Renal physiology part 2

Westmead Primary Exam Lectures

Defence of tonicity

- *The total body osmolality is directly proportionate to the total body sodium and the total body potassium divided by the total body water*
    - *Changes in osmolality of the body fluids occurs when a disproportion exists between the amount of these electrolytes and the amount of water*
    - *When the effective osmolality of the plasma rises then vasopressin secretion is increased, and the thirst mechanism is stimulated*
- *In a healthy person - plasma osmolality ranges from 285 - 295 mosm/kg of H2o*

Vasopressin

- *3 kinds of vasopressin receptor*
    - *V1a —> vasoconstrictor*
    - *V2 —> cGMP mediated —> aquaporin insertion into collecting ducts*
- *Vasopressin is secreted from the posterior pituitary*
    - *Stimulated when osmolality > 285, standing, haemorrhage /decreases ECF via low pressure and high pressure sensors, hypotension,*
    - *Etoh decreases vasopressin secretion*
- *DI = ADH deficiency*

Defence of Volume

- *Volume is determined by the amount of osmotically active solute - the amount of Na is the most important determinant - therefore the mechanisms that control Na secretion are important*
- *RAAS system causes vasoconstriction and Na + water retention*

Defence of H^+ ion concentration

- *Machinery of cells are very sensitive to changes in H^+ concentration*
- *pH of blood is the pH of true plasma because it is in equilibrium with RBC which contain Hb which is an important buffer*

H+ Balance

- *The pH of arterial plasma is 7.40, variations of 0.05 occur with minimal physiological effect and the levels compatible with life are from 7 - 7.7*
- *Amino acids in liver for gluconeogenesis —> produce NH_4^+ and HCO_3*
    - *NH_4^+ becomes urea and the proton becomes*
    - *Some sulphur containing amino acids are metabolised to H_2SO_4, and metabolism of phosphorylated amino acids form H_3PO_4 - these strong acids present a major H^+ burden*
- *CO_2 is formed by metabolism is in large hydrated to H_2CO_3 —> however, most of the CO_2 is excreted by the lungs*
- *Aside from these two sources, common other sources of acid load include DKA, Lactic acidosis*

Buffering

- *The Henderson - Hasselbalch equation*
  - *HA <=>(H^+) + (A^-)*
  - *HA = undissociated acid , A^- = ANY anion*
  - *If an acid stronger than HA is added to this system the equilibrium is shifted to the left - the hydrogen ions that thus tied up in the formation of new acid*
  - *If a base is added H^+ and OH^- react to form H_2O - but more HA dissociates*

Buffers in the blood

- *Proteins - plasma proteins*
- *Hb - has 6 x the buffering capacity of plasma proteins*
- *Carbonic acid - bicarbonate system*
- *H_2PO_4 system*

Renal compensation

- *HCO_3 reabsorption in the renal tubules depends on the filtered load of bicarb (which is a product of GFR and plasma bicarb) but also on the rate of active secretion of H^+ by the tubular cells - since HCO_3 is reabsorbed by exchange for H^+*
- *In respiratory acidosis, renal tubular H^+ secretion is increased:*
  - *This is when PCO_2 is high, the interior of most cells including renal tubular cells is more acidic*
  - *Also bicarb reabsorption is increased*
  - *Cl excretion is increased and plasma Cl falls as plasma bicarb is increased*
- *Conversely in resp alkalosis*

Metabolic acidosis

- $H_2CO_3 \longrightarrow H_2O$ and CO_2 which is rapidly excreted by expiration
- Renal compensation
  - The anions that replace bicarb in the plasma in metabolic acidosis are filtered each with a cation, principally sodium - thus maintaining electrical neutrality
  - Tubular cells secrete H^+ into the tubular fluid in exchange for Na and for each H^+ secreted on Na and one HCO_3 are added to the blood
  - The limiting urinary pH of 4.5 would be reached rapidly this way
  - However H^+ reaction with HCO_3 in the urine to form H_2O and CO_2
    - Also with HPO_4 and NH_3
    - Allowing large amounts of H^+ to be secreted
    - When the acid load is large cations are lost with the anions, causing depletion of cation stores and diuresis