The Adrenal Glands

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Adrenal glands: anatomy

- Two adrenal glands
- Triangle-shaped endocrine glands
- Located superior and medial to the kidneys
Adrenal glands: anatomy

- Two distinct regions
  - Inner medulla
  - Outer cortex
- Functionally two different endocrine organs
- Different embryological origins

Adrenal cortex
- Secretes steroid hormones
  - Glucocorticoid
  - Mineralocorticoid
  - Androgens

Adrenal medulla
- Secretes Catecholamines
  - Adrenaline
  - Noradrenaline
**Function**

- Chiefly responsible for regulating stress response
  - through synthesis of corticosteroids and catecholamines
- Certain diseases (TB / cancer / AIDS) can impact production of these hormones
  - Leading to diseased states

**Adrenal medulla**

- Centre of adrenal gland, surrounded by adrenal cortex
- Synthesize and secrete catecholamines
Physiologic Effects

Medullary Hormones
- Catecholamines → adrenaline and noradrenaline
- Common stimuli for hormone secretion → exercise → hypoglycaemia → haemorrhage → emotional distress

Physiologic responses aid in dealing with stress
- ↑ rate and contraction of heart muscle: adrenaline through β-receptors
- Blood vessels constriction: noradrenaline → ↑ BP
- Dilation of bronchioles: ↑ pulmonary ventilation
- ↑ metabolic rate: ↑ oxygen consumption and heat production
- Stimulation of lipolysis in fat cells: FFA for energy
- Breakdown of glycogen in skeletal muscle: glucose for energy
- Pupil dilatation
- Inhibition of "non-essential" processes: i.e. gastrointestinal secretion and motor activity

**Cortex**

Divided into **3 layers** (zones) of cells → each secrete different steroid hormones

- The zona glomerulosa
  - Outer zone → Secretes **aldosterone**

- The zona fasciculata
  - Middle zone → Secretes **glucocorticoids** and **androgens**

- The zona reticularis
  - Inner zone → Secretes mostly **androgens**, also **glucocorticoids**
Aldosterone (= Mineralocorticoid)

- Principal steroid with mineralocorticoid activity is aldosterone
- Control multifactorial
  - Activation of RAAS (renin-angiotensin-aldosterone system)

  - Net effect: \( \uparrow \text{Na} \downarrow \text{K} \)

Physiologic Effects

- Critical role \( \rightarrow \) \( \text{Na}^+ / \text{K}^+ / \text{H}_2\text{O} \) homeostasis
  - Life-threatening electrolyte / fluid balance abnormalities if absent
- Three primary effects of aldosterone result in:
  - Increased renal resorption of sodium
  - Increased resorption of water \( \rightarrow \) expansion of ESV
  - Increased renal excretion of potassium
**Cortisol (= Glucocorticoid)**

- Majority of glucocorticoid activity from cortisol
- Corticotropin Releasing Hormone (Hypothalamus) → ACTH (Pituitary) → Cortisol (Adrenal)

**Physiological effects (1)**

- Intermediate Metabolism (↑substrates for energy)
  - Stimulation of gluconeogenesis (liver)
  - Inhibition of glucose uptake in muscle and adipose tissue
  - Inhibit insulin secretion
  - Lipolysis of adipose tissue - fatty acids → substrate for energy
  - Breakdown of skeletal muscle → amino-acids
### Physiological effects (2)

- **Electrolytes**
  - Weak mineralocorticoid effect $\rightarrow$ ↑ sodium
  - ↑ ESV $\downarrow$ Potassium
  - Role in calcium homeostasis

- **Immune regulation**
  - Suppresses inflammatory response

- **Psychiatric effects**
  - Maintains emotional balance
  - Suppresses REM sleep

### Pathology

- **Removal of adrenal glands $\rightarrow$ fatal**
  - ↑ potassium in extracellular fluid
  - ↑ urinary excretion of sodium, ↓ sodium in ESV
  - Volume of extracellular fluid and blood decreases
  - Cardiac output declines and shock ensues

- **Direct result of loss of mineralocorticoid activity**
  - Acutely critical for maintenance of life

- **Prevented by replacement of salts and mineralocorticoids**
Pathology

- Pathology can occur within or outside the adrenal glands
  - within pituitary and/or hypothalamus → affect functioning of adrenal glands
- Can result in excess production (hyperfunctioning) or decreased production (hypofunctioning) of secreted hormones

Hyperfunctioning States

- Medulla
  - Phaeochromocytoma
- Cortex
  - Hyperaldosteronism
  - Cortisol excess → Cushing’s syndrome
  - Androgen excess → Hirsutism, virilization
<table>
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<td>■ BILATERAL ADRENAL HYPERPLASIA (BAH)</td>
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## Symptoms and Signs

Hypersecretion of aldosterone may result in:
- Hypernatraemia
- Hypervolaemia
- Hypertension
- Hypokalaemic alkalosis manifested by:
  - Episodic weakness
  - Paraesthesias
  - Transient paralysis
  - Hypokalaemic nephropathy with polyuria and polydipsia
- Low renin levels

## Hyperaldosteronism

- Excess production of aldosterone by abnormal glomerulosa tissue → hyperaldosteronism
- Primary hyperaldosteronism
  - Conn’s adenoma
  - Bilateral adrenal hyperplasia
Conn's Syndrome

- Benign aldosterone producing adenoma
- Originating from the glomerulosa cells of the adrenal cortex
- Unilateral
- Very rarely due to adrenal adenocarcinomas

Adrenal hyperplasia

- Bilateral micro- or macronodular hyperplasia of adrenal glands
- Rarely unilateral
- Often milder form of primary hyperaldosteronism
**Diagnosis**

- Differentiating between Conn’s and BAH
  - Important for treatment decision
- Biochemical differentiation imprecise
  - Similar findings on biochemistry:
    - low K (with alkalosis)
    - high aldosterone
    - low renin
    - high aldosterone / renin ratio

**Imaging**

- CT (adrenal protocol) used to differentiate
  - Diagnosis of adrenal hyperplasia made by excluding presence of adenoma on imaging
  - Sometimes bilateral adrenal nodules and enlargement noted
- Adrenal venous sampling is indicated for further evaluation
  - Adrenal vein samples taken bilateral for aldosterone
    - Adenoma - ↑ adenoma side ↓ opposite
    - BAH - ↑ bilateral
Treatment

- Aldosterone-producing adenoma
  - Surgical removal
    - unilateral adrenalectomy
- Bilateral adrenal hyperplasia
  - managed medically
    - spironolactone

Secondary Aldosteronism

- Increased production of aldosterone by adrenal cortex, due to RAAS stimulation

- Mostly due to:
  - cardiac failure
  - cirrhosis with ascites
  - nephrotic syndrome
  - accelerated hypertension
Primary vs Secondary Hyperaldosteronism

Congenital adrenal hyperplasia

- Group of heritable disorders associated with inability or deficiency in ability to produce cortisol
- Begins early in gestation, manifest at birth
- No negative feedback → excessive secretion of corticotropin-releasing hormone from hypothalamus and ACTH from the pituitary
  - Continued stimulation of the adrenal cortex, leading to hyperplasia
- Involves aldosterone (too little) → results in mild to severe loss of body sodium
Clinical presentation

At birth
- Can involve overproduction of adrenal androgens
  - Affected females → prenatal virilization with an ambiguous or male-like external genitalia
- Hyponatraemia and dehydration

Presentation

Childhood
- Pubarche and advanced growth

Adolescent girls
- Oligo-Amenorrhoea, acne, hirsutism, androgenic alopecia

Adolescent boys
- Early beard growth, acne and early growth spurt

Adult
- Infertility
- Needs glucocorticoid replacement and anti-androgen therapy
Liquorice Intoxication

- Syndrome of water and sodium retention with low plasma potassium due to excessive licorice ingestion
- Liquorice contains glycyrrhizinic acid
  - inhibits an enzyme needed to inactivate cortisol in mineralocorticoid target cells
- Net effect is similar to aldosterone excess

Hypercortisolism
CUSHING’S SYNDROME
Cushing’s syndrome

- First described by Cushing in 1932
  - Constellation of clinical abnormalities due to chronic exposure to excesses of cortisol

Aetiology
- Exogenous (iatrogenic, factitious)
  - ACTH - dependent:
    - Pituitary adenoma 70 - 80%
    - Ectopic ACTH syndrome 15%
  - ACTH - independent:
    - Adrenal adenoma 10%
    - Adrenal carcinoma 5%
    - Bilateral macronodular hyperplasia (rare)

Clinical manifestations

Nonspecific findings:
- Truncal obesity
- Supraclavicular & dorsal fat pads →Buffalo hump
- Hypertension
- Hirsutism, acne
- Amenorrhoea
- Depression
- Growth failure

More specific findings:
- Rounded "moon" facies
- Thin and atrophic skin
- Poor wound healing
- Easy bruising
- Red/purple striae on abdomen
- Facial plethora
- Slender distal extremities
- Muscle wasting & weakness
- Osteoporosis
Moon facies and increased supraclavicular fat pads in Cushing's syndrome

30-year-old woman with Cushing's disease showing round, plethoric "moon" face, facial hirsutism, and increased supraclavicular fat pads.


Buffalo hump in Cushing's syndrome

Side view of a patient with Cushing's syndrome showing a dorsocervical fat pad ("buffalo hump").

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Striae in Cushing’s disease

Axillary and lower abdominal striae in a 21-year-old man with Cushing’s disease. Abdominal obesity is also present.

Courtesy of David N Orth, MD.

Centripetal obesity in Cushing’s syndrome

30-year-old woman with Cushing’s disease showing centripetal obesity, relatively thin limbs, dorsal kyphosis, and thick neck.

Reproduced with permission from Williams Textbook of Endocrinology, 8th ed, Foster, DW, Wilson, JD (Eds), WB Saunders, Philadelphia, 1996.
**Other features**

- Glucose intolerance
- Reduced resistance to infection
- Cessation of linear growth
- Females usually have menstrual irregularities
- In adrenal tumours increased production of androgens in addition to cortisol lead to:
  - Hypertrichosis (hirsutism)
  - Temporal balding
  - Other signs of virilization in the female
Cushings Syndrome

- **Cushings disease**
  - Hypercortisolism due to pituitary ACTH hyper-secretion
- **Always exclude iatrogenic cause first**

**Diagnosis**

**Screening tests for elevated cortisol:**
- Overnight low dose Dexamethasone suppression test (1mg po at 24h00): 8h00 cortisol should be <50nmol/l
- 24h urinary cortisol
- Diurnal rhythm of cortisol 23h00 level (suspect Cushing's if midnight cortisol is > 75% of 08h00 level)

**If Cushing's not excluded:**
- Prolonged low dose dexamethasone suppression test (0.5mg po 6 hourly x 48 hours)

**Evaluate aetiology:**
- Plasma ACTH
- CT-adrenals (if ACTH low)
- Pituitary MRI scan +/- Inferior petrosal sinus sampling (if ACTH high)
Treatment

Pituitary tumour
- Selective transsphenoidal resection
- Radiation therapy
- Cortisol synthesis inhibitors:
  - Ketoconazole
  - Metyrapone
  - Mitotane
- Bilateral adrenalectomy

Adrenal neoplasm
- Resection
- Medical treatment to inhibit steroid synthesis

Ectopic ACTH
- Surgical resection if possible
- Cortisol synthesis inhibitors
- Bilateral adrenalectomy
### Pseudo-Cushing’s syndrome

- Illness
- Depression
- Alcoholism
- Anorexia nervosa

### Adrenal insufficiency

ADDISON’S DISEASE
### Introduction

- Adrenocortical insufficiency due to destruction or dysfunction of entire adrenal cortex
- Can affect both glucocorticoid and mineralocorticoid function
- The onset of disease usually occurs when 90% or more of both adrenal cortices are dysfunctional or destroyed

### Aetiology

- Autoimmune disease most common: 70-90%
- TB: previously common cause, now 7-20% of cases
- Other causes:
  - Infectious diseases: histoplasmosis, blastomycosis, and cryptococcosis
  - Sarcoidosis
  - Infiltrative causes
  - Malignancy: metastatic cancer / lymphoma
  - Adrenal haemorrhage / infarction
  - Drugs
Autoimmune adrenalitis

Idiopathic autoimmune adrenocortical insufficiency:
- Most common cause (70-90% of reported cases)
- Isolated or association with other autoimmune disease
- Often associated with autoimmune destruction of other endocrine glands (50%)
- Autoimmune polyendocrine syndrome type 2 (Schmidt syndrome)
- Autoimmune polyendocrine syndrome type 1

AIDS

Adrenal insufficiency as a result of invasion of:
- MAC
- Cryptococci
- Metastatic Kaposi’s sarcoma
- Peripheral glucocorticoid resistance
- Drugs
Clinical presentation

- Symptoms and signs depend upon:
  - Rate and extent of loss of adrenal function
  - Whether mineralocorticoid production is preserved
  - Degree of stress
- Onset often is insidious and nonspecific
- May go undetected until an illness or stress precipitates an adrenal crisis

Symptoms

- Weakness & fatigue
- Anorexia & weight loss
- Nausea & vomiting
- Dizziness and orthostatic symptoms
- Lethargy, stupor
- Hyponatraemia
- Hyperkalaemia
- Hypotension
- Hypoglycaemia
- Shock & death
- Hyperpigmentation
Hyperpigmentation in primary adrenal insufficiency

Thirty-two-year-old man with Addison’s disease caused by tuberculosis with generalized hyperpigmentation, most marked on areas exposed to sunlight, such as face and neck.

Courtesy of David N Orth, MD.

Buccal hyperpigmentation due to ACTH excess

Lips and gums of a 32-year-old man demonstrating hyperpigmentation of the buccal mucosa along the line of dental occlusion (an area of repeated trauma) and of the gums (in the area of chronic inflammatory periodontal disease). The high plasma ACTH concentrations responsible for the hyperpigmentation were due in this case to primary adrenal insufficiency; similar changes can be seen in patients with ACTH-dependent Cushing’s syndrome or Nelson’s syndrome.

Reprinted with permission from Williams Textbook of Endocrinology, 8th ed, Foster, DW, Wilson, JD (Eds), WB Saunders, Philadelphia, 1996.
Hyperpigmentation in primary adrenal insufficiency

Eighteen-year-old white woman with autoimmune Addison’s disease associated with polyglandular autoimmune syndrome type 2. Panel A: The patient has marked generalized hyperpigmentation compared with the complexion of a normal nurse. Panel B: The hands demonstrate deep pigmentation of the dorsal surface and increased pigmentation of the palmar creases. There is minimal accentuation of the hyperpigmentation over the knuckles, presumably because she is so deeply pigmented elsewhere. There are also small areas of villi near the base of the left thumb, a strong clinical clue that the adrenal disease has an autoimmune etiology.


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Hyperpigmentation of nails in primary adrenal insufficiency

Fingers of a 28-year-old white woman with Addison’s disease (underneath) compared to those of a normal woman (top). There is hyperpigmentation of the skin and increased pigmentation of the distal half of the nails that occurred during the period of adrenal insufficiency. The proximal half of the nails are hypopigmented, a reflection of the reduction in ACTH secretion after the institution of glucocorticoid therapy.

Courtesy of David N Orth, MD.

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Other features

- Dizziness with orthostasis
  - Due to volume depletion and hypotension
  - May lead to syncope
  - Patients may show evidence of dehydration
- Myalgias and flaccid muscle paralysis
  - Due to hyperkalaemia
- Absence of axillary /pubic hair, decreased body hair
  - Due to loss of adrenal androgens
  - Severe GIT symptoms, with clinically acute abdomen

Axial, noncontrast-enhanced CT scan through the upper abdomen of a 42-year-old woman shows irregular peripheral calcification in a soft tissue mass immediately anterior and superior to the right kidney (arrow). These findings are consistent with chronic hemorrhage of the right adrenal gland.

Courtesy of Jonathan Kruskal, MD.
Laboratory confirmation

Three stage process:
- Demonstrate inappropriately low cortisol secretion
- Determine whether it is ACTH dependent or not
- Seek a treatable cause

Low cortisol determination

- Early morning cortisol level
  - <80 nmol/L = adrenal insufficiency
  - > 415 nmol/L (some books: 460): excludes adrenal insufficiency in virtually all patients

- ACTH stimulation test
  - Synthetic ACTH (0.25 mg) is given IM or IV
  - 0, 30 and 60 minutes cortisol taken
Evaluate ACTH

- Distinguish
  - Addison disease → Primary adrenal pathology (high ACTH, low cortisol)
  - from Secondary adrenocortical insufficiency (low ACTH, low cortisol)

Seek cause

- Chest x-ray:
  - Evidence of TB or fungal infection
- CT scan:
  - Often normal
  - May show bilateral enlargement of the adrenal glands or calcifications because of TB, fungal infections, adrenal haemorrhage, or infiltrating diseases
  - In idiopathic autoimmune Addison disease, the adrenal glands are usually atrophic
Management

- Glucocorticoid replacement
- Mineralocorticoid replacement
- Androgen replacement in women
- Illness or surgery
- Pregnancy
- Medic alert bracelet

Treatment of chronic primary adrenal insufficiency

Glucocorticoid replacement
1. Desamethasone 0.5 (2.5-4.75) mg or prednisone 5 (2.5-7.5) mg orally at bedtime, supplemented with hydrocortisone 5-10 mg orally in mid-afternoon if necessary.
2. Alternative therapy is with hydrocortisone 15-20 mg upon awakening and 5-10 mg in early afternoon.
3. Monitor clinical symptoms and morning plasma ACTH.

Mineralocorticoid replacement
1. Fluorocortisone 0.1 (0.05-0.2) mg orally.
2. Liberal salt intake.
3. Monitor lying and standing blood pressure and pulse, edema, serum potassium, and plasma renin activity.

Androgen replacement
1. Deltamethrinolone 25-50 mg orally in women.

Patient education
1. Educate patient about the disease, how to manage minor illnesses and major illnesses and how to inject desamethasone intramuscularly.

Emergency precautions
1. Obtain Multi-effect brucella, bacta, Emergency Medical Information Card, and prefilled syringes containing desamethasone 4 mg in 1 mL saline.

Treatment of minor febrile illness or stress
1. Increase glucocorticoid dose 2 to 3 fold for the few days of illness. Do not change mineralocorticoid dose.
2. Contact physician if illness worsens or persists for more than 3 d.
3. No extra supplementation is needed for most uncomplicated, outpatient dental procedures under local anesthesia. General anesthesia or intravenous sedation should not be used in the office.

Emergency treatment of severe stress or trauma
1. Inject contents of prefilled desamethasone (4 mg) syringe intramuscularly.
2. Get to physician as quickly as possible.
Endocrine hypertension

- Idiopathic (primary/ essential) HT
  - approximately 90% of diagnosed cases

- Identifiable conditions (secondary HT)
  - approximately 10% of hypertensive patients
    - Primary renal disease (vascular / parenchymal)
    - Drug use i.e oral contraceptive use / NSAIDS / sympathomimetics / anabolic or corticosteroids / alcohol
    - Sleep apnoea syndrome / Obesity
    - Congenital / acquired CVD (coarctation of aorta)
    - Pregnancy
    - Excess hormonal secretion → Endocrine HT
### Endocrine hypertension

- Approximately 3% of secondary forms of HT
- States in which hormonal derangements result in clinically significant HT
- Most common causes
  - Mineralocorticoids (Hyperaldosteronism)
  - Glucocorticoids (Cushing's syndrome)
  - Catecholamines (Phaeochromocytoma)
  - Thyroid disorders (hyperthyroidism or hypothyroidism)
  - Hyperparathyroidism
  - Acromegaly

### Catecholamines

- Adrenaline
- Noradrenaline
- Both act by binding to cell-surface adrenergic receptors (α or β) which are widely distributed
- Leads to increase in blood pressure
**Phaeochromocytoma (UK)
Phaeochromocytoma (USA)**

- Tumour in adrenal medulla which secretes catecholamines
  - adrenaline and noradrenaline
- Phaeochromocytoma
  - 80-85% located in adrenal medulla
- Paraganglioma (extra-adrenal phaeochromocytoma)
  - Originate in extra-adrenal sympathetic chain/chromaffin tissue
  - Thoracic, mediastinal, abdominal or pelvic locations
- Ganglioneuroma
  - Behave like paraganglioma biochemically

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**Phaeochromocytoma**

- 0.01-0.1% of hypertensive population
  - Found in 0.5% of those screened
  - 0.05% Prevalence (Autopsy)
- 5% Adrenal incidentalomas
- 50% MEN2
- M = F
- 3rd to 5th decades of life
### Clinical features

- Non-specific, leads to delay (3 yrs) between onset of symptoms and diagnosis
- Symptoms due to direct action of secreted catecholamines
- Results in HT, with classic triad of episodic headaches, sweating and palpitations
- Hypertension
  - Most common presenting feature
  - Usually resistant to conventional treatment
  - Sustained or paroxysmal in nature

### Phaeo: Paroxysms / ‘Spells’

- Due to episodic nature of catecholamine secretion
- 10-60 min duration, frequency: daily to monthly
- Spontaneous or Precipitated:
  - Diagnostic procedures, Contrast, Anaesthesia
  - Drugs Strenuous exercise, movement that increases intra-abdominal pressure (lifting, straining), abdominal palpation
  - Micturition (bladder paraganglioma)
- Episodes may be serious and can potentially be fatal due to cardiovascular complications
### Phaeo: Signs & Symptoms

- Paroxysms of:
  - pallor (or flushing)
  - palpitations
  - sweating
  - headache
  - anxiety / fear of death

### Phaeo: Signs & Symptoms (cont)

- Hypertension (can be paroxysmal); Cardiac dysrhythmia & conduction defects; CCF
- Angina/MI with normal coronaries
- N/V, abdominal pain, severe constipation, megacolon
- Psychiatric disorders, anxiety or panic
- Pallor, visual blurring, flushing, papilloedema, polyuria, polydipsia, dyspnoea, general weakness
- Weight loss; glucose intolerance
- Increased ESR, leucocytosis, rarely secondary erythrocytosis due to overproduction of erythropoietin
### Phaeo: ‘Rule of 10’?

- 10% extra-adrenal (closer to 15%)
- 10% occur in children
- 10% familial (closer to 25%)
- 10% bilateral or multiple (more if familial)
- 10% recur (more if extra-adrenal)
- 10% malignant
- 10% discovered incidentally

### Familial Phaeochromocytoma

- MEN type 2a (Multiple Endocrine Neoplasia)
- MEN type 2b
- Von Hippel-Landau disease
- Neurofibromatosis (Von Recklinghausen's disease)
## Diagnosis

Confirming the diagnosis
- biochemical evidence of inappropriate catecholamine production
  - 24h urine collections
  - Plasma

## 24h Urine Collection

- Traditional:
  Creatinine, catecholamines, metanephrines, vanillylmandelic acid (VMA), +/- dopamine
- Superior test:
  Fractionated metanephrines urine (normetanephrine) and catecholamines
- Positive results (> 2-3 fold elevation)
- Chromogranin A: Insufficient sensitivity /specificity → no added benefit over catecholamines or metabolites for diagnosis
Imaging Procedures

Anatomical imaging studies:
- CT-scan
- MRI

Functional imaging:
- $^{123}\text{I}$ or $^{131}\text{I}$ labelled metaiodobenzylguanidine (MIBG)
- $^{111}\text{In}$odium-pentreotide/ octreotide
- PET Position emission tomography
Management

- Prior to 1951, reported mortality for excision of phaeochromocytoma 24 - 50 %
- Currently, mortality: 0 - 2.7 %
- Experienced & Coordinated team:
  - Endocrinologist, Anaesthesiologist and Surgeon

- Preoperative
- Operative
- Postoperative
Preoperative

- Patients should be adequately prepared to prevent threatening catecholamine induced complications
- Combined $\alpha + \beta$ blockade (*1st $\alpha$ blockade x 1 week!*)
  - Phenoxybenzamine, or:
  - Selective $\alpha_1$-blocker (ex. Prazosin, Doxazosin)
  - $\beta$- Propanolol ($\beta$ blocker)
- Calcium Channel Blocker (CCB)
- Salt load: NaCl 600 mg daily-tds as tolerated
- No Randomized Clinical Trials to compare various regimens!

O.R.

- Admit night before for overnight IV saline
- Arterial line, ECG monitor, CVP line
- Regardless of pre-op medications:
  - Have ready: IV phentolamine, IV nitroprusside, IV esmolol
  - Rx hypotension with crystalloids +/- colloid 1st
  - Inotropes may not work!
- Laparoscopic adrenalectomy if tumour < 8cm
Post-op

- Most cases can stop all BP meds post-op
  - Post-op hypotension: IV crystalloid
  - HTN free:  - 5 years 74%
    - 10 years 45%
- 24h urine collection 2 weeks post-op
- Surveillance:
  - 24h urine collections yearly for at least 10 years
  - Lifelong follow-up

When to screen for 2° HT:

- Clinical signs and symptoms of a specific disorder
- Biochemical features in keeping with an underlying disorder
- Hypertension in young patients
  - = < 30 years
- Refractory hypertension
  - (poorly controlled BP on > 3 anti HT drugs
- Accelerated hypertension
- (HT starting in the elderly)