Hypertension

HISTORY:

Ask about FAMILY:
- **Hypertension**
- **Heart disease or STROKE**
- **Diabetes, GOUT, hyperlipidaemia**
- **Kidney disease**

Ask about LIFESTYLE VICE
- **Salt intake**
- **Fat intake**
- **Obesity**
- **Alcohol use**
- **Tobacco Use**
- **Cocaine**
- **Methamphetamine**
- **Lead Exposure Risk**

Ask about ENDOCRINE DISEASE
- **Diabetes Mellitus**
- **Hyperthyroidism**
- **Hypothyroidism**
- **Hyperparathyroidism**
- **Cushing's Disease**
- **Aldosteronism**
- **Pheochromocytoma**

Ask about CARDIOVASCULAR DISEASE
- **Myocardial Infarct**
- **Claudication**

Medications causing HT
- Decongestants
- Nose drops
- Appetite suppressants
- Thyroid Replacement
- NSAIDs
- Stimulant Medications or drugs
- Ergonomic aids (athletes)
- Cocaine
- Herbals containing Ephedra
- Sodium retaining agents
- Oral Contraceptives (in 5% of users) Estrogen Replacement Therapy
- Licorice
- High Sodium Antacids
- Mineralocorticoids
- Glucocorticoids
- Anabolic steroids
- Antidepressants
- Cyclosporine (significantly raises Blood Pressure)
- Erythropoietin
- Growth hormone
- Herbals Affecting Blood Pressure

PHYSICAL EXAMINATION

Neck Exam
- Thyroid exam
- Carotid Bruits
- Neck vein exam

Chest exam
- Congestive Heart Failure signs
- Palpable intercostal pulses

Cardiovascular Exam
- S4 Gallop rhythm (decreased LV compliance)
- Tachycardia
- Accentuated S2 Heart Sound
- Aortic Insufficiency murmur

Abdominal Exam
- Abdominal bruit
- Abdominal Aortic Aneurysm
- Enlarged or tender kidneys (CVA pain)

Peripheral Vascular Disease
- Femoral bruits
- Symmetrical pulses
- Lower extremity shin Hair Loss
- Peripheral neuropathy
- Radio-femoral delay

!! FUNDOSCOPY !! characteristic changes →

Hypertensive Retinopathy
Grading of Hypertensive funduscopic changes
Grade 1: spasm of vessels
Grade 2: Arteriovenous "nipping"
Grade 3: Hemorrhages, exudates
Grade 4: all of the above + papilloedema

DIFFERENTIAL DIAGNOSES:
- **Primary HT**
- **Secondary HT (renal a. stenosis)**
- **Phaeochromocytoma**
- **Hyperthyroidism**
- **Diabetes**
- **Drug-induced**

TESTS AND INVESTIGATIONS:

*If you measured their blood pressure, you know they're hypertensive. Time to look for **end organ damage***

Urinalysis

→ to **screen for diabetes**,
i.e **microalbuminuria, glucose,**
→ to **screen for chronic renal failure**
i.e **blood, high protein**

Plasma Biochemistry

→ **look at** **Creatinine**
Blood Urea Nitrogen,
Potassium
Glucose
Lipids
Urate

Discretionary Tests:
- **24 hr free cortisol**
  - (cushings)
- **24 hr catecholamines**
  - (phaeochromocytoma)

Plasma aldosterone/renin ratio
- (Conn's syndrome)
- **CT or MRI of thoracic aorta**
  - (Coarctation of the aorta)

Run all the tests you like, but
90% OF HYPERTENSION =
NO IDENTIFIABLE CAUSE
i.e "Essential Hypertension"

FBC

→ its really the **HEMOGLOBIN** that youre interested in;
chronic renal failure results in both hypertension and NC-NC anaemia

Chest X-ray

→ **HEART SIZE (LV hypertrophy expected)**
→ **RIB NOTCHES (coarctation of aorta results in collateral circulation,**
thus expanded costal arteries,
thus notching of the inferior rib surface

12-lead ECG:

- **Broad P waves in limb leads** (left atrial enlargement)
- **Conduction abnormalities,** eg. left bundle branch block
- **Left Axis Deviation** signifying LV hypertrophy
- **ATRIAL FIBRILLATION (!!)**

DISEASE DEFINITION: flavours of hypertension

Optimal BP	120/80	**These are arbitrary outcome-related definitions**
Moderate HT	= 140/90	
SEVERE HT	= 180/120	
Isolated systolic HT	= more than 140 / less than 90	

MANAGEMENT

Western diet =
10x the physiological salt requirement

Mild/Moderate Hypertension

Non-pharmacological treatment: → **NO RESPONSE? Switch to DRUGS**

- **Meditation**
- **Weight loss**
- **WALKING**
- **Reduce salt and fat in diet**

- **Alpha-2 agonists**
- **Alpha-1 ANTAgonists**
- **Beta-1 ANTAgonists**
- **Diuretics esp. THIAZIDE**
- **Vasodilators eg. ACE-inhibitors**

Severe Hypertension → always with drugs.

PHARMACOTHERAPY

1st choice: DIURETICS: thiazides in particular (K+ sparing)

2nd choice: Beta blockers: save lives.

- Not only do they vasodilate and reduce cardiac output, BUT
- They also block the sympathetic stimulation of renin relsease!

Alpha-2 adrenoceptor agonists

- Act centrally @ medulla to decrease sympathetic outflow
- Thus cause an unopposed vagal tone, and reduced sympathetic vasoconstriction
- Thus → reduce cardiac output and thus decreased blood pressure

Alpha-1 adrenoceptor antagonists

- Act peripherally on "capacitance" vessels; dilate these

Vasodilators

- Must use together with other drugs because of reflex responses to reduced arterial pressure
- Eg. tachycardia may result
- Example: MINOXIDIL: opens K+ channles in vascular smooth muscle;
  - thus → hyperpolarisation, no risk of contraction no matter what.
- **ONLY USE IF SEVERE HT!!**

L-type Calcium Channel Blockers

- Very good for decreasing chances of stroke, infarct and sudden death

Angiotension 2 (type 1 receptor) inhibitors

- Disease-Modifying (?) → vascular hypertrophy is mediated via the AT1 receptor.

ACE Inhibitors

- Discussed elsewhere

NITROPRUSSIDE: for absurdly high blood pressure

- Intravenous drug; metabolism liberates cyanide

COLLATERAL MANAGEMENT:

Statins:

- Block cholesterol synthesis @ liver
- Cause Feedback upregulation of LDL receptor expression
- **Thus increase LDL removal**

PHYSIOLOGY: SHORT TERM REGULATION OF BLOOD PRESSURE:

Arterial pressure regulation:

Divided into short-term, intermediate term and long-term mechanisms.

short-term mechanisms are **neural reflexes,**
intermediate mechanisms are **hormonal**,
long-term mechanisms consists mainly of the **renal system**

SHORT TERM: Seconds to Minutes ##

Mainly the Arterial baroreceptor reflex

= spray-type nerve endings lying in the walls of the carotid sinus and aortic arch

CAROTID → GLOSSOPHARYNGEAL N.
AORTIC → VAGUS N.

In the **MEDULLA OBLONGATA**
Nucleus of the solitary tract (NTS)

= are stretch receptors
= respond to changes in arterial wall stretch
= tonic activity @ normal arterial pressure
= CHANGE IN FIRING RATE influences B.P.
= **SENSITIVE RANGE 50 to 160** mmHg
= **ADAPT within 1-2 days** to whatever pressure level that prevails

THUS: mostly used for compensating **CHANGES IN POSTURE and METABOLIC ACTIVITY**

OTHER MECHANISMS= ALSO RUN IN VAGUS + GLOSSOPHARYNGEAL NERVES to (NTS)@ medulla

arterial chemoreceptors (located in the carotid body and aortic arch)
- stimulated primarily by a decrease in the pO_2
- BUT: also affected by massive drops in BP eg. blood loss

REFLEX: chemoreceptor stimulation → VASOCONSTRICTION

ATRIAL RECEPTORS respond to a drop in VOLUME
REFLEX: drop in volume → increase in VASOMOTOR NERVE ACTIVITY @ KIDNEY
→ signal to hypothalamus → PITUITARY RELEASES ANTIDIURETIC HORMONE
→ ADH (vasopressin) causes INCREASED WATER and SALT RETENTION

= Thus, water is retained and blood volume is restored

VENTRICULAR ARRHYTHMIA 5.05

RAPID VENTRICULAR RATE = SHORT DIASTOLE = NOT ENOUGH FILLING + LOW HEART NUTRITION
THUS; → ARRHYTHMIA = DROP IN CARDIAC OUTPUT + MYOCARDIAL ISCHAEMIA
Might be fast enough to have NO OUTPUT **(= CARDIAC ARREST)**
Might have no electrical activity (= **ASYSTOLE**)
Might have uncoordinated 300 beats/min (= **Ventricular Fibrillation)**

CAN BE BRADY OR TACHY
BRADY: Failure of conduction from AV node;
THUS:
Ventricle contracts from normal His-Purkinje rhythm, 40/min
→ Treat with artificial pacemaker

TACHY: depolarisation waves begin in the ventricle (long abnormal QRS)
Might re-direct atrial contraction by reverse conduction through AV node

Abnormal rapid depolarisations = **re-entering waves circling the ventricle**
The refractory period of the ventricular myocardium normally prevents this arrhythmia.
OCCURS IF:
- Scar tissue slows down normal conduction (thus, refractory periods run down before the wave can make it all the way around, and thus the cells become excited again)
- the ventricles are hypertrophied so that longer pathways and conduction times are possible,
- The re-entry electrical wave may become inconstant or break up into multiple uncoordinated re-entry loops, causing ventricular fibrillation.
- **Treatment is by modifying sympathetic tone with beta adrenergic blockade**,
- **by modifying the conduction time and refractory period of the myocardium with anti-arrhythmic drugs,**
- **by interrupting the re-entry loops by electrically depolarising all the ventricular myocardium at once (defibrillation),**
- **by destroying localised potential re-entry pathways surgically.**

A rarer cause of abnormal rapid depolarisations is abnormalities in myocardial cell membrane electrical properties leading to repetitive action potentials. These are likely in acute myocardial ischaemia, some drug intoxications, and inherited tendencies to ventricular arrhythmias.

PATHOGENESIS OF HYPERTENSION 5.05

Primary (essential) and secondary

Factors in the pathogenesis of hypertension relate to
(a) **those important in regulating normal blood pressure** such as the heart, the kidneys, vascular diameter and the venous system
(b) how these systems are influenced by the autonomic nervous system, various circulating hormones (such as catecholamines, ANP, renin, aldosterone and other steroids) and numerous local hormones or autacoids (eg. prostaglandins, nitric oxide and endothelin).
(c) **Various lifestyle and dietary factors**, including exercise, ethanol intake and dietary sodium level.

PATHOPHYSIOLOGY OF HYPERTENSION 5.05

90% of patients = NO KNOWN CAUSE ("Essential" hypertension)

SECONDARY HYPERTENSION = due to:

Chronic renal disease

Renal ischaemia due to renal artery atherosclerosis, either at its orifice or in its stem. ("renovascular hypertension")
Chronic renal parenchymal damage following for example, immunological glomerular injury (glomerulonephritis) or recurrent bacterial infections.

DROP IN RENAL PEREFUSION → Activation of RAAS

Essential hypertension itself, especially when very severe (malignant) may lead to **structural and functional alterations in the kidney** which may activate the renin-angiotensin system.

Adrenal lesions

Adrenal cortex produces gluco and mineralocorticoids, eg. ALDOSTERONE.
Thus, functionally active tumours will produce the same secretions.

PHEOCHROMOCYTOMA:

Tumour of the Adrenal medulla; produces NORADRENALINE (→vasoconstriction)

High Blood Pressure (WHO)=
= **140 over 90**
OR
Receiving medication for high blood pressure

RISK FACTORS FOR HYPERTENSION

- **diet** (primarily salt, but also other dietary components such as potassium and fish oils)
- **obesity,**
- **alcohol consumption,**
- **lack of exercise**
- **stress**

RISK FACTORS FOR HYPERTENSION 5.05

Defining the terms

*"**Screening** is the systematic application of a test or inquiry, to identify individuals at sufficient risk of a specific disorder to benefit from further investigation or directive preventive action, among persons who have not sought medical attention on account of symptoms of that disorder"*

People with high blood pressure have
a 2 to 4 times higher risk of -
- **stroke,**
- **myocardial infarction,**
- **heart failure**
- **peripheral vascular disease**

than people without hypertension

The higher your blood pressure, the higher your risk.

How common is Hypertension in the Australian community?
(DISTRIBUTION)

Large cross-sectional surveys of the general population = best source of evidence on the prevalence of a particular condition.
29% or 3.6 million Australians over the age of 25 had high blood pressure or were on BP medication:
- 31% of men and 26% of women.

The proportion of men and women with high blood pressure increases with age.

Since 1980 the prevalence of hypertension in Australia has decreased.

The prevalence in men (aged 25-64 years) has more than halved from 45% in 1980 to 22% in 1999-2000 and has almost halved in women from 29% in 1980 to around 1999-2000. **Limited data suggests that hypertension is up to three times more common in indigenous Australians.**

What causes hypertension?
(CAUSE)

- positive family history,
- overweight or physically inactive,
- consume excessive amounts of alcohol,
- high dietary salt intakes
- diets marked by low fruit/vegetable intake
- high saturated fat.
- Acute emotional or mental stress can also cause a temporary rise in blood pressure.

How can hypertension be prevented?
(PREVENTION)

primary prevention strategies

- maintaining body weight in a healthy range,
- reducing dietary salt intake,
- undertaking regular exercise for cardiovascular disease.

Its detection and early treatment by screening could be considered one of a number of *secondary prevention* strategies for cardiovascular disease.

Screening for hypertension as a secondary prevention strategy for cardiovascular disease
(MANAGEMENT/EVIDENCE)
It has been shown that each reduction of 10-14 mmHg in SBP and 5-6mmHg in DBP reduces the occurrence of stroke by two-fifths,
of coronary heart disease by one-sixth,
of cardiovascular disease by one-third
.Currently, the RACGP Guidelines for preventive activities in general practice recommends **screening for hypertension every 2 years for *all* Australians from age 15 years and older.**

Global cardiovascular risk tables can be estimated for individual patients using the colour-coded tables developed by the New Zealand guidelines group.

Which screening method is recommended?
Normal sphygmo is fine; **3 visits for obvious, 5 for bordeline (**90 to 95 distolic BP)

What are the potential harms of screening for hypertension?
There is mixed evidence that 'labeling' people as hypertensive temporarily increases absenteeism from the workplace but in general it is felt that **screening produces no adverse effects on psychological well-being.**

What is the probable harm to benefit ratio of screening for hypertension?
(PERSONAL EFFECTS)
The benefits of screening probably outweigh potential harms but the side effects of anti-hypertensive treatments need to be weighed against the potential benefits and global cardiovascular risk for each patient.
How does hypertension currently impact on the Australian community?
(SOCIETAL EFFECTS & RESPONSE)
High blood pressure was the most commonly managed problem by general practitioners in 2000-1, accounting for 6% of all conditions managed. It is estimated that more than 5% of the total burden of disease in Australia is attributable to hypertension. The reason for the decline in hypertension over the past 20 years is possibly due to reduced dietary salt intake but this is not proven. Changes in attitude and salt consumption appear to have been considerable amongst the general community over the past two decades.

RENIN-ANGIOTENSIN SYSTEM:

DROP IN CARDIAC OUTPUT → Reduced salt delivery to the **MACULA DENSA**

Activated **beta-2** receptors @ Juxtoglomerulus (**SYMPATHETIC STIMULATION**)

→ **RENIN IS SECRETED**

Angiotensinogen → **Angiotensin 1** → **Angiotensin-converting enzyme** → **ANGIOTENSIN 2**

ANGIOTENSIN 2

- Constricts ALL arterioles → **Increased BP**
- Increases ADH release → Increased water resorption @ the collecting duct (upregulated aquaporins) → **Increased VOLUME**
- Increases ALDOSTERONE secretion → Increased Na+ and thus H2O resorption @ distal tubule → **Increased VOLUME**

BUT…
chronic ANGIOTENSIN 2 elevation leads to
INCREASED CYTOKINE PRODUCTION and thus
Increased fibroblast and macrophage activity
→ **contribute to cardiac and vascular fibrosis**

Increased VOLUME → **INITIAL BENEFIT !!**
MORE PRELOAD
Thus → more cardiac output as the Frank-Starling sarcomeric mechanism takes effect

→ **RESTORATION OF CARDIAC OUTPUT**

BUT!!

- **Increased AFTERLOAD: THUS →**
  AORTIC VALVE WILL CLOSE PREMATURELY
  THEREFORE: stroke volume will be reduced
  (not as much blood can be ejected past the rapidly closing valve)
  → Reduced stroke volume → Reduced Cardiac Output
- **Increased VOLUME: thus →**
  Peripheral OEDEMA
  → Pulmonary Congestive Symptoms

Stretched ATRIUM:
Results in the release of ATRIAL NATRIURETIC PEPTIDE …which:
- Increases excretion of water and sodium
- Inhibits secretion of **renin and aldosterone**

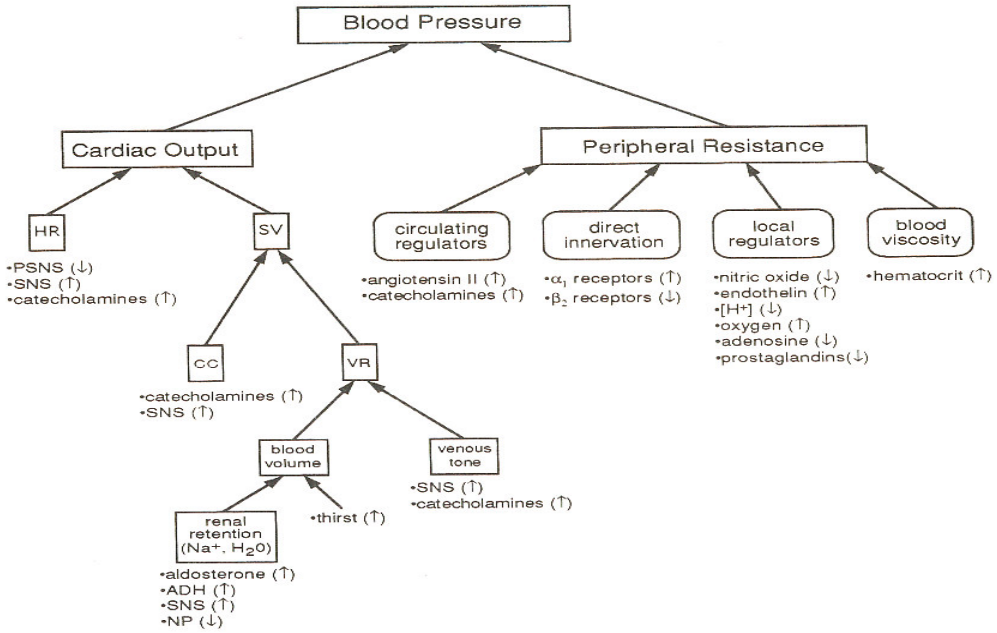

Figure 13.3. **Regulation of systemic blood pressure.** The small arrows indicate whether there is a stimulatory (↑) or inhibitory (↓) effect on the boxed parameters. HR, heart rate; SV, stroke volume; PSNS, parasympathetic nervous system; SNS, sympathetic nervous system; CC, cardiac contractility; VR, venous return; ADH, antidiuretic hormone; NP, natriuretic peptides.

Mechanism of hypertensive pathology 5.05