Learning objectives

By the end of this interactive session students should be able to:

• describe the population distribution of blood pressure and consequences of elevated blood pressure for individuals and communities;
• accurately measure blood pressure, and diagnose hypertension;
• evaluate the hypertensive patient for secondary causes and target organ damage;
• understand the concept and estimation of absolute cardiovascular risk;
• outline the principles and details of lifestyle and pharmacological management of hypertension; and
• discuss approaches to resistant hypertension.
Is hypertension a disease or a condition?
Blood pressure shows a unimodal distribution in the population.

Systolic BP data from 3000 adults in the Victorian Family Heart Study.
High blood pressure predisposes to

- coronary heart disease
- stroke
- cardiac hypertrophy
- heart failure
- kidney failure
Is an average blood pressure associated with CV risk?
The relationship between BP and CV risk is continuous

Above 115/75 mmHg, for each increase of 20 mmHg in SBP the risk of major cardiovascular and stroke events doubles.
Do most of the CV deaths attributable to BP occur in people with high or average levels of BP?
Prevention Paradox

• the majority of deaths attributable to blood pressure occur in people with “normal” BP.
• modest risk in many with average BP accounts for more deaths than high risk in fewer hypertensives
Public Health Prevention

• reducing the community average BP is good for the community
How is hypertension defined?
Hypertension is an operational diagnosis, defined by arbitrary thresholds.
How is the threshold for the diagnosis of hypertension determined?
Hypertension

• hypertension coincides with a level of BP above which the benefits of treatment have been shown to outweigh the side effects.
Clinical implications

- detecting and treating hypertension will benefit individuals
Primary Hypertension

• In 95% of hypertension, no specific cause is identified.
What general groups of factors lead to high blood pressure?
Primary Hypertension

• polygenic
  – sympathetic hyperactivity
  – renin activation
  – susceptibility to salt

• multi-environmental
  – obesity
  – excess salt (especially in elderly)
  – alcohol
Secondary Hypertension

• In 5% of hypertension a specific cause is identified.
Secondary Hypertension

• Kidney disease
  – Acute or chronic

• Renovascular
  – Renal artery stenosis
  – (Coarctation)

• Endocrine: adrenal tumors secreting
  – aldosterone
  – cortisol
  – Catecholamines
  – the pill or hormone replacement therapy!

• Obstructive sleep apnoea
Measuring Blood Pressure

- arm cuff
  - at heart level
  - correct size (large if arm circumference > 32 cm)
- manometer
  - mercury
    - being phased out
  - semiautomated
    - avoids observer error
    - use manually with stethoscope if atrial fibrillation
Why might have some GPs complained that the new semi-automated BP machines give a higher reading for systolic BP than the manual auscultatory method with a mercury manometer?
Possible explanations

• the doctor had hearing impairment
• the deflation was too fast with the mercury manometer
• the doctor tended to “round down” manual readings
• the doctor disregarded higher manual values that might have precipitated discussions about treatment
Diagnosing Hypertension

• BP > 140/90 mmHg
• after 5 minutes seated rest
• 2 readings 2 minutes apart
Confirming Hypertension

• additional visit in 1 to 4 weeks
• 24-hour ambulatory measurements
  – daytime > 135/85mmHg
• home BP measures
Diagnosing Hypertension

• “white coat” hypertension
  – detected by 24-hour ambulatory measurements
• isolated systolic hypertension
  – generally reflects the high pulse pressure (with relatively low DBP) seen in aged and stiff arteries
Patient evaluation

• for diagnosis
• for secondary causes
• for target organ damage
• for management decisions
History

• Family history
  • Hypertension, CKD
  • Cardiovascular disease

• Other CVS risk factors
  • smoking
  • diabetes
  • high cholesterol
  • past coronary or cerebrovascular events

• Other End organ disease
  • Known heart failure or heart failure symptoms
  • CKD or renal disease symptoms
Extra thoughts

• Pregnancy
  – Hypertension and pre-eclampsia
  – GDM

• Obstructive sleep apnoea
  – Obesity, alcohol and sleeping tablets
  – Snoring, witnessed apnoea
  – Sleep quality and somnolence

• Falls history and risks
  – Risk-benefit equation

• Medications history
Examination

• blood pressure and pulse rate/rhythm
• weight & height for BMI
  – *Or measuring tape for girth?*
• Full cardiovascular examination
  – Renal mass or bruits
  – Fundal inspection
• *Stigmata of secondary causes*
What *are* the stigmata of secondary causes?

- Kidney disease (acute or chronic)
- Renovascular
  - Renal artery stenosis
  - Coarctation
- Obstructive sleep apnoea
- Polycythaemia
- Endocrine
  - Adrenal tumours secreting
    - Aldosterone
    - Cortisol
    - Catecholamines
  - *Less common: abnormalities of just about any endocrine system*
    - Hyperthyroidism, acromegaly

If we have time we can discuss these ...
Routine tests

• EUC
  – plasma $K^+$ and creatinine (eGFR)
    • high in advanced renal (kidney) disease
  – low K in aldosteronism
  – Confirm: N sodium, $HCO_3^-/Cl^-$

• fasting glucose
  – associated glucose intolerance

• fasting lipids
  – associated CV risk
Routine tests

• FBE
  – associated anemia of CKD
  – Polycythamia

• LFTs
  – associated fatty liver or drug reaction
  – Serum Albumin

• urine albumin/creatinine ratio
  – evidence of renal damage

• MSU
  – clues as to causes of renal disease

• ECG and echocardiogram
  – to detect coronary disease and cardiac hypertrophy
Who should be tested for kidney disease?

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Recommended Tests</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Established cardiovascular disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of CKD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginal or Torres Strait Islander origin aged over 30 years</td>
<td></td>
<td>Every 1-2 years*</td>
</tr>
<tr>
<td></td>
<td>Urine ACR eGFR Blood Pressure</td>
<td></td>
</tr>
</tbody>
</table>

*yearly for people with diabetes or hypertension

If an individual has multiple risk factors, follow the more frequent regime

When to treat

• Blood pressure
• Cardiovascular risk
BP - when to treat

- SBP > 180 mmHg
- DBP > 110 mmHg
- SBP > 160 mmHg & DBP < 70 mmHg
BP+risk – When to treat

• SBP > 140 mmHg or DBP > 90 mmHg

with

• associated conditions (diabetes, existing CV or renal disease)

or

• high CV risk
High CV Risk

• > 15% CV event risk over 5 y (NHF)
High CV risk by standard risk factors

- Age
- Systolic pressure
- Total:HDL cholesterol ratio
- Smoking
- Diabetes

- \textit{CKD as a CV risk equivalent}
High CV risk by standard risk factors

Heart Foundation of Australia multifactorial risk factor charts

Risk level for 5-year cardiovascular (CVD) risk

- **High risk**: ≥ 30%
- **Moderate risk**: 10–15%
- **Low risk**: < 5%

*Significant risk factors for cardiovascular disease*
The tool is approved by NH&MRC

If:
- CKD 3B ie eGFR <45 mL/min/1.73m² or
- macroalbuminuria (ACR >25mg/mmol men; >35mg/mmol women)

Highest CVD risk and in this case the tool should not be applied

If Michael stops smoking his CV risk score reduces to 15%

(www.cvdcheck.org.au)
High CV risk by end organ damage

- Microalbuminuria or low eGFR
  - renal damage...CKD
- LV Hypertrophy
  - cardiac damage
- High pulse wave velocity
  - stiff large arteries
- Increased intima-media thickness
  - reflects atherosclerosis
Are we effective in the management of high blood pressure?
Unfortunately not

The rules of halves still applies today:

- only $\frac{1}{2}$ of hypertensives are detected
- only $\frac{1}{2}$ of detected hypertensives are treated
- only $\frac{1}{2}$ of treated hypertensives are controlled
Non-pharmacological treatment

• Stop smoking
• Lose weight
• Improve fitness
• Avoid excess salt
• Moderate alcohol
Modifiable Risk Factor - Smoking

• Among individuals with diabetes, those who smoke are more likely to get albuminuria and among those with diabetic kidney disease, smoking accelerates progression to failure [1,2]

• Even among the normal Australian population, smoking has been associated with kidney damage [3]

Lifestyle modification effects on BP

<table>
<thead>
<tr>
<th>Modification</th>
<th>Recommendation</th>
<th>Approx SBP reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>BMI 18-24.9 kg/m²</td>
<td>5-20 mmHg / 10kg lost</td>
</tr>
<tr>
<td>Dietary salt restriction</td>
<td>&lt;100 mmol/day</td>
<td>2-8 mmHg</td>
</tr>
<tr>
<td>DASH* diet</td>
<td>Fruit, vegies, low saturated and total fat</td>
<td>8-14 mmHg</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Aerobic activity for 30mins most days</td>
<td>4-9 mmHg</td>
</tr>
<tr>
<td>Moderate alcohol consumption only</td>
<td>1-2 standard drinks/day</td>
<td>2-4 mmHg</td>
</tr>
</tbody>
</table>

* Dietary Approaches to Stop Hypertension
Which is more important in pharmacological blood pressure reduction: how or how much?
How much
Drug treatments

A. ACE inhibitors, ARBs
B. Beta-blockers
C. Ca antagonists
D. Diuretics
Drug treatment

• Most patients require more than 1 drug
• Wait 2-3 weeks before adding drugs
• Combination drugs are simpler
Drug treatment algorithm

Step 1

A
C
D

Step 2

A+C
A+D

Step 3

A+C+D
Step 1 considerations

- **A**: Preferred step 1 if < 55 years
- **B**: Not preferred because of side effects of increased weight and insulin resistance
- **C**: Preferred step 1 if > 55 years or black
- **D**: Useful step 1 if there is fluid retention
General considerations

A. Useful in coronary disease and heart failure
   Renoprotective in diabetes
   Contra-indicated in pregnancy

B. Useful in coronary disease and heart failure

C. Avoid verapamil & diltiazam in heart failure

D. Thiazide-like drugs have less metabolic side-effects
What should Michael’s target BP be?

Answer

- There is no firm rule about what defines high blood pressure
- For most people, the lower the blood pressure the better
- Current blood pressure targets are as follows:

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Maintain BP consistently BELOW (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuminuria</td>
<td>&lt;130/80</td>
</tr>
<tr>
<td>Diabetes</td>
<td>&lt;130/80</td>
</tr>
<tr>
<td>Chronic Kidney Disease</td>
<td>&lt;140/90</td>
</tr>
</tbody>
</table>
What is the most important cause of “resistant hypertension”?
Poor compliance
Drug treatment algorithm

Step 4

- “resistant hypertension”
- consider adding spironolactone, beta-blocker, centrally-acting agent, alpha-blocker or vasodilator
- question compliance
- check for use of NSAIDs, cold remedies, antidepressants, etc
- consider secondary causes
Your responsibilities

• encourage everyone to know their blood pressure
• measure blood pressure carefully
• make decisions thoughtfully
• act using reliable information
A few words about secondary hypertension
What are the stigmata of secondary causes?

- Kidney disease (acute or chronic)
- Renovascular
  - Renal artery stenosis
  - Coarctation
- Obstructive sleep apnoea
- Polycythaemia
- Adrenal tumors secreting
  - aldosterone
  - cortisol
  - catecholamines
“To screen or not to screen?”

- **Routine screening for 2° causes NOT justified**
  - Expensive
  - Low yield
    - High-false positives
      - Expense
      - Patient distress
    - Delay or distract from optimising BP
    - If easily treated underlying cause *may* be irrelevant

- **Not to be confused with turning your eyes, ears, and brain off!**
Think: at least once

- Kidneys (AKI, CKD)
  - EUC, urine ACR, dipstick +/- MSU (blood)
- Renovascular
  - Coarctation, dissection: bilateral BP, brachio-femoral delay
  - RAS: atherosclerotic or fibromuscular dysplasia (FMD, FM hyperplasia)
    - Listen for abdominal bruit
    - Response to ACEi/ ARBs: see later
      - Whether to investigate and treat or not is a separate debate
- Sleep apnoea
  - Clues:
- Polycythaemia
  - FBE and if polycythaemic look for causes
    - hypoxaemia, tumour, PV, etc
Think: at least once
Endocrine causes

- Cushing’s (hypercortisolism)
  - Physical stigmata
  - Syndrome: obesity, diabetes, bones, (a)oligomenorrhea, sexual dysfunction
- Hyperadrenalism
  - Low K+, low-normal Na, mild oedema
  - Particularly good response to
    - Spironolactone especially
    - ARBs/ACEi
      - this raises issue of whether to treat, esp...
- Phaeochromocytoma
  - Adrenal incidentaloma
  - Classic triad: Headaches, sweating, tachycardia
    - Spells (nonexertional palpitations, sweating, headache, tremor, or pallor)
  - FHx (phaeo, MEN 2, NF, VHL)
Particular clues to secondary HT

- Malignant or accelerated hypertension
  - eg, severe hypertension + end organ
    - Eyes retinal hemorrhages /papilloedema, neurologic disturbance, acute heart failure, or acute kidney injury).
- Severe or resistant hypertension.
  - Persistent hypertension despite 3 agents
    - adequate doses, different classes, including a diuretic.
- Acute rise in BP where previously stable.
- Early onset
  - < 30 y.o in non-obese, negative family history
  - before puberty
Renal artery stenosis

• > 25% eGFR fall after instituting ARB/ACEi
  • **Tolerate a 25% decrease in eGFR**
    – Good outcomes presumably associated with reduced glomerular pressure
    – Continue ACEi or ARB if reduction is < 25% and stabilises within two months of starting therapy
    – Cease ACEi or ARB if reduction > 25% below baseline GFR

• Recurrent acute (flash) pulmonary edema or

• Maybe...
  – +/- refractory heart failure with impaired renal function
  – +/- abdominal bruit that lateralizes to one side.