General Surgery Grand Rounds

General Management and Resuscitation in Acute Brain Injury

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Severe Brain Injury: Learning Objectives

- Epidemiology
- Classifications based on severity and type
- Clinical presentations
- General outcome predictors
- On Site Hospital TX
- General conclusions
- Future directions
- Questions
Traumatic Brain Injury
Definition & Epidemiology

- NIH NTCDB = GCS < 9 48 hrs post injury
- 500,000 cases of head injury per year
- 10% die before reaching the hospital
- Mortality = 17/100K pre hosp, 6/100K hosp
- 80% mild
- 10% moderate
- 10% severe
- 100,000 sig LT disability
TBI: Epidemiology (cont)

- Mechanism: MVA > Falls > Firearm > other assault
- High risk: young, male, low income, unmarried, ethnic minority, city dweller, sub abuse, prev TBI
- Male to female incidence ratio 2.8:2, mortality ratio 3.4:1
- Age: occurring most commonly age < 25 with a bi-modal distribution rising again at age > 65
- Cause of death in 45-50% of all trauma
- 40-80% association with sig systemic trauma (thoracoabdominal, ortho,)
- Alcohol: implicated in 50-70%
Classifying Brain Injury

- Based on GCS
- Mild: GCS 13-15
- Moderate: GCS 9-12
- Severe: GCS 8 or less
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<tr>
<th>Eye</th>
<th>Voice</th>
<th>Motor</th>
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<td>oriented</td>
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<td>4 spontaneous</td>
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TBI Exam Findings

- Depressed level of consciousness not clearly due to EtOH, drugs, metabolic, cardio-pulmonary abnormalities etc.
- Focal neurologic findings (pupillary changes, false localizing, not all have mass lesion e.g., DAI)
- Penetrating skull injury or depressed fracture
- Raccoon eyes, Battle’s sign, CSF rhinorrhea an/or otorrhea
TBI Presentation (con’t)

- SZ
- Decorticate, deceribrate posturing
  Cushing’s Triad:
    - Inc BP W/ widened PP, and proportionally greater inc in SBP over that of DBP
    - Bradycardia (SR)
    - Irregular Breathing
    - Usually heralds brain damage/herniation sec/2 inc ICP/poor perfusion.
TBI Outcome predictors

Choi et al, correlated:

- Fixed pupil
- Low GCS motor
- Age > 60

Strongly predictive of death or significant disability
Types of Traumatic Brain Injury

- Forces = compressive, tensile (stretch), shearing (slide)
- Skull fractures
- Concussion
- Diffuse axonal injury
- Contusion
- Subarachnoid hemorrhage (SAH)
- Subdural hematoma (SDH)
- Intraparenchymal hemorrhage (ICH)
- Epidural hematoma (EDH)
- Intraventricular hemorrhage (IVH)
Skull Fractures

- Linear
- Depressed
- Comminuted
- Compound
- Basilar

Significance: 10% of fractures have lesions requiring emergent surgical intervention
Linear Depressed Comminuted Skull Fx
Linear Depressed Comminuted Skull Fx
Concussion

- Reversible transitory neurologic deficit. Associated with rotational shear stress.
- Considered mild form of DAI
- Three grades; mild, moderate and severe, proportional to retrograde and anterograde amnesia
Diffuse Axonal Injury

- Most common traumatic brain injury
- Shearing of axons
- Temporary and permanent loss of cellular function
- Occurs as a clinical and radiographic spectrum, with 3 grades on CT
- Mortality 30-40%,
- Good outcomes 20-30%

Grade 1: Parasagittal WM
Grade 2: Grade 1 + CC (deeper)
Grade 3: Grade 2 + cerebral peduncle / brain stem / IVH
Diffuse Axonal Injury (con’t)

- Imaging:
  - CT: petechial hemorrhage (hyperdense) along gray white junctions, symmetric or asymmetric edema, intraventricular / cisternal hemorrhage
  
  MRI: hemorrhage as above, high signal on T2
Diffuse Axonal Injury (con’t)

- Overall Prognosis
  - Related to:
    - CT imaging
    - Control of intracranial pressure
    - Post resuscitation GCS
Diffuse Axonal Injury (con’t)

CT Brain Grade 2 DAI

T2 MRI Brain Grade 2 DAI
Cerebral Contusion

- Second most common brain injury
- Coup = small-moderate direct impact
- Countercoup = high energy with translational dissipation of energy
- Essentially a bruise on the brain with hemorrhage from torn pial vessels, evolution of localized edema
- Location: temporal > frontal > parasagittal, > occipital (convexities)
- 20% will have delayed hemorrhage
Cerebral Contusion (con’t)
Cerebral Contusion (con’t)
Subarachnoid Hemorrhage

- MC traumatic intracranial hemorrhage
- Associated with DAI when caused by trauma
- Can also result from bleeding saccular aneurysm and/or AVM, fusiform and mycotic aneurysms, fibromuscular dysplasia, coagulopathies, moyamoya disease, infection, neoplasia, psuedo aneurysm, substance abuse.
- Outcomes inversely proportional to clinical and radiographic grades
Subarachnoid Hemorrhage (con’t)

Hunt and Hess grading system (predicts clinical outcomes)

- Grade 1 - Asymptomatic or mild headache
- Grade 2 - Moderate-to-severe headache, nuchal rigidity, and no neurological deficit other than possible cranial nerve palsy
- Grade 3 - Mild alteration in mental status (confusion, lethargy), mild focal neurological deficit
- Grade 4 - Stupor and/or hemiparesis
- Grade 5 - Comatose and/or decerebrate rigidity
Subarachnoid Hemorrhage (con’t)

Fisher scale: Radiographic Grade (predicts degree of vasospasm)

- Grade 1 - No blood detected (LP + Xantho)
- Grade 2 - Diffuse deposition of subarachnoid blood, no clots, and no layers of blood greater than 1-3 mm
- Grade 3 - Localized clots and/or vertical layers of blood 3 mm or greater in thickness
- Grade 4 – Diffuse, or no subarachnoid blood, with intracerebral or intraventricular clots are present
Subarachnoid Hemorrhage
(con’t)
Subarachnoid Hemorrhage (con’t)

CT Showing Traumatic SAH

CT Showing Aneurysmal SAH
Subdural Hematoma

- Hemorrhage under the dura
- Caused by torn bridging veins and/or bleeding contusions (pial vessels)
- Occur in 10-35% of severe head injuries
- 10-50% are associated with skull fractures
- Cortical atrophy is RF for occurrence, but Pt’s with normal brain vol at higher risk for LT disability
- Also associated with coagulopathies
- Often associated with other brain injuries
- Acute, sub acute and chronic (density on CT) depending on the age of SDH
Subdural Hematoma (con’t)

- Prognosis:
  - Mortality is 40-70%
  - Complete recovery in 8%
  - Severe disability in 74-84%

- Imaging:
  - CT scan: crescentic (concaved) shaped hematoma
  - Crosses sutures but not dural insertions, mass effect with shift, edema
Subdural Hematoma (con’t)

CT Showing Acute SHD

CT Showing Acute on Chronic SDH
Intraparenchymal Hemorrhage

- Hemorrhage in the substance of the brain also called intracerebral hemorrhage (ICH)
- Associated with HTN, anerysmal SAH, amyloid, cerebral contusions and DAI
- Occur most commonly in frontal and gray white junctions of brain when caused by trauma, vs vascular ICH’s due to HTN, which occur in distinct vascular distributions
- Commonly expand (re-bleed) within first 24 hours
- Often cause localized edema and elevated intracranial pressure
- Outcomes inversely proportional to size of ICH, and Pt age
Intracerebral Hemorrhage (con’t)

CT Brain Showing Traumatic ICH and Skull Fx
Intracerebral Hemorrhage (con’t)
Epidural Hematoma

- Hemorrhage between inner table of skull and dura
- Often caused by severed meningeal artery or torn large venous sinus
- Obeys suture lines (coronal, lambdoid)
- Occur in 3-5% of head injuries
- Peak incidence 10-30 years old
- Rare in those <2yrs or > 60yrs
- 85-90% are associated with skull fractures
Epidural Hematoma (con’t)

- Often present with lucid interval
- Commonly occur with other brain lesions
- Mortality and Morbidity 5% - 20%
- Higher rates are associated with the following:
  - Advanced age
  - Intradural lesions
  - Temporal location
  - Increased hematoma volume
  - Rapid clinical progression
  - Pupillary abnormalities
  - Increased intracranial pressure (ICP)
  - Lower Glasgow coma scale (GCS)

In the US: EDH occurs in 1-2% of all head trauma cases and in about 10% of patients who present with traumatic coma.

CT appearance: convex hyperdensity, swirl sign, obeying suture lines
Epidural Hematoma (con’t)
GSW & projectile TBI

- Missile vs nonmissle
- Energy dissipation = $\frac{1}{2}$ projectile mass \times velocity$^2$ (velocity and blast proximity)
- Associated with all forms of traumatic hemorrhage
- Outcomes related to velocity, location of entry and exit, post resuscitation GCS
GSW & projectile TBI (con’t)
“Type A” Neuro Pt = Over-Achiever
On Site Hospital TX/RX

- Airway and breathing assessment in the awake and directable neurological patient

GCS 10-15, maintains Sao2 96-100%, NOT rapidly deteriorating neurologically, non-agitated/combative, and cardio-pulmonary status is stable:

1) Nasal Cannula, Non Re-breather
2) Bag and Mask Valve, as a bridge in some cases
3) Serial Neuro exams and Chest auscultation
4) CXR, and serial ABG’s
5) “Big 3” BAT R/O
On Site Hospital TX/RX (con’t)

- Tracheal Intubation Criteria in the Neuro Pt:
  1) Depressed LOC GCS < 8-9
  2) Rapidly deteriorating neurologic exam (minutes-Hrs)
  3) Immediate need for HV w/ target PCO2 30-36
  4) Severe maxillofacial trauma or airway edema, w/ impending loss of airway protection &/or patency
  5) Need for pharmacological sedation, SZ control &/or paralysis
  6) Cardiovascular instability (MI, CHF, Sepsis, shock)
  7) Primary pulmonary instability (Edema, Asp, Apnea, Stridor)

a) In all cases, intubation should be based on emergent clinical criteria, rather than lab values and/or radiographic studies. When in doubt, elect to intubate the acute Neuro Pt., early and/or prior to any transport.
Difficult Airways: clinical presentations/syndromes in the Neuro Pt:

- Potential or proven cervical spine injury, also RA, Down’s
- Basilar skull Fx
- Maxillofacial trauma &/or burns
- Receding chin (micrognathia)
- Prominent incisors (buck teeth)
- Short plethoric &/or muscular neck
- Morbid obesity, acromegaly, scleroderma
- Prev Trac, head/neck surgx &/or radiation
- Pregnant, Illeus/SBO/full stomach
On Site Hospital TX/RX (con’t)

- Tx Hypertension in the Neuro Pt with SAH, ICH, EDH, Trauma, & some strokes, ie, Pt’s having intracranial mass effect/inc ICP:
  1) Control ICP
  2) Intubate/Sedate
  3) Control any SZ
  4) Nipride gtt: 1-10 mic/kg/min (SE’s: cardiopulm shunt, CN-thio tox ?? Inc ICP)
  5) Esmolol gtt: 25-200mcg/kg/min (SE’s: bradycardia, heart block, bronchospasm, unopposed alpha)
  6) Labatolol: 2.5-20 mg q 1 hr PRN (SE’s: bradycardia, heart block, unopposed alpha)
  7) Hydralazine: 2.5-20 mg IV q 1hr PRN (SE’s: reflex tachy, > AAA)
  8) Nicardapine : (SE’s: Inc Myocardial O2 demand)
On Site Hospital TX/RX (con’t)

- Hypotension in the Neuro Pt:
  - Causes: Cardiogenic = Hypovolemic > Acidosis > Neurogenic > Vasogenic (sepsis/anaphylaxis)
  - Dx:
    - IBP monitoring, 12 lead EKG, Trop-I, Blood gas, CXR, CVP or PAP monitoring, Blood Cultures, +/- BAT R/O
  - Tx:
    1) Fluid Challenge in face of inc ICP W/ 500 cc 5% Albumen, or blood (repleats intravase vol and inc oncotic pressure)
    2) Dopamine gtt: 5-20mic/kg min titrate SIBP 100-120
    3) Neosyephrine gtt: 0.5-5mic/kg/min
    4) Vasopressin gtt: 0.01-0.1 u/min
On Site Hospital TX/RX (con’t)

- Control of Presumed Elevated ICP:
  - (Trauma, SAH, ICH, SDH, EDH, Large stroke, Tumor)
    1) Head of bed ~ 30 deg (inc JV outflow)
    2) Intubate (PCO2 30-36, PEEP ~ 5), ET fastened w/o IJ occlusion, keep head forward & neck straight after intubation
    3) Light- heavy sedation inversely proportional to GCS
    4) Avoid IJ Location for central venous catheter (Subclavian)
    5) Mannitol 1.5 G/kg IV, check Na+/serum osmo afterwards if time permits, (+/-) Hypertonic saline
    6) Lasix 10-20 mg IV ½ hr after Mannitol, check K+
    7) Minimal- Euvolemic total fluids (MIVF + Rx + TF) ~ 40-100cc/hr
    8) Ventricular drainage &/or ICP monitor
    9) Early Crainectomy
On Site Hospital TX/RX (con’t)

Control of Seizure

1) A-B-C’s
2) Ativan 2-10 mg IV push
3) Phosphenytoin 18-20mg/kg IV load < 50 mg/min (SE’s bradycardia and hypotension)
4) Depakon 1000-1500 mg IV load < 20 mg/min
5) Phenobarbital 200-500mg IV load < 60 mg/min, spike Dopamine &/or Neo gtt (SE’s cardiopulm, immune syst suppression) Deploy PA cath, titrate to burst – suppression on EEG
6) Propofol gtt, titrate to burst – suppression on EEG
7) Lamictal 1400 mg IV load
On Site Hospital TX/RX Overview

- Kept position head up > 30 degrees (max V outflow, min A hydrostatic head) at all times
- CVP/ IBP / PA cath monitoring, (no IJ lines)
- Allowed Low normothermia – core T 35-36.5 or mild hypothermia
- Loaded Dilantin 18-20mg/kg slowly (< 50mg/min)
- Normalized coags (Novo-7, FFP, Cryo-PTT, Vit K)
- Normalized Platelet count/function (# > 100K, DDAVP)
- (+/-) Steroids
- (+/-) Nimodapine in extensive and/or traumatic SAH
- Instituted Spinal Precautions, Spinal radiographs
- Cerebral vascular studies ( angio, CTA, CTV, MRA, MRV)
- Serial CT, (+/-) MRI scans
On Site Hospital TX/RX Overview

- Intubated, sedated, (Narcs, Benzos, Propofol, Presidex) +/- paralytic gtt (1-0/4 train of 4) or Barbs /24 hr EEG
- Maintained MAP 80 -100, Pressors (dopa, neo, vaso) or Anti-HTN Rx, with CVP or PA catheter monitoring in many Pt’s
- Given Mannitol 1.5g/kg load, can be followed by Lasix (synergy), albumen & blood, HCT 30-33, Euvolemia-Hypovolemia in acute phase, if tolerated
- Targeted PcO2 30-36, minimum PEEP / PS if possible, PaO2> 80-90
- Placed ICP monitor, goal ICP < 20, with CSF drainage via EVD if necessary and CPP > 60-70 in pt’s with ICP sustained > 20
- PbTO2, and Brain Temp monitoring (Licox) target PbTO2 > 20
- (+/-) Lumbar drainage, (+/-) ENT consult for some CSF leaks
- Started Abx prophylaxis esp: pneumocephaly, CSF leaks, EVD
- (+/-) Emergent surgical evacuation and possible crainectomy
Timing of Craniotomy

- “Four hour rule” Seelig et al. 1981 N Engl J Med: 82 patients with acute subdural, operation in < 4 hours = 30% mortality, if > 4 hours 90% mortality

- “Six hour rule” Citow 2001
  Operation in < 6 hours = 30% mortality, if > 6 hours, 95% mortality
Timing of Craniotomy (con’t)

- Wilberger et al 1990 & 1991, found no statistical significance to earlier evacuation, rather statistical outcome variables were: presenting neuro exam, and post op ICP
- Hatashita et al 1993 found no statistical advantage in decompressing before ten hours post injury
Conclusion

- Severe brain injuries are associated with high mortality and morbidity
- GCS, Hunt & Hess, Fisher Grades, and age are strong predictors of outcome
- Timing of surgical repair and repair likely does have prognostic significance
- Periop care of the TBI Pt impacts outcome via: systemic control of airway, ICP, MAP, Volume status, coagulopathy, and possibly CBF
Future Directions

- Invasive brain tissue oxygenation monitoring
- Invasive CBF monitoring
- Invasive brain T monitoring
- Invasive brain Dialysis Catheter monitoring
- Minimization of early apoptosis via control of excitatory AA (glutamate, aspartate), control of NMDA receptor agonists, ....? control of presynaptic endogenous opioid peptides
Questions
Severe Spinal Injury
Epidemiology
Severe Spinal Injury
Epidemiology

- Cervical
- Thoracic
- Lumbar
- Sacral
SCI R/O

- All victims with significant mechanism of trauma
- Trauma Pt’s with LOC
- Minor trauma Pt’s with neck or back pain, or sensory motor, or vasomotor findings on PE
- SCI may mask other injuries
Instability

Segmental instability is a loss of spinal motion segment stiffness, such that force application to that motion segment produces greater displacement(s) than would be seen in the normal structure, resulting in a painful condition, the potential for progressive deformity and neurologic structures at risk.

John W. Frymoyer
Instability and Treatment

- Acute SCI with potential for healing to stability
- Acute SCI with low potential for healing to stability
- Chronic (glacial)
- Etiology
  - Osseous
  - Ligamentous
- Risk/benefit
Spinal Biomechanics

- Cartesian System
- Two motion types
  - Translations
  - Rotations
- Coupling
- Kinematics vs biomechanics
Upper Cervical Instability

- Visible occipital condyles
- Widened C1 vs C2 on AP > 7mm
- **Rupture of transverse ligament**
- Type II odontoid fracture w/ > 6mm displacement
- Flex/ext range > 11 degrees (C1-3)
- > 50% loss of facet contact
- Interspinous widening
- C2-C3 z axis translation > 3mm
Upper Cervical Instability (con’t)

- Occipito-atlantal dislocation
- C1 injuries
- C1-C2 dislocations
- C2 fractures
- C1-C2 combination injuries
- Odontoidectomy
Occipito-atlantal Dislocation

- High mortality at scene
- Very unstable (immediate halo fixation)
- Floating condyles
- Power’s ratio
- CT vs MRI
Occipito-atlantal Dislocation Treatment

- NO TRACTION!
- Backboard immobilization
- Immediate halo
- Move pt to OR with halo
- Urgent internal fixation
Occipito-atlantal Dislocation
C1 (atlas) Injuries

- **Osseous**
  - Ring fractures (Jefferson)
  - Lateral mass fractures

- **Ligamentous**
  - Transverse ligament

- 45% will have C2 injury
Atlas Injury
Atlas Injury
Diagnosis

- C spine plain film with odontoid view, showing overlap of C1 on C2 on AP > 7mm
- CT with 1 mm resolution and 3d reconstruction, showing predental space > 3mm
- MRI for transverse ligament
Atlas Injury Treatment

- Dependent on transverse ligament and potential for healing to stability
- Ring fractures - external immobilization
- Transverse ligament incompetence
  - Osseous basis - possible nonsurgical management
  - Pure ligamentous injury - surgery
C1-C2 Dislocations

- Transverse ligament injury
- Rotatory subluxation
Transverse Ligament Disruption
C1/2 Fusions
C1/2 Transarticular Fixation
C1/2 Rotatory Subluxation
C1/2 Rotatory Subluxation

- Normal transverse ligament
  - External reduction
    - Reducible
    - External immobilization
  - Irreducible
- Transverse ligament disrupted
  - ORIF
C2 Fractures

Odontoid Type I

Odontoid Type II

Odontoid Type III

Hangman's Fracture

Miscellaneous C2 Fracture
Odontoid Fractures

Type I
- <6mm displacement
  - Halo

Type II
- >6mm displacement or comminuted
  - ORIF

Type III
- Halo
Odontoid Fracture by Displacement

- Overall nonunion rate = 28%
- Nonunion below 6.0 mm, 5/50 = 10%
- Nonunion 6.0 mm/above, 14/18 = 78%

**KEY:**
- Anterior dislocation: X
- Posterior dislocation: O
- Lateral dislocation: □
- Nondisplaced: △
Odontoid Fracture by Age

- Overall nonunion rate = 28%
- Nonunion below 6.0 mm, 5/50 = 10%
- Nonunion 6.0 mm/above, 14/18 = 78%

Key:
- Anterior dislocation: ⌂
- Posterior dislocation: ●
- Lateral dislocation: □
- Nondisplaced: △
- Nonunion denoted by circle ○
Odontoid Fracture
Hangman’s Fracture

Type I
• Non-displaced
• Minimally displaced

Type II
• Angulated > 11 deg
• Sublux > 4mm

Type III
• Disrupted C2/3 facets

Reducible
Rigid brace

Irreducible or recurrent subluxation
Halo

ORIF
Complex Upper Cervical Fractures

C1 - Odontoid Type II
C1 - Odontoid Type III
C1 - Miscellaneous Fractures
C1 - Hangman's Fracture
Operative Intervention

- Poor immobilization or recurrent deformity/malalignment
- Nonunion after nonsurgical treatment
- Ligamentous injury
- Above criteria for odontoid and Hangman’s fractures
Transoral Odontoidectomy
Transoral Odontoidectomy
Stability

![Graph showing translation in X, Y, and Z axes for normal and post-operative conditions.]

- X Axis
- Y Axis
- Z Axis

Translation (mm)

- Normal
- Post-operative
Three Column Model of Thoracolumbar Spine

- Anterior: anterior vertebral body + disc + anterior longitudinal ligament
- Middle: posterior vertebral body + disc + and posterior longitudinal ligament
- Posterior: facet joints/capsules + supraspinous/intraspinous ligaments + ligamentum flavum
Posterior Column

Middle Column

Anterior Column
Hospital Care of Acute Non Traumatic Ischemic Stroke
Definitions

- **Stroke**: Any vascular injury to the brain
- *Ischemic stroke* is a persistent clinical deficit at 24 hours.
- A TIA lasts less than 24 hours *and clears completely*.
- This distinction is a continuum with damage proportional to the severity and duration of ischemia.
Definitions (con’t)

- 80% are ischemic
- 20% are hemorrhagic (SAH, IPH, IVH)
Stroke in the United States

- 750,000 new strokes a year
- 4 million stroke survivors
- #1 cause of major neurologic disability
- #3 cause of death
Stroke risk factors

- HTN 6X
- Diabetes 3X
- Asymptomatic bruit 3X
- Rheumatic Atrial fib 17X
- Paroxysmal Atrial fib 6X
- Lipids 2X

- Smoking 2X
- Prior CVA/TIA 10X
- Obesity 1.5X
- Age increases 10X/20 years of age
Ischemic stroke S/Sx

Unilateral paralysis—Weakness, clumsiness, or heaviness, usually involving 1 side of the body

Unilateral numbness—Sensory loss, tingling, or abnormal sensation, usually involving 1 side of the body

Language disturbance—Trouble understanding or speaking (aphasia) or slurred speech (dysarthria)

Monocular blindness—Painless visual loss in one eye, often described as a curtain dropping

Vertigo—Sense of spinning or whirling that persists at rest. Isolated vertigo is also a common symptom of many nonvascular diseases; therefore, at least one other symptom of TIA or stroke should also be present.

Ataxia—Poor balance, stumbling gait, staggering, incoordination of one side of the body
Stroke Mimics

- Systemic infection
- Brain tumor
- Toxic-metabolic
- Positional vertigo
- Syncope/ MI
- Trauma (post stroke)
- Seizure
- Dehydration / hyperosmolality
Brief exam for stroke

- Grimace (CN 7)
- Repeat a sentence (aphasia)
- Hold arms up with eyes closed (pronator drift)
ED / in Hospital evaluation

- Blood work
  - Complete blood count (CBC)
  - Serum electrolytes
  - PT (INR) / PTT
  - Blood glucose
  - Cultures

- Electrocardiogram / R/O MI

- Pulse oximetry / blood gas

- Chest x-ray

- Stat CT of head (non-contrast)
### Emergency treatment of ischemic stroke

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<tr>
<th>Step</th>
<th>Treatment and Notes</th>
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<tr>
<td>1.</td>
<td>Intravenous fluids</td>
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<td>2.</td>
<td>Blood sugar</td>
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<td>3.</td>
<td>Thiamine</td>
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<td>4.</td>
<td>Oxygen</td>
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<td>5.</td>
<td>Acetaminophen</td>
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<td>6.</td>
<td>NPO</td>
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<td>7.</td>
<td>Cardiac monitor</td>
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D5W indicates 5% dextrose in water; So2, oxygen saturation.
Blood pressure and acute ischemic stroke

- Transient and volatile elevation in blood pressure is common
- Usually lasts several days after the stroke
- Patients' blood pressure may be very sensitive to medications
Blood pressure and acute ischemic stroke (con’t)

- No benefit to aggressively reducing blood pressure in acute ischemic stroke, in other words, allow the pt to be HTN
- AHA: treat only if MAP > 130 or SBP > 220, DBP > 140
Algorithm for Emergency Antihypertensive Therapy for Acute Stroke

1. If Diastolic BP is >140 mm Hg on two readings 5 minutes apart, start infusion of sodium nitroprusside (0.5-10 mg/km/min).

2. If systolic BP is >220 mm HG and/or diastolic BP is 121-140 mm Hg on two readings 20 minutes apart, give 20 mg labetalol IV for 1-2 minutes. The labetalol dose may be repeated or doubled every 10-20 minutes until a satisfactory BP reduction is achieved or until a cumulative dose of 300 mg has been administered. (Labetalol is avoided for patients with asthma, cardiac failure, or severe cardiac conduction abnormalities.)
Medications used to treat stroke

- Heparin
- Warfarin
- TPA
- Aspirin
  - Ticlopidine (Ticlid)
- Clopidogrel (Plavix)
- Dipyridamole (Persantine/Aggrenox)
Acute use of heparin and Stroke

- Given IV without bolus
- Controversial
- Little evidence for benefit in most patients with completed stroke outside of pt’s with known A-fib/transmural thrombus/ sig carodit and post circ stenosis
- Not recommended in new AHA guidelines
Warfarin therapy and stroke

- Valvular disease and valve replacement
- Atrial fibrillation / ventricular thrombus / high grade carotid and post circ stenosis
- Unclear benefit vs aspirin in other settings (WARSS trial favors aspirin)
- Risk, cost, complications
Systemic thrombolytic treatment

- Intravenous tissue plasminogen activator (TPA)
  - 3 hour window to treat from onset of symptoms
  - Many contraindications
  - Risk of hemorrhage
TPA contraindications (ACLS)

Evidence of intracranial hemorrhage on pretreatment evaluation
Suspicion of subarachnoid hemorrhage on pretreatment evaluation
Recent (within 3 months) intracranial or intraspinal surgery, serious head trauma, or previous stroke
History of intracranial hemorrhage
Uncontrolled hypertension at time of treatment (see “Management of High Blood Pressure”)
Seizure at stroke onset
Active internal bleeding
Intracranial neoplasm, arteriovenous malformation, or aneurysm
Known bleeding diathesis, including but not limited to
  —Current use of oral anticoagulants (eg, warfarin sodium), an international normalized ratio $>1.7$, or a prothrombin time $>15$ seconds
  —Administration of heparin within 48 hours preceding the onset of stroke and an elevated activated partial thromboplastin time at presentation
  —Platelet count $<100,000/mm^3$

tPA indicates tissue plasminogen activator.
TPA complications

- No difference in mortality at 3 months between the r-TPA and placebo
  - 17% TPA
  - 21% placebo
- Higher incidence of symptomatic hemorrhage in the r-TPA group
  - 10X hemorrhage rate (6% with TPA)
  - 3% of TPA patients died from hemorrhage
TPA Results

- 12% absolute increase in patients with good outcome at three months with r-TPA
- No difference for age, race, sex, stroke location, or stroke mechanism
Intraarterial thrombolytics

- Dx benefit of a diagnostic angiography
- Mechanical disruption of the clot + locally directed therapy
- Limited evidence
- Limited availability in some institutions
- Results at UCHSC excellent on NSS
Aspirin therapy and stroke

- Effective in secondary prevention of stroke and TIA
- Heart and peripheral vascular disease benefits
- Well understood (cyclo-oxegonase) / known safety profile
- Cheap
- Side effects directly related to dose
- No effective measure of aspirin effect – yet
- 325 mg/day American Heart Assoc.
Clopidogrel & Ticlopidine

- Inhibit the platelet ADP pathway
- Clopidogrel better tolerated than ticlopidine
- Marginally more effective than aspirin
- $
Common complications of stroke

- Seizures
- Aspiration
- DVT/ pulmonary embolism
- Appendage dislocation
Summary

- New approach to stroke
- Time is critical
- Simple interventions – glucose control, moderate IVF, temperature, blood pressure
- R/O stroke mimics prior to use of thrombolytics