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Technology: Inside the Black Box -- Managing Neuro Patients

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Technology: Inside the Black Box

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Disclosures

- Neuro Critical Care Society
  - Vice President/Board of Directors
- Honorarium
- Bard
- Scientific Advisory Board/Stock Options
  - Cerebrotech
  - Neuroptics
  - Ceribell

Objectives

- Identify the pathophysiology and cellular processes related to brain injury responses
- Describe the indications, functions, data analysis, and suggested interventions with various monitors of the brain
- Apply this technology to the care of the critically injured neuro patient using evidenced based protocols

4 Factors

- CBF
- Pressure
- Oxygen
- Metabolism

Physiology: Cerebral Blood Flow

- CBF = CPP / CVR
- Optimal CPP > 60 mm Hg and sometimes higher in TBI
- Normal CPP does not ensure CBF is adequate to meet the needs of the injured brain

If you put in the Gizmo....You have to know the Physiology too!
Physiology: Cerebral Blood Flow

- **Oxygen**
  - Low PaO2 creates vasodilation of cerebral blood vessels
  - High PaO2 (200-400 mm Hg) creates vasoconstriction

- **Carbon Dioxide**
  - Low PaCO2 creates vasoconstriction
  - High PaCO2 creates vasodilation

CBF: Indirect/Non-Invasive Transcranial Dopplers

- Non-invasive study using ultrasound to detect changes in the velocity of blood in the arteries of the brain

- **Arteries**
  - Extra-cranial ICA
  - Middle Cerebral Artery
  - Anterior Cerebral Artery
  - Posterior Cerebral Artery
  - Basilar/Vertebral Arteries

CBF: Invasive

- **Thermal diffusion probe**
  - Uses 2 thermisters 5mm apart/embedded on catheter
  - Heats distal thermistor to measure difference in temperature between 2 sites on catheter
  - Absolute flow measurements ml/100gm/min
  - Normal CBF: > 70 ml/100g/min

Myocardial Stunning: “tako tsubo”

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>PbtO2</th>
<th>CBF</th>
</tr>
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<tbody>
<tr>
<td>2</td>
<td>23</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>16.5</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>11</td>
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<td>8</td>
<td>9.5</td>
<td>9.5</td>
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<tr>
<td>10</td>
<td>8.3</td>
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<td>14</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>16</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

Impact of Nimodipine on PbtO2 and CBF

Pressure

Impact of Nimodipine on PbtO2 and CBF
Physiologic Changes: Intracranial Pressure

- Theories on Brain Compartment
  - 80% brain
  - 10% blood
  - 10% CSF
- If one increases the other two decrease
- Compensatory mechanisms

ICP Placement

- Procedure
  - Prep, cleanse scalp and drape
  - Incision right frontal made & twist drill used to gain access
  - Dura is opened with blunt stylet/irrigate
  - EVD catheter passed into the ventricle
  - Confirm CSF flow
  - Incision is closed with suture
  - CSF system connected to drainage system

ICP Systems: Types of transducers: Fluid vs Fiberoptic

ICP: Nursing Implications CSF Drainage

- Level CSF drainage system with zero reference point
- Pressure Transducer
  - Transducer position at phlebostatic axis (PMH: point of maximal impulse
    4th ICS mid chest)
  - For every inch (2.5 cm) the heart is offset from the reference of the transducer, a 2 mm Hg of error is introduced

Physiology: Intracranial Pressure

- Normal range
  - 0-15 mm Hg
- Abnormal ranges > 20 mm Hg

Cerebral Perfusion Pressure

MAP – ICP = CPP

Optimal CPP in TBI

2016: 60-70 mm Hg
Intracranial Pressure

Non Invasive ICP

Pupils: Assessing the “Beat” of the Brain

- Pupillary exam is vital to monitor potential increases in ICP
- High inter-examiner variability (up to 39%) and a severe lack of reliability is reported.

Inter-rater Reliability of Pupillary Assessments

Results: From 2329 paired assessments, the inter-rater reliability between practitioners was only moderate for pupil size (k = 0.54), shape (k = 0.62), and reactivity (k = 0.40). Only 33.3% of pupils scored as non-reactive by practitioners were scored as non-reactive by pupillometry.

Considerations

- Medications
  - Fentanyl affects pupillary reflex dilation
  - Morphine affects pupillary size/constriction velocity (bolus dose)
  - Symmetrical changes
  - Midazolam can affect constriction velocity but it is symmetrical
  - Paralytics no impact
  - Propofol slows but symmetrical
  - Barbiturates
    - Makes them big and non-reactive

SICU – New Admits with Neuro Diagnoses q 1h

- 49 yr old female admitted post op following clipping of a cerebral aneurysm. Pupillometer Assessment @2000 shows normal NPI and Constriction Velocity.

| Right Eye Pupil Reactivity | 4.5 |
| Left Eye Pupil Reactivity | 3.75 |
| Right Eye Min. Aperture | 3.25 mm |
| Left Eye Min. Aperture | 3.1 mm |
| Right Eye Percent Change | 30.9% |
| Left Eye Percent Change | 26.5% |
| Right Eye Constriction Velocity | 1.25 mm/sec |
| Left Eye Constriction Velocity | 0.86 mm/sec |
Careful reassessment by our Night Shift RN reveals the following:

- The NPI changed 1 hour before the pupil blew. MD was notified 3 times with the pupilometer changes!!! Patient went to CT and OR for emergent craniectomy.

**Oxygen**

- Regional Detection Penumbra Area
- Global Measurement Contralateral to Injury

**Physiology: Brain Tissue Oxygen (PbtO2)**

- Normal: 20-40 mm Hg
- Risk of death increases
  - < 15 mm Hg for 30 minutes
  - < 10 mm Hg for 10 minutes
- PbtO2 < 5 mm Hg
  - High mortality
- PbtO2 < 2 mm Hg - neuronal death

**BP and Oxygen in the Brain**

- Guiding MAP according to PbtO2
  - Hgb 6.9 – Tx with 2 u Packed RBCs 9-10am
**Brain Perfusion: BP and ECG Relationship**

- ICP 3-15 mm Hg
  - It is NOT a intracranial pressure problem!
- ECG rhythm changes...brady- tachy- pauses... Pacer wire accessed

**Focal Vasospasm**

- Vasospasm: Stat TCDs ordered: ↑ TCD velocity

**Cerebral Autoregulation**

- Autoregulation Prx
  - ICP and CPP Relationship
    - • Correlation (-1 to 0)
      - • As CPP increases, ICP decreases
      - • Indicates intact cerebrovascular reactivity
    - • + Correlation (>0 to 1)
      - • As CPP increases, so does ICP
      - • Indicates the loss of cerebrovascular reactivity
      - • Pressure passive dilatation

**Autoregulation**

- • Concept of Prx measurement as a dynamic assessment of cerebral autoregulation

- PRx show the U-shape relationship with mean CPP (200 patients).
  - This indicate that for low CPP and CPP above 90 mm Hg both autoregulation and pressure reactivity are defective.

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**Outcome and PRx opt during first 48 hours after SAH**

- [Graph showing outcomes and PRx optimisation during first 48 hours after SAH]

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**Slide credit:** NCS Multimodality Monitoring, Marek Czosnyka & Chad Miller
Metabolism

Microdialysis Technology

Microdialysis – Key Parameters

Electrophysiology and Seizures

Electrophysiology

- Monitoring for seizure activity
- Continuous EEG (detecting non-convulsive status)

Brain glucose is a very important marker due to the increasing evidence in showing lower glucose levels in seizure activity. Tight glucose control can be achieved through a combination of genuine treatment and early detection through monitoring.

Table 1: Data illustrates the values of metabolic parameters important in the management of patients with subarachnoid hemorrhage.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Max Post hemorrhage</th>
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<tbody>
<tr>
<td>Blood glucose</td>
<td>124 mg/dl</td>
<td>23 mg/dl</td>
</tr>
</tbody>
</table>

Heldb et al, Neurocrit Care, 2010;12(3):317-33
Non-convulsive Seizures (NCS) or Non-convulsive Status Epilepticus aka NCSE

- Prospective observational study/ Adult NICU
- Patients with altered mental status
- Data collected EEG
- 21% of patients (36 of 170) had NCS/NCSE

EEG with Ceribell

- Ceribell EEG
  - Disposable head band with pocket size EEG recorder
  - 10 channel EEG device that records EEG and transforms EEG waves into sounds

Spreading Depolarizations

Mission – Status Case

Mission – No Seizure Case
Putting it All Together
CASE EE

Mechanism: Pre-hospital

• 20 year old female driver single car into center divider wall of freeway
  • Car rolled then went airborne
  • GCS at scene 5
  • BP 104/p HR 80 R 28

Trauma Admit 1727

• GCS 1-2-1 with R pupil 4 sluggish and L pupil 4 deviated outward with no reaction
  • BP 139/75 HR 104 RR 9
• Intubated with 7.5 ET tube/OG placed/Foley
• Labs
  • H/H: 9.7/29 Plt 360,000
  • Na 137 K 3.0 Cl 105 CO2 20
  • PT 11.6 INR 1.1 PTT 28 sec Fibrinogen 327
• Radiology
  • Multiple facial fractures/skull fractures
  • Bilateral cerebral contusions, cerebral edema with shift

2045 Hypothermia Initiated

• ICP 70s and PbtO2 13.8
  • 2045 Iced saline 2 liters given
  • 2130 Arctic sun on
  • Mannitol given
• ICP 70s
  • ICP remains > 70 mmHg with PbtO2 low
  • To CT scan
  • Numerous calls to neurosurgery

CT Scan Admission 8:58pm
To OR for ICP/PbtO2

CT 9:58pm
ICP 73 mm Hg
OR Decompressive Craniectomy 2247

- 2316 Cut time
  - ICP in high 90s and PbtO2 5 mm Hg
- 2317 3% saline given
- 2330
  - Fluid bolus – 3 bottles albumin/Packed RBCs
- 2338 Temp 33 degrees
- 0008
  - Closure
  - Rapid infuser for blood products
  - 2 grams Ca Cl given

To SICU at 0055

- ICP 96 mm Hg on arrival
- Neurosurgeon discusses condition with parents
- Next 7 hours delicate balance...
  - ICP 90s down to 50 mm Hg by 7am
**Coma Emergence**

- GCS Improves during Days 13-21
- 7–9 - 10–11
- ICP/PbtO2 removed on Day 15
Coma Emergence

- SICU x 30 days
- Tx to Acute Rehab Unit
- ARU stay x 22 days
  - Wernicke's aphasia
  - Improves throughout ARU stay
- Cranioplasty Day 54
- Discharged home on Day 59 after TBI
- Home/Out pt Tx

Home Life

- Outpatient speech cognitive therapy for fluent dysphasia (Wernicke's aphasia) which is improving
- Walking - 10 weeks post
- ADLS on own

1 year
Back to UCI

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