Stroke	
Ischemic (85%)	Hemorrhagic (15%)
(1) Thrombotic (80%) (2) Embolic (20%) (3) Lacunar (rare) (4) Global 2/2 Shock (other) (5) Carotid Stenosis (other)	(1) ICH (75%) (2) SAH (25%) (3) Epi/Subdural Hematomas (other)

General

- Epidemiology: 1st leading cause of disability and 3rd leading cause of death
- Definitions
    i. Stroke = acute brain disorder, of vascular origin, accompanied by neurologic dysfunction (usually unilateral and as such LOC is not usually seen, except when hemorrhagic stroke or post ischemic stroke edema), that persists longer than 24hrs and thus is irreversible (each minute of ischemia 2 million neurons die), Evolving Stroke = one that is worsening vs Completed Stroke = maximal deficit has already occurred
    ii. RIND (Reversible Ischemic Neurologic Deficit) = reversible lasting >24hrs but <2wks
    iii. TIA (Transient Ischemic Attack) = reversible lasting <24hrs usually 30min (important to note b/c there is a 10% risk of stroke per yr) note that TIA and strokes are indistinguishable early on, reversible b/c reperfusion occurs 2/2 (1) collateral via Circle of Willis or External Carotid thru Ophthalmic Artery or (2) clot breakup, TIA are usually 2/2 embolism however severe carotid stenosis can cause TIAs
- DDx: carotid dissection, migraines, vasospasm, drug abuse, HTN encephalopathy, psychogenic, vasculitis, subclavian steal syndrome (stenosis of sublcavian artery proximal to origin of vertebral artery, when pt exercises left arm a reversal of blood flow occurs such that the arm steals blood away from vertebral circulation resulting in symptoms of posterior fossa insufficiency), venous vein thrombosis (Dx w/ MRV, 2/2 hypercoagulable state, S/S: focal deficits, intracranial HTN)
- Posterior Reversible Encephalopathy Syndrome (PRES)
    i. Etiology: unclear but associated w/ new onset malignant HTN, glomerulonephritis, various cytotoxic/immunosuppressive meds
    ii. Mech: capillary leakage and acute disruption of BBB
    iii. S/S: HA, AMS, seizures, vision changes
    iv. Dx: MRI (white matter edema uniquely in the posterior parietal/temporal/occipital cortex)
    v. Tx: if underlying cause is corrected then PRES is reversible after 2wks

Algorithm

- **Suspect ischemic vs hemorrhagic based on hx regardless immediately get a non-contrast CT (50% sensitive for ischemic and 97% for hemorrhagic) to rule out hemorrhagic (you can't really rule-in ischemic b/c low sensitivity), CT can also discriminate stroke from nonvascular lesions like tumors**
- **If ischemic regardless of exact cause administer thrombolytics if meets checklist or anti-platelet if fails part of checklist then determine thrombotic vs embolic based on hx, echo, US, etc**
- **If hemorrhagic then NS consult**

Ischemic

(1) Thrombotic Infarct

a. Etiology
    i. Atherosclerosis
b. Location
    i. Big Cranial Vessels (Cerebrum/Cerebellum) esp IC, MCA, VA, BA
c. Symptoms
    i. Progressive symptoms
    ii. Pt usually wakes up with deficits
    iii. Pt usually had similar deficits in past 2/2 TIA b/c thrombi are always at the same spot
d. Diagnosis
    i. CT (infarction ~ air therefore darker aka hypodense vs. hemorrhage ~ bone therefore lighter aka hyperdense, sometimes you don't see anything but hyperdense vessels suggesting thrombus, loss of Grey/White jxn, effacement of cerebral sulci)
        1. <50% sensitive at <24hrs from onset of Sx w/ false negative due to (1) early <1d (earlier with MRI) (2) small <1cm (3) posterior fossa
    ii. MRI (opposite)
        1. 90% sensitive at <24hrs from onset of Sx
    iii. *** the data is now showing that CTA is very helpful***
e. Prognosis
f. Treatment/Complications (despite the emphasis on primary treatment most pts actually succumb to the complications therefore the focus should be on managing these problems, 25% of pts worsen during the first 2d after stroke and there is no way of predicting which one, goal is to prevent penumbra from becoming infarction)
    i. Primary

1. Thrombolytics if meets checklist (Yes to All Inclusion Criteria and NO to All Exclusion Criteria)
    a. all based on one landmark study, 12% morbidity and 4% mortality reduction with 5% ICH increased incidence, despite this the FDA approved its use
        i. Tissue plasminogen activator for acute ischemic stroke: The National Institute of Neurological Disorders and Stroke r-tPA Stroke Study (NINDS). N Eng J Med 1995;333:1581-1587
    b. of the 700,00 strokes/yr, 600,000 were ischemic, 12,000 meet criteria and received t-PA (only 1.5% of strokes), only 1,400 benefited from t-PA (only 0.2% of strokes)
    c. it used to be that tPA could only be given <3hrs from onset of Sx but a recent study (European Cooperative Acute Stroke Study – ECASS) showed its effectiveness up to 4.5hrs w/ improved outcome and no change in mortality b/w placebo and tPA and no increase in ICH risk compared to the 3hr standard
    d. after thrombolytic therapy start anti-platelet the following day as noted below
    e. 6.5% r/o hemorrhagic conversion vs 0.5% w/o tPA (w/ half of each dyeing but note that overall mortality was 5.4% for tPA and 6.4% for placebo hence tPA is used)
    f. ICH usually occurs w/in first 24hrs (+RFs: bigger the size of stroke, edema, and shifting)
    g. not as good for carotid and basilar than for branches of carotid
    h. consider IA rt-TPA or rPro-UK if <6hrs (NB not FDA approved)
        i. more effective and less ICH therefore some are arguing that IA is the best administrative route
    i. venous/arterial access and nasogastric tubes restricted during the first 24hrs
    j. if pt worsens (like HA, AMS, etc) always suspect bleeding as a complication to thrombolysis and d/c infusion, obtain blood for coag tests, obtain surgical consult, consider transfusions/cryo/platelets
2. Anti-Platelets: Aggrenox/Plavix/Aspirin if fails to meet checklist (No to Any Inclusion Criteria and YES to Any Exclusion Criteria) and/or 24hours after tPA
    a. reduces mortality, morbidity, and recurrence
    b. continue forever
    c. Prevention Regimen for Effectively Avoiding Second Strokes (PRoFESS) showed no difference b/t Aggrenox and Plavix
3. Anti-Coagulants
    a. recent studies indicate little or no benefit from full anticoagulation in progressive ischemic stroke EXCEPT IN POSTERIOR CIRCULATION CVAs
4. Penumbra (clot sucking device) vs Merci (coil retrieval device)
5. SAMMPRIS Trial is looking at stenting

ii. ABCs
1. Oxygen Supplementation or Intubation if decreasing mental status so as to avoid hypoxia
2. Neuro Checks Q15min x2hrs, Q30min x6hrs, Qhr
3. HOB

iii. Appropriate Blood Pressure
1. If thrombolytics used: SBP<185 and DBP<100 but keep MAP>110
2. If thrombolytics NOT used: SBP<220 and DBP<120 but keep MAP>110
3. Labetalol (Trandate) 10mg IVP prn above parameters Qhr up to three doses holding if HR<55 and if you are finding the pt needs a lot then do labetalol (Trandate) 2-8mg/min IV or nicardipine (Cardene) 5-15mg/hr IV to reach above parameters
    a. difficult to manage b/c appropriate blood pressure is a delicate balance between hypotensive ischemia and hypertensive hemorrhage
    b. 5% of ischemic strokes will spontaneously develop symptomatic hemorrhagic transformation
    c. CPP (cerebral perfusion pressure, nl >60) = MAP (mean arterial pressure, nl >80) – ICP (intra cranial pressure, nl >20)
        i. ischemic tissue has impaired autoregulation such that CPP is a fxn of only MAP

iv. Fever
1. Aceteminophen (Goal: afebrile)
    a. seen in 50% of strokes >1d after onset of Sx
    b. degree of fever is proportional to degree of morbidity and mortality
    c. search for infection
    d. Mechanism: lower temp → lower metabolic activity → less oxygen/glucose demand

v. Tight Glucose Control
1. Insulin/Glucose (Goal: Glu 100-300)
    a. hyperglycemia is common after a CVA and is associated with a poor outcome, however, the causal relationship is unproven
    b. associated w/ increased r/o hemorrhage
    c. hypoglycemia is obviously a bad thing b/c the CNS solely relies on glucose for energy

vi. Cytotoxic Edema → Increased ICP

1. Refer to CNS trauma section
2. seen in 15% of pts, occurs >3d after stroke

vii. Seizures
1. either they occur during the first day or several years after stroke
2. 2/2 irritating effect of blood on cortex
3. intermittent seizures do not alter overall prognosis but status epilepticus can
4. little data AED prophylaxis until then if pt is seizure free then no AED but once pt has one seizure then give phenytoin for future prevention

viii. Immobility → DVT, Pneumonia, Contractures, Ulcers
1. Anticoagulation, Passive FROM Exercises, Frequent Bed Turning

ix. Nutrition
1. Ensure good swallowing function

x. Control RFs
1. Highest risk for 2nd stroke is w/in 1st month esp 1st few days
2. Check FLP and start statin
3. Smoking cessation
4. etc

(2) Embolic Infarct

a. Etiology
i. Heart: Afib (atrial thrombus), MI (ventricular thrombus), Endocarditis (valve vegetation)
ii. Aorta/Carotid: mobile thrombus
iii. Paradoxic: DVT thru septal defect
iv. Cryptogenic

b. Location
i. MCA (b/c direct continuation of carotid) > PCA > ACA

c. Symptoms
i. Sudden symptoms
ii. Pt usually is exercising when deficits develop
iii. Pt usually had different deficits in past 2/2 TIA b/c emboli lodge at different spots

d. Diagnosis
i. Cardiac Exam
ii. CT
iii. ECG
iv. TEE
v. Carotid US

e. Prognosis

f. Treatment
i. Primary
1. 1º Anticoagulation
2. 2º Antiplatelet
ii. Similar Complications to Thrombotic Ischemic Strokes

(3) Lacunar Infarct aka "lacunes"

a. Etiology
i. Chronic HTN resulting in progressive narrowing of vessel

b. Location
i. Small Cranial Vessels
1. MCA branches (lenticulostriates) lacunar infarcts of subcortex
2. SCA branches (penetrating branches) lacunar infarcts of cerebellum
3. Basilar branches (paramedian) lacunar infarcts of brainstem

c. Symptoms (pure motor, pure sensory, ataxia or clumsy hand-dysarthria syndrome, INO)

d. Diagnosis

e. Prognosis

f. Treatment
i. Primary
ii. Similar Complications to Thrombotic Ischemic Strokes

(4) Global Ischemia (occurs at watershed regions (b/t ACA and MCA))

(5) Carotid Thrombosis

a. NB much more likely to have an MI than a CVA!!!

b. Etiology
i. Emboli (more common but less severe – refer above)
ii. Stenosis (less common but more severe)
iii. Spontaneous Dissection

c. Location
    i. @ Carotid Sinus where the Carotid Bifurcates into External and Internal specifically at postero-lateral side
d. S/S
    i. Typical CVA/TIA Motor and Sensory Deficits
    ii. Amaurosis Fugax (monocular hemianopsia 2/2 embolus to retinal artery, "curtain coming down" lasting 15min-1hr)
    iii. Hollenhorst (plaque is a shiny, golden, refractile object lodged at retinal artery bifurcations)
    iv. Horner's Syndrome (suspect carotid dissection)
    v. Examine Bifurcation = Angle of Mandible
        1. Asymmetric Pulses
        2. Bruits
            a. differentiate bruits transmitted from coronary vascular disease, valve dz, brachiocephalic occlusive dz, thyrotoxicosis, etc. from true bruits by moving stethoscope downward, if it gets louder then likely 2/2 other problem and not carotid stenosis, also if bilateral then likely not carotid disease
            b. poor correlation b/t intensity of bruit and degree of stenosis
            c. very advanced stenosis (>80%) DOESN'T produce a bruit
            d. important to note that the presence of bruits doubles the stroke risk in asymptomatic pts
e. Diagnosis
    i. Carotid Duplex Ultrasonography (diagnostic test for screening though not accurate if <50% stenosis)
    ii. CTA/MRA of extra/intracranial vessels
    iii. Cerebral Angiography (gold standard but 4% r/o neurologic complications and 1% r/o death)
f. Treatment
    i. Spontaneous Dissection
    ii. Embolization (refer above)
    iii. Thrombosis
        1. Medical Therapy w/ Aggrenox and Plavix
            a. "Clopidogrel and Aspirin for Reduction of Emboli in Symptomatic Carotid Stenosis" (CARESS) showed that in symptomatic pts the use of combo ASA+Clopidogrel reduces asymptomatic embolization more when compared to ASA alone
            b. "European Stroke Prevention Study" (ESPS) showed that in symptomatic pts the use of combo ASA+Dipyrimadole reduces stroke and/or death more when compared to ASA alone
        2. Carotid EndArterectomy (CEA)
            a. First performed by in 1954 by Eastcott, Pickering, and Rob
            b. The first studies included the CASANOVA and MACE trials but they were considered suboptimal due to poor study design
            c. Common and External Carotids are clamped and a shunt is used for cerebral protection if back pressure from internal carotid is <50mm Hg, EEG shows changes after clamping, or there is a change in neural status (procedure is done under regional cervical block in order to assess status during procedure by talking to pt)
            d. Contraindications: ***totally occluded carotid***, disabling stroke, severe medical illness
            e. Complications: MI, Stroke, Death, CN10/12 injury, hematomas, hypoTN, hyperperfusion syndrome (impaired autoregulation after restoration of blood flow presenting as motor seizures, TIAs, and ICH)
        3. Carotid Artery Stenting (CAS)
            a. Angioplasty w/o stenting has poor results and multiple complications
            b. Initial trials comparing CEA and CAS where equivocal but two new studies are confirming CAS efficacy nevertheless only pts in studies can get this procedure done
                i. "Stent Supported Percutenous Angioplasty of the Carotid Artery versus Endarterectomy" (SPACE) showed that in symptomatic pts the use of CSA and CEA where comparable in their endpoints of stroke and/or death
                ii. "Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy" (SAPPHIRE) showed that in asymptomatic pts w/ >80% stenosis and symptomatic pts w/ >50% stenosis the use of CSA and CEA where comparable in their endpoints of stroke, MI and/or death
                iii. "Carotid Revascularization Endarterectomy versus Stent Trial" (CREST)
                iv. "Carotid Revascularization Using Endarterectomy or Stenting Systems (CaRESS)
            c. Consider in pts w/ difficult surgical anatomy, severe cardiopulmonary disease, recurrent stenosis after CEA, and radiation induced stenosis
            d. Complications: increased r/o distal embolization, in-situ thrombosis, dissection
        4. Asymptomatic
            a. <60, 99-100% occlusion = Medical Treatment

b. 60-99% occlusion = CEA as long as risk of surgical mortality or stroke is <3%

5. Symptomatic

a. <50, 99-100% occlusion = Medical Treatment

b. 50-99% occlusion = CEA as long as risk of surgical mortality or stroke is <6%

Hemorrhagic

(1) IntraCerebral Hemorrhage (ICH)

a. Epidemiology: 2nd most common stroke but most deadly
b. NB consider hemorrhagic conversion of ischemic stroke
c. Etiology
    i. Old
        1. HTN (most common)
        2. Amyloidosis (second most common, not related to systemic amyloidosis)
        3. Bleeding Diathesis, AC, etc
        4. AVM/Aneurysm
        5. Tumors
    ii. Young
        1. Cocaine
        2. Charcot Bouchard Aneurysm
d. Location
    i. Small Sized Cranial Vessels (Basal Ganglia, Thalamus, Cerebellum, Brainstem and can spread into ventricles)
e. Symptoms
    i. Abrupt onset of neurologic deficits which worsens over 30min-3hrs
    ii. S/S of increased ICP
f. Diagnosis (consider CTA if you think aneurysm)
    i. CT
        1. 100% sensitive at <24hrs from onset of Sx
g. Prognosis
    i. very high mortality (50% die w/in 30d) and morbidity esp if greater than golf ball size
    ii. 2/3 develop into SAH
    iii. Prognostic factors include volume of hemorrhage and level of consciousness
h. Treatment/Complications
    i. Primary
        1. Surgical Evacuation if cerebellar hemorrhage or cerebral hemorrhage w/ mass effect otherwise no other surgical intervention for hemorrhages in other locations
        2. Tx of HTN like ischemic strokes
    ii. Similar Complications to Thrombotic Ischemic Strokes and SAH Hemorrhagic Strokes
    iii. There are some studies looking at PROcoagulants specifically factor VIIa

(2) SubArachnoid Hemorrhage (SAH)

a. Etiology
    i. Sporadic
    ii. Acquired
        1. Trauma
        2. Hypertension
    iii. Congenital Aneurysm
        1. Saccular Berry
        2. FMD
        3. AVM
        4. APCKD
        5. Marfans/Ehlers-Danlos
    iv. Mycotic Aneurysm
        1. NB the term “mycotic” was used by William Osler b/c the aneurysm looked like a mushroom on gross anatomy not because it was a fungal infection emphasizing that the dx of mycotic aneurysm is made by imaging
        2. Can be an aneurysm that becomes secondarily infected or infection that then causes an aneurysm
            a. IE w/ septic embolic to vaso vasorum
            b. Trauma
            c. Local infection
        3. NB cannot only cause CNS aneurysms but also systemic aneurysms in fact… 1° femoral/ab aorta 2° MCA
        4. Primary complication is bleeding
        5. Usually a bacterial infection despite being called “mycotic”
            a. 1° Staph
            b. 2° Salmonella (especially in old pts)

c. 3° TB
d. 4° Syphilis
e. NB ¼ of time the pathogen is NOT found

6. S/S: depend on location (cerebral = CVA, systemic = painful, pulsatile, enlarging mass w/ constitutional Sx)
7. Dx: classic type and location of aneurysm seen on imaging specifically mushroom shaped and located at distal branch points but also highly suggestive if a aneurysm + blood Cx, leukocytosis, fever, etc
8. Tx: 4-6wks of abx (follow APRs to determine exact duration) ± surgery esp if rupture occurs

b. Location
   i. Big Cranial Vessels (Cerebrum/Cerebellum) esp at Bifurcations of Circle of Willis w/ 85% in anterior circulation
c. Symptoms
   i. Severe Sentinel Headache (2/2 aneurysm dilation but only seen in 1/2 of pts) → Instantaneous Excruciating Classic Headache aka "thunderclap" (2/2 aneurysm rupture) →
   ii. LOC (2/2 blood around entire brain)
   iii. Mengismus
   iv. Focal Neurologic Deficits esp CN 4,6 but also sensory/motor deficits
   v. Retinal Hemorrhage
   vi. N/V
   vii. AMS
d. Diagnosis
   i. CT/MRI
      1. 93% sensitive at <24hrs from onset of Sx w/ 7% due to (1) early <1d (earlier with MRI), (2) small <0.5cm, (3) posterior fossa, (4) not actively bleeding during scan
      2. finger like hemorrhage as it extends between gyri, no hemorrhage IN the parenchyma
      3. if neuroimaging equivocal then do an angiogram if high suspicion or LP if low suspicion (don't do LP if high suspicion b/c the high CSF pressure acts as to tamponade and thus removal of CSF could decrease pressure precipitating rebleeed)
   ii. Angiogram
   iii. LP
      1. looking for xanthochromia = represents not RBC but RBC lysis

Day	% +CT	% +Xanthochromia
0	93	100
7	50	100
14	30	70
21	0	70

e. Prognosis
   i. very high mortality (50% die w/in 30d) and morbidity
f. Treatment/Complications
   i. Primary (need angiography to determine location, if no bleed but incident aneurysm then consider Tx the larger and more posterior the aneurysm otherwise just watch)
      1. Open Surgical Clipping
      2. Endovascular Coiling
   ii. Cerebral Vasospasm (50%)
      1. spasming 2/2 surrounding blood usually occurring 4-12d following SAH
      2. new ischemic CVA Sx
      3. nimodipine
      4. hypervolemia with colloid infusion and hypertension with phenylephrine so that MAP overcomes the vasospasm
   iii. Recurrent Hemorrhage (30%)
      1. most SAH stop bleeding when dx nevertheless rebleeding can occur w/in the first few days and is associated with 50% mortality, this can be obviated via clipping/coiling
   iv. DVT
      1. anticoagulation
   v. Myocardial Ischemia/Infarction 2/2 hypovolemia
      1. monitor with ECG and cardiac markers, usually transient requiring no intervention if no prior CAD
   vi. Hydrocephalus → Increased ICP
      1. 2/2 blood clotting in the subarachnoid space
      2. refer to CNS trauma section
   vii. Seizures
      1. either they occur during the first day or >2wks after onset of Sx
      2. 2/2 irritating effect of blood on cortex
      3. Phenytoin for prophylaxis

viii. Pulmonary Edema
ix. HypoNa 2/2 SIADH
x. DI
xi. Stool Softeners

(3) Epidural Hematoma

a. Etiology
   i. trauma (eg. hit with a hammer) to temporal bone resulting in laceration of middle meningeal artery (fast hemorrhage)
   ii. associated coup contusion
b. Location
   i. White (blood) with convex/convex ("lenticular") hematoma that does NOT cross suture lines
c. Symptoms
   i. Brief LOC → Lucid Interval → Coma w/ Herniation (refer) (this classic pattern only seen in 20% of pts)
d. Diagnosis
   i. CT (refer above)
e. Prognosis
f. Treatment
   i. Rapid Surgical Decompression

(4) Subdural Hematoma

a. Etiology
   i. trauma (eg. deceleration MVA or trip and fall) resulting in traction tearing of bridging veins (slow hemorrhage) esp in pts who have decreased brain mass (alcoholics and elderly)
   ii. associated contra-coup contusion
b. Location
   i. Grey (blood+CSF) with concave/convex hematoma that crosses suture lines
c. Symptoms
   i. Acute: LOC w/ in hours-days after a distinct injury
   ii. Chronic: AMS over several days after a subtle injury
d. Diagnosis
   i. CT (refer above)
e. Prognosis
f. Treatment
   i. Acute: surgery
   ii. Chronic: nothing depending on size