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Kidneys

- Acid Base Balance
  - Body pH 7.40 +/- 0.05
  - Acidosis
    - Metabolic Acidosis
      - Low pH, Low HCO3-
        - Due to increased production of organic acids.
          - Lactic acid (shock, infection, hypoxia)
          - Ketones (DM, alcohol)
            - Urate (renal failure)
            - Drugs / toxins
        - Due to loss of HCO3- or ingestion of H+
          - Renal tubular acidosis
          - Diarrhoea
          - Drugs
          - Addison's
          - Ammonium chloride ingestion
    - Respiratory Acidosis
      - Low pH, High CO2
        - Any lung, neuromuscular or physical cause of respiratory failure
  - Alkalosis
    - Metabolic Alkalosis
      - High pH, HighHCO3-
        - Vomiting
        - K+ depletion (diuretics)
        - Burns
        - Ingestion of base
    - Respiratory Alkalosis
      - High pH, Low CO2
        - A result of hyperventilation
          - CNS Causes: stroke SAH, meningitis
          - Other causes: anxiety, altitude, drugs (salicylates)
- AKI
  - Classification
    - Pre Renal (Usually normal dipstick)
      - Appropriate renal response to poor perfusion - when prolonged kidney damage can occur
      - Hypovolaemia
      - Hypotension / Vasodilators
      - Renal artery stenosis
      - Sepsis
      - ACE-I / ATII Receptor Antagonists / Renin Inhibitors
    - Renal (Abnormal dipstick)
      - Vascular
      - Glomerular Nephritis
      - Interstitial Nephritis
      - Tubular Necrosis (major cause)
        - Ischaemia
        - Toxins
        - Pigment
    - Post Renal (Ultrasound)
      - Urinary obstruction - increasing incidence with age
      - Extra renal: prostatic disease, pelvic cancer
      - Intra renal deposition: urice acid nephropathy, Ca oxalate crystalluria (anti freeze)
  - Treatment
    - Prevention - EWS
    - Treat the underlying cause
    - Restore adequate circulating volume & BP
    - Control fluid intake
    - Renal replacement therapy
  - RIFLE Classification
    - Risk
    - Injury
    - Failure
    - Loss
    - End Stage Renal Disease
- CKD
  - Staging
    - Stage 1: eGFR > 90 + evidence of renal damage
    - Stage 2: eGFR 60 - 89 + evidence of renal damage
    - Stage 3: eGFR 30 - 59 (moderate)
    - Stage 4: eGFR 15 - 29 (severe)
    - Stage 5: eGFR < 15 (established)
- Renal Replacement Therapy
  - Haemodialysis
    - Access via ateriovenous fistula
    - Solute transfer by diffusion; removal of excess fluiod by -ive transmembrane pressure - ultrafiltration
    - Problems: disequilibration syndrome, hypotension, arrhythmias, fistula (thrombosis & stenosis)
      - DS: rapid changes in plasma osmolality resulting in cerebral oedema causing headaches, nausea, altered conciousness and coma
  - Haemofiltration
    - Convective process removing water, ions and small molecules while maintaining cells and proteins - the same volume is replaced with healthy fluid
    - More expensive and takes longer than haemodialysis; greater haemodynamic stability
  - Peritoneal Dialysis
    - Access via Tenchkoff catheter
    - PD fluid is introduced into peritoneal cavity; uraemic solutes diffuse across the peritoneal membrane; ultrafiltration is achieved by adding osmotic agents (glucose) to the PD fluid
    - Problems: peritonitis (60% staph, 20% gram neg, 5% fungi), exit site infection, catherter malfunction)
- Renal Artery Stenosis
  - Causes: atherosclerosis (80%), fibromuscular dysplasia (10%)
  - Clinical features: HT resistant to treatment, poorer renal function after ACE-I therapy, sudden onset pulmonary oedema
  - Tests: ultrasound (affected kidney is smaller), CT/MR angiography, renal angiography (gold standard but invasive)
  - In bilateral RAS cicrulating angiotensin II constricts efferent arteriole more than the afferent arteriole maintaing glomerular filtration pressure - ACE I / ARB therapy can cause a significant fall in GFR
    - PVD exam should be done before prescribing these drugs
- Anatomy / Physiology
  - Kidneys lie at T12 / L3
  - Renal Corpuscle
    - Production of filtrate
  - PCT
    - Reabsorption of water, ions & organic nutrients
  - Loop of Henle
    - Further reabsorption of water (descending) and Na &Cl (ascending)
  - DCT
    - Secretion of ions, drugs & toxins. Variable reabsorption of water & Na (aldosterone control)
  - Collecting Ducts
    - Variable reabsorption of water and reabsorption / secretion of Na, K, H & HCO3
  - Papillary Duct
    - Delivery of urine to minor calyx
- Hormonal Regulation
  - Renin Angiotensin System
    - Renin release by juxtaglomerular apparatus
    - Triggers
      - Decline in blood flow at JGA
      - Sympathetic stimulation
      - Decline in osmotic conc at macula densa
    - Renin > angiotensinogen > angiotensin I > angiotensin II
      - Increased sympathetic activity
      - DCT Na+ reabsorption in exchange for K+ / H+ (overall water retention)
      - Increased aldosterone secretion
      - Arteriolar vasoconstriction
      - ADH secretion
  - ADH
    - Impacts on collecting ducts increasing water retention
  - Aldosterone
    - Acts on receptors of DCT
    - Increases Na+ reabsorption in exchange for K+ / H+ (overall water retention)