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Key Points

• Hypertension is a disease of blood pressure regulation.

• Hypertension is a risk factor for atherosclerosis.

• Blood pressure measurement is important and requires attention to technique.

• Treatment decisions made in the context of overall risk factor burden.

• Secondary forms of hypertension are infrequently encountered and are usually recognized by resistance to treatment and distinctive biochemical features.
Hypertension

= high blood pressure
≠ being “hyper”, anxious
Systolic (upper #)

Diastolic (lower #)

“Normal” is less than 140/90 mmHg
<table>
<thead>
<tr>
<th>BP Classification</th>
<th>SBP mmHg</th>
<th>DBP mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120–139</td>
<td>80–89</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>≥160</td>
<td>≥100</td>
</tr>
</tbody>
</table>
Hypertension: Ethnic Variation (United States)

40% greater relative prevalence in African-Americans
Blood pressure regulation

• Hemodynamic (descriptive)

• Sympathetic nervous system (short-term)

• Renal pressure natriuresis (long-term)
Blood pressure regulation

Hemodynamic

Mean arterial blood pressure = Cardiac output $\times$ Peripheral vascular resistance

$\text{MAP} = \text{C.O.} \times \text{TPR}$

See discussion in Lilly hypertension chapter
Blood pressure regulation

Sympathetic nervous system

Source: Undetermined
Blood pressure regulation

Renal pressure natriuresis

Chronic BP Regulation

Sodium Intake or Output (fold increase)

"Normal" Na Intake

Mean Arterial Pressure (mmHg)
Sequelae of Essential Hypertension

- Hypertension
  - Heart Failure
  - Myocardial Ischemia and Infarction
  - Stroke
    - Nephrosclerosis and Renal Failure
  - Retinopathy
Cardiovascular Disease Risk by BP Status in Persons Aged 35–64 Years
Framingham Heart Study 36-Year Follow–Up

<table>
<thead>
<tr>
<th>Disease</th>
<th>Men</th>
<th>Women</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary Heart Disease</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Stroke</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Peripheral Arterial Disease</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>4</td>
<td>3</td>
<td>10</td>
<td>3</td>
</tr>
</tbody>
</table>

Risk ratio: Rate in HTN/Rate in Normals
Excess risk: Rate in HTN - Rate in Normals

Risk ratio: 2  3  4  2  4  3
Excess risk: 23 12 9 4 5 5 10 4
Trait level affects risk of disease (risk factor)

Total burden of risk factors affects disease severity
Cornary Heart Disease Mortality vs Usual BP by Age


### Systolic Blood Pressure

<table>
<thead>
<tr>
<th>Age at risk:</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>80-89</th>
</tr>
</thead>
<tbody>
<tr>
<td>120</td>
<td>256</td>
<td>128</td>
<td>64</td>
<td>32</td>
<td>16</td>
</tr>
<tr>
<td>140</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>160</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>180</td>
<td></td>
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</table>

### Diastolic Blood Pressure

<table>
<thead>
<tr>
<th>Age at risk:</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>80-89</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>256</td>
<td>128</td>
<td>64</td>
<td>32</td>
<td>16</td>
</tr>
<tr>
<td>80</td>
<td></td>
<td></td>
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<tr>
<td>90</td>
<td></td>
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</tr>
<tr>
<td>100</td>
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<tr>
<td>110</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

IHD Mortality (floating absolute risk and 95% CI)
Components of CVD Risk Stratification in Patients With Hypertension

Major Risk Factors

- Smoking
- Dyslipidemia
- Diabetes Mellitus
- Age >60 years
- Gender (men and postmenopausal women)
- Family history of early onset Coronary Heart Disease:
  - women <65 years
  - men <55 years

Source: JNC VI. Arch Intern Med. 1997;157:2413
The “Metabolic Syndrome” is a Cluster of “Diseases of Civilization”

- Hyperlipidemia
- Diabetes Mellitus
- Hypertension
- Obesity
Rate of CHD in Hypertension According to Risk Factors

Adapted with permission from Kannel WB. *JAMA*. 1996;275:1571
Injury to Endothelium Causes Endothelial Dysfunction

LDL cholesterol  Diabetes  Hypertension  Smoking  Aging

Endothelium

Vasoconstriction  Dysfunction  Lipid deposition and clearance

Blood-cell adhesion and/or infiltration  Proliferation, growth, and migration of smooth muscle cells

Atherosclerosis and Cardiovascular Events

Blood Pressure Measurement

- Patients should be seated with back supported and arm bared and supported at heart level.
- Patients should refrain from smoking or ingesting caffeine for 30 minutes before measurement.
- Measurement should begin after at least 5 minutes of rest.
- Appropriate cuff size and calibrated equipment should be used.
- Both SBP and DBP should be recorded.
- Two or more readings should be averaged.
24-h BP Profile
Typical Medical Student

<table>
<thead>
<tr>
<th>Time of day</th>
<th>23:00</th>
<th>02:00</th>
<th>06:00</th>
<th>10:00</th>
<th>14:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure (mm Hg)</td>
<td>160</td>
<td>140</td>
<td>120</td>
<td>100</td>
<td>80</td>
</tr>
</tbody>
</table>

- **Awakening**
- **Sleep**
- **Awake**

- SBP
- DBP
“White Coat” or “Office” Hypertension

Office: 160/90 mmHg

Home: 120/80 mmHg, 110/70 mmHg
Impact of “Normal” BP on CV Disease Risk In Men

- Cumulative Incidence (%)
- Time (years)
- 130-139/85-89 mm Hg
- 120-129/80-84 mm Hg
- <120/80 mm Hg

Objectives of the Initial Evaluation of Hypertensives

• To identify other risk factors or disorders that might guide treatment

• To assess presence or absence of target organ damage and cardiovascular disease

• To identify known causes
Evaluation Components

- Medical history
- Physical examination
- Routine laboratory tests
- Optional tests
Medical History

• Duration and classification (stage)
• Patient history of cardiovascular disease
• Family history
• Symptoms suggesting causes of hypertension
• Lifestyle factors
• Current and previous medications
Hypertension Runs in Families

Relative Risk for Hypertension

- ≥ 2 before age 55y
- ≥2 affected
- 1 before age 55y
- 1 affected

Age of hypertension onset in offspring

- 20-39 y
- 40-49 y
Physical Examination

- Blood pressure readings (two or more).
- Verification in contralateral arm.
- Height, weight, and waist circumference.
- Funduscopic examination.
- Examination of the neck, heart, lungs, abdomen, and extremities.
- Neurological assessment.
Objectives of the Initial Evaluation of Hypertensives

• To identify other risk factors or disorders that might guide treatment

• To assess presence or absence of target organ damage and cardiovascular disease

• To identify known causes (secondary HTN)
Causes of Hypertension

• “Essential” 90-95%
• Renal 3-5 %
  – Chronic renal failure
  – Renovascular disease
• 1° aldosteronism < 1%
• Pheochromocytoma < 1%
• Hypertension of pregnancy
Identifiable Causes of Hypertension

- Renovascular disease
- Primary aldosteronism
- Pheochromocytoma
- Pseudopheochromocytoma
- Sleep apnea
- Drug-induced or related causes
- Chronic kidney disease
- Chronic steroid therapy and Cushing’s syndrome
- Coarctation of the aorta
- Thyroid or parathyroid disease
Atherosclerotic Renovascular Disease
Renin-Angiotensin-Aldosterone System

- **Angiotensinogen** (From liver)
- **Angiotensin I**
- **Angiotensin II**
- **Renin** (From kidney)
- **ACE**
- **Sodium & fluid retention**
- **Vasoconstriction**
- **Aldosterone secretion**
Atherosclerosis is a systemic disease
Fibromuscular Renovascular Disease (FMD)

- Frequently bilateral
- May be associated with cerebral arterial FMD
Clinical Clues Suggesting Renovascular Hypertension

• Onset of hypertension under age 25 or over age 55
• An abdominal bruit, particularly in diastole
• Refractory, accelerated, or malignant hypertension or worsening of previously controlled hypertension
• Undiagnosed renal failure, with or without hypertension (particularly with normal urine sediment)
• Acute renal failure precipitated by hypertension treatment, particularly with ACE inhibitors
• A unilateral small kidney (by any prior investigational procedure)
Aldosterone: Important Component of Renin-Angiotensin-Aldosterone System

Angiotensinogen → Ang I → Ang II → Aldosterone

- Renin
- ACE
- Non-RAAS Stimulators

Pathophysiologic Effects on Cardiovascular System

Na^+ / H_2O Retention, K^+, Mg^{++} Loss
Stimulators of Aldosterone

RAAS
Angiotensin II

Non-RAAS
Potassium
Adrenocorticotropic Hormone
Norepinephrine
Endothelin
Serotonin

Aldosterone

1° Aldosteronism
Aldosterone secretion independent of normal regulators

RAAS = renin-angiotensin-aldosterone system
Pheochromocytoma

- Tumors of chromaffin cells (adrenal or extra-adrenal)
- “Rule of 10s”
  - 10% are extra-adrenal
  - 10% of extra-adrenal are extra-abdominal
- “5 Ps”
  - Pressure, palpitations, perspiration, pallor, pain
# Secondary Hypertensions

<table>
<thead>
<tr>
<th>Pheochromocytoma</th>
<th>1° Aldosteronism</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pl. free metanephrine 99% sensitive and 89% specific</td>
<td>• Plasma aldosterone-renin ratio (ARR)</td>
</tr>
<tr>
<td></td>
<td>PRA (ng/mL/hr)</td>
</tr>
<tr>
<td></td>
<td>Plasma aldosterone (ng/dl)</td>
</tr>
<tr>
<td></td>
<td>• ARR &gt; 30 suggests 1° Aldosteronism</td>
</tr>
</tbody>
</table>
Norepinephrine
Epinephrine

Pheochromocytoma = Tumor
Pseudopheochromocytoma = Physiological hyperactivity
Primary Prevention

• Primary prevention offers an opportunity to interrupt the costly cycle of managing hypertension.

• Lifestyle modifications have been shown to lower blood pressure.

• A population-wide approach may reduce morbidity and mortality; trials are lacking.

• Most patients with hypertension do not sufficiently change their lifestyle or adhere to drug therapy enough to achieve control.
Goal of Hypertension Prevention and Management

• To reduce morbidity and mortality by the least intrusive means possible. This may be accomplished by
  – Achieving and maintaining SBP < 140 mm Hg and DBP < 90 mm Hg.
  – Controlling other cardiovascular risk factors.
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Slide 12: A. Weder
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