Urgent decompressive craniectomy – How to best care for your patient

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Disclosures

None

Learning Objectives

- Illustrate the varied mechanisms leading to intracranial hypertension (IH)
- Review circumstances requiring urgent decompressive craniectomy and perioperative management of the patient
- Address learned falsehoods regarding anesthetic agents and their impact on intracranial pressure and brain relaxation

Summary of IH mechanisms

- Venous obstruction – Sinus or jugular veins
- Increased brain volume - tumor
- Increased blood volume – AVM, hypercapnia
- Mass effect – Sub/epidural hematoma, empyema
- Cerebral Edema
  - Cytotoxic – ischemic stroke
  - Vasogenic – tumor, encephalopathy
  - Transependymal - SAH
  - Osmotic - DKA

Case #1

- 24 yo primigravid female 29 weeks gestation presents with headache, hypertension and proteinuria
  - Dx – pre-eclampsia
- Within the next 24 hours she developed:
  - Seizures
  - Left-sided hemiparesis

Choy et al 2023

What is in your DDx?

- Migraine
- Infection
- Inflammatory condition (MS, PML)
- Seizure
- Concussion/TBI
- Tumor
- Stroke
  - Ischemic
  - Hemorrhagic
  - Cerebral Venous Sinus Thrombosis

What imaging modality is MOST appropriate?

A) CT angiogram
B) CT venogram
C) MR angiogram
D) MR venogram

Case #1 continued

- CT venogram demonstrated a superior sagittal sinus thrombosis and right frontal hematoma.
  - Nifedipine, labetolol and IV heparin were started.
- 12 hours later, GCS decreased to 10 with sluggish pupils and repeat CT showed right frontal hematoma enlargement with midline shift
- Taken to OR for decompressive craniotomy and cesarean section
- 1 week post-operatively, left hemiparesis remained severe
- 4 months post-operatively, her left leg showed 4/5 strength

Choy et al 2023

Intracranial Hypertension Signs and Symptoms

- Headache – throbbing or bursting in nature
- Nausea and vomiting – refractory to medications
- Diplopia – CNVI palsy
- Decreased level of consciousness – correlated to degree of midline shift
- Papilledema – can be delayed
- Pupillary dilation – CNIII palsy
- Downward deviation of the eyes – sunset eyes from brainstem dysfunction
- Cushing's triad – late and ominous sign of herniation
  - Severe HTN
  - Bradycardia
  - Irregular respiration

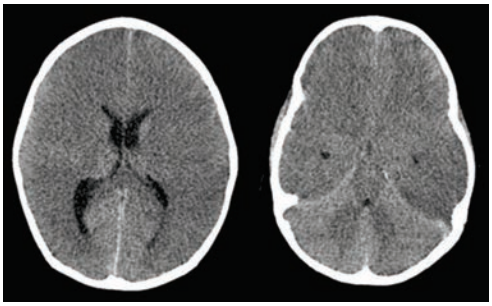
Midline shift

- Greater than 5mm an indication for surgery
- Automated measurements from CT scan
- Three locations assessed
  - Septum pellucidum
  - Third ventricle
  - Pineal gland

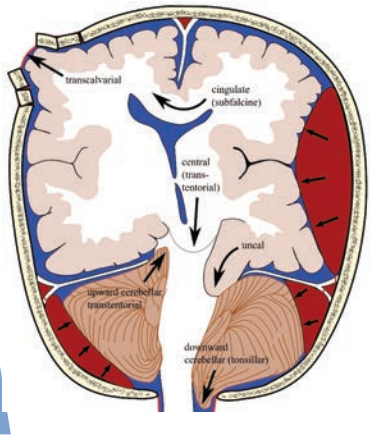
Case #2

- 4 yo male was admitted for fever, difficulty feeding, and impaired walking. Exam revealed meningeal signs and CSF analysis supported a diagnosis of meningitis
- On HOD#3, he became drowsy and less responsive then had onset of focal seizures.
- One day later, he had sudden tachycardia (150 bpm) and hypertension (170/100) with non-reactive pupils
- CT scan showed diffuse cerebral edema and uncal herniation

Monteiro et al 2023

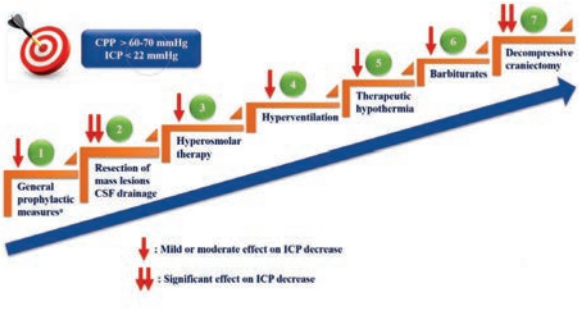


Global effacement
Loss of gray/white matter differentiation



Which of the following is the MOST effective means to decrease ICP in a closed cranium?

A) Administer hyperosmolar therapy
B) Drain cerebrospinal fluid
C) Initiate hyperventilation
D) Administer propofol/thiopental



Schizodimos et al 2020

Prophylactic measures

- Intubation and mechanical ventilation
- Avoid hypoxemia and hypercapnia
- BP-CPP optimization - **benefit**
- HOB to 30 degrees
- Patient's head face midline
- Fever control - **benefit**
- Glycemic control - **benefit**
- Seizure prophylaxis

Medical management of ICH

- Hyperosmolar therapy
- Hyperventilation
- Hypothermia
- Barbiturates/Propofol

Hyperosmolar therapy

- Mannitol
- Hypertonic saline (HTS)
  - Varied concentrations

Hyperosmolar Therapy Comparison

	Sodium Concentration (mEq/L)	Osmolarity (mOsm/L)	Typical Initial Bolus Dose
Mannitol 20%	n/a	1,098	0.5-1.5 g/kg*
Mannitol 25%	n/a	1,375	0.5-1.5 g/kg*
0.9% Sodium Chloride	154	308	n/a
3% Sodium Chloride	513	1,026	150–250 mL
23.4% Sodium Chloride	4,004	8,008	30 mL

Source: Adapted from References 3, 9.
Note: IV bolus doses may differ based on patient presentation and weight.
** Dose based on actual body weight.*

Peters et al, 2018

More On Hyperosmolar Therapy

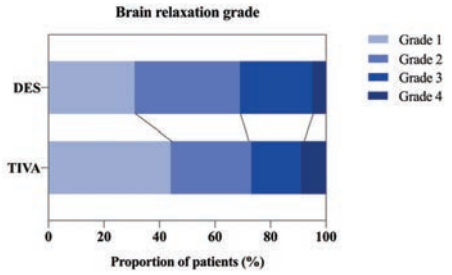
- Both mannitol and HTS
  - Draw brain interstitial fluid into the vasculature and result in pial arteriolar constriction
  - No clear differences on mortality/morbidity
- Mannitol
  - 20-60 minutes for peak effect lasting 4-6 hours
  - Adverse effects: hypotension, AKI
- HTS
  - Rapid onset, as quick as 5 minutes lasting up to 12 hours
  - Adverse effects: hypernatremia, hypokalemia

Brain relaxation

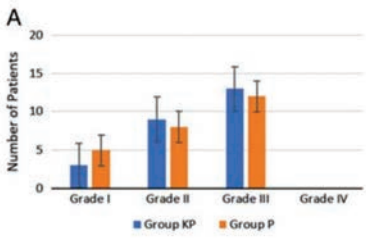
- Relationship between volume of intracranial contents and capacity of intracranial space
- Operating conditions
- Retraction injury
- Focal cerebral ischemia
- Subjective assessment during surgery
- Four predictors of poor intraoperative relaxation (Rasmussen et al 2004)
  - Glioblastoma multiforme
  - Brain metastatic disease
  - Midline shift of preoperative imaging
  - Elevated subdural pressure (closed dura, objective measure)

Administration of which of the following is MOST likely to prevent adequate brain relaxation?

A) Volatile anesthetic
B) Ketamine
C) Propofol
D) Nitroprusside



Jiang et al 2023



Maheswari et al 2023

Final Thoughts

- Decompressive craniectomy is most often a last resort for refractory IH, regardless of mechanism
- Unfortunately, mortality or morbidity benefits are not observed after decompressive craniectomy in most instances
- Our anesthetic care should focus on optimization of cerebral perfusion pressure, temperature control, and glycemic control

Anesthetic Management of the Patient with Traumatic Brain Injury

Benjamin K. Scott, MD
Associate Professor of Anesthesiology and Critical Care
February 28th, 2024

Disclosures

None

Learning Objectives

- Understand the impact of Traumatic Brain Injury
- Understand the pathophysiology of traumatic brain injury (TBI) in the perioperative period
- Review current guidelines and the latest evidence regarding physiologic, surgical, and intensive care management of TBI
- Apply these concepts to the anesthetic management of patients with acute, subacute, and chronic traumatic brain injury

The good news...

Patients can recover from moderate to severe TBI

TRACK-TBI McCrea et al. observed a cohort of 484 patients with mod-severe TBI for one year

Findings
- By 12 months post injury, ~ 50% of patients with severe TBI and 75% with moderate TBI recovered the ability to function independently at home for at least 8 hours per day.
- Among participants in a vegetative state at 2 weeks, 77% recovered consciousness and 25% regained orientation by 12 months.

Meaning
- Acute severe impairment did not universally portend poor functional outcomes
- Clinicians should refrain from making early, definitive prognostic generalizations

McCrea MA, Giacino JT, Barber J, et al. Functional Outcomes Over the First Year After Moderate to Severe Traumatic Brain Injury in the Prospective, Longitudinal TRACK-TBI Study. *JAMA Neurol.* 2021;78(8):982–992.

Disease trajectories may be modifiable

Question TBI is a heterogeneous entity. Are there clinical signals that might predict and thereby eventually improve outcomes?

Findings In a prospective observational study using cluster analsysis, Akerlund et al. observed 4509 patients from initial injury, through 7 days of ICU treatment

- Explores the relationship between primary injury, clinical variables, biomarkers and Glasgow Outcome Scores
- Strongest signals associated with glucose variability and elevated biomarkers

Meaning Identifying illness trajectories may allow us to identify illness phenotypes and improve outcomes

Åkerlund CAI, et al.; CENTER-TBI participants and investigators. Clinical descriptors of disease trajectories in patients with traumatic brain injury in the intensive care unit (CENTER-TBI): a multicentre observational cohort study. Lancet Neurol. 2024 Jan;23(1):71-80.

The bad news…

TBI is a chronic disease that increases the risk of long-term neurocognitive disorders

- **Functional outcomes after TBI can improve or deteriorate even many years after the initial injury**
  - Emotional, cognitive, behavioral changes
  - Functional Limitations
  - Increased risk of premature death
- **There is an emerging consensus that TBI increased post-injury risk of neurogenerative conditions**
  - Dementia
  - Parkinson's
  - ? ALS

Wilson L, et al.; The chronic and evolving neurological consequences of traumatic brain injury. Lancet Neurol. 2017 Oct;16(10):813-825.

Extracranial surgery is associated with worse outcomes in TBI patients

Question Do patients with TBI undergoing extracranial surgery have increased risk of poor outcome? Using the TRACK-TBI database, Christopher et al. compared 486 patients with TBI who were exposed to extra-cranial surgery and anesthesia with 1349 who were not

Findings Patients undergoing EC surgery had significantly worse Glasgow Outcome Scores at 2 and 6 months as well as Trail Making Test performance versus patients who did not

Meaning We do not know if this is relationship is causal, but protecting patients with TBI who require other surgical interventions should be a clinical and research focus

Roberts CJ et al.; Clinical Outcomes After Traumatic Brain Injury and Exposure to Extracranial Surgery: A TRACK-TBI Study. JAMA Surg. 2023 Dec 13:e236374.

Implications for informed consent and surgical planning…

- Traumatic brain injury should be considered (for now) a non-modifiable risk factor for post-operative neurocognitive disorders in patients undergoing extra-cranial surgery in the acute phase
- Although many extracranial surgeries in this population are likely also traumatic and may be urgent, consideration should be given to postponing or delaying elective procedures during at least the acute phase of injury

Collins CR, Campbell A. Surgery, Anesthesia, and TBI Outcomes—Unraveling the Complex Interplay. *JAMA Surg.* Published online December 13, 2023.

So how to we modify risk and improve outcomes?

There is minimal evidence favoring one sedation regimen over another

- Russo et al, 2023
- Retrospective observational study of 262 patients across 14 Trauma Centers (EU, UK, Aus)
- Propofol most common sedative followed by midazolam
- No differences in 60 day morality

Russo G, et al.; TBI Collaborative Investigators. Early sedation in traumatic brain injury: a multicentre international observational study. Crit Care Resusc. 2023 Oct 16;24(4):319-329. doi: 10.51893/2022.4.OA2. PMID: 38047010; PMCID: PMC10692594.

Some data to support the use of dexmedetomidine

- Liu et al, retrospective observational design (Premier Database)
- 19,751 Patients with mod-severe TBI requiring MV within 2 days of ICU admission
- After propensity matching, early exposure to dexmedetomidine (within 2d) associated with: lower hospital mortality, increased likelihood of liberation from MV, and reduced LOS
- No increased risk of hemodialysis, vasopressor exposure or hospital resource utilization

Liu SY, et al.; Association of Early Dexmedetomidine Utilization With Clinical Outcomes After Moderate-Severe Traumatic Brain Injury: A Retrospective Cohort Study. Anesth Analg. 2024 Feb 9. doi: 10.1213/ANE.0000000000006869. Epub ahead of print. PMID: 38335145.

Regional analgesia as an adjunct is safe, but does not appear to impact outcomes

- Manzanera et al, RCT
- 76 patients with polytrauma requiring MV, included a TBI group
- Randomized to IV analgesia versus continuous regional analgesia (CRA)
- No difference in sedation requirements/opioid use
- CRA was not associated with any increased risk

Manzanera J, et al.; Continuous peripheral nerve blocks for analgesia of ventilated critically ill patients with multiple trauma: a prospective randomized study. Anaesth Crit Care Pain Med. 2023 Apr;42(2):101183. doi: 10.1016/j.accpm.2022.101183. Epub 2022 Dec 8. PMID: 36496124.

There is no data to support the superiority of any one anesthesia technique over another when it comes to neurocognitive outcomes

- Many of these studies have compared general anesthesia with epidural anesthesia, focusing on the potential for hypotension
- Few studies measure/report thorough pre- and post-operative neurocognitive testing
- Trauma populations are often younger, and have fewer medical comorbidities which may benefit from more specific study

Collins CR, Campbell A. Surgery, Anesthesia, and TBI Outcomes—Unraveling the Complex Interplay. *JAMA Surg.* Published online December 13, 2023.

All that we know definitively summarized in one slide!

All that we know definitively summarized in one slide!

LEVEL I
The use of steroids is not recommended for improving outcome or reducing ICP. In patients with severe TBI, high-dose methylprednisolone was associated with increased mortality and is contraindicated.

Carney N, et al.; Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery. 2017 Jan 1;80(1):6-15.

Brain Trauma Foundation Guidelines 4th Edition, 2016

- Reviewed 189 publications using an evidence grading approach
- 18 Domains Considered
- In only one of these 18 was there enough high-quality evidence to warrant a Level 1 Recommendation

Carney N, et al.; Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery. 2017 Jan 1;80(1):6-15.

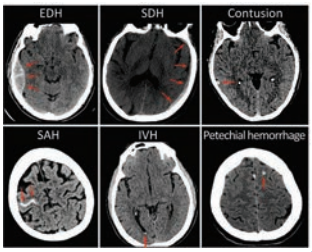
Brain Trauma Foundation Guidelines 4th Edition, 2016

- The foundation has since moved to a "living guideline" format without regularly planned updates
- The only significant formal update came in 2020 and addressed the role of decompressive craniotomy

Carney N, et al.; Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery. 2017 Jan 1;80(1):6-15.

Pathophysiology of TBI: Primary Injury

- Compression
- Edema
- Hypoxia/Ischemia
- Metabolic impairment
- Shearing forces
- Cellular destruction



Credit: Manley Lab/UCSF

Yuh EL et al., Pathological computed tomography features associated with adverse outcomes after mild traumatic brain injury, JAMA Neurology, July 19, 2021.

Pathophysiology of TBI: Secondary Injury

Macro

- Intracranial hypertension
- Hydrocephalus and CSF Dynamics
- Ischemia and malperfusion
- Edema and hyperemia
- Infection

Micro

- Glutamate toxicity
- BBB disruption
- Neuronal dysregulation/loss
- Mitochondrial dysfunction
- Complement activation/inflammation
- Oxidative Stress

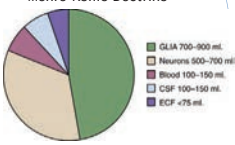
Intracranial Pressure

ICP derived from CBF and CSF circulation

- $ICP = ICP_{vasc} + ICP_{CSF}$

- CSF Component
  - ICP_{CSF} = (CSF formation rate x R_{CSF}) + sagittal sinus pressure
  - Avg CSF formation rate = 0.3-0.4 ml/min (approx 500ml/day)
  - Normal CSF resistance = 6-10 mmHG
  - Normal Sagittal Sinus pressure = 5-8 mmHG
- CBF Component
  - Pulsatile fraction of CBF
  - Magnitude depends on vascular tone, heart rate, autoregulation, etc.

Monro-Kellie Doctrine

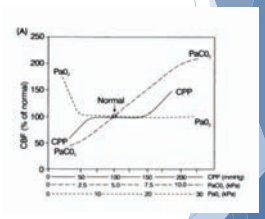


Cerebral Blood Flow

- 15% of cardiac output
- Approx 50 ml blood per 100g brain tissue per minute
- Ischemic threshold < 18-20 mL/100g per minute
- CBF = CPP/cerebral vascular resistance
- CVR depends on
  - Metabolic factors (CMRO2 associated changes)
  - Viscosity
  - Autoregulatory vascular tone (mechanism unclear)
  - PaCO2 and PaO2
  - Neural feedback (cholinergic, adrenergic, serotonergic, VIP-ergic)
  - Calcium homeostasis

Cerebral Blood flow and CO2

- Exponential relationship between CBF and PaCO2 from 25-65 (4% per mm HG)
- Minimal effect over 70 or less than 20 mm HG
- Flow changes occur within 2 minutes of a change in PaCO2 and plateau at about 12 minutes
- Mediated by
  - Peri-vascular pH around smooth muscle
  - Neurogenic reflexes and catecholamine concentrations
  - Endothelium derived relaxing factor
- By 36 hours, adaptation will return blood flow to baseline



Cerebral Perfusion Pressure

- Driving pressure across cerebrovascular bed
- Cerebral arterial pressure – venous pressure at the bridging veins
- Pressure in bridging veins difficult to measure, can be approximated by ICP

$$CPP = MAP - ICP_{mean}$$

Implications of CPP abnormality

- Too low = ischemia
- Too high = hyperemia
- Lower CPP has been associated with worse outcomes in TBI
- Optimal CPP remains somewhat debated: Lund Hypothesis versus Anglo-American Consensus

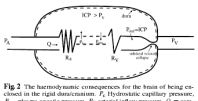


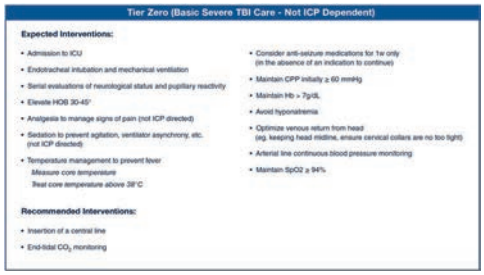
Fig 2 The haemodynamic consequences for the brain of being enclosed in the rigid duracranium. P_c Hydrostatic capillary pressure, P_{coh} plasma oncotic pressure, P_A arterial inflow pressure, Q = cerebral blood flow, R_A arterial resistance, R_V venular resistance, P_{ICP} pressure retrograde to the venous collapse, P_V venous outflow pressure

Per Olaf Grande, Intensive Care Med 2006 (32):1475

Our goal is to protect our patients from avoidable secondary injury…

By preserving normal physiology.

Seattle International Severe TBI Consensus Conference (SIBICC)



Tier Zero (Basic Severe TBI Care - Not ICP Dependent)

Expected Interventions:

- Admission to ICU
- Endotracheal intubation and mechanical ventilation
- Serial evaluations of neurological status and pupillary reactivity
- Elevate HOB 30-45°
- Analgesia to manage signs of pain (not ICP directed)
- Sedation to prevent agitation, ventilator asynchrony, etc. (not ICP directed)
- Temperature management to prevent fever
  - Measure core temperature
  - Treat core temperature above 38°C
- Consider anti-seizure medications for 1w only (in the absence of an indication to continue)
- Maintain CPP initially ≥ 60 mmHg
- Maintain Hb > 7g/dL
- Avoid hyponatremia
- Optimize venous return from head (eg. keeping head midline, ensure cervical collars are no too tight)
- Arterial line continuous blood pressure monitoring
- Maintain SpO2 ≥ 94%

Recommended Interventions:

- Insertion of a central line
- End-tidal CO_2 monitoring

Hawryluk GWJ, et al. A management algorithm for patients with intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC). Intensive Care Med. 2019 Dec;45(12):1783-1794.

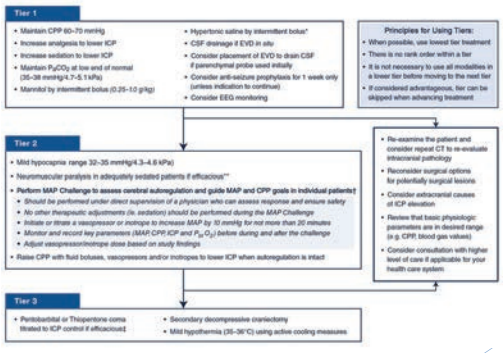
Treatment NOT RECOMMENDED by SIBICC When Only ICP is Monitored:

Mannitol by non-bolus continuous intravenous infusion
Scheduled infusion of hyperosmolar therapy (e.g., every 4–6 h)
Lumbar CSF drainage
Furosemide
Routine use of steroids
Routine use of therapeutic hypothermia to temperatures below 35 °C due to systemic complications
High-dose propofol to attempt burst suppression
Routinely decreasing P_aCO_2 below 30 mmHg/4.0 kPa
Routinely raising CPP above 90 mmHg

CPP **cerebral perfusion pressure,** ***ICP*** **intracranial pressure,** ***kPa*** **kiloPascals,** P_aCO_2 **arterial partial pressure of carbon dioxide**

Hawryluk GWJ, et al. A management algorithm for patients with intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC). Intensive Care Med. 2019 Dec;45(12):1783-1794.

SIBICC Algorithm for ICP-based Management



Tier 1

- Maintain CPP 60–70 mmHg
- Increase analgesia to lower ICP
- Increase sedation to lower ICP
- Maintain P_aCO_2 at low end of normal (30–38 mmHg/4.7–5.1 kPa)
- Mannitol by intermittent bolus (0.25–1.0 g/kg)
- Hypertonic saline by intermittent bolus*
- CSF drainage if EVD in situ
- Consider placement of EVD to drain CSF if parenchymal probe used initially
- Consider anti-seizure prophylaxis for 1 week only (unless indication to continue)
- Consider EEG monitoring

Principles for Using Tiers:

- When possible, use lowest tier treatment
- There is no rank order within a tier
- It is not necessary to use all modalities in a lower tier before moving to the next tier
- If considered advantageous, tier can be skipped when advancing treatment

Tier 2

- Mild hypocapnia range 32–35 mmHg/4.3–4.6 kPa)
- Neuromuscular paralysis in adequately sedated patients if efficacious**
- **Perform MAP Challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients†**
  - *Should be performed under direct supervision of a physician who can assess response and ensure safety*
  - *No other therapeutic adjustments (ie. sedation) should be performed during the MAP Challenge*
  - *Initiate or titrate a vasopressor or inotrope to increase MAP by 10 mmHg for not more than 20 minutes*
  - *Monitor and record key parameters (MAP, CPP, ICP and P_aO_2) before during and after the challenge*
  - *Adjust vasopressor/inotrope dose based on study findings*
- Raise CPP with fluid boluses, vasopressors and/or inotropes to lower ICP when autoregulation is intact
- Re-examine the patient and consider repeat CT to re-evaluate intracranial pathology
- Reconsider surgical options for potentially surgical lesions
- Consider extracranial causes of ICP elevation
- Review that basic physiologic parameters are in desired range (e.g. CPP, blood gas values)
- Consider consultation with higher level of care if applicable for your health care system

Tier 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious‡
- Secondary decompressive craniectomy
- Mild hypothermia (35–36°C) using active cooling measures

Hawryluk GWJ, et al. A management algorithm for patients with intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC). Intensive Care Med. 2019 Dec;45(12):1783-1794.

Intraoperative Management

- Transport and positioning
  - **Clamp EVD** for transport and during positioning
  - **Elevate HOB** and avoid tight c-collars, constrictive trach ties, etc.
- Access
  - Low threshold for **arterial line** and strict SBP 90-140, MAP>65
  - Maintain **2 reliable peripheral IVs** (quality>quantity unless resuscitating)
  - Subclavian access is preferred if necessary but risk of IJ impacting venous drainage is acceptable and should be considered when central line is indicated

Intraoperative Management for Acute TBI

- **RSI for airway protection**
  - Etomidate reasonable for induction when hypotension is a concern
  - Ketamine is probably a safe choice and may be a good choice
  - Propofol for normo to hypertensive patients
- **Mechanical Ventilation**: Lung protective vent strategy
  - Target **low normocapnia**
  - Hyperventilation may be considered as a temporizing measure, avoid PaCO<25
  - Maintain adequate oxygenation (SpO2 > 94, PaO2> 65)
  - Physiologic PEEP is OK
  - Elevated Peep Should be Considered if necessary for oxygenation

Intraoperative Management

Transfusion and Coagulation Management

- Transfuse to keep hgb>7
- TEG-based coagulopathy monitoring in acute trauma
- Treat thrombocytopenia in conjunction with neurosurgery threshold
- In subacute stages, SQH is safe in most cases after 24h of stable brain imaging
- **TXA appears safe, may improve outcomes** (CRASH-3, Yokoburi et al. 2022)

CRASH-3 trial collaborators. Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH-3): a randomised, placebo-controlled trial. Lancet. 2019 Nov 9;394(10210):1713-1723. doi: 10.1016/S0140-6736(19)32233-0. Epub 2019 Oct 14. Erratum in: Lancet. 2019 Nov

Yokobori, S., *et al.* Efficacy and safety of tranexamic acid administration in traumatic brain injury patients: a systematic review and meta-analysis. *J intensive care* **8**, 46 (2020). https://doi.org/10.1186/s40560-020-00460-5

Intraoperative Management

- Prophylactic AED (Keppra or phenytoin)
- ABX PPX per local protocol
- Mannitol/HTS in collaboration with neurosurgeon, avoid Lasix in acute severe TBI
- Consensus has progressed toward **goal euvolemia**
- But avoid fluid overload!
- Choice of fluid:
  - Consider avoiding albumin based on worse outcomes in TBI subgroup analysis of the SAFE trial (N Engl J Med 2007; 357:874-884)
  - Consider balanced salt solution (esp PlasmaLyte or Normosol) due to concern regarding risks of large volume normal saline exposure

Intraoperative Goals for any TBI:

- Euvolemia
- Enforced Normothermia
- Low Normocarbia
- Normal oxygenation
- Avoid severe anemia
- Euglycemia
- Minimize metabolic demand (Rx pain, agitation, vent dys-synchrony, seizure, shivering)

Questions?

Thank You!

CRASH
Guiding the future of patient care

Diagnosis and Management of Spinal Epidural Abscess

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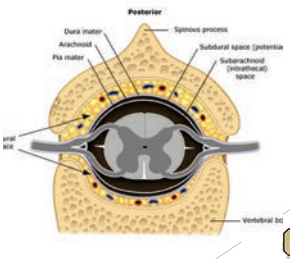
Anesthesiology
UNIVERSITY OF COLORADO
ANSCHUTZ MEDICAL CAMPUS

Learning Objectives

- Review the types of spine infections
- Understand the pathology of spinal epidural infections (SEI)
- Examine the indications for urgent spine surgery
- Discuss the anesthetic management of SEI in the OR

Types of Spine Infections

- Vertebral osteomyelitis
- Disk space infection
- Canal space infection
- Adjacent soft-tissue infection



Pathogenesis of SEA

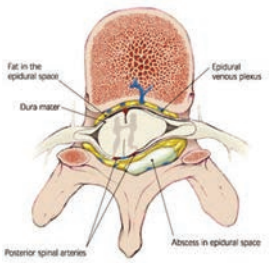
Source	Number of patients, percent
No source identified	30
Skin and soft tissue	22
Spinal surgery or procedures	12
Injection drug use	10
Other sources, including epidural catheters	8
Bone or joint	7
Urinary tract	3
Upper respiratory tract	3
Sepsis	2
Abdomen	2
Intravascular catheter associated	<1

Epidemiology

- Incidence
  - 5 cases per 10,000 admission
- Predisposing comorbidities
  - Bacteremia
  - Vertebral osteo
  - Immunosuppression
    - EtOH, HIV, DM

Clinical Manifestations

- Back pain
- Fever
- Neurologic deficits
  - Back pain, nerve root pain, motor/sensory loss, paralysis



Labs and Imaging

- WBC, ESR, CRP
- MRI vs CT
- Cultures

https://www.nature.com/articles/s41394-021-00437-y

DDx

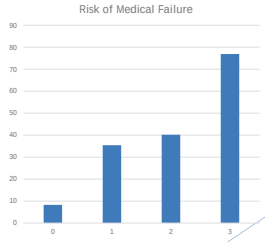
- Degenerative disk disease
- Tumor
- Vertebral discitis or osteomyelitis

Treatment

- Medical vs combined approach
- How to choose?
  - No risk factors for poor outcome
  - No neuro deficits or SEA resulted in complete SCI for >48 hr
  - Infecting organism is known
  - Medically unstable

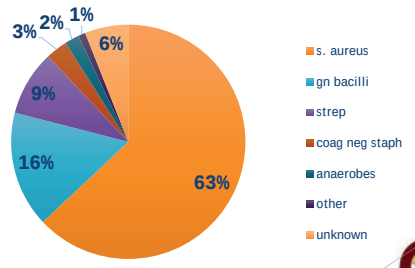
Predictive model

- Patient Risk Factors
  - Diabetes
  - WBC >12.5
  - CRP > 115
  - Positive blood cx



Patel et al., Spinal epidural abscesses: risk factors, medical versus surgical management, a retrospective review of 128 cases. Spine J. 2014

Microbiology

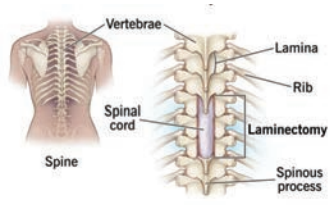


Sexton et al., Spinal Epidural Abscess, uptodate.com, 2024

Antibiotic Therapy

- Vancomycin and Ceftriaxone
- If cephalosporin intolerant; meropenem or moxifloxacin
- If vanco intolerant; linezolid, bactrim or daptomycin
- Plus/minus nafcillin or oxacillin for MSSA
- Switch to targeted therapy ASAP
- Four to eight weeks

Surgical Approach



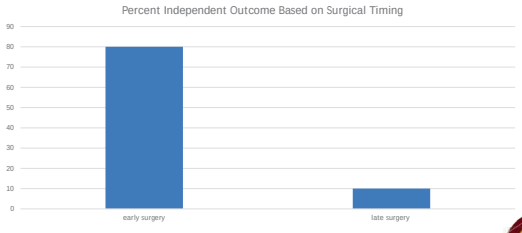
- Focal laminectomy
- Skip laminectomies
- Apical laminectomies
- Different approaches if VO or disk space infection or HWR needed

https://my.clevelandclinic.org/health/procedures/10895-laminectomy

Prognosis

- 5% mortality from SEA complications
- Irreversible paraplegia 4-22%
- Depends on timing of diagnosis and therapy

Prognosis



Liem et al., Thoracic epidural abscess. J Spinal Disord. 1994 Oct;7(5):449-54

Prognosis

Parameter	Patients with Diagnostic Delay (n = 47)	Patient without Diagnostic Delay (n = 16)
% of all patients	75	25
Multiple ED visits (%)	68	N/A
Admission delay (%)	66	N/A
Neurologic deterioration during "delay" (%)	57	N/A
"Classic triad" present at admission (%)	9	13
Residual weakness at discharge (%)	45	13

Davis et al. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med. 2004

Anesthetic Management

- Airway
- Induction
- Lines
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

Anesthetic Management

- Airway
- Induction
- Lines
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- Extubate?

- Myelopathic
- Possibly unstable
- C spine precautions prn

Anesthetic Management

- Airway
- **Induction**
- Lines
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- Distributive shock
  - Septic and/or neurogenic
- Acidotic
- Cardiogenic shock

Anesthetic Management

- Airway
- Induction
- **Lines**
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- 2 PIV
- Likely arterial line
- Possible central line
- Changes with positioning and response to induction

Anesthetic Management

- Airway
- Induction
- Lines
- **Positioning**
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- Prone or combined
- T10
- Pins vs prone view
- 0, 90 or 180

Anesthetic Management

- Airway
- Induction
- Lines
- Positioning
- **NM considerations**
- Analgesia
- Hemodynamics
- Extubate?

- TIVA vs HIVA
- Residual function?
- Guide for positioning and hemodynamic mgmt.
- How to reverse with sugammadex

Anesthetic Management

- Airway
- Induction
- Lines
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- PO multimodal
  - APAP, NSAID, neuropathic
- Methadone
- Hydromorphone
- Ketamine
- Precedex
- Lidocaine

Anesthetic Management

- Airway
- Induction
- Lines
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- SBP at baseline or above
- Raise MAP to 100 mmHg prn
- Neo vs norepi vs vaso
- Hg 8-10 in the OR
- 2:2:1 with RBC:FFP:CaCl

Anesthetic Management

- Airway
- Induction
- Lines
- Positioning
- NM considerations
- Analgesia
- Hemodynamics
- Extubate?

- Usual considerations plus…
- Neuro exam if extubated
- Empiric MAP pushes if intubated
  - May need central line

Summary

- SEA is becoming more common
- Good outcomes depend on prompt diagnosis and treatment
- Cases present unique challenges in terms of anesthesia management
- Expect to have serial washouts, wound vacs, increased pain requirements and exhausted patients

Thanks

- CU Neuroanesthesia Faculty
- Peter Witt, MD
- CJ Kleck, MD

References for further review

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- https://my.clevelandclinic.org/health/procedures/10895-laminectomy
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