

Cerebral Dynamics

1. What percent of ATP depletion leads to changes in neurologic cortical signs: speech, behavior, and memory. P. 1
 - When ATP levels fall to around 55% of normal, we see difficulty with conduction.
2. At what temperature will brain cell death occur because of ATP depletion? P. 3 This is not to say death can't occur at lower temperatures but at 106 or higher it is deadly.
 - Hyperthermia results in a 10% increase in metabolic demand for every one degree elevation centigrade in temperature over normal. At a temperature of 106 F, the brain rapidly advances to vegetation and brain death due to rapid atp depletion
3. In terms of a time frame, when does seizure activity become a medical emergency? P. 2 & 3
 - Seizure activity that is ongoing more than 20 to 30 minutes is a medical emergency due to rapid atp depletion
4. Review the changes in blood flow during seizure activity (increased or decreased). P.2 & 4
 - The patient who can will attempt to increase cerebral blood flow as much as 250% to 300% in an attempt to meet the metabolic demands of seizure activity
5. What is the primary energy source for brain function? P.1
 - The primary source of energy for the brain is glucose, which is converted to adenosine triphosphate (ATP).
6. Review cerebral blood flow. P. 5 What effect would an increase or decrease in blood pressure have on the diameter of cerebral arteries? See page 6, pressure regulation.
 - The supply of glucose and oxygen, and therefore atp, is maintained by cerebral blood flow. Normally, the brain receives 750 ml of blood per minutes or 15% to 20% of the total resting cardiac output.
 - A decrease in blood pressure results in vasodilation protecting the brain from subflow states and subsequent infarction
 - When systemic arterial pressure or intracranial pressure increase, the arterioles vasoconstrict to safeguard the brain from a hyperemic flow state.
7. Review the metabolic factors and pressure changes that affect the brain's ability to auto-regulate (see Autoregulatory Failure). P. 7
 - Increased levels of PaCO₂, carbon dioxide acts as a potent dilatory agent as the brain attempts to remove the products of cellular breakdown as efficiently as possible. In the rang of PaCO₂ from 20 mm Hg to 80 mm Hg, CBF is halved if PaCO₂ is halved, and doubled if PaCO₂ is doubled
 - Decreased levels of PaO₂, a PaO₂ below 60 or an O₂ saturation below 90 results in hyperemic flow state.
 - Head injuries, 90% of all moderately to severely head injured patients have ischemic brain tissue on post-mortem exam. Within ischemic tissue is contained massive quantities of waste the majority of which are vasodilatory agents.
 - Intracranial bleeds, Intracranial bleeds like subarachnoid hemorrhages can result in a loss of autoregulation. Blood contains many substances like calcium and free iron which are tolerated poorly in the ventricular system and the subarachnoid space of the brain.
 - when autoregulation is lost after SAH=stroke

8. Know how to calculate MAP. p. 8 I typically use $MAP = SBP - DBP/3 + DBP$. In other words, subtract the DBP (diastolic blood pressure) from the SBP (systolic BP) and divide that number by 3. Then add to that number the DBP and that equals the MAP. For example, for a BP of 120/70, the difference would be 50 and $50/3 = 16.7$. Then $16.7 + 70 = 86.7$ for MAP.
- $MAP = SBP - DBP/3 + DBP$
 - In normally normotensive patients a map <50 mm hg results in poorly perfused cerebral tissue with ischemic changes
 - In these same patient a $MAP > 170$ mm Hg causes a loss in the ability of the arterioles to constrict, which causes hyperemia and increased ICP
9. Know that $CPP = MAP - ICP$.
- The normal range for CPP is 50-150 mm Hg with an average of 80-100 mm Hg.
 - CPP of less than 50 result in ischemia and values of greater than 150 result in hyperemia
 - $CPP < 50$ mm Hg= ischemia
 - $CPP > 150$ mm Hg-hyperemia
 - $CPP < 30$ is incompatible with life and results in neuronal hypoxia and cell death
 - When the MAP equals the ICP, the CPP is 0 and all CBF stops.
10. What would classify an ischemic stroke as being embolic, thrombotic or sub-flow? P. 2-3
- Ischemic stroke is classified as embolic or thrombotic. Thrombotic strokes are more common than embolic strokes and occur in both small and large arteries. They result from an obstruction of blood flow in an artery due to a pathological process within that artery
 - Embolic strokes are caused when blood clots or atheromatous debris form outside the brain and become lodged in the cerebral circulation. Emboli commonly originate in the heart or in the proximal arteries that supply the brain, the internal carotid or vertebral systems.
 - Hemorrhagic stroke accounts for the remaining twenty percent of strokes and occurs when a blood vessel supplying the brain ruptures.
- When would t-PA be contraindicated in a patient with an ischemic stroke? See page 61. With reference to time frame, note the time frame has been extended from a maximum of 3 hours to 4.5 hours from onset of symptoms to latest time to receive tPA. recently revised to >4.5 for subset of patients
- Contraindicated when:
- Symptoms minor or rapidly improving
 - Seizure at onset of stroke
 - Another stroke or previous head trauma within the past 3 months
 - Major surgery within 14 days
 - Known history of intracranial hemorrhage
 - Sustained systolic blood pressure > 185 mm Hg
 - Sustained diastolic blood pressure > 110 mm Hg
 - Aggressive treatment required to lower blood pressure
 - Symptoms suggestive of SAH
 - Gastrointestinal or urinary hemorrhage within 21 days
 - Arterial puncture at noncompressible site within 7 days
 - Received heparin within 48 hours and has elevated PTT
 - $PT > 15$ sec or International Normalized Ratio (INR) >1.7
 - Platelet count <100,000 uL

- Serum glucose <50 or >400 mg/dl

11. What is the usual cause of a subarachnoid hemorrhage? See page 6. How would a patient typically describe the headache associated with subarachnoid hemorrhage?

- The usual cause is a rupture of a cerebral aneurysm. When an aneurysm ruptures blood enters the subarachnoid space encircling the brain. The result can be an increase in intracranial pressure. Furthermore, the blood can act as an irritant to the brain generating vasospasm.
- Headache is many times the defining feature of SAH, occurring 85% of affected patients. Headache is usually characterized by the patient as sudden, intense, of no distinctive location or quality, and different than previous headaches. SAH should always be suspected if the patient describes the headache as the "worst headache of my life" or when headache is followed by a transient loss in consciousness.

Renal System:

1. Review anatomy and primary functions of the nephron. P.1
 - Each kidney contains approximately 1 million nephrons. Each nephron consists of a glomerulus (coiled capillaries within Bowman's capsule), proximal convoluted tubule, loop of Henle and distal convoluted tubule.
 - The specific functions of the nephron are GFR filtration, tubular reabsorption, and tubular secretion and excretion
2. Blood flow to the kidneys accounts for what percent of cardiac output? P.4
 - Blood flow to the kidney is about 20-25% of cardiac output (4-6L/min)
 - Usual range is about 1100-1200 ml of blood >kidney/min.
3. What is the normal GFR/minute or per day. P.4
 - About 15-20% of plasma is filtered > Bowman's capsule/min
 - For example 625 ml. plasma x 20% = 125 ml (GFR)/min
 - 125 ml x 60 min = 7.5 L/hr or 180 L/day
 - oliguria is defined as urine output that is body wt (kg)/hr .5cc/kg/hr or less
4. What is the blood threshold for the appearance of glucose in the urine? p.4
 - glucose will begin to spill into the urine at a blood glucose of approximately 180 mg per cent (180 mg/100ml)
5. Know the normal and abnormal urinary findings. P.5
 - Ph 4.5-8.0
 - Specific gravity 1.003-1.030
 - Osmolality 300-1200 mosm/kg
 - Protein 30-150 mg/24 hr
 - Sodium 27-287 meq/24 hr
 - Creatinine 1-2 gm/24 hr
 - Urea 6-17 g/24 hr
 - Myoglobin absent/negative
 - Rbcs 0-5
 - Wbcs 0-5
 - Bacteria none-few
 - Casts none-few