NOTES
HYPERTENSION & HYPOTENSION

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Changes of blood pressure above (*hyper-*) or below (*hypo-*) normal (120/80mmHg)

CAUSES

- *Mostly idiopathic, but can include various causes:* impaired regulatory pathways (hormonal/neurologic disorders), heart disease, kidney disease, medications etc.

SIGNS & SYMPTOMS

- Can be asymptomatic/include symptoms according to underlying cause, degree of change in blood pressure
  - *Hypertension:* range from headache, dyspnea, to blurred vision, oliguria, seizures
  - *Hypotension:* range from fatigue, pallor to syncope

DIAGNOSIS

OTHER DIAGNOSTICS

- Blood pressure cuff (sphygmomanometer) or arterial catheter

TREATMENT

- *Hypotension:* generally requires no treatment
- *Hypertension:* treated according to degree, treat underlying cause if present

MEDICATIONS

- *Hypertension:* beta blockers, diuretics, ACE inhibitors, calcium channel blockers, etc.

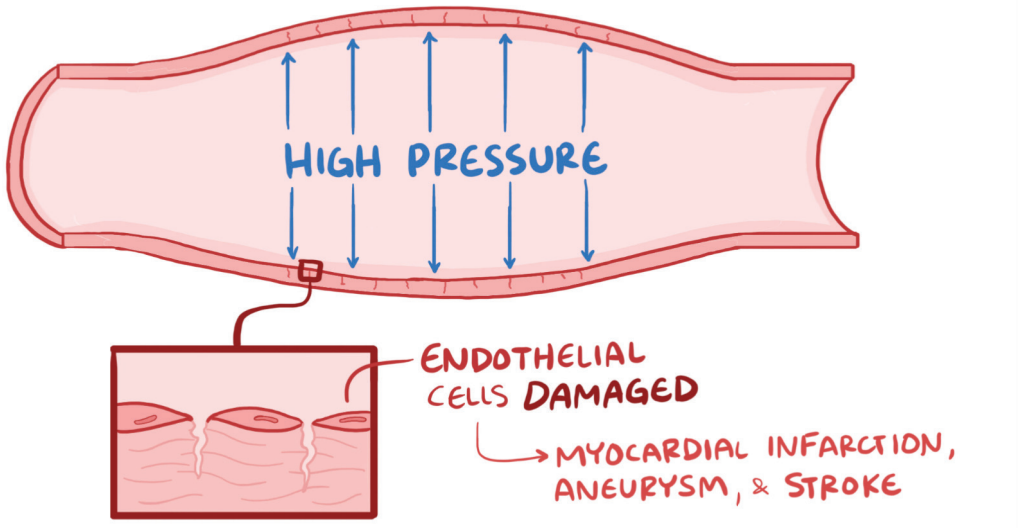

Figure 11.1 Illustration depicting endothelial damage caused by hypertension.

HYPERTENSION

osms.it/hypertension

PATHOLOGY & CAUSES

- Condition in which blood pressure is regulated maladaptively, elevating blood pressure over 140/90mmHg
- *Isolated systolic hypertension:* systolic blood pressure is elevated, diastolic is not
- *Isolated diastolic hypertension:* diastolic blood pressure is elevated, systolic is not

CAUSES

Primary/essential hypertension

- Most cases (90%)

Secondary hypertension

- Known etiology, often reversible
- *Renovascular hypertension:* anything partially obstructing blood flow to kidneys (e.g. atherosclerosis, vasculitis, fibromuscular dysplasia).
  - Decreased blood flow to kidneys → kidneys secrete renin → renin converts angiotensinogen to angiotensin I → angiotensin converting enzyme converts angiotensin I to angiotensin II (active) → angiotensin II effects:
  - *Vasoconstriction:* directly increases blood pressure
  - *Stimulation of sodium reabsorption:* increases water reabsorption
  - Stimulation of adrenal cortex to release aldosterone from adrenal cortex → aldosterone increases reabsorption of sodium + water → increased volume → high blood pressure
- *Primary hyperaldosteronism:* increased aldosterone → increased reabsorption of sodium + water → increased volume → increased blood pressure
- *Nonsteroidal anti-inflammatory drugs (NSAIDs)* → inhibit cyclooxygenase in kidneys → ↓ production of PGE-2 (renal vasodilator) → vasoconstriction of afferent arterioles in kidneys → ↓ renal blood flow, glomerular filtration rate → ↓ secretion of sodium + water → ↑ volume → ↑ blood pressure
- *Preeclampsia/eclampsia in pregnancy:* mechanism unknown
- *Coarctation of the aorta*: low pressure past coarctation → low renal perfusion → activation of renin angiotensin-aldosterone system (RAAS) → secondary hypertension
- *Cushing's syndrome:* combination of several pathophysiological mechanisms (e.g. elevated cortisol) that regulate plasma volume, cardiac output, peripheral vascular resistance
- *Chronic kidney disease:* fluid overload, sodium retention

MNEMONIC: RHNECCK

Causes of Secondary hypertension

Renovascular hypertension
1° **H**yperaldosteronism
NSAIDs
Pre-**E**clampsia / **E**clampsia
Coarctation of the aorta
Cushing's syndrome
Kidney Disease (chronic)

RISK FACTORS

Primary hypertension

- Risk increases with age, biological male, obesity, stress, sedentary lifestyle, family history of hypertension
- *Race (in decreasing order of risk):* African descent > white European descent > Asian descent
- *Diet:* excessive sodium, alcohol intake
- Abnormal lipid panel (high low-density lipoproteins, low high-density lipoproteins, high triglycerides)

Secondary hypertension

- *Atherosclerosis:* Elderly biological males
  - Atherosclerosis → renal stenosis → less blood flow to renal arteries → activation of RAAS → renovascular hypertension

COMPLICATIONS

- Increased risk of atherosclerosis, arteriosclerosis
- *Arteriolar rarefaction:* loss of arterioles
- Coronary artery disease, left ventricular hypertrophy, atrial fibrillation, stroke, hypertensive nephropathy, retinopathy, aortic dissection, aneurysms

SIGNS & SYMPTOMS

- Vast majority of cases asymptomatic
- May experience headache, dyspnea
- Renal bruit in secondary hypertension due to renal artery stenosis
- Hypertensive retinopathy

DIAGNOSIS

OTHER DIAGNOSTICS

- Non-invasive/invasive blood pressure monitoring
  - *High blood pressure:* at least 2 separate measurements with blood pressure > 140/90mmHg

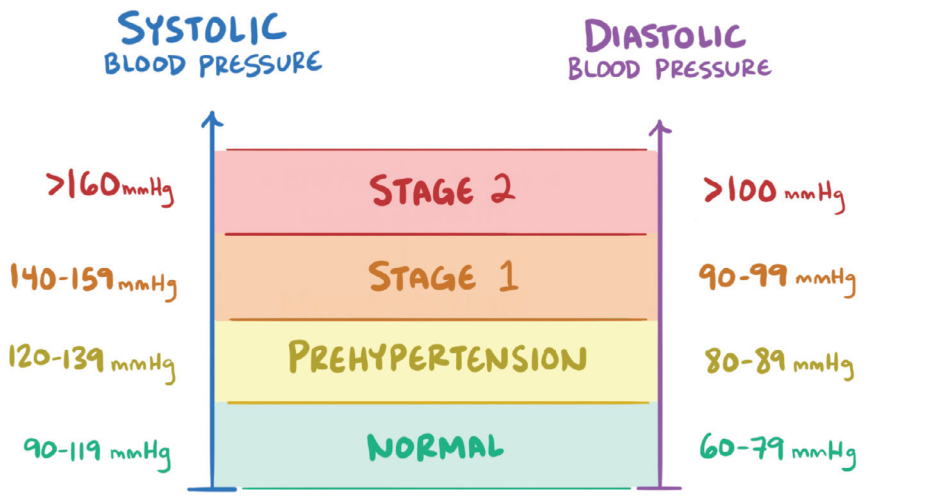

Figure 11.2 Illustration depicting stages of hypertension.

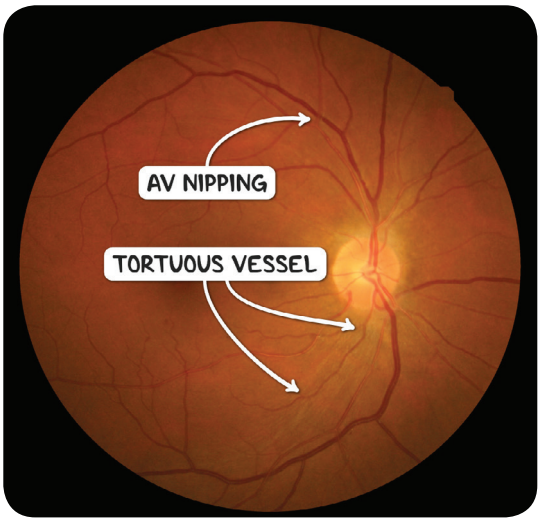

Figure 11.3 Retinal photograph demonstrating changes of hypertensive retinopathy (AV nipping and tortuous vessels).

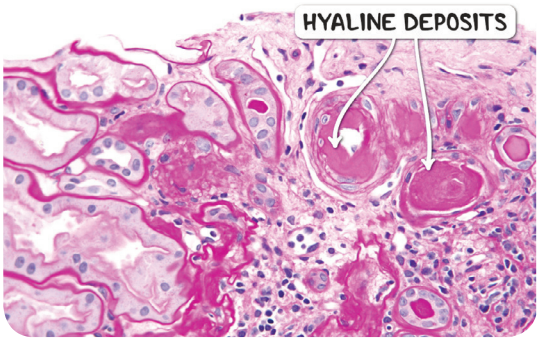

Figure 11.4 Histological appearance of renal artery hyalinosis; a manifestation of hypertensive renal disease.

TREATMENT

- Reduce BP <140/90mmHg with lifestyle modification first, then medical treatment

MEDICATIONS

Monotherapy/together

- *Thiazide-type diuretics:* reduce blood volume by increasing excretion of sodium, water
- *Angiotensin-converting enzyme (ACE) inhibitors:* block ACE from converting angiotensin I to angiotensin II → blocks RAAS → dilates arteries, decreases blood volume
  - Lower levels of angiotensin II → vasodilation
- *Dihydropyridine calcium channel blockers:* disrupt movement of calcium through calcium channels in blood vessel walls → vasodilation

Other agents

- *Angiotensin II receptor blockers (ARBs):* prevents the vasoconstrictive effects of angiotensin II by blocking its receptors → lowering blood pressure
  - Used when individuals get a cough from ACE inhibitors
- *Beta receptor blockers:* decrease contractility, heart rate
- *Alpha-2 agonist:* stimulates alpha-2 receptors → decreases sympathetic activity → decreased blood pressure, heart rate
- *Renin inhibitor:* aliskiren
  - Lower levels of angiotensin I
- *Hydralazine:* elicits direct vasodilation of vascular smooth muscle, useful in pregnancy

OTHER INTERVENTIONS

- Low sodium diet, exercise, quit smoking, limit alcohol, maintain healthy weight

HYPERTENSIVE EMERGENCY

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PATHOLOGY & CAUSES

- Elevated blood pressure > 180/120mmHg with signs of acute end organ damage (e.g. encephalopathy, stroke, papilledema, myocardial infarction, heart failure, microangiopathic hemolytic anemia, etc.)
- Complication of poorly managed hypertension

RISK FACTORS

- Kidney failure, renovascular hypertension, stimulant abuse, medication non-adherence
- More common in young adults, particularly those of African descent

COMPLICATIONS

- Neurological complications (stroke, seizures), myocardial infarction, kidney failure, permanent blindness, pulmonary edema

SIGNS & SYMPTOMS

- Blood pressure > 180/120mmHg, signs of end-organ damage
  - Blurred vision, altered mental state, chest pain, headache, nausea, vomiting, numbness in extremities, oliguria, seizure, dyspnea, weakness, papilledema

DIAGNOSIS

OTHER DIAGNOSTICS

- Sphygmomanometer
  - Blood pressure > 180/120mmHg
- Evaluation to identify at-risk target organ
  - Electrocardiography (heart)
  - Chest X-ray (heart, lungs)
  - Urinalysis (kidneys)
  - Serum electrolytes, serum creatinine (kidneys)
  - Cardiac enzymes (heart)
  - CT scan of brain (if brain suspected → neurologic symptoms, retinopathy)
  - Contrast-enhanced CT scan of chest (if aortic dissection suspected)

TREATMENT

- Treatment varies case-by-case, dependent on affected organ

MEDICATIONS

- Unwise to lower blood pressure too quickly/too much, as this can lower cerebral perfusion excessively
  - *Most cases:* mean arterial pressure (MAP) should be reduced using intravenous medication 10–20 % in first hour, then 5–15% over the following 23 hrs. Specific medications used dependent on case

OTHER INTERVENTIONS

- *Exceptions to most cases:* acute phase ischemic stroke (not lowered unless specific conditions met)
  - Acute aortic dissection (rapid lowering), intracerebral hypertension (variable)

HYPOTENSION

osms.it/hypotension

PATHOLOGY & CAUSES

- Condition in which arterial blood pressure drops below 90/60mmHg
- Physiological in some cases (professional athletes); considered pathologic if symptomatic
- Not a distinctive disease, but a manifestation of various conditions
- *Orthostatic hypotension:* hypotension caused by standing up from a sitting/lying position

CAUSES

Hypovolemia

- Fluid loss (hemorrhage, diarrhea, vomiting), low fluid intake (starvation, oligodipsia), endocrine disorders, anemia

Heart disease

- Cardiomyopathies, heart valve disease (e.g. mitral stenosis), congestive heart failure, myocardial infarction, arrhythmias

Medications

- Most commonly cause orthostatic hypotension
- Excessive use of diuretics, alpha/beta blockers, nitrate preparations, calcium channel blockers, angiotensin II (AT1) receptor blockers, antidepressants

Neurological disorders

- Spinal cord injury resulting in ↓ sympathetic output or ↑ parasympathetic output
- Dysautonomia (intrinsic autonomic system dysfunction), Parkinson's disease

COMPLICATIONS

- Ischemia
- If severe, can lead to shock

SIGNS & SYMPTOMS

- Lightheadedness, fatigue, pallor, confusion
- Significant hypotension → syncope

DIAGNOSIS

OTHER DIAGNOSTICS

- Evaluation of blood pressure with sphygmomanometer/arterial catheter
  - Systolic blood pressure < 90mmHg
  - Diastolic blood pressure < 60mmHg
  - Mean arterial pressure < 65mmHg
- Orthostatic hypotension
  - Drop in 20mmHg of systolic pressure/10mmHg of diastolic pressure when standing up from a sitting/lying position

TREATMENT

- Asymptomatic hypotension does not require treatment
- Treat underlying cause