Stanford HEALTH CARE

Feeling the Pressure

(as in Intracranial Pressure….)

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Disclosures

- Financial Disclosures:
  - None relevant to the clinical content being presented
  - Intermittent Stroke reviewer for The Joint Commission

- Unapproved/Usage Disclosure:
  - None

Outline

- Intracranial hemodynamics
  - CBF – Cerebral blood flow
  - CPP – Cerebral perfusion pressure
  - ICP – Intracranial pressure
- Causes of increased ICP
- Signs and Symptoms of ICP
- Treatment
  - Emergency Neurological Life Support:
    - Intracranial Hypertension and Herniation Protocol
  - Hyperosmolar Therapy
  - Decompressive Craniectomy for Malignant MCA Infarcts

Intracranial Pressure

- Skull is a fixed volume vault; skull by nature is non-compliant

- ICP = sum of 3 components to total a fixed volume in the cranial vault

CSF ~10% + Intravascular Blood ~5% + Brain Tissue ~ 85% = ICP

- Non-compressible, but partially displaceable

Intracranial Pressure

Monro-Kellie Doctrine

Sum of the intracranial volumes of blood, brain, CSF, and other components is **constant**, and that an **increase in any one of these must be offset by an equal decrease in another**, or else pressure increases.

Normal:

5 - 15 mmHg

Intracranial hypertension:

ICP > 20mmHg sustained for more than 5 minutes

Cerebral Dynamics: Cerebral Perfusion Pressure

Cerebral perfusion pressure (CPP)

Difference between the force driving blood into the brain and the force resisting movement of blood into the brain

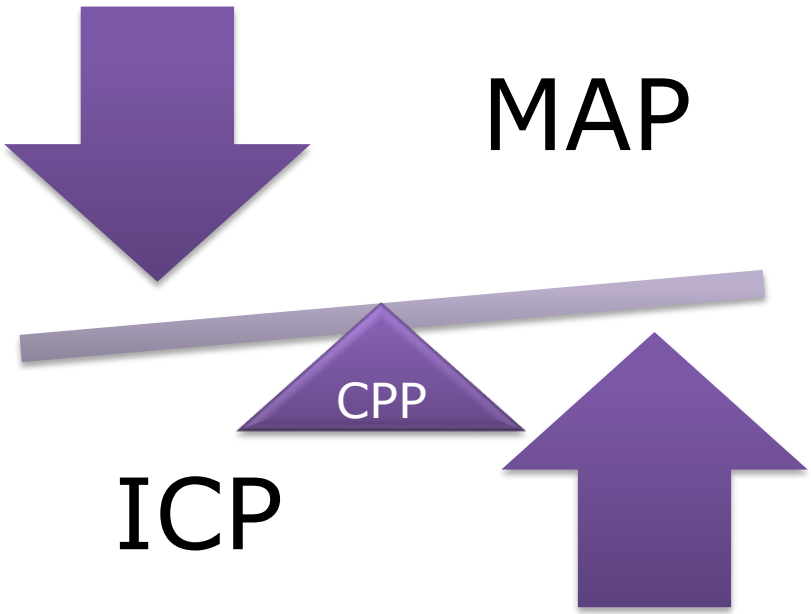
CPP =

MAP – ICP

Normal: 70-100mmHg

< 50 mmHg: Cerebral ischemia

< 30 mmHg: Brain death



Cerebral Dynamics: Cerebral Blood Flow

Cerebral Blood Flow (CBF)

Amount of blood passing through 100g of brain tissue in 1 minute

CBF =

$$\frac{\text{Cerebral perfusion pressure}}{\text{Cerebral vascular resistance}}$$

750ml/minute

~15% of cardiac output

50ml/min per 100g of brain tissue

Average: 50

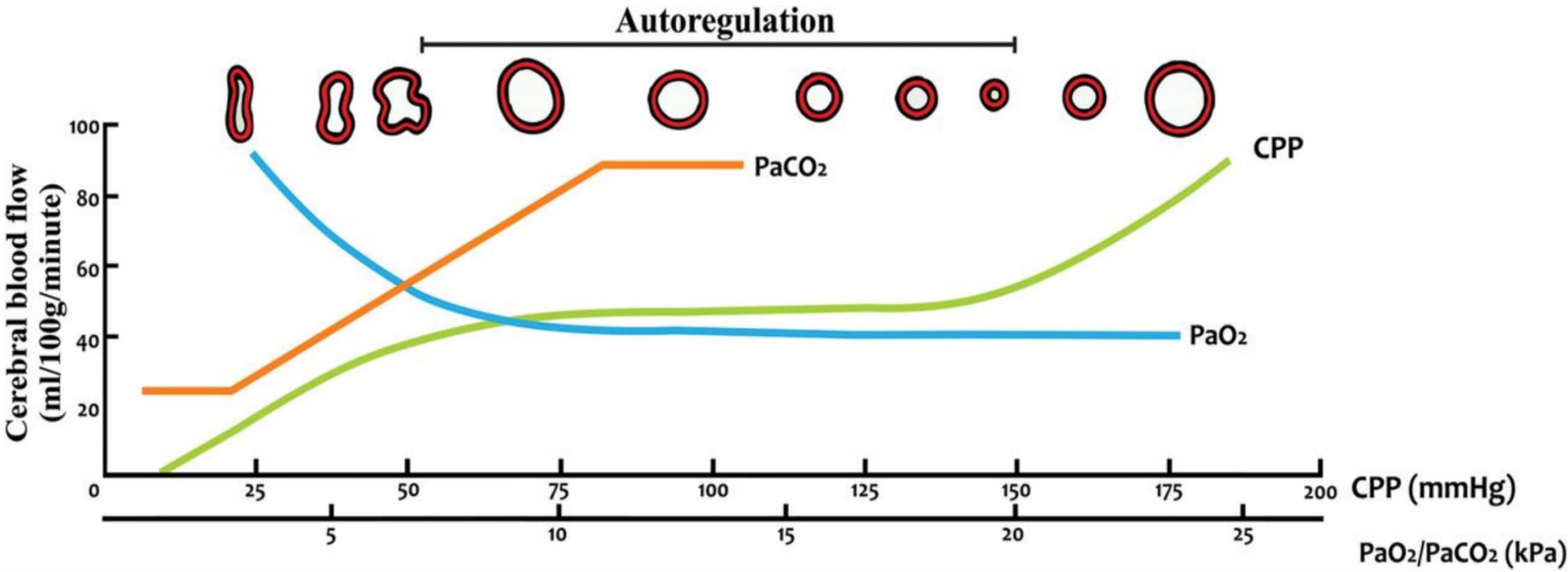
Ischemia: < 18 – 20

Tissue death: < 8 – 10

Hyperemia: > 55 – 60

Autoregulatory mechanisms maintain a relatively constant CBF, despite changes in systemic parameters

Cerebral Dynamics: Cerebral Blood Flow



Tameen et al., 2013

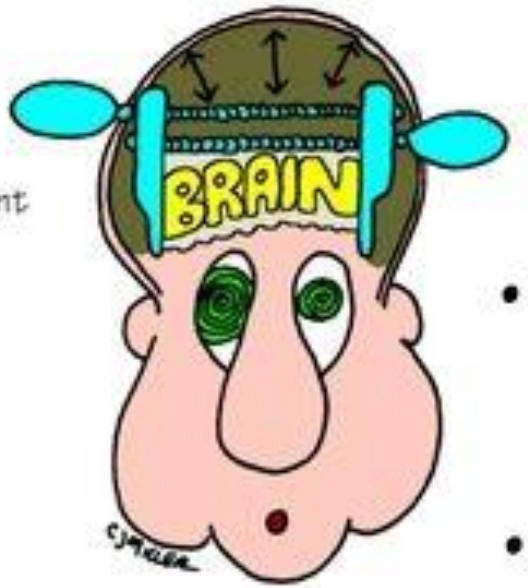
Causes of Increase ICP

Intracranial (primary)	Extracranial (secondary)	Postoperative
Tumor	Airway obstruction	Mass lesion (hematoma) edema
Tramua (Epidural & Subdural hematomas & contusions)	Hypoxia or hypercarbia	Increased cerebral blood volume (vasodilation)
Non-traumatic intracranial hemorrhages	Posture (head rotation)	Disturbances of CSF
Ischemic stroke	Hyperpyrexia	
Hydrocephalus	Seizures	
Idiopathic or benign intracranial hypertension	Drug and metabolic derangements	
Other (eg, pseudotumor cerebri, pneumoencephalus, abscesses, cysts)	Others (eg, high-altitude cerebral edema, hepatic failure)	

Rangel-Castello, et al., 2008

INCREASED INTRACRANIAL PRESSURE

- Changes in LOC
- Eyes
  - Papilledema
  - Pupillary Changes
  - Impaired Eye Movement
- Posturing
  - Decerebrate
  - Decorticate
  - Flaccid
- Decreased Motor Function
  - Change in Motor Ability
  - Posturing

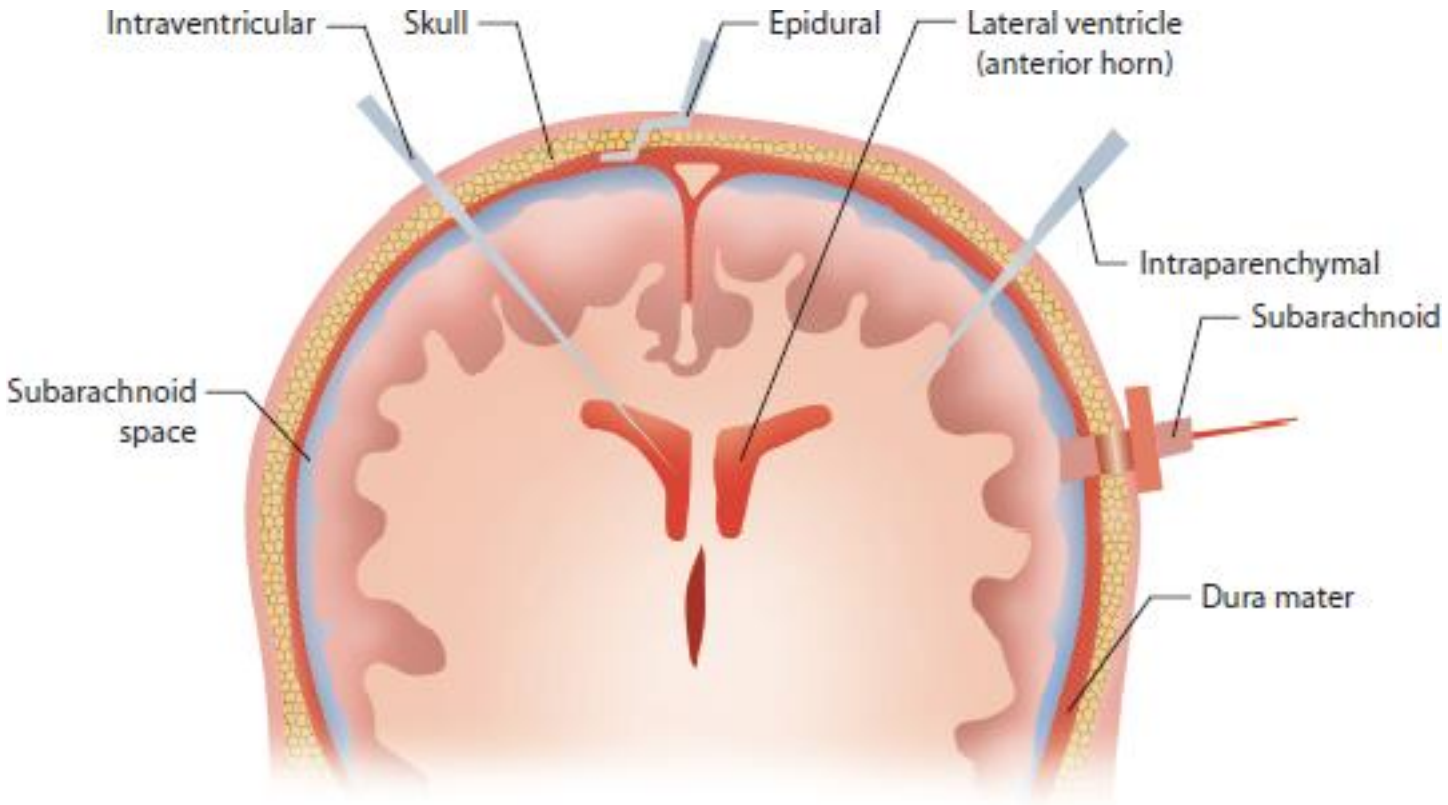


- Headache
- Seizures
  - Impaired Sensory & Motor Function
- Changes in Vital Signs:
  - Cushing's Triad:
    - ↑ Systolic B/P
    - ↓ Pulse
    - Altered Resp Pattern
- Vomiting
- Changes in Speech
- Infants:
  - Bulging Fontanels
  - Cranial Suture Separation
  - ↑ Head Circumference
  - High Pitched Cry

Signs of Increased ICP

"EARLY" →	"LATE"
Headache	Changes in level of consciousness or ↓ GCS or FOUR Score ≥ 2 points
Irritability	Ipsilesional change in pupillary size, shape and light-responsiveness
Vomiting	Contralesional hemiparesis (new or worsening)
Photophobia, nystagmus, diploplia	Contralesional change in pupillary size and ipsilesional hemiparesis (Kernohan's phenomenon)
Lethargy	Cushing's triad: ↑ SBP (widened pulse pressure), bradycardia, irregular respirations
Seizure	

How do we measure ICP?

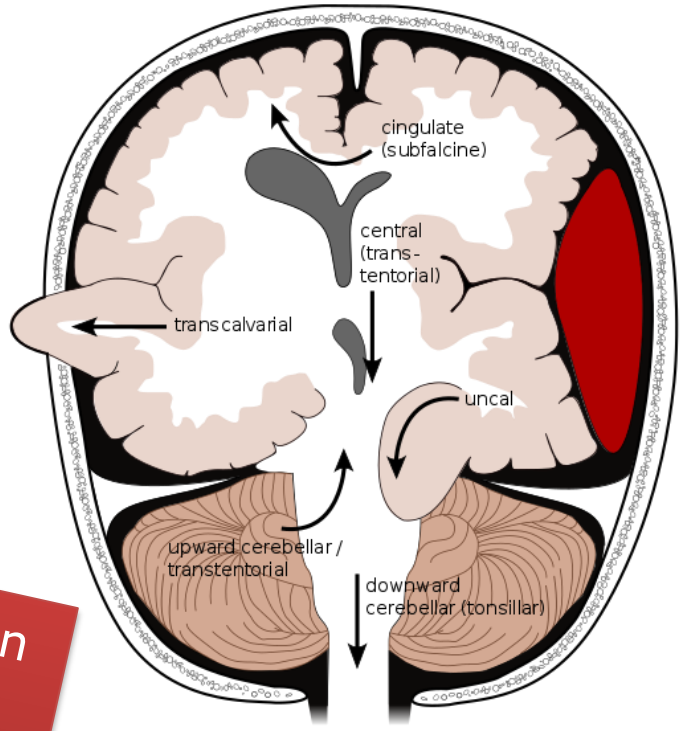


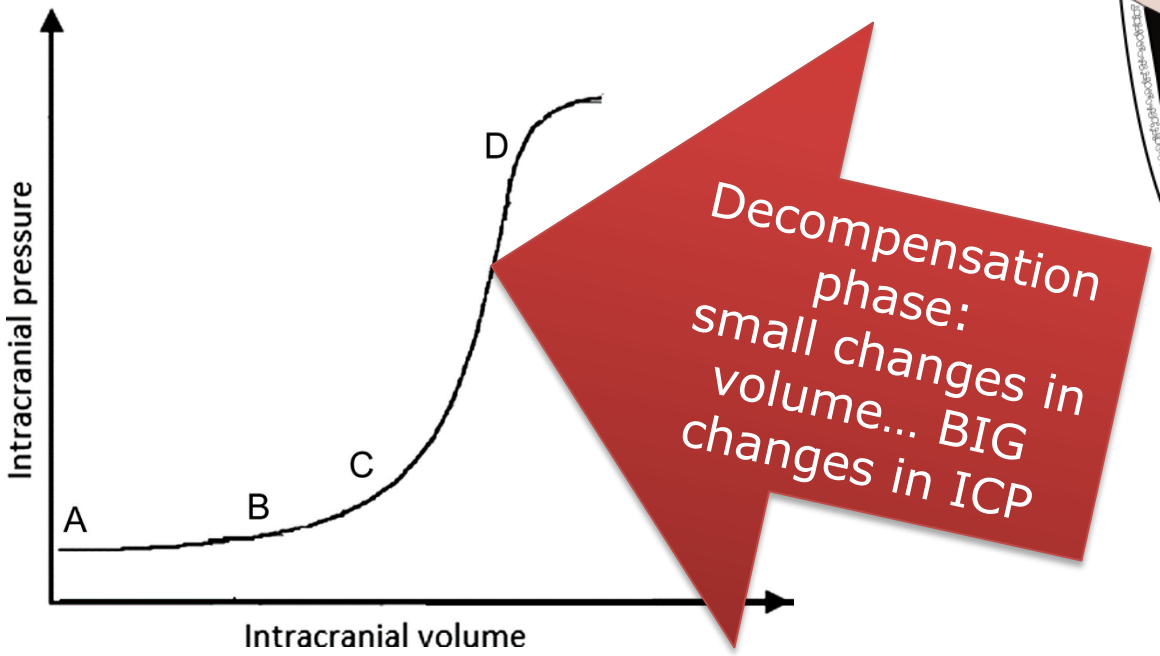
Source: Jesse B. Hall, Gregory A. Schmidt, John P. Kress: *Principles of Critical Care*, 4th Edition: www.accessmedicine.com

http://accessmedicine.mhmedical.com/data/books/1340/hall4_ch86_fig-86-16.png

Herniation

- Increased intracranial compartmental pressure causing to tissue shifts that compress or displace the brainstem, cranial nerves, or cerebral vasculature

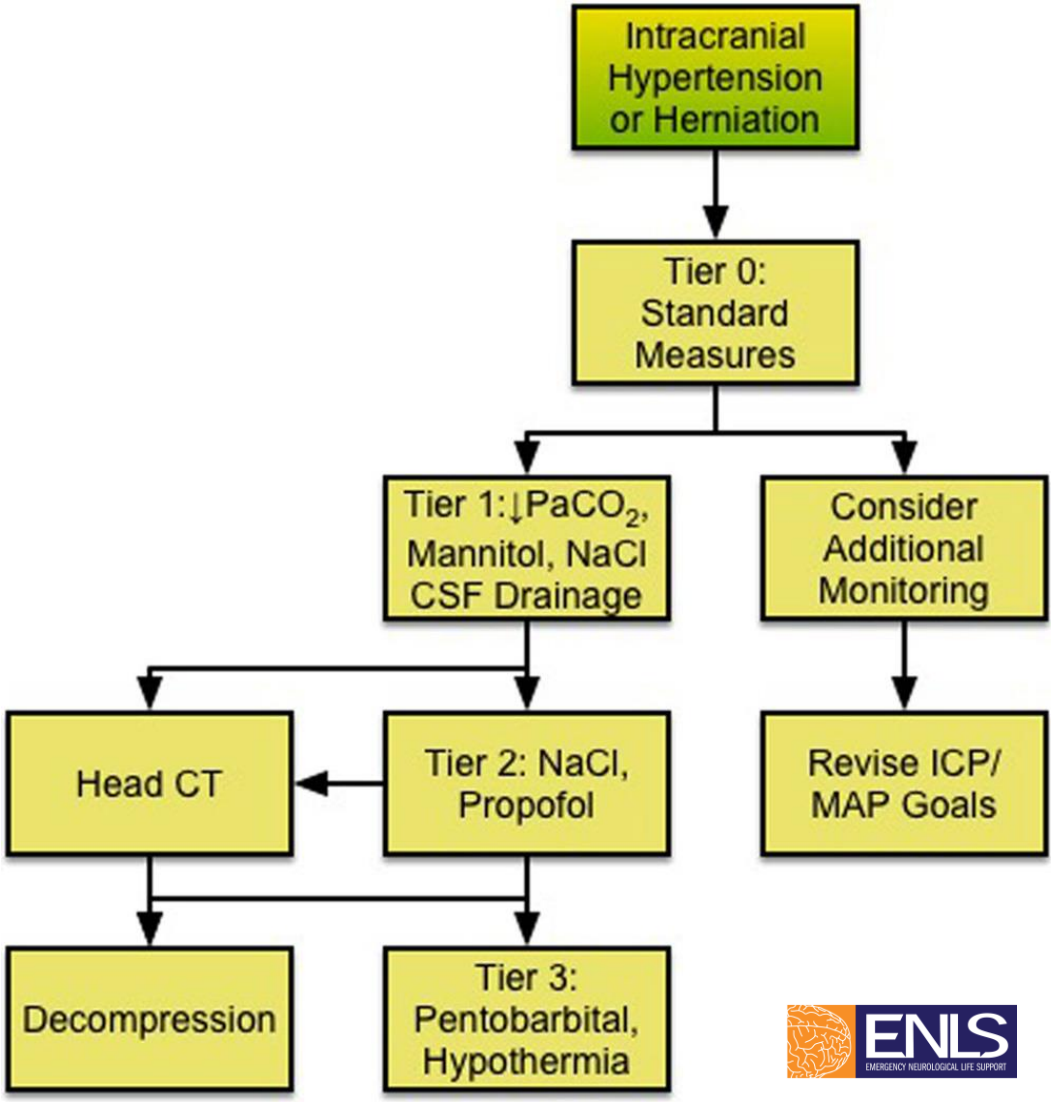




Tameen et al., 2013

Treatment of Intracranial Pressure

- Think BIG – heterogeneous population
- Step wide approach



ENLS: Tier 0 – Standard Measures

ABCs

(avoid hypotension and hypoxia)

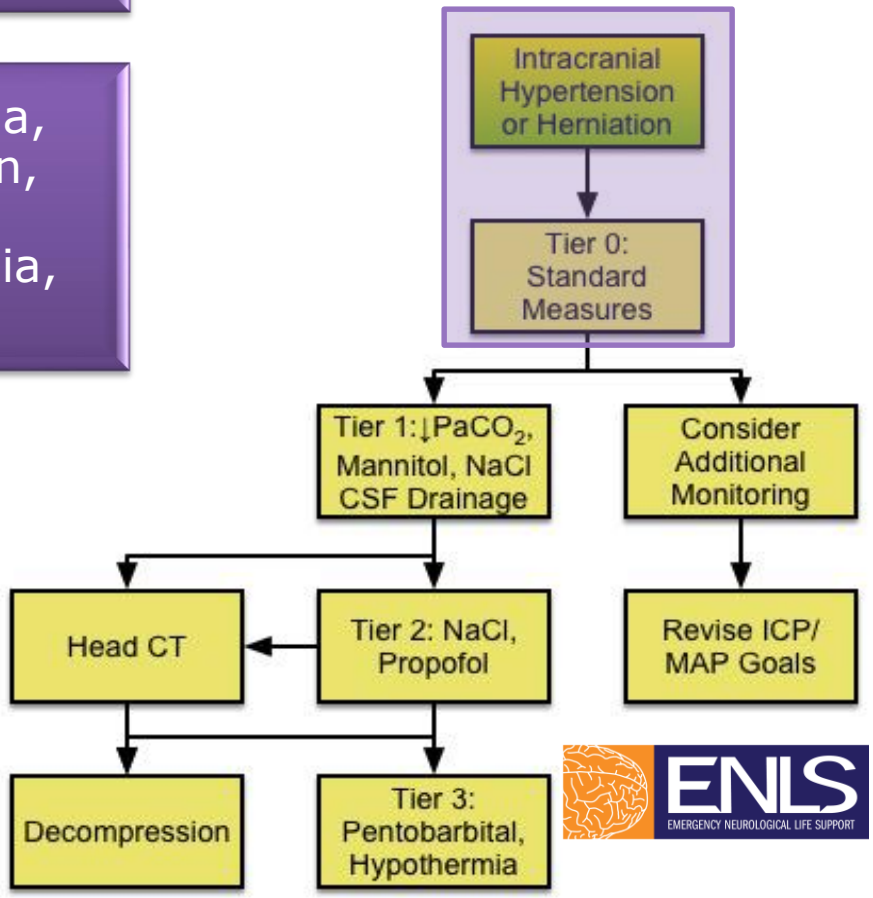
Head of bed elevated > 30 degrees and midline

(increase venous return)

Minimize stimuli or adequately sedate and provide pain relief

Normothermia, normotension, euvolemia, normonatremia, euglycemic

Treat vasogenic edema (steroids for tumors)



ENLS: Tier 1

Hyperosmolar therapy:

- Mannitol 0.5-1g/kg (Serum osmolality q4-6 hours)
- Hypertonic saline (Serum Na levels q4-6 hours)

Placement of external ventricular drain (EVD)

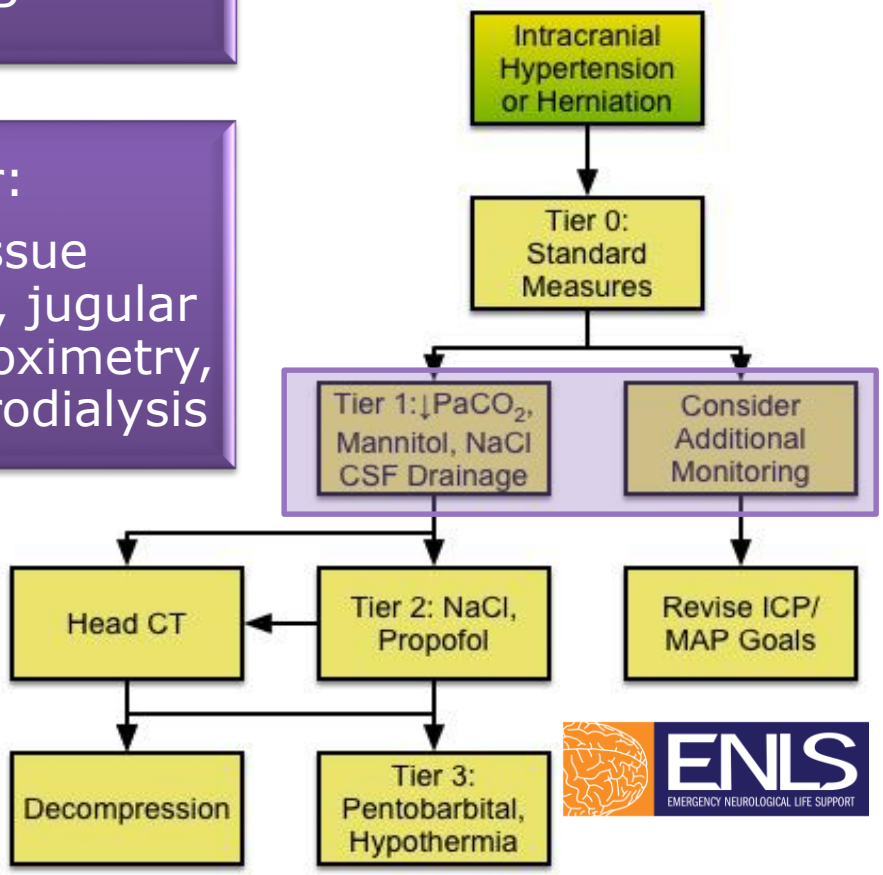
- Drain for acute rises in ICP

Hyperventalation:

Consider BRIEF (<2 hours) (PaCO2 30-35 mmHg) as temporizing measure

Other:

Brain tissue oxygenation, jugular bulb venous oximetry, cerebral microdialysis



Hyperosmolar Therapy

Mannitol:

- Osmotic diuretic, drawing water out of edematous brain tissue
- Typically given as bolus of 0.25-1g/kg
- Caution/Contraindicated: hypovolemia, hypotension, renal failure, pulmonary edema
- Over time "opens" blood brain barrier and mannitol crosses, losing efficacy
- Monitoring: Serum osmolality q4-6 hours (< 320)

Hypertonic saline:

- Causes an osmotic gradient, drawing water out of edematous brain tissue
- Can be given as bolus or infusion, ranging from 3-23.4%
- Can result in plasma volume expansion (increases blood pressure and CPP) – can be used with hypotension/hypovolemia
- Requires an intact blood brain barrier
- Overtime can lead to electrolyte abnormalities such as hyperchloremic acidosis
- Monitoring: Serum Na levels q4-6 hours (<160)

Which is better???

Difficult question limited by small number and size of trial

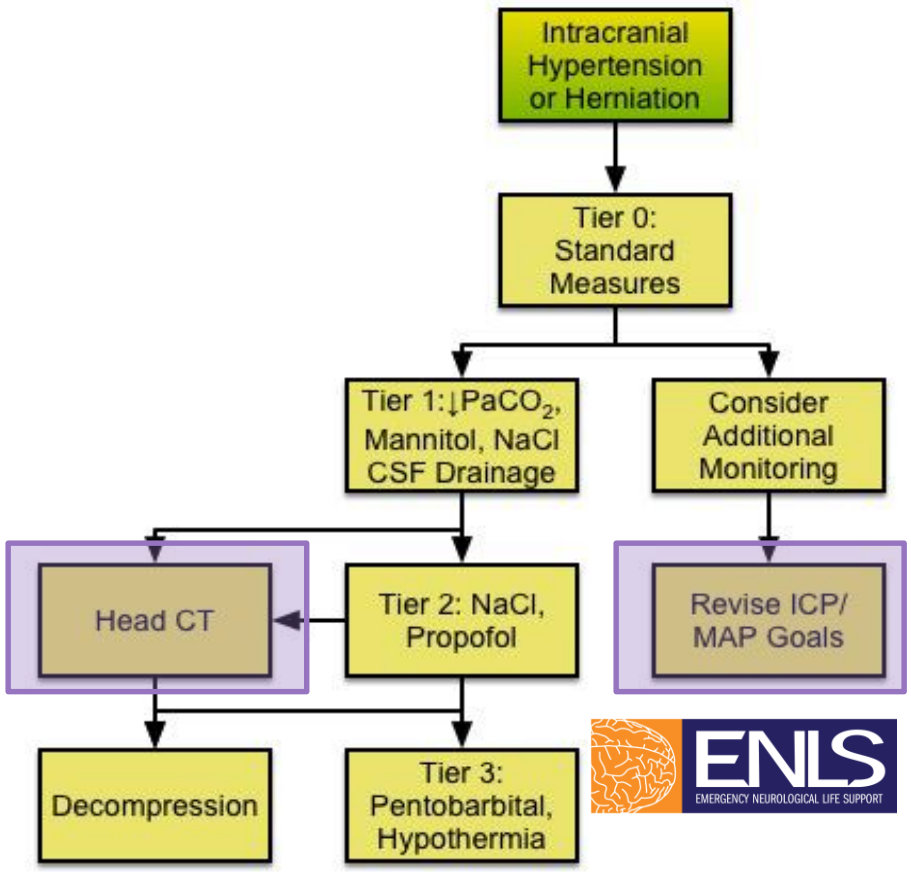
In a true emergency, whichever you can obtain/administer the quickest!

ENLS: Post Tier 1

If ICP stabilized with Tier 1 → obtain a head CT

If not, move to Tier 2 → obtain head CT

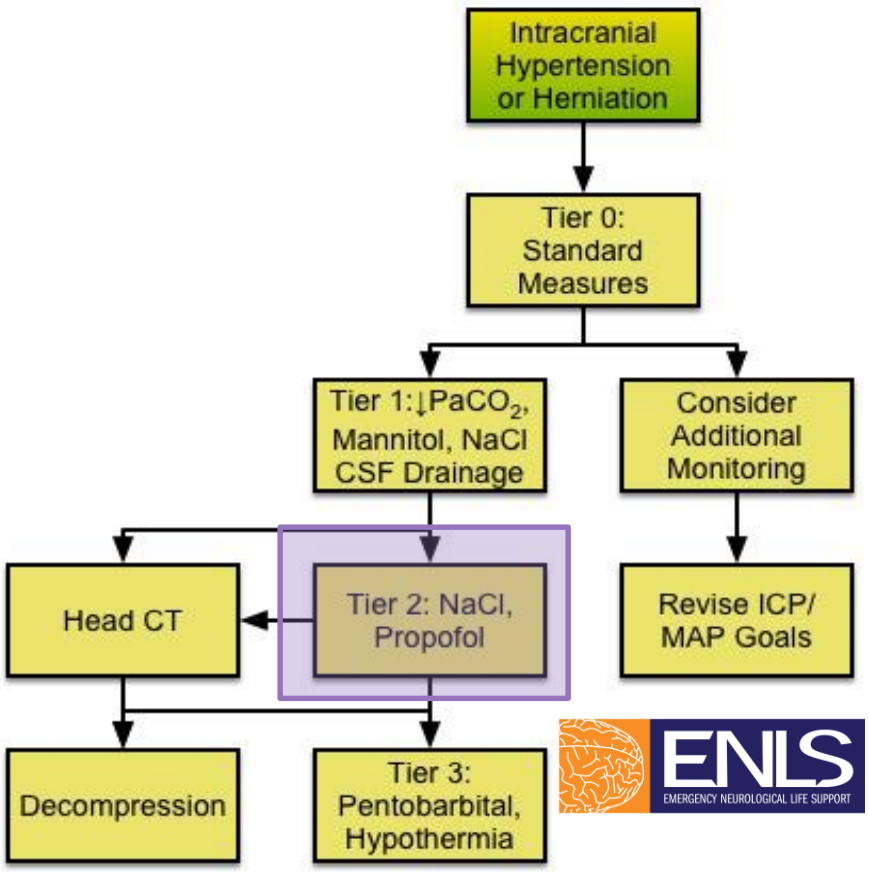
Consider adjusting ICP, MAP and CPP based on clinical context



ENLS: Tier 2

Increase Na goal (~160mmol/L)

Increase sedation

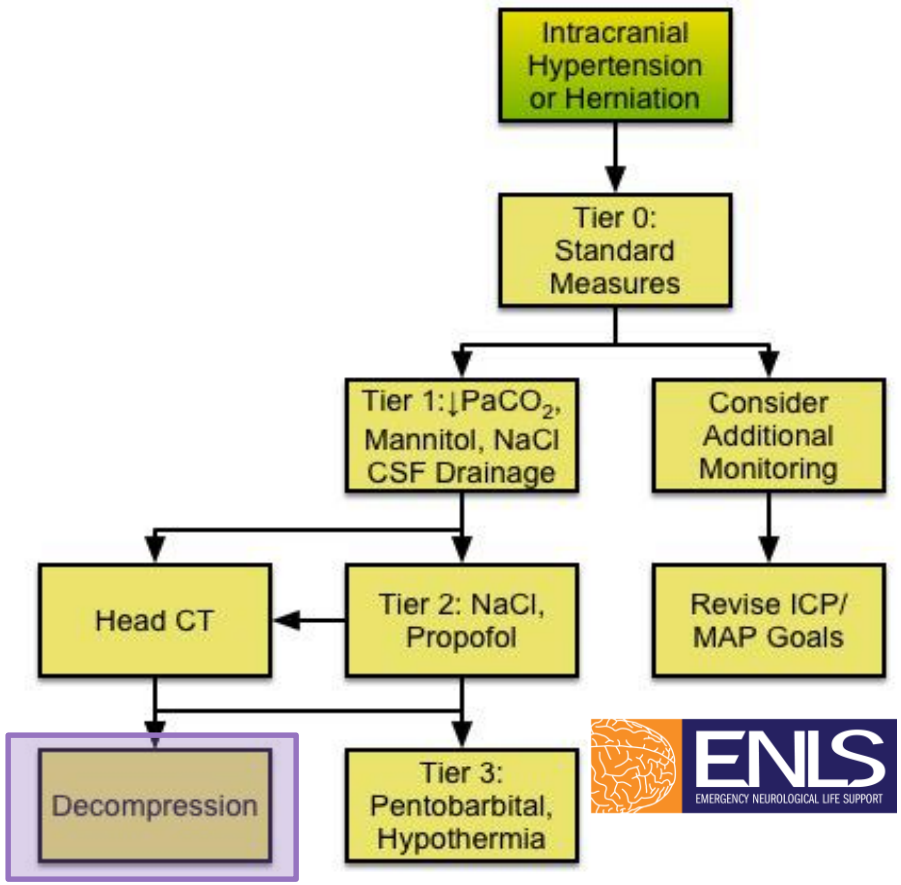


Decompression

If failing medical management:

- Review surgical options
- Evacuation of mass lesion or decompression craniectomy

If the patient is ineligible for surgery or too unstable for brain imaging, move to Tier 3



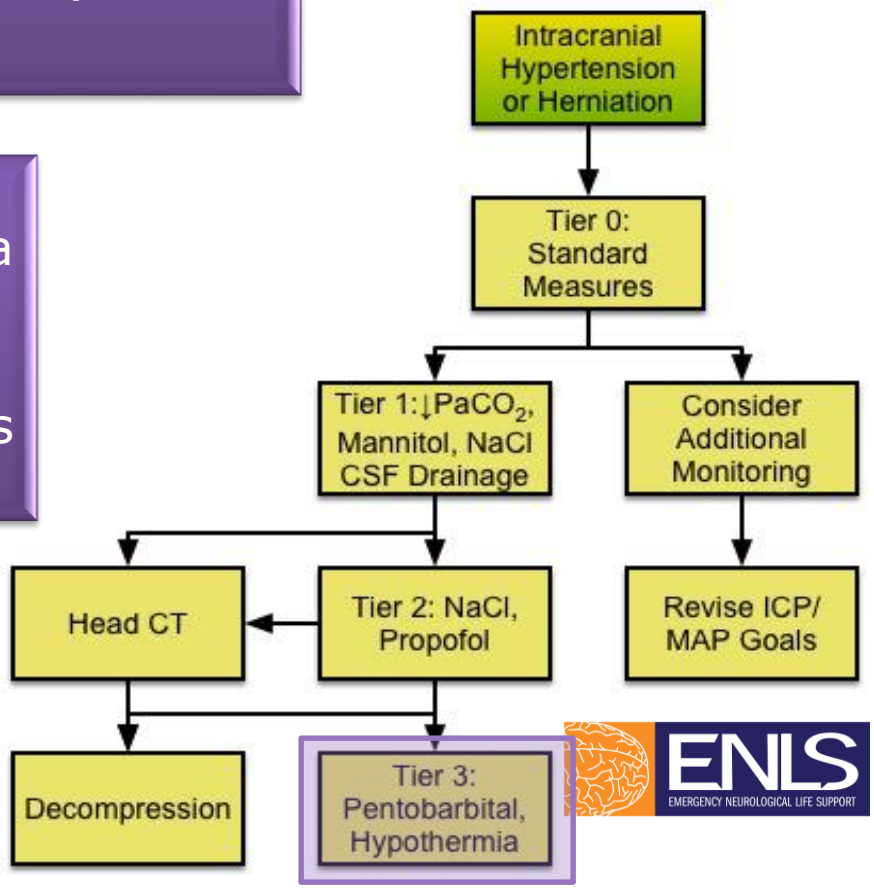
ENLS: Tier 3

Pentobarbital infusion (cEEG) 24-96 hours

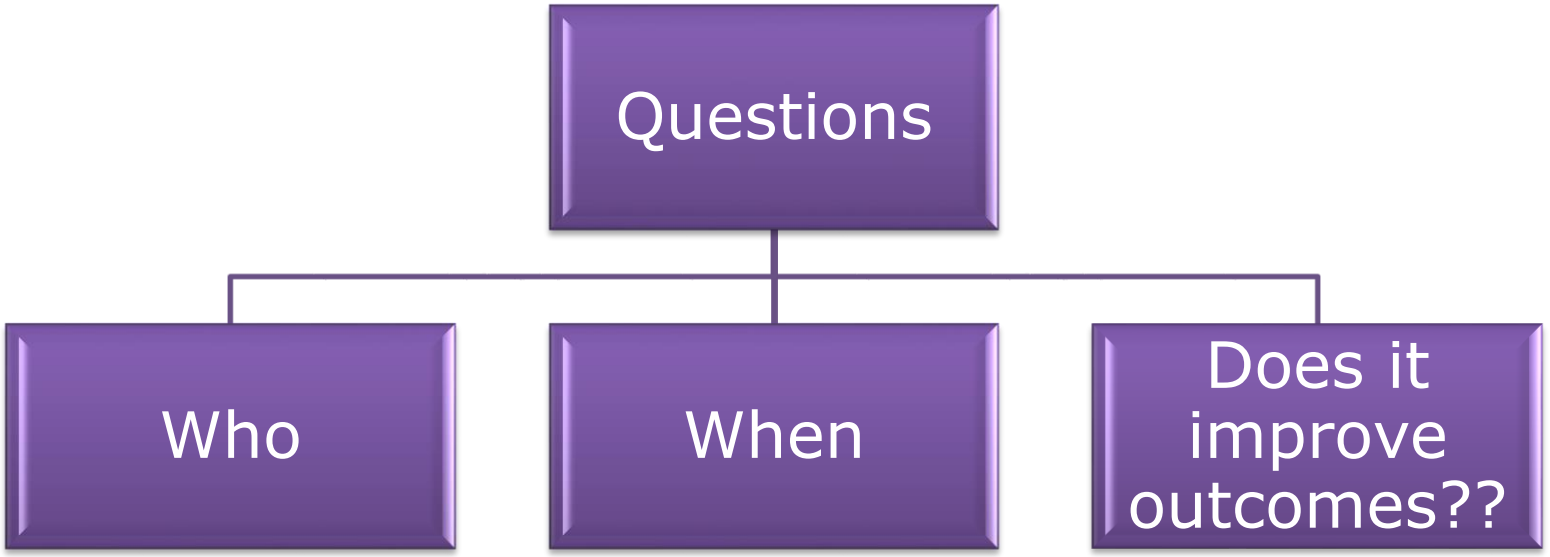
Moderate hypothermia (32-34 degrees Celsius)

Hyperventilation to achieve mild to moderate hypocapnia (PaCO2 25-30mmHg)

Ideally with cerebral oxygen monitoring and for < 6 hours duration



Decompressive Craniectomy in Ischemic Stroke



- Malignant Middle Cerebral Artery Infarct
  - Distal ICA or proximal MCA trunk occlusion leading to a large MCA infarction (+/- ACA or PCA involvement) and poor collateral compensation
  - Mortality of 78%, due to transtentorial herniation and brain death, range 2-5 days

Hacke et al., 1996

Demographic & Clinical Predictors
Malignant MCA Infarct

Predictor	# patients	Odds ratio or Sens/Spec	Studies
Younger age	192	OR 0.4 95% CI 0.3-0.6 p<0.0001	Jaramillo et al Neurology 2006
Female sex	192	OR 8.2 95% CI 2.7-25.2 p = 0.0003	Jaramillo et al Neurology 2006
NO prior infarcts	192	OR 0.2 95% CI 0.05-0.7 p= 0.01	Jaramillo et al Neurology 2006
History of HTN	201	OR 3.0 95% CI 1.2-7.6 p=0.02	Kasner et al, Stroke 2001
History of CHF	201	OR 2.1 95% CI 1.5-3.0 p=0.001	Kasner et al, Stroke 2001
Admission NIHSS >20 [>15 for non-dom hemisphere]	28	100% sens 78% spec	Oppenheim et al, Stroke 2000
Nausea and vomiting 1st 24 hours	135	OR 5.1 95% CI 1.7-15.3 p=0.003	Krieger et al, Stroke 1999

Adapted from Wartenberg, 2012

Radiographic Predictors of Malignant MCA

Predictor	# patients	Odds ratio or Sens/Spec	Studies
Hypodensity on initial head CT > 50% MCA territory	135 201 36	OR 6.1, 95% CI 2.3-16.6, p=0.0004 OR 6.3, 95% CI 3.5-11.6, p = 0.001 OR 14.0, 95% CI 1.04-189.4, p=0.047	Krieger et al, Stroke 1999 Kasner et al, Stroke 2001 Manno et al, Mayo Clin Proc 2003
CT Hyperdense MCA sign	36	OR 21.6, 95% CI 3.5-130, p < 0.001	Manno et al, Mayo Clin Proc 2003
CT Anteroseptal shift ≥ 5 mm on follow up head CT <48 hrs	135	OR 10.9; 95% CI 3.2-37.6	Barber et al, Cerebrovasc Dis 2003
MRI DWI volume >145 mL within 14 hours	28	100% sens, 94% spec	Oppenheim et al, Stroke 2000
MRI DWI volume >82 mL within 6 hours of onset	140	52% sens, 98% spec	Thomalla et al, Ann Neuro 2010

Adapted from Wartenberg, 2012

2007 Pooled DECIMAL, DESTINY, HAMLET Analysis

DECIMAL, DESTINY, HAMLET trials pooled their data prior to each individual results completed and published

- 93 pts randomized <48 hours

Conclusions:

Significantly more patients met the primary outcome measures mRS0-4 at one year in the surgical group, ARR 51%, p<0.0001

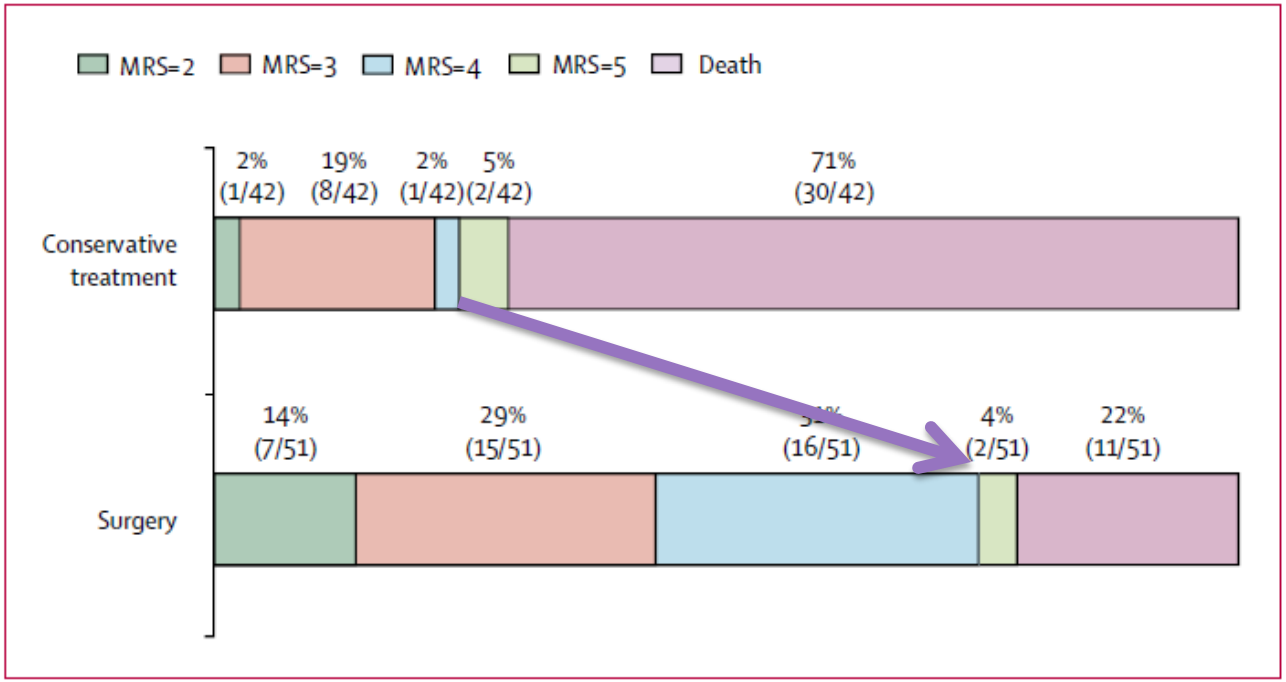


Figure 1: Distributions of the scores on the mRS and death after 12 months for patients treated with or without decompressive surgery

Vahedi al., 2007

2014 AHA/ASA Recommendations for the Management of Cerebral and Cerebellar Infarction With Swelling

Neurosurgical Options: Recommendations

1. In patients <60 years of age with unilateral MCA infarctions that deteriorate neurologically within 48 hours despite medical therapy, decompressive craniectomy with dural expansion is effective. The effect of later decompression is not known, but it should be strongly considered (*Class I; Level of Evidence B*).
2. Although the optimal trigger for decompressive craniectomy is unknown, it is reasonable to use a decrease in level of consciousness and its attribution to brain swelling as selection criteria (*Class IIa; Level of Evidence A*).
3. The efficacy of decompressive craniectomy in patients >60 years of age and the optimal timing of surgery are uncertain (*Class IIb; Level of Evidence C*).
4. Suboccipital craniectomy with dural expansion should be performed in patients with cerebellar infarctions who deteriorate neurologically despite maximal medical therapy (*Class I; Level of Evidence B*).

Timing – 48 vs. 72 hours

Conclusions:

When evaluated dichotomously, the odds of discharge to institutional care and of a poor outcome did not differ at 48 hours after hospital admission, but increased when surgery was pursued after 72 hours.

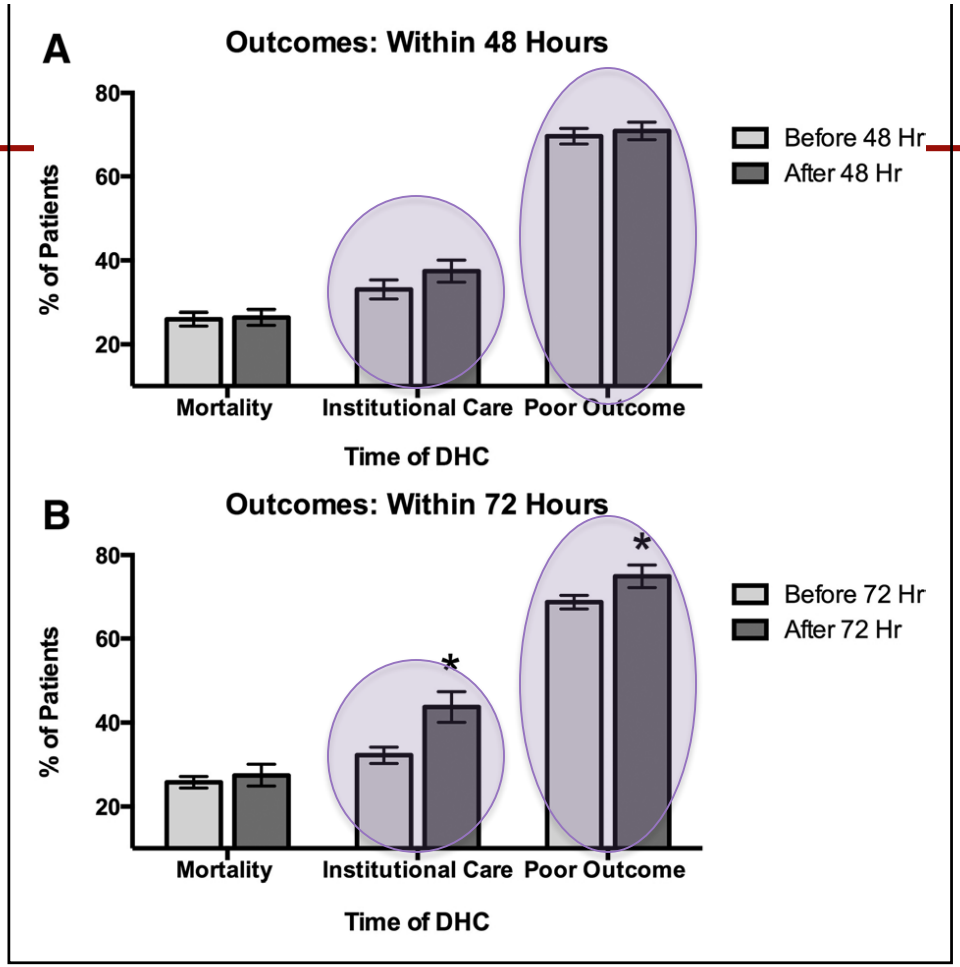


Figure. Differences in the crude rates (and SE) in mortality, discharge to institutional care, and of a poor outcome stratified by the timing of surgery: within 48 hours (**A**) and within 72 hours (**B**). A poor outcome was defined using the Nationwide Inpatient Sample-subarachnoid hemorrhage outcome measure (death, tracheostomy and gastrostomy placement, or discharge to institutional care). Statistically significant differences from multivariable logistic regression are designated with an asterisk. DHC indicates decompressive hemicraniectomy.

Dasenbrock et al., 2017

Age: Greater than 60 years old????

Conclusions:

Hemicraniectomy increased survival without severe disability among patients 61 years of age or older with a malignant middle cerebral artery infarction.

The majority of survivors required assistance with most bodily needs.

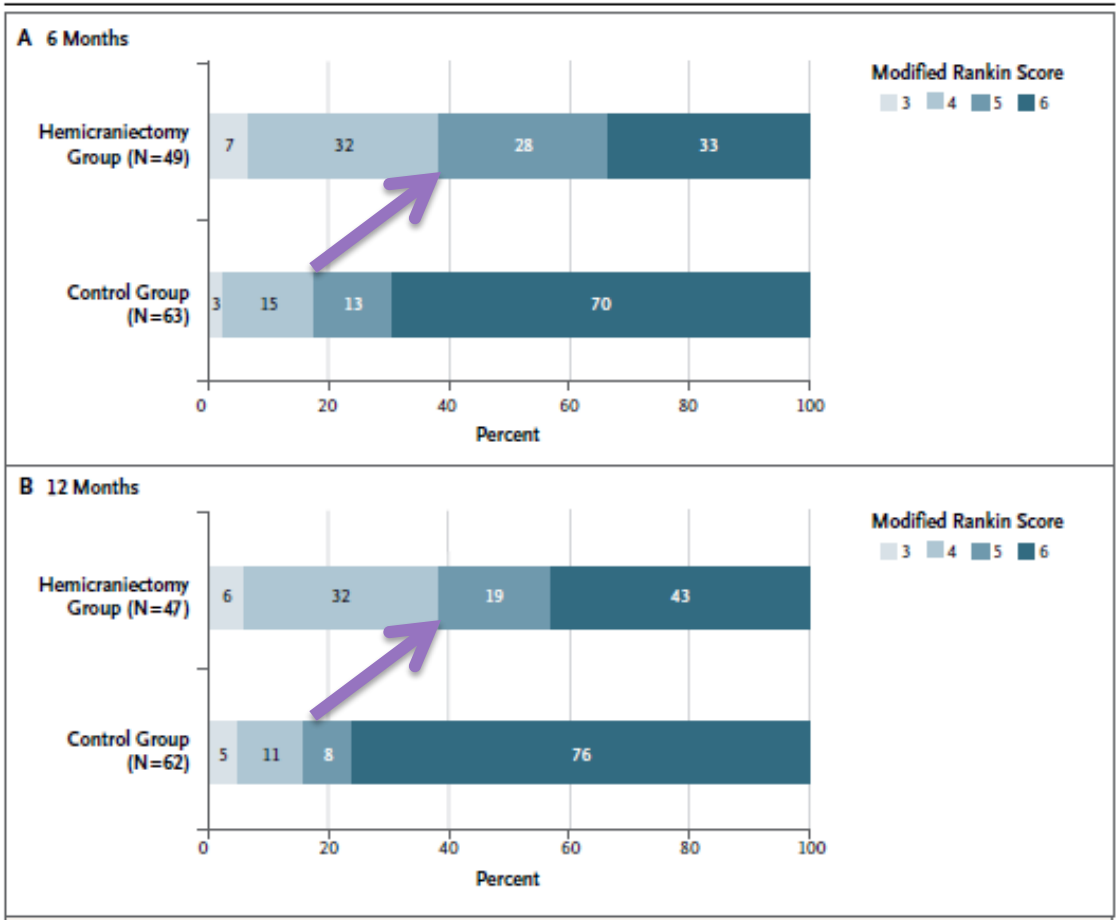


Figure 1. **Functional Outcome after Hemicraniectomy and after Conservative Treatment Alone According to the Modified Rankin Score.**

The primary end point was survival without severe disability, defined as a score of 0 to 4 on the modified Rankin scale (range, 0 to 6, with 0 indicating no symptoms and 6 indicating death). The results shown are the probability estimates for all patients who underwent randomization (the intention-to-treat population). Panel A shows the bias-corrected distribution of scores on the modified Rankin scale at 6 months. Panel B shows the raw distribution of Rankin scores at 12 months.

Jutter et al., 2014

Quality of Life Outcomes

Health state preferences and decision-making after malignant middle cerebral artery infarctions

Adam G. Kelly and Robert G. Holloway

Neurology 2010;75;682; Published online before print July 14, 2010;

Overall analysis found that DH had more quality adjusted life-years compared to medical therapy alone

Quality of Life Following Hemicraniectomy for Malignant MCA Territory Infarction

Alexander G. Weil, Ralph Rahme, Robert Moumdiian. Alain Bouthillier. Michel W. Bojanowski

Can. J. Neurol. Sci. 2011; 38: 434-438

Despite moderate to severe disability including dominate hemisphere strokes, 7/8 patients had no regret for completing DH

Cognition after malignant media infarction and decompressive hemicraniectomy - a retrospective observational study

Holger Schmidt[1,2*], Trutz Heinemann[3], Judith Elster[1], Marija Djukic[4], Stefan Harscher[5,6], Katja Neubieser[1], Hilmar Prange[1], Andreas Kastrup[7] and Veit Rohde[8]

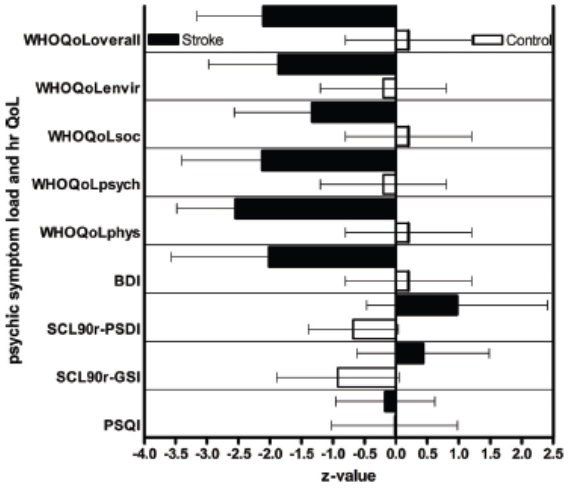


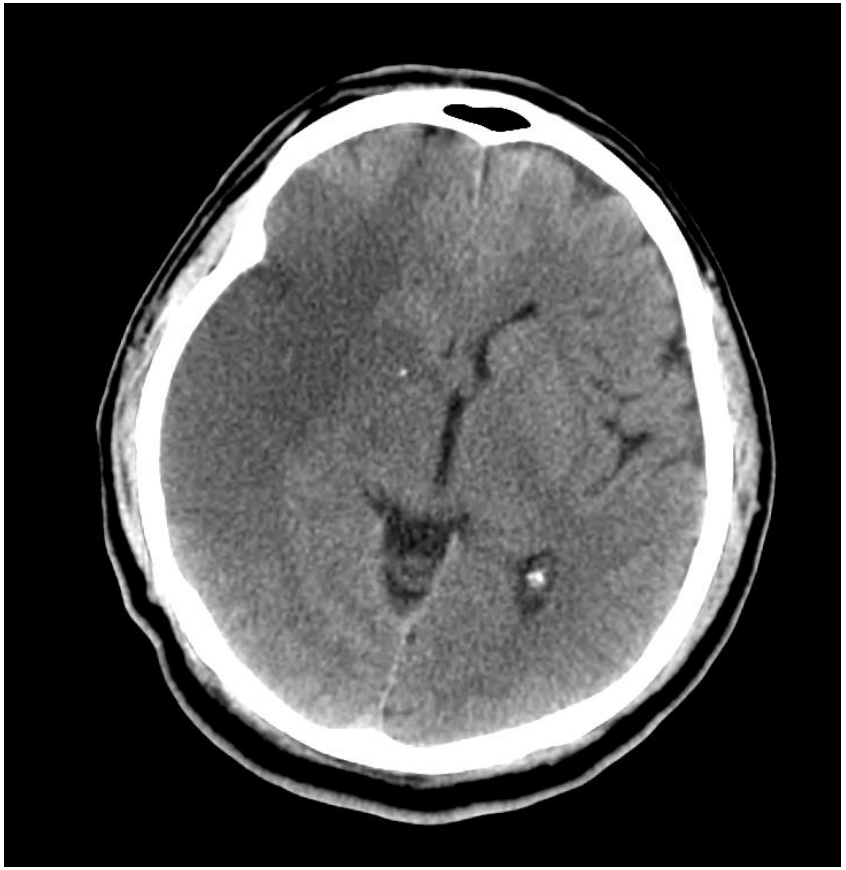
Figure 2 Domain mean ± SD z values of quality of life, sleep quality and mental symptom load (all domains of the patients group are significantly different from the control group).

So…. What does this look like in practice?

Case Study

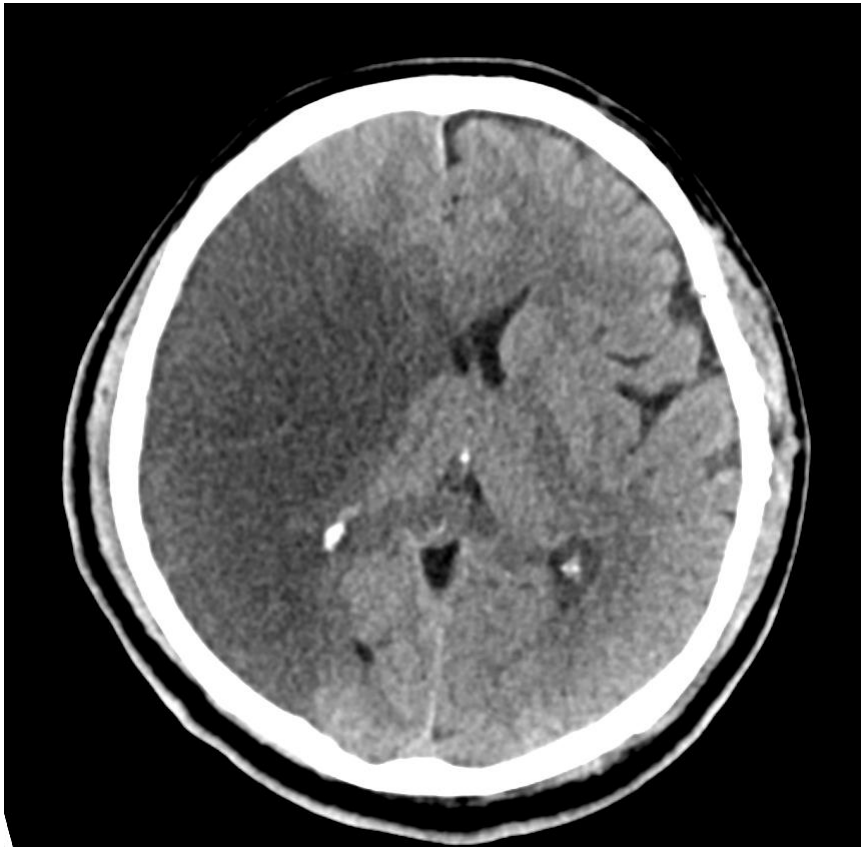
Case Study - OSH Presentation

- 59 year old male with history of Afib (on AC), HTN, BPH, GERD presenting to outside hospital with right MCA syndrome 16 hours from LKN. Transferred for higher level of care.
  - Likely stroke etiology: Cardio embolic (History of Afib, with non-compliance on Xarelto, related to recent PNA)



OSH – Non-Contrast Head CT
12/21 @ 6:36pm

Case Study - Arrival

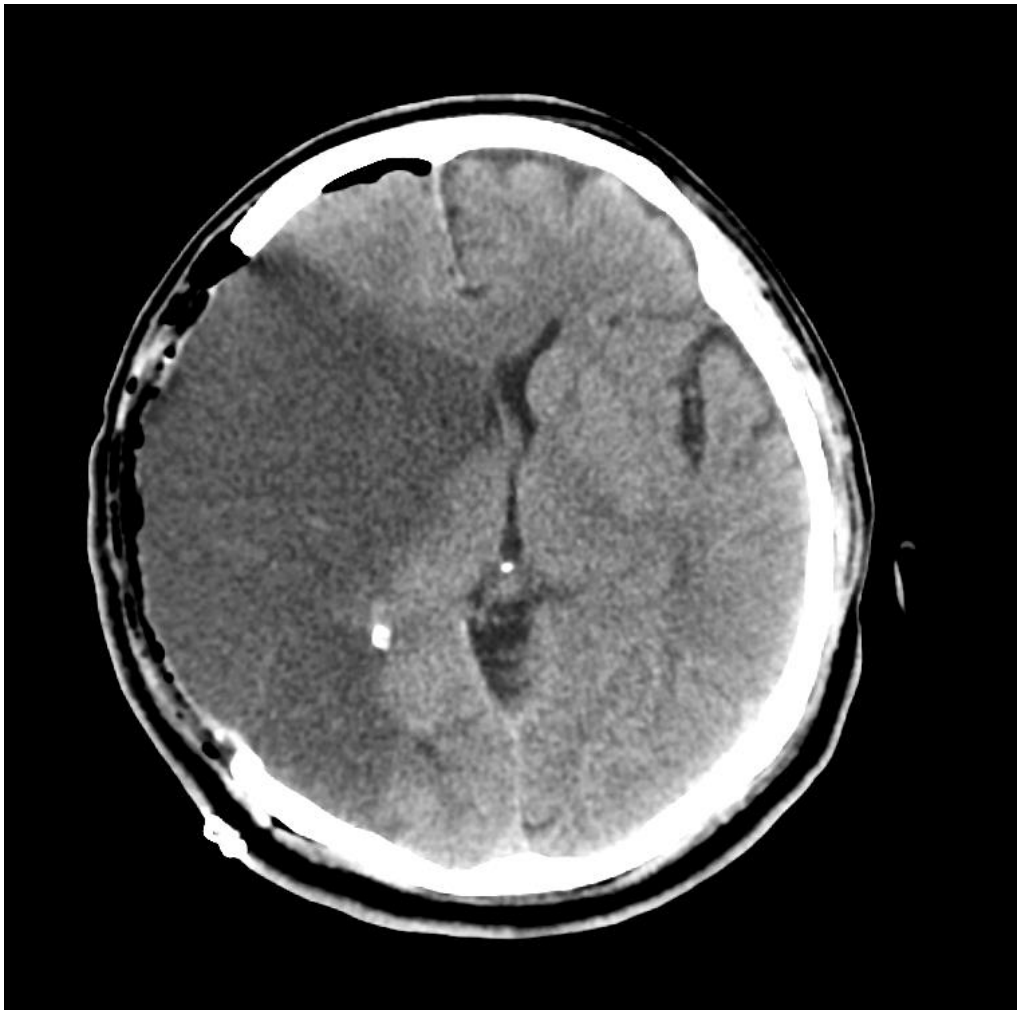


CT angiogram head and neck with and without contrast: 12/23/2016 2:38 am

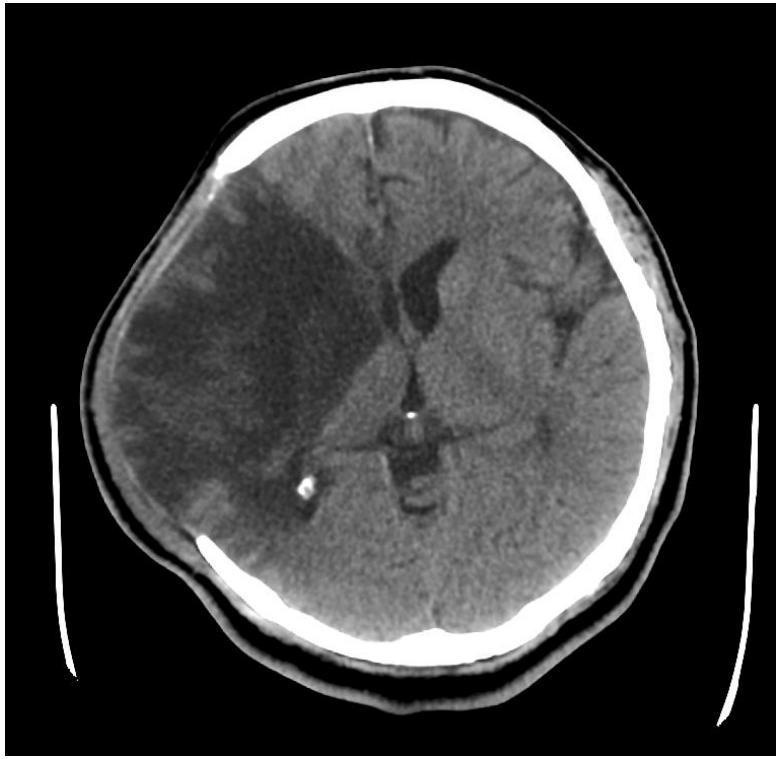
- Arrived at 12:30am
- NIHSS at arrival: NIH of 17 (1 right gaze, 2 left VF, 2 left face, 4 left arm, 4 left leg, 2 sensory, 1 dysarthria, 1 neglect)
- Initial plan: 23% boluses q4h for goal Na of > 150 with q4 hour monitoring, OR in AM
- Around 3:30a exam deteriorated with and was taken for decompression ~ 20-24 hours post stroke

Post Op

CT head without contrast : 12/23/2016 11:17 am



Follow up



CT head with and without contrast: 1/30/2017 7:16 pm

Stroke Clinic:

- 5 months post stroke – living at SNF with bi-weekly rehab, will soon be moving in with son
- Full PO diet, not using PEG
- NIHSS: 11 (1 left VF, 1 left face, 4 left arm, 3 left leg, 1 sensory, 1 dysarthria)

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THANK YOU!