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Adrenals

- Anatomy
  - Cortex
    - Produce steroids
      - Glucocorticoids (fasciculata)
        - Increase / stimulate: gluconeogenesis, glycogen deposition, protein catabolism, fat deposition, Na retention, K loss, free water clearance, uric acid production
        - Decrease / inhibit: protein synthesis, response to infection, lympocyte transformation, delayed hypersensitivity
      - Mineralocorticoids (glomerulosa)
        - Control Na and K balance
          - Not ACTH sensitive
          - Sensitive to K and angiotensin II levels
      - Androgens (reticularis)
        - Sex hormones
        - Weak effect until peripheral conversion
  - Medulla
    - Produce catecholamines
      - Conversion of tyrosine into the adrenaline, noradrenaline, and dopamine (chromaffin cells)
- Physiology
  - Circadian rhythm + stress > hypothalamus > CRH > pituitary > ACTH > adrenals > cortisol (negative feedback)
  - Investigations
    - Basal levels - 9am for peak of circadian rhythm
    - Suppression test for suspected excess cortisol
      - Dexamethasone suppression (overnight, low dose, high dose)
      - Normal - prompt feedback supression of CRH, ACTH and cortisol
      - Cushing's syndrome - cortisol will not be suppressed
      - Conditions that cause false positives: depression, obesity, alcohol excess, inducers of liver enzymes that increase the rate of steroid metabolism
    - Stimulation test for suspected deficient cortisol
      - ACTH (synacthen) stimulation (short, long)
      - Normal - stimulates cortisol production
      - Can help exclude primary adrenal failure (Addison's)
    - Random cortisol of no value due to diurnal, stress and illness variation
- Addisonian Crisis
  - Causes: long term prednisolone suppression of pituitary adrenal axis, sepsis, metastatic cancer
  - Signs / Symptoms: shock (tachycardia, peripheral vasoconstriction, hypotension, oliguria, weak, confused, coma), hypoglycaemia
  - Precipitating factors: infection, trauma, surgery
  - Immediate management: bloods for cortisol & ACTH > hydrocortisone sodium succinate IV > fluids (plasma expander then saline > monitor BMs > blood & urine for culture > antibiotics
  - Further management: glucose IV (by BM) > IV fluids (by clinical state) > IV steroids then oral steroids after 72 hrs > identify cause
- Addison's Disease
  - Causes
    - Primary insufficiency - destruction of adrenal cortex (rare)
      - Autoimmune (commonest UK cause), TB (commonest worldwide case), adrenal metastases, opportunistic infection in HIV, adrenal haemorrhage
    - Secondary insufficiency - iatrogenic (common - through suppression of pituitary adrenal axis)
  - Symptoms / Signs
    - Symptoms: often diagnosed late, fatigue, weight loss, dizziness, myalgia, depression, nausea, vomitting, abdo pain, diarrhoea, constipation
    - Signs: hyperpigmentation (palmar creases & buccal mucosa), postural hypotension (due to low Na)
      - Due to raised ACTH which cross reacts with melanin receptors
  - Treatment
    - Hydrocortisone 15 - 25 mg daily
      - 2 - 3 divided doses
      - Higher dose on waking to reflect natural variation
      - Avoid giving late in day - insomnia
    - Fludrocortisone (mineralocorticoid)
      - Give if postural hypotensive, low Na, high K, raised plasma renin
  - Prognosis is good with treatment
  - Steroids
    - Warn against abrupt cessation
    - Other doctors, surgeons and dentists must know of steroid therapy
    - Steroid card (blue) / bracelet
    - Add 5 - 10mg before strenuous activity
    - Double steroid intake with illness, injury or stress
    - IM hydrocortisone in case vomiting prevents oral intake
    - Gradual decrease after 3 weeks of therapy
- Hyperaldosteronism
  - Excess prodction of aldosterone, independent of renin angiotensin system
    - Increased Na and water retention
  - Features: hypertension, hypokalaemia, alkalosis
    - Alkalosis - aldosterone works on Na channels that retain Na in exchange for K or H+ (can result in alkalosis)
  - Usually asymptomatic, but may have features of low K: weakness, cramps, paraesthesia
  - Causes
    - 2/3 Aldosterone producing adenoma (Conn's syndrome)
      - 1/3 Bilateral adrenocortical hyperplasia
  - Treatment
    - Hyperplasia: spironolactone
      - Conn's: adrenalectomy & spironolactone pre-op to treat HT and low K
- Cushing's Syndrome
  - Symptoms / Signs
    - Symptoms: weight gain, depression, insomnia, amenorrhoea, thin skin, easy to bruise, muscle weakness, psychosis
    - Signs: moon face, thin skin, bruising, hypertension, pathological fractures, dorsal fat pad, osteoporosis, proximal myopathy
  - Causes
    - ACTH dependent: pituitary dependent (Cushing's disease), ectopic ACTH producing tumour, exogenous ACTH (iatrogenic)
    - Non-ACTH dependent: adrenal adenoma / carcinoma, glucocorticoid administration
  - Treatment
    - Iatrogenic
      - Stop / limit medications (if possible)
    - Cushing's disease
      - Selective removal of pituitary adenoma
      - Pituitary radiotherapy (esp. children and in preventing Nelson' syndrome)
        - Nelson's syndrome - increased skin pigmentation due to excess ACTH from an enlarging pituitary tumour after bilateral adrenalectomy remove -ive feedback
      - Bilateral adrenalectomy if source cannot be located
    - Ectopic Tumour
      - Surgery if located and not spread
    - Adrenal tumour
      - Adrenalectomy (curative for adenoma, but rarely for carcinoma)
      - Radiotherapy
    - Medical treatment
      - Reduce cortisol secretion pre surgery or awaiting radiotherapy
      - Metyrapone
        - Competitive inhibitor of 11 beta-hydroxylation in adrenal cortex
        - Inhibition of cortisol and to some extent aldosterone production
  - Prognosis
    - Untreated: poor due to CV complications
    - Treated: good (esp. physical features and psychological disorders) although osteoporosis, glucose intolerance, DM, HT may persist
- Phaeochromocytoma
  - Rare catecholamine producing tumours - dangerous but treatable
  - 90% sporadic, 10% hereditary
  - Clinical features
    - General: sweating, heat intolerance, pyrexia, apprehension
    - Neuro: headaches, visual disturbances
    - CV: palpitations, chest tightness, dyspnoea, faints
    - GI: abdo pain, nausea, constipation
  - Investigations
    - Screening: 3 x 24 hr urine for catecholamines
    - CT / MRI
  - Complications: heart failure, dilated cardiomyopathy, arrhythmias, stroke, hypertensive crisis
  - Treatment
    - Alpha and beta blockade pre op
      - Alpha block first - to avoid crisis from unopposed alpha adrenergic stimulation
    - Surgery