. Not a solid based of data to back these guidelines up.

Here are the big patient groups:
1. Patient is a reperfusion candidate (tPA and/or reperfusion intervention)
2. Patient is not a reperfusion candidate and has no special medical conditions that mandate BP control

For the latter group management of hypertension in patients with acute ischemic stroke, 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**,
- **mean arterial pressure (MAP) is greater than 130 mm Hg**

For the groups where aggressive lowering of BP is indicated, such as when fibrinolytic therapy is planned (See Box 91.2):
- **BP must be <185/110**

If patient is eligible for treatment with rtPA or other acute reperfusion intervention:
- Check blood pressure level
- If 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, etc.) 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 rtPA 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.
- If BP 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
- If BP 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 may requiring lowering if specific medical indications are present. These medical indications include:

- **Acute myocardial infarction**
- **Aortic dissection** (systolic goal 100-120 mmHg)
- **Hypertensive encephalopathy** (10-15% reduction)
- **Severe left ventricular heart failure.** (amelioration of heart failure and improvement in pulmonary edema, which can often be achieved with a 10 to 15 percent reduction in blood pressure)

**12)** What are the inclusion criteria for fibrinolytic therapy in stroke?

**586 Stroke** February 2016

Table 4. Inclusion and Exclusion Characteristics of Patients With Ischemic Stroke Who Could Be Treated With Intravenous rtPA Within 3 Hours From Symptom Onset

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
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<tbody>
<tr>
<td>Diagnosis of ischemic stroke causing measurable neurological deficit</td>
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<tr>
<td>Onset of symptoms &lt;3 h before treatment begins</td>
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<tr>
<td>Age ≥18 y</td>
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**Endovascular therapy:**

- Usually performed after tPA for strokes caused by proximal large vessel occlusions within 3 hours of onset.

**13)** What are the AHA exclusion criteria for fibrinolytic therapy in stroke?
a) How do they compare with the NEW alteplase prescribing information (2015)
   i) Same inclusion criteria for consideration of fibrinolytic therapy
   ii) Removed many exclusion criteria:
      (1) These are the only exclusion criteria:
         (a) Bleeding diathesis (vague with no specifics)
         (b) Current severe, uncontrolled HTN
         (c) SAH
         (d) Pregnancy
      (2) These are the "warning points"
         (a) Current GI or GU bleeding
         (b) Pediatrics
         (c) Recent ICH
14) What are the inclusion and relative exclusion criteria for acute ischemic stroke in the 3-4.5 hr time window?

See 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

15) What is the dosing strategy for rtPA?

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).

16) Describe blood pressure targets in hemorrhagic stroke and indications for surgical intervention

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.

Don't forget reverse any pre-existing coagulopathy:
- **Patient on warfarin:**
  - Vitamin K 10 mg IV
  - FFP 2-4 units or prothrombin complex concentrate (PCC)
- **Patient on dabigatran**
  - Idarucizumab
- **Patient on apixaban, rivaroxaban**
  - PCC
- **Patient on ASA or Clopidogrel:**
  - No current evidence to support platelet transfusion, unless plt count < 30,000

Surgery indications:
- Neurosurgery consultation for patients with clinical or radiographic evidence of elevated ICP
  - Question re: benefits of an external ventricular drain (EVD) placement or hematoma evacuation
A few things to know about:

- Surgical evacuation is most helpful for cerebellar hemorrhage within 48 hrs of onset - assuming they have a sizable lobar hemorrhage that is close to the cortical surface with associated progressive neurological deterioration
- Non-cerebellar ICH is rarely intervened on
- People with intraventricular hemorrhage or posterior fossa hemorrhages may benefit from a ventricular drain

17) **List three potential complications of ischemic stroke**

- Cerebral edema progressing to increased ICP and deterioration (needing ICU care)
- Hemorrhagic transformation
- GI bleeding
- CHF
- Hospital related complications: DVT, PE, UTI's, pneumonia
- Post-stroke seizures
- Post-stroke delirium and depression

18) **What are poor prognostic factors for ICH? What is a short formula for calculating ICH volume?**

**ICH score predicting mortality:**

- Scored 0-6.

<table>
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<th>Points</th>
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<tr>
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</table>

- *No patients in study score 6, but estimates 100%


**ICH formula:**
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).

**FORMULA**

Volume of Hemorrhage = \( A \times B \times C \times \text{Slices} / \text{Hemorrhage Shape} \)

- A = Length
- B = Width
- C = Slice Width
- # Slices with Hemorrhage Present

Hemorrhage Shape: If Round/Ellipsoid: 2; Otherwise: 3. (See references below for this update to the formula.)
Of note, the volume of an ellipsoid is \( \frac{4}{3} \times \pi \times (ABC/2) \); when you estimate \( \pi \) to be \(~3\), the volume becomes \( ABC/2 \).


**Wisecracks**

1. **List 8 risk factors for stroke in young patients**

- **Big categories**
  - Hypercoagulable states
  - Vasospasm
  - Post-infectious
  - Traumatic
  - Connective tissue disorders
- **Specific situations**
  - Pregnancy
  - Use of oral contraceptives
  - Antiphospholipid antibodies
  - Protein C and S deficiencies
  - Sickle cell anemia
  - Polycythemia
  - Migraine syndromes
  - Recreational drugs
    - **Cocaine**
    - Amphetamines
  - Recent infection from varicella or fungal meningitis
  - Carotid / vertebral trauma leading to dissection
    - Spinal manipulation / cough / yoga / vomiting

2. **Differentiate between Wernicke’s and Broca’s aphasia**

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).

**Sidebar:**

<table>
<thead>
<tr>
<th>Aphasia / dysphasia</th>
<th>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.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysarthria</td>
<td>A motor deficit of the mouth and speech muscles; the dysarthric patient articulates</td>
</tr>
</tbody>
</table>
3. List 5 early CT findings consistent with ischemic stroke

Most findings don’t appear on routine CT scans till 6-12 hrs post infarction. However, subtle, early ischemic changes have been noted in up to 67% of noncontrast CT scans within the first 3 hours. These early ischemic changes include:

1. Hyperdense artery sign (acute thrombus in a vessel)
2. Sulcal effacement (due to edema)
3. Loss of the insular ribbon
4. Loss of gray-white interface,
5. Mass effect
6. Acute hypodensity (Fig. 91.4)

Great video: https://radiopaedia.org/articles/loss-of-the-insular-ribbon-sign

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, imaging with MRA or CTA is indicated.
4. What were the exclusion criteria in ECASS III?

https://www.wikijournalclub.org/wiki/ECASS_III

**Exclusion Criteria**

- Intracranial hemorrhage on CT or MRI
- Symptoms of subarachnoid hemorrhage even without signs on CT
- Major ischemic infarct on CT or MRI
- NIHSS score >25
- Seizure at onset of stroke
- Unknown onset of symptoms or greater than 4.5 hours prior to drug administration
- Symptoms minor or rapidly improving prior to administration
- Stroke or head trauma within 3 months
- History of combination of previous stroke and diabetes
- Heparin within past 48 hours and PTT greater than upper level of normal
- Oral anticoagulant therapy
- Platelets <100k
- SBP >185, DBP >110; or aggressive anti-hypertensives required to reduce BP below these ranges
- Blood glucose <50 or >400mg/dl
- Major surgery or trauma within 3 months
- Other disorders associated with increased risk of bleeding

Bottom line: patients with severe strokes were excluded from the trial

5. What did the NINDS trial show?

https://www.wikijournalclub.org/wiki/NINDS

**Bottom Line**

In patients with ischemic stroke within 3 hours, tPA administration significantly improved NIHSS scores but did not confer survival benefit.

6. List inclusion criteria and exclusion criteria for treatment of rtPA in ischemic stroke

The inclusion criteria are easy:

1. Age > 18 yrs
2. Ischemic stroke with a measurable neuro deficit
3. Onset of symptoms in 3-4.5 hrs before beginning treatment

The exclusion criteria are a beast, let’s break them down into “absolute” and “relative”
Absolute

1. History
   a. Ever
      i. ICH
      ii. Brain neoplasm, AVM, aneurysm
   b. Recent
      i. Head trauma / prior stroke in last 3 months
      ii. Intracranial or intraspinal surgery
   c. Today
      i. Use of warfarin, DOAC AND evidence of its bleeding diathesis effect

2. Exam
   a. Symptoms suggest SAH
   b. BP > 185/110
   c. Active internal bleeding
   d. Bleeding diathesis

3. Investigations
   a. Blood glucose < 2.7 mmol/L
   b. Plt count <100,000
   c. Elevated aPTT
   d. INR > 1.7 or PT > 15 sec
   e. CT showing multilobar infarction

Relative (for the 3 and 3-4.5 hr window)

1. History
   a. Any oral anticoagulant
   b. Older > 80
   c. Hx of DB and prior ischemic stroke
   d. Major surgery or serious trauma within 14 days
   e. GI or GU hemorrhage in the past 21 days
   f. MI in the last 3 months

2. Exam
   a. Severe stroke (NIHSS > 25)
   b. Minor or rapidly improving stroke symptoms

3. Labs

The aggregate risk of symptomatic ICH is about 6% in trials and observational studies, for people who received tPA. (but this takes all comers into consideration - probably less in the young patient and higher in the older patient).