Neuro Critical Care

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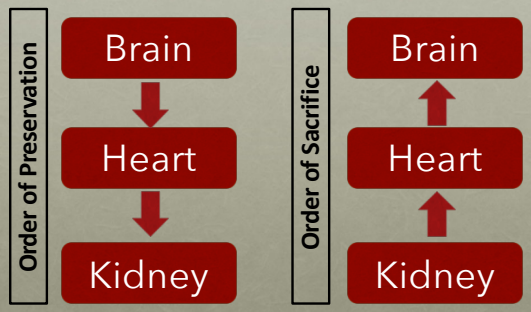
Hierarchy of the body



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Definition of Neurocritical Care

- Intracerebral and intraventricular hemorrhage
- Subdural and epidural hematoma

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Definition of Neurocritical Care

- Subarachnoid hemorrhage
- Cerebral aneurysms
- Cerebral and spinal vascular malformations
- Brain tumors

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Definition of Neurocritical Care

- Status epilepticus
- Meningitis and encephalitis
- Neuromuscular disorders in crisis (myasthenia gravis, Guillain-Barre syndrome) and acute myelopathies

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Goal of neurocritical care is Neuroprotection

- Assessment of end organ perfusion
  - No direct laboratory measurements
  - Physical exam
  - MRI/CT imaging
- Interval to end-organ failure under adverse conditions can be rapid.
- Further injury to even small regions of brain can have devastating consequences.

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Important Physiology

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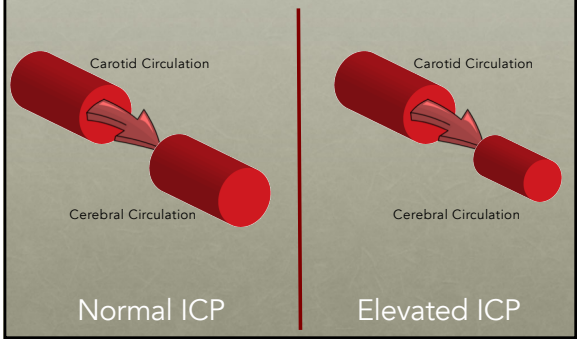
Cerebral Perfusion Pressure



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Cerebral Perfusion Pressure

- MAP = 1/3(systolic - diastolic) + diastolic
- ICP 0 - 10 torr (20 upper limit)
- CPP 80 - 100 torr
- CPP drops below 60 torr = ischemia
- CPP <30 torr - incompatible with life

CPP = MAP - ICP

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Cerebral Perfusion Pressure

- CPP correlated with CBF
- Decrease in MAP
- Increase in ICP
- Hypotension defined as systolic BP <90 mmHg
- Hypoxia
  - apnea
  - cyanosis
  - SpO2 <90% = PaCo2 of 60 torr

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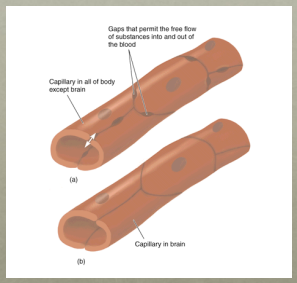
Physiology

- CSF - 25 ml/hour
- Blood brain barrier
  - tight capillaries
  - limit movements of solutes and water
  - glucose, oxygen, carbon dioxide, and lipid soluble substances (nicotine, caffeine, narcotics)



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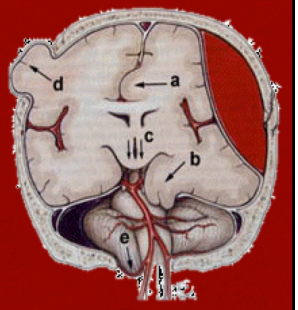
Monro-Kelli Doctrine

The volume in the rigid skull is equal to the sum of the brain (90%) + CSF + blood

A change in one compartment must be balanced by a change in another



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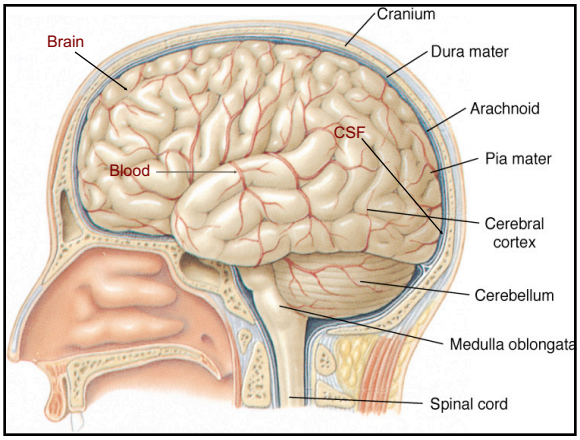

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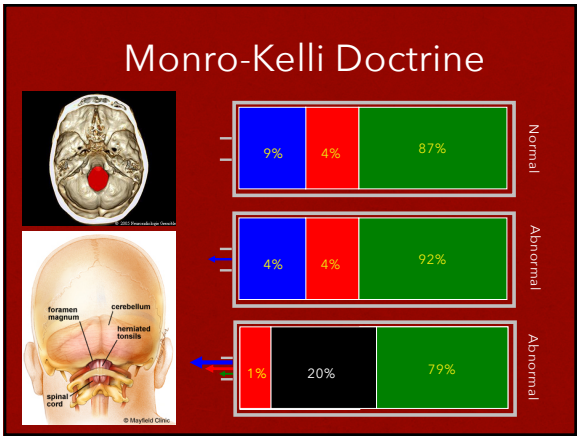

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Monro-Kelli Doctrine

- Reduce brain size – dehydrate ECF
  - Mannitol
  - Hypertonic saline
- Reduce CSF
- Reduce cerebral blood volume
- Reduce mass (tumor, hematoma)

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PATHOPHYSIOLOGY

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Cerebral Infarction

- General Considerations
  - Thrombotic or embolic occlusion of major vessel results in cerebral infarction
    - Thrombotic believed to be most common
  - Resulting deficit dependent upon which vessel and extent of collateral circulation

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Clinical Findings

- Onset usually abrupt
- Progression is usually absent
  - Brain edema may cause new signs/symptoms

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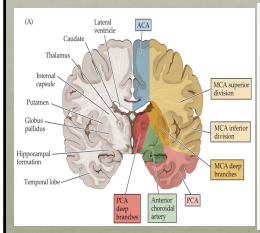

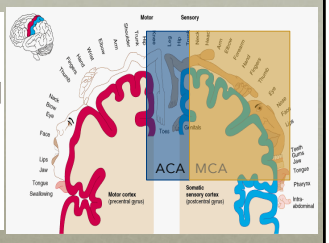

- ACA Lesion affects lower body
- MCA Lesion affects upper body

Both on contralateral (opposite) side

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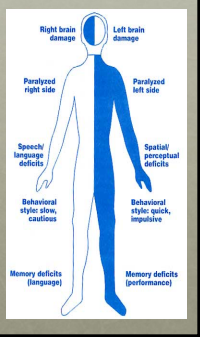
Location of the lesion

- Aphasia and/or seizures
  - Suggest hemispheric cortical lesion
- Ataxia suggests cerebellar lesion



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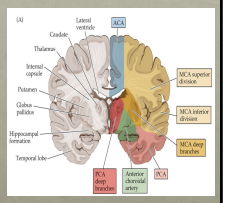
Clinical Findings

- Findings depend upon vessel involved
  - Obstruction of Carotid Circulation
    - Carotid Obstruction
      - Usually asymptomatic secondary to collaterals
    - Anterior Cerebral Artery
      - Weakness and cortical sensory loss in contralateral leg
        - Sometimes arm weakness
      - Anterior communicating artery provides collateral



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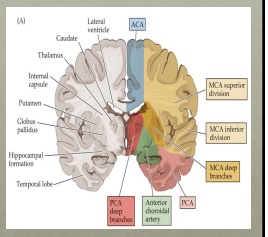
Clinical Findings

- Middle Cerebral Artery
  - Contralateral:
    - Hemiplegia
    - Hemisensory loss
    - Homonymous hemianopia
      - bilaterally symmetric loss of vision in half of the visual fields
    - Eyes deviated to the side of the lesion
  - +/- Global aphasia



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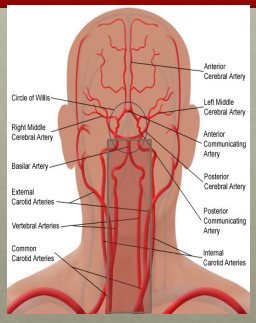
Clinical Findings

- Obstruction of Vertebrobasilar Circulation
  - Supplies:
    - Thalamus
    - Cerebellum
    - Brainstem
  - Occlusion of both vertebral arteries or basilar artery:
    - Coma with pinpoint pupils
    - Flaccid quadriplegia
    - Sensory loss
    - Various cranial abnormalities



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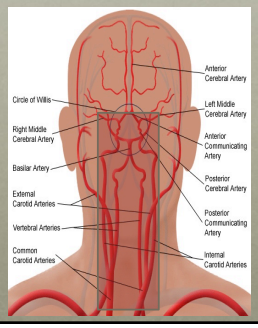
Clinical Findings

- Coma
  - Infarction in either carotid or vertebrobasilar system may cause loss of consciousness



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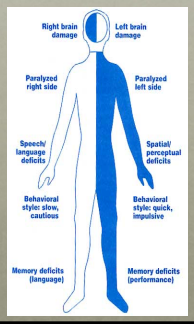
Location of the lesion

- Cerebral hemispheres
  - Paralysis involves the contralateral face, arm, and leg
    - If arm > leg, suspect lesion in distribution of MCA
    - If arm = leg, suspect a deep hemisphere lesion
    - If leg > arm, suspect anterior cerebral territory lesion



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Stroke Care 2019

- Cath Lab no longer means just the Heart
  - Neurovascular stent retrievers.
    - first alternative to clot-busting drugs to treat emergency stroke patients
    - wire-caged device can now be threaded through a patient's blood vessels to catch and remove clots from the bloodstream.

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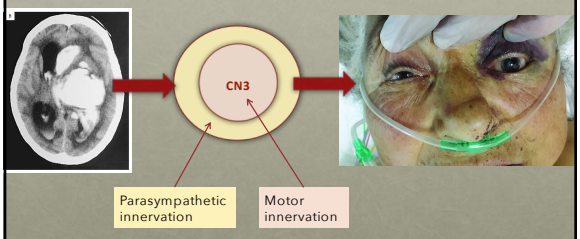
Acute catastrophic neurologic injury

- Major insult culminating with cerebral edema, elevated ICP, and ultimately herniation.



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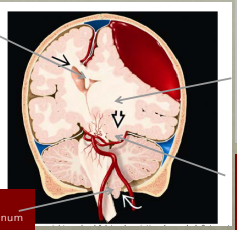
Herniation syndromes

Subfalcine Herniation
Cerebral cortex under falx
- Ipsi/contra leg weakness
- ↓ mental status

Upward Herniation
Brainstem up through tentorium
- ↓ mental status
- Dilated pupil (CNIII), ophthalmoplegia
- Ipsi paresis/posturing

Tonsillar Herniation
Cerebellar tonsils in foramen magnum
- Awake, quadriparesis
- Arrhythmia/cardiac arrest
- Respiratory arrest



Central Herniation
Brainstem down through tentorium
- ↓ mental status
- Dilated pupil (CNIII), ophthalmoplegia
- Ipsi paresis/posturing (contra cerebral crus)
- Basilar stroke

Uncal Herniation
- ↓ mental status
- Uncus over tentorial notch
- Dilated pupil (CNIII), ophthalmoplegia
- Ipsi paresis/posturing (contra cerebral crus)
- PCA stroke

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Stroke Alert

Medical Interventions

Reduce Cranial Contents:
Blood - vasodilation to constriction of
- Venous Return
- **Hyperventilation**
- Reduction of $CMRO_2$

Brain water
- Osmolar therapy for edema

Airway: O2 sat>90%
Breathing: normal CO2
Circulation CPP> 60mmHg

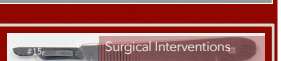
Head of Bed:
- 30 degree, midline

Hyperventilation:
- pCO2 30 +/- 2 mmHg

Hyperosmolar therapy
- Mannitol IV 1 gm/kg IV
- Hypertonic saline (CVL)
  - 3% NaCl or 23.4% NaCl

Normothermia/?Hypothermia
Pharmacologic Coma

Surgical Interventions



- Drain CSF
- Surgical removal of mass
- Break the rigid skull… craniectomy

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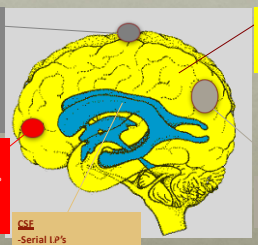
Increased ICP by source



Venous blood
- HOB up
- Neck straight
- No EJ lines, do not lay flat for lines
- Do no use venodilating BP agents

Arterial blood
- Hyperventilate
- Avoid hyperemia: MAP target 60, Pa02>50
- Decrease metabolism: sedation, cooling

Brain parenchyma
-Osmotherapy (mannitol, hypertonic saline)
-Steroids only if appropriate

CSF
-Serial LP's
-IV shunt placement

Lesion
-Blood, tumor, pus -> surgery
-Air-> 100% NRB, surgery

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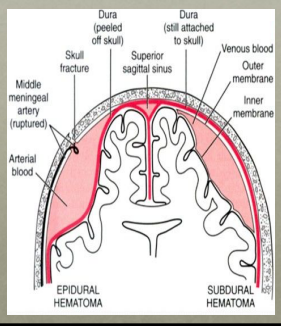
EPIDURAL HEMATOMA



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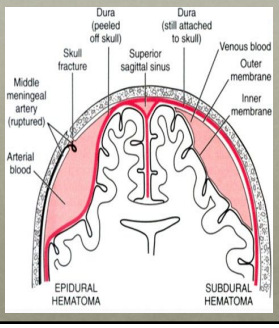
SUBDURAL



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CONTUSION

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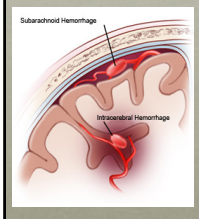
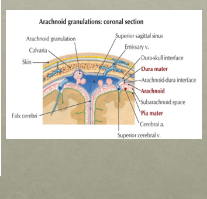
Subarachnoid Hemorrhage





"Worst Headache of my life"

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Rx of Intracerebral Hemorrhage

- Supportive
- Surgical removal of hematoma appropriate in some cases

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SECONDARY INJURY

- Events after the primary insult that exacerbate the brain injury and worsen outcome
- Leading cause of in-hospital deaths from traumatic brain injury
- **We impact this . . . TBI treatment begins in the field . . . 1st critical link**

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SECONDARY INJURY

- Reliant on appropriate early management
  - ABC's
  - AVOIDANCE OF HYPOTENSION AND HYPOXEMIA

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SECONDARY INJURY

- Trauma Coma Data Bank (TCDB)
  - 717 patients (prospective)
  - prehospital hypotension (SBP<90) and hypoxemia (apnea, cyanosis, Sat<90, PaO_2<60) were among the five most powerful predictors of outcome and statistically independent of other major predictors (age, adm GCS, adm GCS motor, intracranial diagnsosis, pupillary status)

single episode of hypotension . . .
40% INCREASE IN MORTALITY

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SECONDARY INJURY

Oxygen Saturation	Mortality	Severe Disability
>90%	14.3% (3/21)	4.8% (1/21)
60-90%	27.3% (6/22)	27.3% (6/22)
<60%	50% (3/6)	50% (3/6)

Stocchetti et al. Journal of Trauma 1996

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SECONDARY INJURY

Outcome by Secondary Insult at Time of Arrival at Traumatic Coma Data Bank Hospital for Mutually Exclusive Insults[a]

Secondary Insults	Number of Patients	% Total Patients	Outcome (%)		
			Good or Moderately Disabled	Severely Disabled or Vegetative	Dead
Total cases	699	100%	43	20	37
Hypoxemia[b]	78	11	45	22	33
Hypotension[b]	113	16	26	14	60
Neither	456	65	51	22	27
Both	52	8	6	19	75

Chestnut et al. 1993. The role of secondary brain injury in determining outcome from severe head injury. Journal of Trauma Feb;34(2):216-22.

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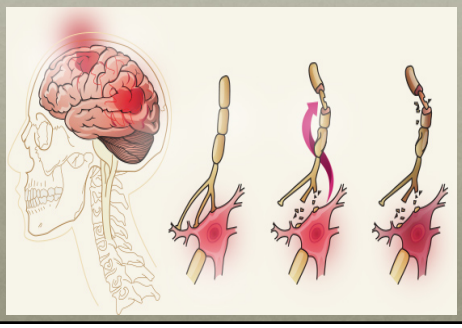
DIFFUSE AXONAL INJURY



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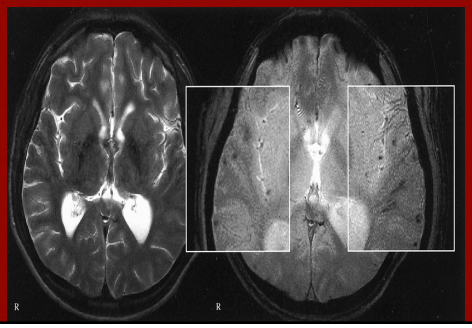
DIFFUSE AXONAL INJURY



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Cerebral Blood Flow and Traumatic Brain Injury

- Systolic BP must be maintained at >80 mmHG
  - Severe brain injury – SBP > 110 mmHg
- Maintain CPP at >70 mmHg
- Cerebral Perfusion Pressure (CPP) drives Cerebral Blood Flow (CBF)
- CBF found to be near ischemic threshold after injury
- CBF near areas of contusions and hematomas reduce even further

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Cerebral Blood Flow and Traumatic Brain Injury

- Studies have shifted from ICP to CBF
- Hypotension has been shown to increase ICP in patients with intact autoregulation
- Decrease in BP = cerebral vasodilation (as high as 65% increase in vessel size) = increase in ICP
- CPP <60 mmHg = mortality of 95%

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ICP and BP

- An increase in systolic BP by 30 mmHg changes the ICP by an average of only 4 mmHg
  - Normal ICP is 0-10mmHg, ICHTN is 20-25mmHg
- In three cases, the ICP decreased with an increase in BP
- Moderate increase in BP to maintain CPP should not cause an increase in ICP
- Cerebral edema and ICP increases occur in 40% of TBI

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Hyperventilation: Good or Bad?

- CBF during first day after TBI is less than half that of normal CBF
- Hyperventilation reduces ICP by reducing CO2 and causing vasoconstriction
- Vasoconstriction will also reduce CBF

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Hyperventilation: Good or Bad?

- Aggressive hyperventilation = PaCO2 of less than 30 mmHg
- Will reduce CBF but has not been found to consistently reduce ICP - what it is intended to do
- May cause loss of autoregulation
  - BP increase = vasodilation = >ICP
  - BP decrease = vasoconstriction = <CBF

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Hyperventilation: Good or Bad?

- PaCO2 <25 torr = no better outcome
- Hyperventilation used to decrease PaCO2 by 15 to 20 mmHg caused a 40% reduction in CBF
- For every 1 torr change in PaCO2 the CBF changes by 3%
- Some patients lost autoregulation with hypocapnia and preserved with normocapnia

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Hyperventilation Guidelines

- No evidence of herniation - 12/minute
- Evidence of herniation = 20/minute
  - unilateral or bilateral dilated pupil
  - asymmetric pupil reactivity
  - motor posturing
- Evolution of one or more signs is evidence of ICP - systemic resuscitation is key to survival

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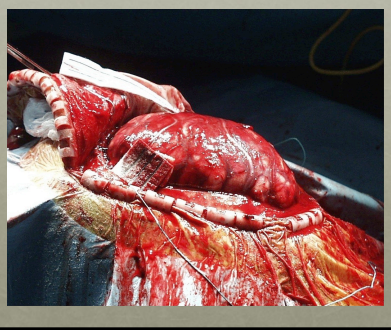
Edema Formation

- Vasogenic
- Cytoxic



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INITIAL MANAGEMENT

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Cerebral Resuscitation

- Reverse hypotension
- Maintain BP
- Reverse hypoxia
- Maintain adequate ventilation and oxygenation
- What about fluids in trauma?

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Hypertension

- How much is too much?
  - SBP of > 180 mmHg
  - MAP of > 130 mmHg
- Choices
  - Labetelol
  - Nicardipine (Cardene)
- How fast do you lower it?
  - As fast as necessary to get under 180 mmHg or 130 mmHg (as above) but no more than 20% in 24 hours

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PREHOSPITAL MANAGEMENT

- Airway
- Ventilation
- Oxygenation
- Fluid resuscitation

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AIRWAY

- Endotracheal intubation is recommended for patient with GCS ≤ 8

Prehospital endotracheal intubation and outcome in severe head injury patients (Winchell)[9].

	Intubated	Not Intubated
All Patients - Mortality	26%	36.2%
Isolated TBI - Mortality	22.8	49.6

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AIRWAY

Field GCS score and the need for prehospital endotracheal intubation in TBI patients (Hsiao)[10].

	GCS Score			
	3-5	6-7	8-9	10-13
Field intubation	27%	27%	8%	2%
ED intubation	73	45	53	18
CT scan positive	73	36	62	23

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VENTILATION

- Routine hyperventilation is NOT recommended and detrimental to the injured brain
- Normocapnia is most prudent
  - (PCO_2 35 to 40)

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FLUID RESUSCITATION

- Support oxygen delivery and avoid hypotension
- Most common cause of hypotension in the trauma patient is hemorrhagic shock
- Euvolemia
  - Mainstay of therapy is isotonic crystalliod solution
- Hypertonic saline . . . ?

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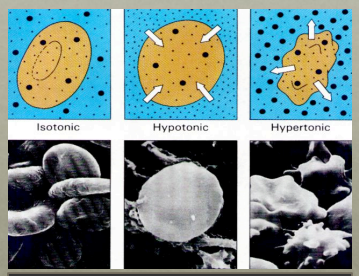
HYPERTONIC SALINE



- Beneficial in animal studies
- Ongoing clinical trials with mixed results
- May have a benefit in hypotensive trauma patients with GCS ≤ 8

Restore perfusion in hypovolemic head-injured patients with less volume

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OTHER THERAPIES

- Mannitol
  - not routinely recommended due to concern diuretic effect
- Lidocaine with RSI
  - Believed to prevent increase in intracranial pressure (ICP) with intubation
  - no study; thus far, has shown an impact on transient increases in ICP on patient outcome

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OTHER THERAPIES

- Nimodipine for SAH
  - Day 1 to 21
- Lidocaine with RSI
  - Believed to prevent increase in intracranial pressure (ICP) with intubation
  - no study; thus far, has shown an impact on transient increases in ICP on patient outcome

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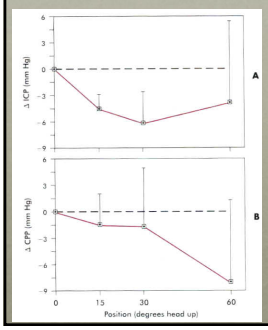
OTHER THERAPIES



- Sedation and analgesia
  - treat hypotension, hypoxemia, hypoglycemia,and patient discomfort first
  - data on the use of pharmacologic agents in the prehospital setting is lacking
- head elevation
  - no more that 30 degrees

Do not forget spinal precautions

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DEFINITIVE MANAGEMENT

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EMERGENCY DEPARTMENT

- ABC's with C-spine immobilization
- Recognition and control of exsanguinating injuries
- Neurologic assessment
- Immediate CT Head after initial stabilization
  - characterizes anatomic injury
  - identifies surgical lesion

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CT (Computed Tomography)

- Presence of abnormalities on initial CT
  - 90% of pts with severe TBI
  - normal CT has better prognosis
    - outcome related primarily to extracranial injuries

40% of patients with normal CT will develop lesions

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INTRACRANIAL PRESSURE

- Normal ICP 0 – 10mmHg
- Intracranial hypertension (ICH) usually defined as ICP > 20 – 25mmHg

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ICP Management

- AVOIDANCE OF HYPOXEMIA AND HYPOTENSION WITH ASSURANCE OF EUVOLEMIA (consider CVP monitoring)
- Early measures
  - HOB 30° upright or reverse Trendelenberg . . . Avoid HOB > 45° (decreases CPP)
  - appropriate analgesia and sedation ± neuromuscular paralysis

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Other therapies

- Seizure prophylaxis
- Avoidance of hyperthermia
- Tight glucose control
- Barbiturate coma
- Surgery

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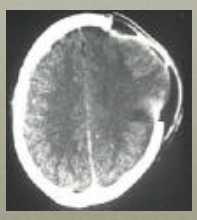
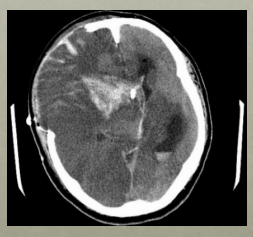
Decompression Surgery





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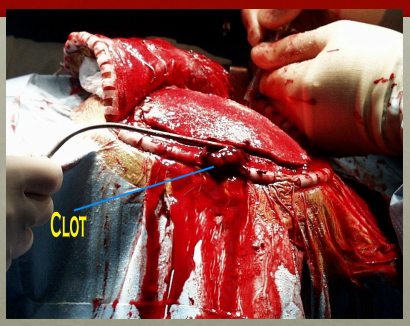
Decompression Surgery



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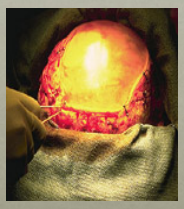
Decompression Surgery



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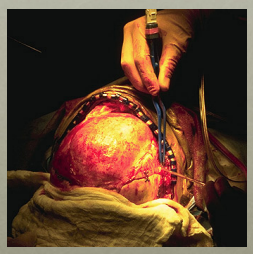
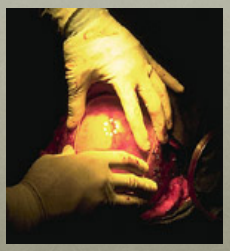
Cranioplasty





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THANK YOU!!

For handouts or further information

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