CHRONIC RESPIRATORY ALKALOSIS

What controls ventilation?

- **Thoracic neural receptors**: present in the airways, lungs, chest wall and pulmonary vessels; two types—slow adapting pulmonary stretch receptors that respond to changes in lung volumes and rapidly adapting irritant receptors that respond to changes in lung volume and chemicals such as histamine, prostaglandins and local chemical environment. These receptors travel through the vagus nerve to stimulate an increased respiratory rate, or stimulate cough, bronchoconstriction and mucous production.

- **Peripheral chemoreceptors**: mainly carotid receptors that sense decrease in PaO2 (<75), hypercapnea and acidosis; account for 15% of ventilation (if you denervate, PaCO2 rises 5-10 mmHg.) Impulses travel through CN IX to medulla to increase respiratory rate. Also, there are also other undefined peripheral receptors that regulate ventilation in response to pH in extracellular fluid around muscles during exercise.

- **Central chemoreceptors**: present in ventral surface of the medulla and midbrain, major role in maintaining acid-base homeostasis by instantaneously adjusting ventilatory rate to changes of pH in the CNS environment (note that CO2 is lipid soluble, so crosses blood brain barrier rapidly, allowing rapid ventilatory response). Acetylcholine and parasympathetic nerves help determine the response to CO2 stimulation. Note, that this occurs at a constant body temperature.

- **Central pattern generator cells**: present in the medulla, integrate signals from above receptors to determine respiratory frequency, inspiratory and expiratory time. Usually under mild inhibitory input form cortex (loos of inhibition results in Cheyne-Stokes pattern)

What is the appropriate compensation for respiratory acid-base disorders:

- Compensation occurs in two stages: initially (minutes to hours) cellular buffering is main mechanism; renal compensation takes 3-5 days, this accounts for the different HCO3 responses in the acute or chronic setting.

- Respiratory acidosis: for every 10 mmHg of CO2 there should be an increase of 1 mEq of HCO3 acutely or 3.5 mEq HCO3 chronically.

- Respiratory alkalosis: for every 10 mmHg CO2 there should be a drop of 2 mEq of HCO3 acutely or 4 mEq HCO3 chronically.

- Although it is rare, IT IS POSSIBLE for severe, chronic respiratory alkalosis to result in compensatory levels of HCO3 as low as 10. (same mechanism, decrease in HCO3 reabsorption and ammonium excretion). However, when get below 16, look for a primary metabolic acidosis.

What can cause a respiratory alkalosis?

- Hypoxemia: high altitude
- Pulmonary disorders: ILD, pneumonia, PE, pulmonary edema (even without hypoxia)
- CNS disorders: anxiety, trauma, meningitis, encephalitis
- Drugs: salicylates, nicotine, xanthines
- hepatic encephalopathy
- early sepsis

What is so bad about respiratory alkalosis?

- Clinically, patients present with panic, weakness, paresthesias around hands and feet, muscle cramping.

- Alkalemia results in a fall of free Ca++ due to increased binding of calcium to proteins may lead to tetany and seizures; also hyponatremia and hypokalemia may be seen.

- Cerebral vasoconstriction may lead to cerebral hypoxia.
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