Contents

Practical Anaesthesia

Patient on Steroids

- lower dose steroid supplementation now recommended

- if <3/12 since stopped steroids ⟹ Rx as if on steroids
- >3/12 since stopped then assume normal HPA axis
- specifics:
  - <10mg/day ⟹ normal HPA axis. No cover
  - >10mg/day ⟹ Rx based on surgery:
    - minor = routine preop steroids & 25mg Iv hydrocort at induction
    - moderate = as minor but cont hydrocort 6hrly for 24hr
    - major = as moderate but cont hydrocort for 48-72hrs
  - high dose immunosupression eg 60mg pred ⟹ convert to IV hydrocort until can resume PO
    - ↳ 10mg PO pred = 40mg IV hydrocort; 1.5mg dex
    - ↳ 20mg hydrocort = 0.05mg fludrocortisone

HPA Axis Suppression

- endogenous cortisol = 25-30mg/24hr in circadian rhythm
- during stress can ↑ to 75-100mg/day & stay high for up to 72hrs post major stress
- prednisone:
  - glucocorticoid action ~ x3 more potent than hydrocort
  - mineralcorticoid = much less active
  - ↳ ∴ useful for chronic conditions as avoids water retention issues
- measure using synacthen test:
  - 250mcg IV
  - serum cortisol measured at 0,30 & 60min
  - norm peak cortisol = 420-700
- fludrocortisone only available in oral form

By Disease

Diabetes Mellitus

Preoperative

= progressive pancreatic beta-islet cell failure resulting in -> either decreased insulin secretion or insulin resistance

- insulin:
  - stimulates: glucose uptake and lipid synthesis at adipose & mm cell only
  - inhibits: lipolysis, proteolysis, gluconeogenesis, glycogenolysis and ketogenesis

- DM I (20%) = immune mediated beta-islet cell destruction
- DM II (80%) = insulin resistance

- significant increase in perioperative morbidity with high sugars ie HbA1c >69 (increased length of stay, increased wound infections, increased mortality, worse long term prognosis, prothrombotic state, cardiac sympathetic overactivity, dehydration)

HISTORY

- Symptoms:
  - polyuria
  - thirst
  - polydipsia
  - dehyrdration
- control: diagnosis, recent trends, hypoglycaemic attacks
- complications:
  - Macro:
    - HTN
    - stroke
    - Renal failure
    - IHD & HF
    - PVD
  - Micro:
    - retinopathy,
    - nephropathy,
    - neuropathy: peripheral, autonomic
- co-morbid conditions: PVD, IHD, CHF, infections, ulcers, amputations, HT, CVA, GORD

- Medications:

1. Insulin sensitisers + control hepatic glucose production = biguanides, thiazolidineiones
2. ↑insulin release = sulfonylureas, meglitinides)
3. ↓absorption of starch = alpha-glucosidase inhibitors
4. ↑insulin production and ↓ glucose production = incretin mimetics, DDP 4 inhibitors)
5. Insulin

EXAMINATION

- BSL
- observations

AIRWAY: glycosylation of TMJ or neck joints -> decreased ROM, neck movement
GI - delayed gastric emptying
CVS: postural BP, resting HR, pulses, heart size, JVP
RESP: creps
NEURO: peripheral neuropathy, eyes

INVESTIGATIONS
- BSL
- ECG - silent MI
- Blds -
- urine for ketones & protein
- glucose
- HbA1c (<7% or <69mmol/ml) - if higher refer to DM team preop
- IHD investigation (particularly if has silent ischaemia)

MANAGEMENT
Key points:
- Tight perioperative control:

1. keep basal insulin going:
    - 50% amount during fasting (use local guideline to give equivalent dose of protaphane)
    - 100% when eating
2. IV sliding scale insulin (VIII):
    - Used for all major surgery & long fasting (>1 missed meal or NBM >6hrs)
    - glucose/insulin given through same cannula with pump & anti reflux valve
    - add 20mmol KCL to litre bags if K <4.5mmol
3. Subcut insulin:
    - stop regular short acting insulin
    - if prolonged fast or major surgery ⟹ VIII
    - give short acting for minor surgery with ↑BSL peri-op:
        - use standardised s/c bolus regimes
        - Example dosing bolus dose for ↑BSL:
            - Type 1DM - give 1unit ⟹ ↓BSL by 3
            - Type II DM - give 0.1U/kg ⟹ recheck BSL 1 hr later

- avoidance of hypoglycaemic attacks
- prevent ↓K, ↓Mg, ↓PO4

- if brittle -> admit to hospital night before
- first on list
- stop oral hypoglycaemics once fasting (don't resume until on full diet)
- continue basal insulin (esp type I) - use 50% dose if fasting
- stop meal bolus insulin once starving
- avoid carbohydrate drinks pre surgery in ERAS
- keep SC pump going on basal rate
- glucose 5-10mmol/L
- monitor BSL's closely
- monitor for ketones
- consider an insulin-dextrose infusion (1 U/hr insulin, 40mL/hr 10% dextrose)
- must not drive on day of surgery
- early IV access

Intraoperative
- final check preop of BSL:
    - if >12mmol - check urinary ketones. If ketones >+++ then defer surgery
- continue controlled BSL's throughout OT
- give insulin and dextrose through same cannula so insulin not given without dextrose
- check BSL 1 hourly
- RSI if indicated
- RA may be appropriate
- have pressors ready c/o autonomic dysfunction
- treat hypoglycaemia aggressively (<4mmol/L)
    - no signs in anaesthetised pt
    - 150mL of 10% glucose (2ml/kg) over 10mins
    - 1mg glucagon IM or IV
    - 2-4 teaspoons of sugar in mouth/NGT

- if K+<4.5 -> add 10mmol KCl to dextrose bag

Postoperative

- continue control as above
- coming off variable IV insulin infusions:
  - oral hypoglycaemic agents:
    - restart once when ready to eat & drink
    - ↓sulphonylurea if not on full diet
    - metformin should be restarted only if GFR >50
  - subcut insulin:
    - wait until E&Ding with no vomiting
    - switch at next meal with normal s/c insulin due
    - restart normal preop regime (may need titrating up due to stress response)
    - stop VRIII 30-60min after s/c injection & meal
  - prev on long acting subcut insulin:
    - resume to 100% when eating
  - prev BD fixed mixed regime:
    - stop VIII at appropriate insulin mealtime
- consult endocrinology if concerned

Pregnancy

- more aggressive control (glucose 4-6mmol/L)
- may require massive doses of insulin
- run 5% dextrose around time of delivery if not eating (stops ketosis)
- insulin infusion if required
- halve insulin infusion rate once baby born and continue dextrose in DM I

Acromegaly

Preoperative

= rare clinical syndrome caused by hypersecretion of GH from anterior pituitary

HISTORY

- ↑ICP signs eg morning headaches & vomiting
- neck stiffness
- N+V
- visual disturbance
- growth issues
- looks funny

- associated conditions: HTN (30%), IHD, cardiomyopathy, heart failure and valve disease (AR), OSA, DM (25%)

- drugs:
  - somatosatin analogues (octreotide, lanreotide) - side effect of D&V
  - bromocriptine = long acting dopamine agonist ⟹ can ↓GH levels but can lead to severe postural hypotension

EXAMINATION

- AIRWAY: large jaw, head, jaw, tongue and lips, hypertrophy of larynx and trachea -> vocal cord thickening and chondrocalcinosis of larynx, OSA and look for enlarged thyroid with tracheal displacement
- CVS: murmurs, BP, heart failure

INVESTIGATIONS

- ECG: if indicated
- CXR: if indicated
- ECHO: for murmurs
- BSL: 25% are DM

- U+E: imbalanced c/o electrolyte regulation altered
- Bloods for endocrine axis:
    - GH, TFT, Short Synacthen Test, LH, FSH

MANAGEMENT
- discussion with endocrinologist (optimization and perioperative management of hormone therapy)
- discussion with neurosurgeon (approach, position, blood loss)
- BSL cares
- prehydration cares

Intraoperative
- large facemasks
- large blades
- AFOI if indicated
- pressure cares
- protect nerves as are particulary @ risk (ulna elbow, median wrist & peroneal at fibula head)
- OSA cares

Postoperative
- ICU/HDU if indicated

Special Points
- Pan-hypopituitarism patients eg post resection of adenoma or defunctionning adenoma should be referred to endocrinologist for hormone replacement:
    - hydrocortisone $\Longrightarrow$ ACTH adrenal insufficiency
    - thyroxine $\Longrightarrow$ TSH hypothyroidism
    - testosterone $\Longrightarrow$ GnRH male hypogonadism
    - Oestradiol + progesterone $\Longrightarrow$ GnRH femal hypogonaisdm
    - Growth hormone - injection
    - DDAVP nasal spray $\Longrightarrow$ ADH central diabetes insipidus

HyperThyroid Disease

Causes
- Graves disease
- Toxic multi nodular Adenoma
- Toxic Adenoma
- Amiodarone/iodine induced

Preoperative
History
- sweating
- anxiety
- palpitations
- eyes protruding (Graves disease)
- weight loss
- insomina
- hyperphagia
- tremor
- heat intolerance
- diarrhea
- vomiting

- if goitre present: stridor (>50% compression), positional dyspnoea, dysphagia, SVC obstruction, hoarseness

Other complications - atrial fibrillation, high output heart failure, myocardial infarction

- when thyroid disorder diagnosed and how it presented
- treatments
- whether symptoms are currently controlled
- recent endocrinology assessment
- previous surgery to neck
- other endocrine/autoimmune diseases:
    - adrenal hyperplasia, acromegaly, SLE, RA, addisons, IHD
- amiodarone use - can cause either hyper/hypothyroidism
- socially - smoking history – risk factor for thyroid carcinoma, ET-OH history

EXAMINATION

- euthyroid =
    - HR <80
    - no hand tremor
    - ↳ delay all surgery if possible if not euthyroid

- airway assessment - thyroid palpation for size, tenderness, nodules, displacement of trachea, stridor

- signs of SVC obstruction – facial plethora, pembertons sign, distended neck veins, SOB

- eye lid retraction
- expophthalmos
- conjunctivitis

- thyroid examination – inspection (swallow H2O), palpation from behind, bruit

- focussed CVS, RESP examination – pulse, BP (hypertension), JVP, HS, praecordium and chest signs - for signs of heart failure and pleural effusions, arrhythmias

INVESTIGATIONS

- BLOODS – TFT's (recent), FBC – anaemia with hypothyroidism, U+E – electrolyte abnormalities, thyroid anti-bodies (graves disease)
- CXR – tracheal compression and position
- NECK CT/MRI – assessment of goiter and impingement of airway
- RADIOLABELLED STUDIES – see where nodules are
- PRE-OT – quantification of vocal cord function (damage to recurrent laryngeal nerve can take place)
- ECG: AF or sinus tachycardia

MANAGEMENT

- drugs:
    - antithyroid medications: carbimazole 30-45mg daily for 6-8wks,
    - beta-blockers (propanolol = non selective)
        - given if assoc tremor
        - ß1 block symptoms of ↑HR
        - ß2 block periph conversion of T4 to T3
    - Lugol's iodine – decreases vascularity if give 1/52 course prior to operation
- aim for HR 80/min and no tremor
- operation being planned
- positioning of patient

Intraoperative

- standard care

Postoperative

- keep beta-blockers going

Thyroid Storm

- monitor for **Thyroid Storm**:

CLINICALLY

- life threatening exacerbation of hyperthyroid state with 1 or more organ dysfunction
- 20-30% mortality
- 6-24 hours post surgery with:
    - fever
    - sweating
    - HR >140/min
    - coma
    - D&V

MANAGEMENT

Supportive

- IVF (saline & glucose)
- cooling cares
- paracetamol (no NSAIDS or asprin -> displaces thyroxine from proteins)
- propanolol increments (1mg IV) or esmolol boluses -> infusion (50-100mcg/kg/min). Target HR <90

Specific

- hydrocortisone 200mg IV QID
    - action:
        - adrenal insufficiency
        - decreases T4 release and conversion
- propylthiouracil
    - 1g load PO or via NGT then 250mg QID
    - action:
        - inhibit thyroid hormone release
        - decrease peripheral conversion from T4-T3)
- then give either:
    - sodium iodide 500mg tds IV
    - potassium iodide 5 drops via NGT
    - Lugol's iodine 5-10drops via NGT

HypoThryroidism

Causes

- iodine deficiency
- Congenital defect
- Autoimmune thyroiditis (Hashimoto)
- CNS tumour
- Acquired:
    - Thyroid tumour
    - Amiodarone
    - lithium
    - Surgery

Preoperative

= commonly autoimmune thyroid destruction

Problems

- CVS: ↓blood volume, ↓CO, ↓HR, ↓bp, IHD, pericardial effusions
- Haem: ↓Hb, ↓BSL, ↓Na
- Impaired hepatic drug metabolism

HISTORY
- fatigue
- depression
- hypersomulence
- cold intolerance
- OSA
- pulmonary hypertension

EXAMINATION
- weight gain
- thin hair
- decreased blood volume
- bradycardia
- hypotension
- pleural effusions

Airway Assessment - thyroid palpation for size, tenderness, nodules, displacement of trachea, stridor

Signs of SVC obstruction – facial plethora, pembertons sign, distended neck veins, SOB

Focussed CVS, RESP examination – pulse, BP (hypertension), JVP, HS, praecordium and chest signs- for signs of heart failure and pleural effusions, arrhythmias

INVESTIGATIONS
- TFT's :
  - low Thyroid hormones in sick people are not reliable
  - euthyroid sick syndrome = false low T3/T4
- as per clinical assessment
- BLOODS – TFT's (recent), FBC – anaemia with hypothyroidism, U+E – electrolyte abnormalities, thyroid anti-bodies (graves disease)
- CXR – tracheal compression and position
- NECK CT/MRI – assessment of goiter and impingement of airway
- RADIOLABELLED STUDIES – see where nodules are
- PRE-OT – quantification of vocal cord function (damage to recurrent laryngeal nerve can take place)

MANAGEMENT
- delay elective surgery until euthyroid
- liaise with endocrinologist
- start thyroxine 25-50mcg increasing to 100-200mcg/day over weeks
  ↳ use half measures in elderly due to risk of ↑myocardial work
- if surgery urgent can use liothyronine (T3) 10-50 mcg IV slowly with ECG monitoring -> 25mcg TDS IV

Intraoperative
- give all drugs slowly
- have pressors ready - refractory ↓bp
- actively warm
- IPPV
- drug metabolism can be slow
- ↓relaxant & opioid dose

Hypothyroid Coma
- post op problem
- rare decompensated hypothyroidism - mortality 15-20%

CLINICALLY
- coma

- hypoventilation
- bradycardia
- hypotension
- severe dilutional hyponatraemia

- precipitants: infection, trauma, cold, CNS depressants

MANAGEMENT
- IV glucose + saline
- supportive care ABC
- passive slow external warming (to avoid extreme periph vasodilation
- IV levothyroxine 200-400mcg then 100mcg next day
  ↳ smaller dose if CVS disease
- hydrocortisone 100mg IV QID
- IV liothyronine (T3) 0-50mcg if surgery urgent
- ICU

Parathyroid Disease

- PTH actions:
  - bones - release of Ca into ECF/serum via osteoclast activity
  - Kidney - retain Ca, lose PO4
  - ↑vit D activation

HyperParaThyroid Disease

- classified by cause:
  - primary
  - secondary
  - tertiary

Primary Hyperparathyroid

Preoperative

= usually adenoma causing increased secretion of PTH
- assoc with familial multiple endocrine neoplasia (MEN) type 1

HISTORY
- asymptomatic
- anorexia
- dyspepsia
- nausea
- vomiting
- constipation
- polydipsia
- polyuria
- poor memory
- drowsiness
- 'bones, stones, groans and psychic moans'

EXAMINATION
- hypertension
- rarely palpable

INVESTIGATIONS
- shortened QTc
- high serum Ca2+
- low phosphate

- to adjust Ca2+ for albumin: add 0.1mmol/L to Ca2+ for each 5g/L that albumin below 40g/L

MANAGEMENT
- often need methylene blue 1mg/kg to locate glands
- hydrate
- anti-calcium drugs (pamidronate)

- normalise Ca2+ and proceed (aim <3mmol/L)
- if any of:
  - Ca >3
  - abnormal ECG
  - CVS or renal impairement

  ↳ postpone op (if able) until after treatment

Intraoperative
- monitor response to neuromuscular blockade

Secondary Hyperparathyroid

Preoperative
= associated with chronic renal failure causing compensatory parathyroid hyperplasia due to low Ca ⟹
- high PTH,
- normal or low Ca2+
- high phosphate

HISTORY
- chronic renal failure
- excessive bone resorption -> radial aspect of middle phalanx of second digit
- soft tissue calcification of the vascular and soft tissues (including kidneys, heart, lung and skin)

EXAMINATION
- soft tissue Ca2+ deposition

INVESTIGATIONS
- Xray changes
- high PTH
- normal or low Ca2+
- high phosphate

MANAGEMENT
- medical Treatment (fails in 15%):
  - dietary phosphate restriction
  - dietary or supplemental Ca2+ & vitamin D
- parathyroidectomy
- dialyse prior to surgery

Intraoperative
- renal failure cares

Postoperative
- post-parathyroidectomy -> monitor for:

1. bleeding
2. recurrent hyperparathyroidism
3. hypoparathyroidism
4. injury to recurrent laryngeal nerve
5. hypocalcaemia

6. hypomagnesaemia

Tertiary Hyperparathyroid

Preoperative

= parathyroid hyperplasia progresses to autonomous secretion behaving like an adenoma
- continues despite correction of renal failure
- only few require op

HISTORY

- CRF
- symptoms of hypercalcaemia (stones, bones, groans and psychic moans)

Intraoperative

- standard cares

Postoperative

- standard cares
- see primary hyperparathyroidism

Hypercalceamic Crisis

- most commonly in elderly with undiagnosed hyperparathyroidism & malignancy
- dehydration ⟹ anorexia & D&V ⟹ exacerbation
- other features = weakness, lethargy, mental changes, coma
- Ca >4.5 = life threatening
- First line Treatments:
  - IVF - 4-6litres fluid may be required
  - pamidronate - 60mg in 500ml saline over 4 hrs
  - calcitonin:
    - 4unit/kg IV then 4unit/kg s/c bd
    - rapid but temp ↓Ca & PO4 release from bone
- Other treatments:
  - hydrocortisone 200-400mg IV daily - in malignancy
  - IVF & diuresis with furosemide 40mg IV (↓prox tubular reabsorption of Ca)
  - dialysis - reserved for renal failure

HypoParaThyroid

Preoperative

= under active parathyroid gland
- usual causes:
  - post parathyroidectomy,
  - radiotherapy to neck
  - idiopathic

HISTORY

- carpopedal spasm
- tetany

EXAMINATION

- Chvostek and Trousseau positive
- hypotension

INVESTIGATIONS

- dysrhythmias
- low Ca2+ (corrected) <2.2

    - trough level post prathyroidectomy occurs at 20hrs & normalises by day 2-3
- low PTH
- ECG: prolonged PR and QT

MANAGEMENT
- calcium gluconate 10mL of 10% over 10min
- replace Mg2+
- vitamin D
- oral Ca2+
- replace prior to OT

Intraoperative
- standard care
- monitor neuromuscular blockade

Postoperative
- monitor Ca2+

Adrenocortical Insufficiency

= destruction of the adrenal cortex –
- Glomerulosa = Mineralocortiocids - aldosterone
- Fasiculata = Glucocorticoids - cortisol
- Reticularis = androgens - DHEA, testosterone

Classification

Primary (Addison's Disease)
- causes:
1. autoimmune destruction (80%)
2. infection (TB)
3. septicaemia
4. AIDS
5. haemorrhage
6. metastases
7. surgery

- both glucocorticoid **and** mineralocorticoid deficiency

Secondary
- due to insufficient ACTH to stimulate adrenal cortex
    ↳ caused by pituitary suppression by:
        - exogenous steroids
        - generalised hypopituitarism - tumour
- **only** see glucocorticoid deficiency

Acute Crisis
- crisis ether =
    - chronic insufficiency without adequate steroid cover
    - acute adrenal haemorrhage
    - pituitary apoplexy
        ↳ = sudden neuro impairment usually due to vasc process eg bleed or infarction

Preoperative

HISTORY
- chronic insufficiency symptoms:
    - weakness
    - fatigue (100%)

- skin hyperpigmentation - primary only (90%)
- postural symptoms - pronounced in primary (90%)
- D&V
- weight loss (60%)
- myalgia
- joint pain
- salt craving - primary only
- pale skin - secondary only

EXAMINATION
- postural hypotension
- pallor (secondary)
- pigmentation (primary)

INVESTIGATIONS
- low glucose
- low Na+ (90%)
- raised K+ (70%)
- raised U and Cr (primary only)
- raised Ca2+ (primary only)
- early morning low cortisol (<165nmol/L - primary or <100nmol/L - secondary)
- short synacthen test - peak cortisol <500nmol/L (30mins post 250mcg synacthen)

Table 8.5 Biochemical diagnosis of adrenal insufficiency

Test	Normal range	Definite adrenal insufficiency	
		1°	2°
Early morning cortisol	165–680nmol/L	Cortisol <165nmol/L and ACTH >22.0pmol/L	Cortisol <100nmol/L
Early morning ACTH	1.1–11.0pmol/L		Not diagnostic
Standard short Synacthen® test[1]	Peak cortisol >500nmol/L	Peak cortisol <500nmol/L	Peak cortisol <500nmol/L
Insulin tolerance test[2]	Peak cortisol >500nmol/L		Peak cortisol <500nmol/L

MANAGEMENT
- hydrocortisone 20mg mane, 10mg nocte PO
- fludrocortisone 0.1mg PO (primary only)
- give all medication morning of surgery

Intraoperative
- hydrocortisone 25mg IV @ induction then 5mg/hr as infusion
  ↳ alternatively 100mg IM
- see perioperative steroid guideline

Postoperative
- Q4 hourly glucose
- daily U+E's
- consult endocrinologist but rough steroid cover:
  - should repeat induction steroid strategy 6hrly until eating & drinking
  - then double normal oral steroid dose for:
    - major surgery = 48hr
    - minor surgery = 24hr
  - then return to normal steroid regime
  - any issues return to high dose oral steroids
- replacing mineralocorticoid:

- 20mg of hydrocortisone = 0.05mg fludrocortisone
- ∴ once giving >50mg of hydrocortisone you don't need to worry about fludro supplementation.

Acute Adrenal Crisis (Addisonian Crisis)

- classical presentation:
  - bp
  - ↓Na, ↑K, ↓BSL
  - abdo pain
- looks like hypovolaemic shock but can mimic septic shock ie fever, vasodilation, high CO
- if DM type 1: may present as deterioration of glycaemic control with recurrent hypo's
- Rx:
  - supportive care
  - IVF resus
  - take baseline cortisol & ACTH then give…..
    (↳ if unable then can give 4mg IV dex as doesn't interfere with testing)
  - hydrocort 200mg IV then 100mg qds
  - treat precipitant

Cushing's Syndrome

Preoperative

= syndrome due to excess plasma cortisol

- causes:

1. iatrogenic steroid administration (commonest)
2. pituitary adenoma (80% of remainder)
3. ectopic ACTH (small cell carcinoma) (15% remainder)
4. adrenal adenoma (4%) /carcinoma (rare)

CLINICAL

- appearance: moon face, trunchal obesity, proximal myopathy
- tissue: Osteoporosis, easy bruising, thin skin
- co-morbidities: DM, OSA, GORD, peptic ulcer disease
- CVS: hypertension, LVH, IHD

INVESTIGATIONS

- Diagnosis:
  - dex suppression test (8mg)
  - ACTH level:
    - normal/high ≈ pituitary
    - low ≈ adrenal, ectopic cortisol admin
    - very high ≈ ectopic ACTH
  - plasma cortisol - high with loss of diurnal variation
  - ↑urinary 17-OH-steroids
- Assoc findings:
  - bloods: ↑ Na+,↑K, ↓Ca
  - ↑ HCO3-
- ECG - high voltage QRS, inverted T waves
  ↳ diff to distinguish from IHD but directly related to Cushings
- bp - 80% bp

MANAGEMENT

- treat cause
- DM cares
- steroid supplementation perioperatively if indicated

Intraoperative
- difficult veins
- careful airway assessment
- peptic ulcer prophylaxis
- monitor electrolytes
- pressure area cares

Postoperative
- standard care

Conn's Syndrome

Preoperative
= excessive secretion of aldosterone from either:

1. an adenoma (60%)
2. benign hyperplasia (35-40%)
3. adrenal carcinoma (rare)

- results:

- ↑Na+ reabsorption & ↑K+ excretion ⟹ serum ↑Na, ↓K
- increase in ECF H2O
- tubular secretion of H+ and Mg2+ -> metabolic alkalosis

CLINICALLY
- hypertension
- hypervolaemia
- metabolic alkalosis
- muscle weakness or paralysis from low K+ [late sign]
- polyuria (nephrogenic DI from tubular damage
  ↳ opposite to what might think
- impaired glucose tolerance (50%)

INVESTIGATIONS
- low K+ (<3.5mmol/L)
- low serum aldosterone
- adosterone:renin ratio >400
- imaging: adrenal vein sampling, CT/MRI

MANAGEMENT
- spirinolactone (inhibits aldosterone) – up to 400mg/day
- aim for normal K+ and HCO3-
- exclude end-organ damage from HT
- anti-hypertensives (Ca2+ channel blockers or ACE-I)
- may need adrenalectomy

Intraoperative
- adrenalectomy (open or laparoscopic): phentolamine, MgSO4, BSL monitoring, invasive monitoring
- ↓K intra-op causes:
  - prolonged action of NMBs
  - ↑BSL
  - Suppress baroreceptors
- If bilat adrenalectomy - need to replace mineralocorticoids & glucorticoids

-

Postoperative

- IV hydrocortisone -> PO prednisone + PO fludrocortisone
- monitor and manage BP

Anaesthesia for pt with Conn's syndrome for Non-Adrenal Surgery

- pt has bilat hyperplasia of zona glomerulosa
- HTN more severe ⟹ ↑ing doses of antiHTN required - ACEI useful
- aim restore K prior to theatre

Carcinoid Tumours

Preoperative

= syndrome produced from vasoactive peptide secretion from carcinoid tumours

- derived from argentaffin cells -> produces peptides and amines
- found in: GI tract (75%), bronchus, pancreas and gonads
- Tumours:
  - most benign
  - of malignant ones: only 25% release vasoactive substances into systemic circulation ⟹ carcinoid syndrome
  - mediators metabolised in liver ∴ only produce syndrome if:
    - tumour with hepatic metastasis
    - non-portal venous drainage

  ↳ ∴ about 10% with carcinoid tumours have syndrome
- vasoactive substances: serotonin, bradykinin, histamine, substance P, prostaglandins, vasoactive intestinal peptide
- Summary:
  - asymptomatic carcinoid tumour have simple disease & no specific anaesthetic issues
  - it is carcinoid syndrome ⟹ major problems

Carcinoid Syndrome

- affects 10% with carcinoid tumours
- symptoms from:
  - mass effect from primary tumour eg bowel obstruction, pulmonary symptoms eg haemoptysis
  - vasoactive peptides:
    - flushing 90% (esp head/neck/torso)
    - diarrhoea 78% ⟹ dehydration & electrolyte imbalance
    - bronchospasm 20%
    - CVS: ↓↑bp, ↑HR, R heart failure (endocardial fibrosis of PV & TV)
    - ↑BSL ↳ mediators metab'ed in lung prior to reaching L heart

INVESTIGATIONS

- dependent on symptoms
- blds: FBC, UEs, LFTs, coag
- cross match
- ECG ?RV hypertrophy
- ECHO
- CXR
- spirometry

MANAGEMENT

- symptomatic: antidiarrhoeals, bronchodilators, hydration, replace electrolytes, heart failure
- octreotide 100mcg SC tds for 2 weeks prior to surgery (prevents mediator release)

- avoid factors that may produce crisis = stress, catecholamines, histamine releasing drugs (morphine)

Intraoperative

- octreotide 100mcg IV @ induction (if not already treated)
- tertiary referral
- be ready for
    - severe hypo/hypertension
    - explosive bronchospasm
    - electrolyte problems
    - fluid shifts
- monitor electrolytes
- invasive monitoring
- consider epidural:
    - ↓stress response from surgery ⟹ ↓carcinoid crisis
    - careful LA top up as ↓bp ⟹ bradykinergic crisis
- prevent pressor response to intubation
- sux has be used safely
- TIVA or volatile
- octreotide boluses 10-20mcg boluses for severe hypotension
- avoid all histamine releasing drugs
- labetalol, esmolol good for hypertension

Postoperative

- ICU or HDU
- may wake very slowly (thought 2nd to serotonin)
- PCA fentanyl or epidural
- be ready for hypotensive episodes ⟹ IV octreotide 10-20mcg
- wean octreotide over 7 days post resection

Gastrinoma

= excess production of gastrin by benign adenoma, malignancy or hyperplasia for D pancreatic islet cell
- gastrin ⟹ ↑acid production from parietal cells

CLINICALLY

- may produce
    - Zollinger-Ellison syndrome,
    - severe peptic ulceration
    - diarrhoea
- GI bleeds
- GI perforation
- electrolyte disturbance
- volume depletion

INVESTIGATIONS

- FBC
- coag's (vit K may not be absorbed)
- LFT's

MANAGEMENT

- PPI
- H2 antagonists
- octreotide
- invasive monitoring

VIPoma

= rare tumour secreting vasoactive intestinal peptide (VIP) – inhibits gastrin release

CLINICALLY

- profuse watery diarrhoea
- intestinal ileus
- abdominal distension
- confusion
- drowsiness
- tetany

INVESTIGATIONS

- ↓K, ↓Cl, ↓Mg2+
- ↑BSL
- metabolic alkalosis

MANAGEMENT

- 60% become malignant with liver mets so all warrant resection
- give H2 antagonists prior to surgical excision c/o rebound gastric hypersecretion
- IVF
- octreotide - if fails ⟹ methylprednisolone & indomethacin
- invasive monitoring
- monitor electrolytes
- monitor ABG's

Insulinoma

= rare tumour of beta cells of pancreas that secrete insulin

CLINICALLY

- symptoms of hypoglycaemia

INVESTIGATIONS

- low plasma glucose (<2.2mmol/L)
- increased insulin
- increased C peptide
- absence of sulphonylurea in plasma

MANAGEMENT

- if surgery fails try diazoxide (= non-diuretic benzothiazide which inhibits release of insulin) -> unpredictable efficacy
- monitor glucose and treat

Glucagonoma

= tumour of alpha cells of pancreas -> increased glycogenolysis, gluconeogenesis and increased plasma glucose -> DM

- ketogenesis rare as insulin also increased

CLINICALLY

- rash (necrotising migratory erythema in groin/perineum and migrates to distal extremities)
- weight loss
- glossitis
- stomatitis
- anaemia
- diarrhoea
- liver met

INVESTIGATIONS
- BSL
- MRI
- glycogen level

MANAGEMENT
- debulk tumour
- somatostatin analogues
- increased risk of DVT -> prophylax

Electrolytes

Hypokalaemia

- Hypokalaemia = K+ <3.5

Mild - 3.0-3.5
Moderate – 2.5-3.0
Severe - <2.5

Causes:

- decreased intake
- increased loss – vomiting, diarrhoea, N/G loss, diuretics, RTA, hyperaldosteronism, Mg depletion, leukaemia
- intercompartmental shift – insulin, salbutamol, steroids, alkalosis

CLINICALLY

- weakness, tetnay, ileus
- ECG – T wave flattening and inversion, prominent U waves, ST depression, prolonged PR interval
- dysrrhythmias, decreased contractility
- decreased insulin, GH and ALD secretion

MANAGEMENT

- check U+E & glucose
- Must replace Mg before K
- exclude Cushings (overnight dexamethasone suppression and 24hr cortisol) and Conns (aldosterone and cortisol)
- oral or IV replacement - aim for K >4 if arrhythmia:
    - Max conc
        - peripheral IV = 40mmol/L
        - central IV = 40mmol in 100ml saline/1hr
    - K depletion of 0.3mmol/L = total body loss of ~100mmol from total store
- ?on digoxin ⟹ aim for K >4 as ↑risk of dig toxicity at low K levels
- risk of arrhythmia
- prolongation of NMB

- preop requirements:
    - elective = K>3 - otherwise postpone (although controversial and should consider acute change in K and other co-morbidities)
    - emergency = K >3.5.
        - Aim to replace K in 24hrs prior to surgery
        - check HCO3 - if ↑ed then likely chronic ↓k ≈ low intracellular K ⟹ may take days to replace

Hyperkalaemia

K+ > 5.5 mmol/L

Mild – 5.5-6.0
Moderate – 6.0-7.0
Severe >7.0

Causes

- increased intake – IV administration or rapid blood transfusion
- decreased urinary excretion – renal failure, adrenocortical insufficiency, drugs (K+ sparring diuretics, ACEI, cyclosporine)
- intercompartmental shift – acidosis, rhabdomylosis, trauma, MH, sux, familial periodic paralysis, tumour lysis syndrome
- pseudohyperkalaemia ie lab error/haemolysis

CLINICALLY
- nausea, vomiting, diarrhoea
- ECG: peaked T waves -> widen QRS -> prolonged PR -> loss of P waves -> loss of R wave amplitude -> ST depression -> VF -> asystole
- mm weakness >8

MANAGEMENT
- treat cause
- treat urgently if K >6.5 or ECG changes:
    - insulin
        - 10U in 150mL of 10% dextrose over 30min
        - fastest onset of action
        - shifts K into cells
        - rebound occurs within 2hrs
    - salbutamol: response by ~30min. DUration action >2hrs
    - calcium
        - gluconate 10% 10mL, chloride 10% 5mL
        - stabilises myocardium by ↑threshold potential
        - rapid onset, short lived effect
    - HCO3- if acidotic (50mmol)
    - calcium resonium - oral 15g PO 8hrly to bind K in gut
- haemofiltration/dialysis
- avoid saline - acid load has bigger effect on serum K than that in Plasmalyte
- avoid sux
- avoid hypothermia
- avoid resp acidosis
- monitor K+

Hyponatraemia
- = Na+ < 135:
    - Mild 125-134
    - Moderate 120-124
    - Severe <120
- ECF volume proportional to total body Na content
- renal Na excretion controls ECF volume ∴ total body Na

CLINICALLY
- speed of onset more important than level
- 125-130 = mostly Gi symptoms is D&V
- <125 =
    - neuropsychiatric symptoms
    - muscular weakness
    - headache
    - lethargy
    - psychosis
    - raised ICP
    - seizures
    - coma

DIAGNOSIS
- diagnose based on fluid status:
    - Hypovolaemic:
        - Urinary Na <30mmol/L ≈ extrarenal cause ie D&V, burns, pancreatitis, trauma
        - Urinary Na >30mmol/L ≈ primary renal problem ie diuretic/osmotic fluid losses, mineralcorticoid deficiency (addisons), salt wasting nephropathy, prox renal tubular acidosis
    - Euvolaemic (psychogenic polydipsia, hypotonic IVF, bowel prep, SIADH)
    - Hypervolaemic (renal failure, hepatic cirrhosis, CHF, nephrotic syndrome, TURP syndrome)

Drugs!

MANAGEMENT
- treat cause
- acuteness of change important rather than actual level
- rare to have signs >125mmol/L
- no elective surgery until
    - Na+ > 120mmol/L **and**
    - asymptomatic

- **asymptomatic** -> fluid restrict to 1L/day

- **asymptomatic hyponatraemia** (often chronic)
    - fluid restrict to 1L/d
    - treat cause

- **acute and symptomatic** (<48hrs)
- raise by 2mmol/L/hr until symptoms resolve
- hypertonic saline (3%)
    - 1-2ml/kg/hr & measure Na hourly
    - if severe neuro symptoms ⟹ ↑rate to 4-6ml/kg/hr
- if fluid excess then give frusemide 20mg IV

- **chronic symptomatic** (>48 hrs or unknown duration)
- correct Na+ by 5-10mmol/L/day
    ↳ faster correct ⟹ central pontine myelinolysis, subdural haemorrhage, cardiac failure
- if hypovolaemia: correct with 0.9% NSL
- if hypervolaemic:
    - fluid restrict
    - frusemide 20mg IV
- if SIADH:
    - fluid restrict
    - give demeclocycline 300mcg daily
- monitor electrolytes ev 12hrs

Anaesthesia
- postpone elective surgery <120 or symptomatic
- if emergency consider risks/benefits & consult endocrinologist

Hypernatraemia

- = Na+ > 145
    - Mild 145-150
    - Moderate 151-160
    - Severe >160

CAUSES
- define on fluid status:
    - hypovolaemic =
        - renal - diuretics, intrinsic renal disease, post obstruction
        - extra-renal - D&V, burns, excessive sweating, fistulae
    - euvolaemia = DI, insensible losses
    - hypervolaemic:
        - Salt ingestion/hypertonic saline, Conn syndrome, Cushings syndrome

CLINCALLY
- CNS symptoms if >155 - due to hyperosmolar state & cell dehydration:
    - thirst, confusion, seizures, coma

MANAGEMENT

- treat cause
- correct over 48 hours
- use oral route if possible - give free water
- specifics:
    - hypovolaemia (Na deficiency) ⟹ balanced crystalloid until corrected
    - euvolaemia (water depletion) ⟹ estimate TBW deficit ⟹ give 5% glucose
    - hypervolaemia (Na excess) ⟹ diuretics & 5% glucose +/- dialysis
    - DI: replace urinary losses, desmopressin 1-4mcg daily

Anaesthesia

- no elective surgery until Na+ <155 or normovolaemic
- invasive monitoring