Episode 02: Basic Neuroanatomy and Physiology Shownotes

Barash CH 37, Pg 1009

HY Anatomy Facts

- Brain Compartments
    - Supratentorium
        - Composed of cerebral hemispheres and dicenphalon (thalamus + hypothalamus)
            - Cerebral hemispheres
                - Four Lobes (Frontal / Temporal / Parietal / Occipital)
                    - Primary somatosensory and motor cortex adjacent to central sulcus in parietal and frontal lobes
    - Infratentorium
        - Brainstem and Cerebellum
            - Brainstem composed of:
                - Midbrain
                - Pons
                - Medulla
                - Consciousness via reticular activating system
            - Brainstem contains CN nuclei for 3-12
            - Midbrain controls autonomic functions (respiratory / cardiovascular)
                - As well as reflexes (cough / gag, pupillary reflexes)
- Brain receives 70% of blood supply from two internal carotids, 30% from two vertebral arteries
- Average CSF in adults is 150 cc
    - CSF is produced mostly in choroid plexus of lateral and third ventricles
    - CSF is reabsorbed in dural venous sinuses (primarily sagittal sinus via arachnoid villi and granulations)
        - Some goes into foramen magnum --> subarachnoid space
    - Average production of CSF is 15-20 cc / hr

- Spinal Cord

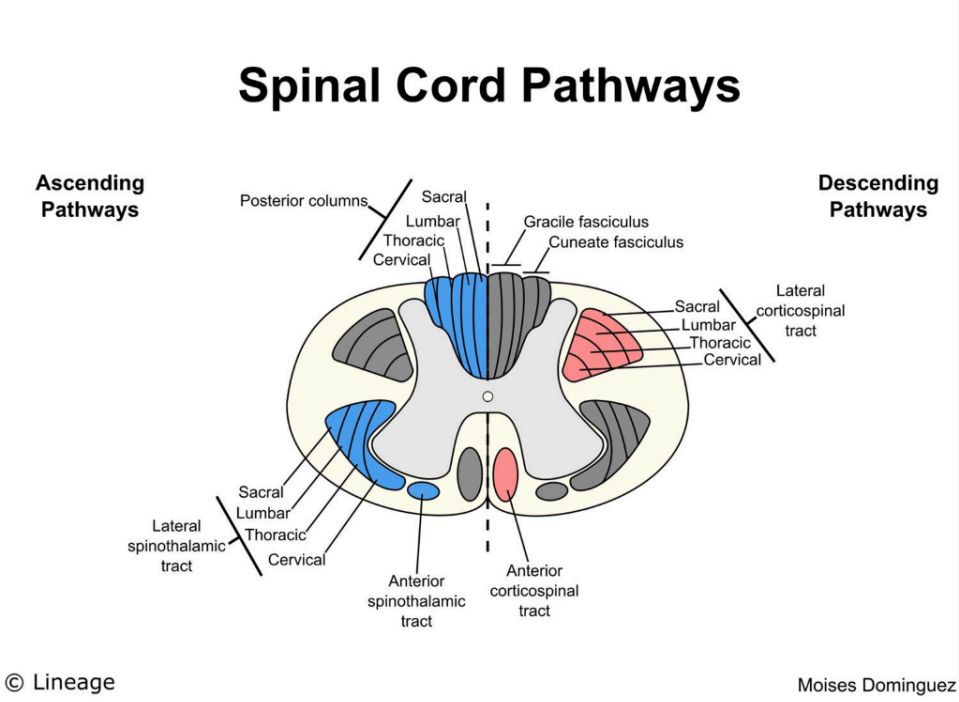

Fig 1: Spinal Cord Pathways (Medbullets)

- Dorsal Columns
    - Propioception and Light Touch
- Lateral Spinothalamic Tract
    - Paiun and Temp
- Outer white matter = lateral corticospinal tracts
- Blood supply
    - One anterior spinal artery
        - Arises from 6-8 major radicular arteries
            - HY PIMP QUESTION
                - Largest radicular artery is Artery of Adamkiewicz
                    - occurs at T11 or T12
                    - Supplies T8 to conus medullaris
                    - Supplies 2/3 of spinal cord
    - Two Posterior spinal arteries
        - Originates from posterior spinal arteries
        - Supplies dorsal horns and white matter
    - This is why tight blood pressure control is very important!

NEUROPHYSIOLOGY

- Brain Metabolic Needs

        - 20% of total body O2 consumption
            - Cerebral Metabolic Rate of O2 Consumption (CMRO2)
                - Normal: 3 to 3.8 cc / 100 g / min
        - 25% of body's glucose consumption
        - Brain requires continuous supply of O2 and glucose --> irreversible injury after 4-5 minutes of global ischemia
- Cerebral Blood Flow Regulation via Flow-metabolism Coupling and Autoregulation
        - Occurs very quickly
        - Normal Cerebral Blood Flow --> 50 cc / 100 g / min (or 750 cc / min)
            - Brain receives 15% of cardiac output
        - Factors affecting Cerebral Autoregulation
            - MAP
                - Maintained between 60 – 160 mmHg
                    - Dynamic parameters depending on the person
            - PaCO2
                - CBF linearly associated between 20 – 80 mHg
                    - Change in PaCO2 of 1 mmHg correlates to similar change in CBF of 1-2 cc / 100 g / min
                    - Below 20 --> maximal cerebral vasoconstriction
            - O2 Tension
                - Minimal effect on CBF
            -
            - Fig 2. Deranged Physiology

- Cerebral Perfusion Pressure (CPP) = MAP – ICP
        - Spinal Cord Physiology very similar to brain
            - Spinal Cord Perfusion Pressure (SCPP) = MAP – SSSP (spinal arachnoid space pressure)
- Effects of Anesthetic Drugs on CBF
        - Propofol, etomidate, benzos, thiopental decrease CBF --> decreases CMRO2
        - Opioids have little effect on CMRO2, CBF, autoregulation, or PaCO2 responsiveness
        - HY Board: Ketamine increases CBF and CMRO2, little effect on autoregulation
        - Volatile anesthetics (iso / sevo / des) are direct cerebral vasodilators
            - Low effects at low doses
            - High doses (I.e. > 1 MAC), suppresses CMRO2
        - Nitrous Oxide
            - Direct cerebral vasodilator causes minimal effect to CMRO2
        -

PATHOPHYSIOLOGY

- Monro-Kellie Doctrine
    - Skull contains three main things: brain (1400 cc), CSF (150 cc), Blood (150 cc)
    - "Increase in volume of an intracranial compartment leads to rise in ICP unless matched by equal reduction in the volume of another compartment
- ICP
    - HY: Normal = 7-15 mmHg
        - Poor Neurologic outcome with prolonged ICP above 20-25
    - Cerebral-Elastance Curve
        - Elastance: E = dP / dV
            - Could be monitored with invasive cerebral and spinal monitoring
        - Three parts of the curve
            - 1: initial flat part = intracranial elastance low --> non pathologic states, changes are compensated easily
            - 2: Elbow of curve = can't compensate, elastance increases, small increases in volume --> rapid increase in ICP
            - 3: Super elastance, very sensitive
        -
    - Fig 3 Veterian Key
-
    - Most common cause of increased ICP is cerebral edema
        - Three Types of Edema:
            - Cytotoxic
                - Increase in intracellular water
                    - Cerebral ischemia, ionic pump fails to to work and accumulates ions
            - Vasogenic
                - Loss of BBB integrity --> accumulates extracellular water
                - Occurs in regions surrounding tumors
                    - Decadron effectibe at decreasing only vasogenic edema
            - Interstitial
                - Occurs in patients with hydrocephalus --> CSF goes into intersitial space
    - Other factors that cause intracranial hypertension
        - Increased CBV
            - Increased arterial inflow
                - Vasodilatory drugs

                - Hypercapnia
                - Severe hypoxemia
                - Acidosis
            - Decreased venous / CSF drainage
                - Decreased CSF absorption at arachnoid villa
                - Mass effect from tumors
    - Tying Things Together: CPP
        - Cerebral Perfusion Pressure (CPP) = MAP – ICP
            - You could control CPP by tight control of MAP (hence arterial line), and management of ICP through the flow metabolism curve and other drugs (e.g. decadron, mannitol, etc.)
- Clinical Presentation
    - Headache
    - Nausea / Vomiting
    - Papilledema
    - Cushing's Triad
        - Hypertension
            - Remember: CPP = MAP – ICP
                - To keep CPP the same, increases in ICP requires increase in MAP
        - Bradycardia
        - Irregular Respiration
    - Feared Complication:
        - Pupillary dilation, oculomotor weakness, absent pupillary light reflex, cardiopulmonary arrest