HYPERTENSION

PULSE WAVE CONCEPT

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HYPERTENSION-PULSE WAVE CONCEPT

- What is the definition of hypertension?
- Dr. Moser’s definition of hypertension.
- Korotkoff sounds
- Anatomy of the artery.
- Pulse wave theory.
- End organ damage from hypertension.
- Wide pulse pressure hypertension.
- Cerebral perfusion pressure.
- Treatment options.

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HYPERTENSION-PULSE WAVE CONCEPT

- In the old days (1960's or so) we did not know much about hypertension.
- Physicians were taught to take the patient's age and add 100. This then would be the systolic pressure that we thought was normal.
- In 1870, Frederick Mahomed was a medical resident at Guy's Hospital In London and started taking a lot of blood pressure readings of the public and first described hypertension.
- In 1977 the National High Blood Pressure Coordinating Committee established a new Joint National Committee on the Detection, Evaluation and Treatment of High blood pressure.
- Initial guidelines were 140/90 (JNC 8). 2021 guidelines remain the same.

HYPERTENSION-PULSE WAVE CONCEPT DR. MOSER'S DEFINITION OF HYPERTENSION.

- Hypertension is that blood pressure reading in any individual patient that when sustained will ultimately create the signs and symptoms classically seen in a patient with uncontrolled high blood pressure.

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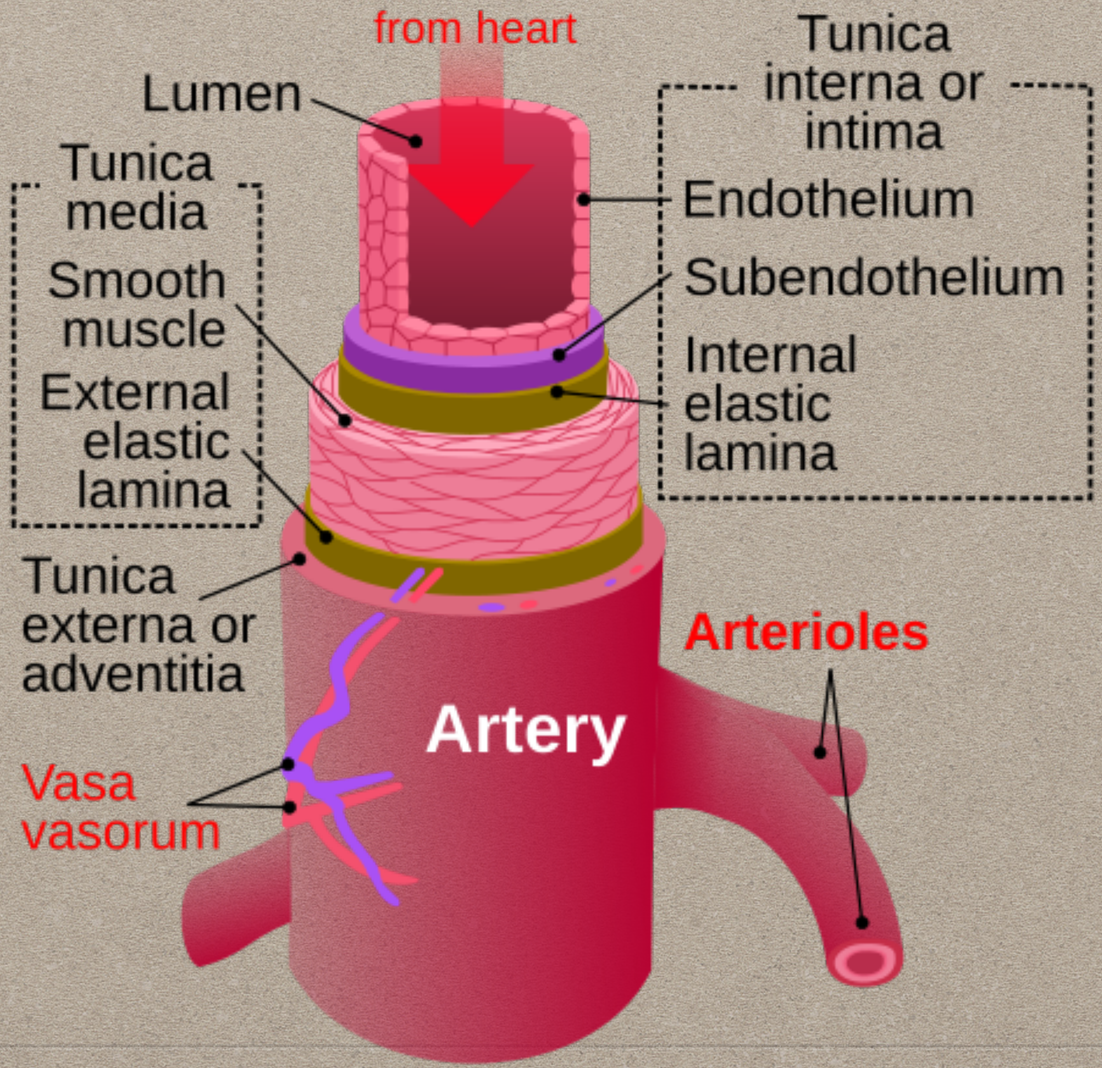
HYPERTENSION-PULSE WAVE CONCEPT THE 5 KOROTKOFF SOUNDS

- Phase I: The first appearance of faint, repetitive, clear tapping sounds which gradually increase in intensity for two consecutive beats is the systolic blood pressure.
- Phase II: A brief period may follow during which the sounds soften and acquire a swishing quality.
- Phase III: The return of sharper sounds which become crisper to regain, or even exceed the intensity of phase I sounds.
- Phase IV: The distinct, abrupt muffling of sounds, which become soft and blowing in quality.
- Phase V: The point at which all sounds finally disappear completely is the diastolic pressure.
- Nikolai Korotkov. Was a Russian physician who first described these sounds in 1905.

HYPERTENSION-ANATOMY OF AN ARTERY



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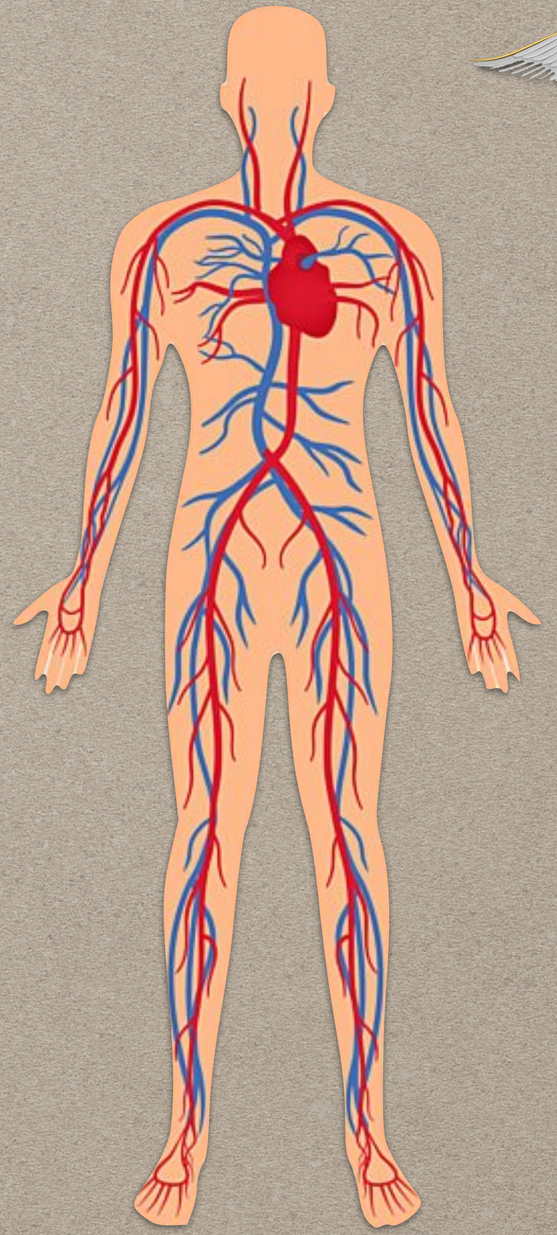
HYPERTENSION
PULSE WAVE CONCEPT

- It is felt that the entire arterial tree contributes to the forward flow of blood.
- The average stroke volume of a 70 Kg male is 70 ml.
- As the stroke volume is ejected from the left ventricle, the aorta expands as well as all of the other arteries.
- Elastic fibers in the artery then collapse behind this volume of ejected blood helping to PUSH the blood on down stream.
- This occurs throughout the entire arterial tree.



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HYPERTENSION
PULSE WAVE CONCEPT

- The incoming blood being returned to right heart helps push out the upcoming cardiac ejection supercharging it in a sense.
- This action then helps maintain the forward flow of blood.
- Due to years of high blood pressure, the muscle cells in the wall of the arteries begin to develop hypertrophy and thicken the wall of the artery.
- This results in a decrease in the luminal diameter of the artery.

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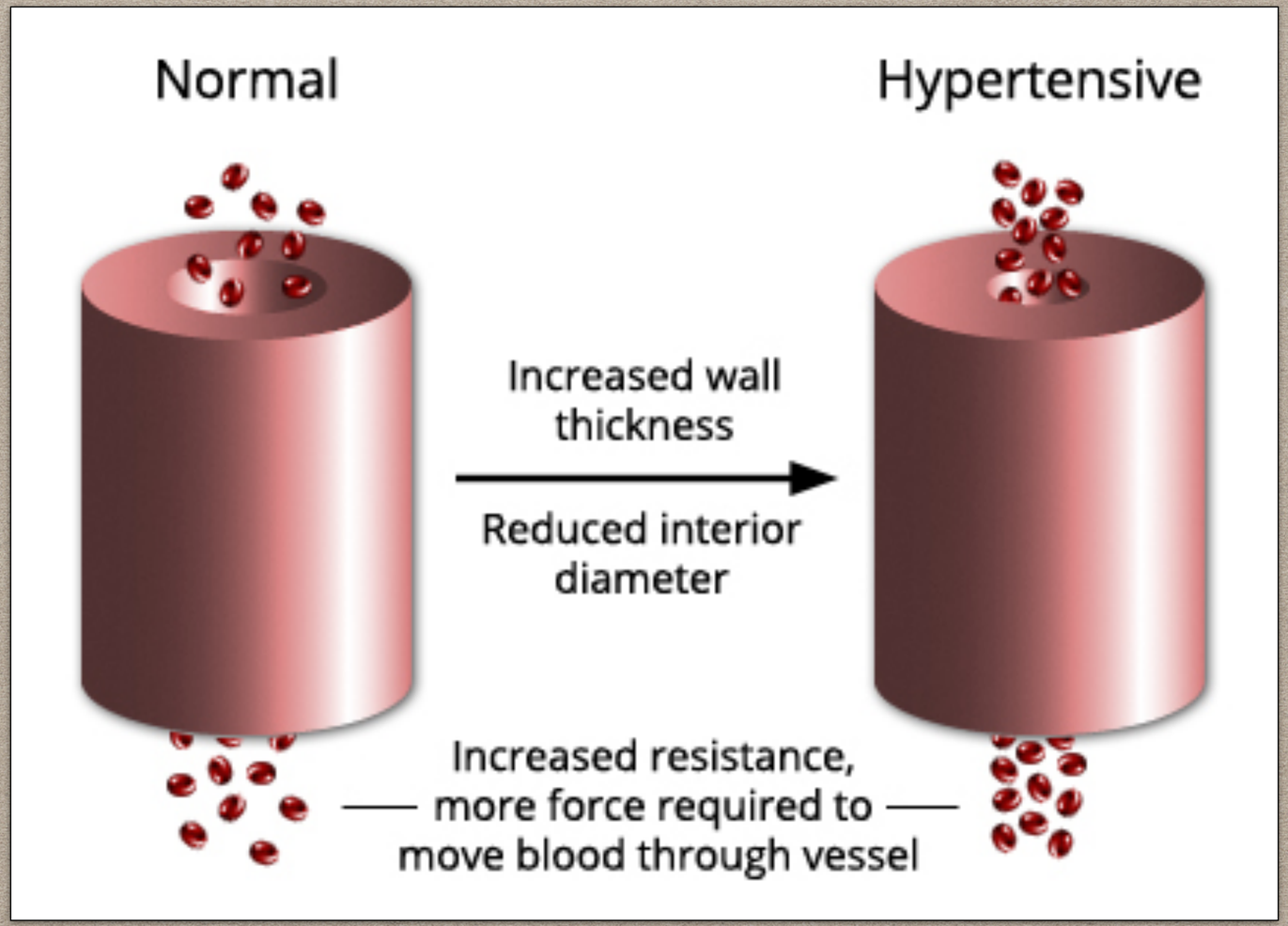
HYPERTENSION
PULSE WAVE CONCEPT



HYPERTENSION-PULSE WAVE CONCEPT

- The decreased luminal diameter of the artery causes decreased perfusion and oxygen delivery to the peripheral tissues.
- This stimulates the nervous system to increase cardiac contractility to increase the cardiac output to guarantee good oxygen delivery to all cells in the body.
- This results in a rise in the systolic blood pressure.
- The arterial muscle cell hypertrophy also hardens the vessel (hardening of the arteries) and reduces the elastic recoil of the vessel.
- This results in a decrease in the force of blood being returned to the right heart and causes the diastolic blood pressure to drop.
- This then causes a reduced “supercharging” of the outgoing ejection compounding the problem.

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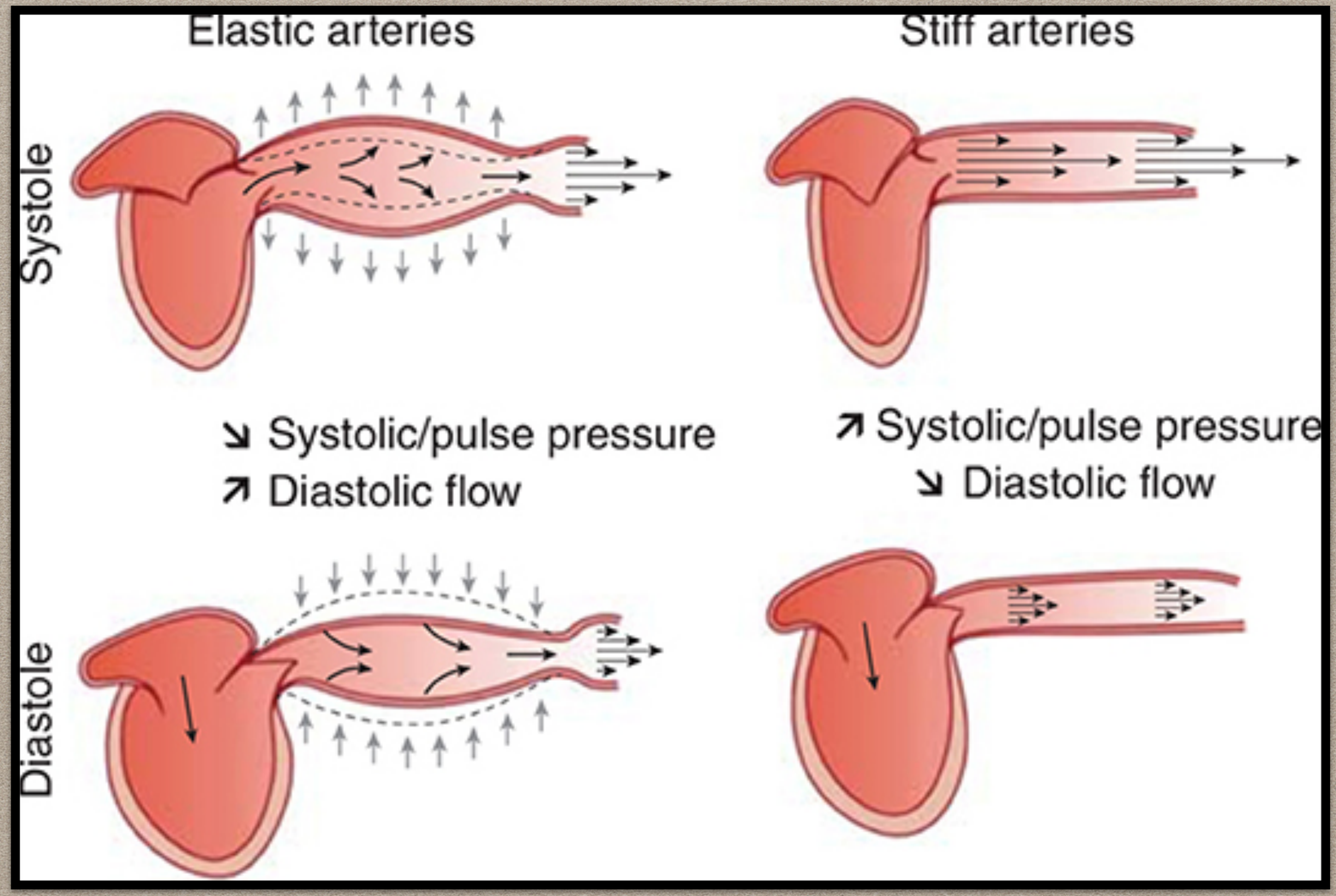
HYPERTENSION-WIDENING OF THE PULSE PRESSURE.



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HYPERTENSION
WIDENING OF THE PULSE PRESSURE.

- The rise in the systolic blood pressure and the decrease in the diastolic blood pressure then result in widening of the pulse pressure.
- Pathophysiology that can result in a widened pulse pressure.
  - Arteriosclerosis (hardening of the arteries)
  - Aortic regurgitation (especially with hypothermia)
  - Aortic sclerosis.
  - Patent Ductus arteriosus.
  - Severe iron deficient anemia.
  - Hyperthyroidism.
  - Severe hypovolemia.

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HYPERTENSION
WIDENING OF THE PULSE PRESSURE.

- Concomitant increases in systolic and diastolic blood pressure occur until age 50 to 55 years.
- Subsequently systolic and diastolic blood pressures begin to diverge.
  - Systolic pressure continues to rise.
  - Diastolic blood pressure stabilizes and then begins to decrease.
    - These changes ultimately result in widening of the pulse pressure and ultimately result in isolated systolic hypertension.
- Once this develops, it can not be reversed

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE.
SIGNS AND SYMPTOMS

- Ankle or foot swelling.
- Shortness of breath and difficulty with breathing.
- Dizziness.
- Facial flushing.
- Fainting.
- Headaches.
- Heart palpitations.
- Generalized weakness.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. DIAGNOSIS AND WORKUP

- Systolic pressure minus the diastolic pressure (normal pulse pressure is 40mm/Hg)
- Stiff radial pulse with inability to compress the artery.
- Cholesterol and triglycerides and/or lipid panel.
- Comprehensive metabolic profile.
- EKG
- Exercise cardiac stress test.
- Echocardiogram.
- Doppler ultrasound. Ultrasonographer comments that the vessels are non compressible.
- Ankle-brachial index.
- Coronary calcium scan.
- Coronary angiography.
- Take the pulse with two fingers.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TYPICAL END ORGAN DAMAGE

- Vasculopathy.
  - Endothelial dysfunction.
  - Vascular remodeling.
  - Generalized atherosclerosis.
  - Atherosclerotic stenosis.
  - Aortic aneurysm.

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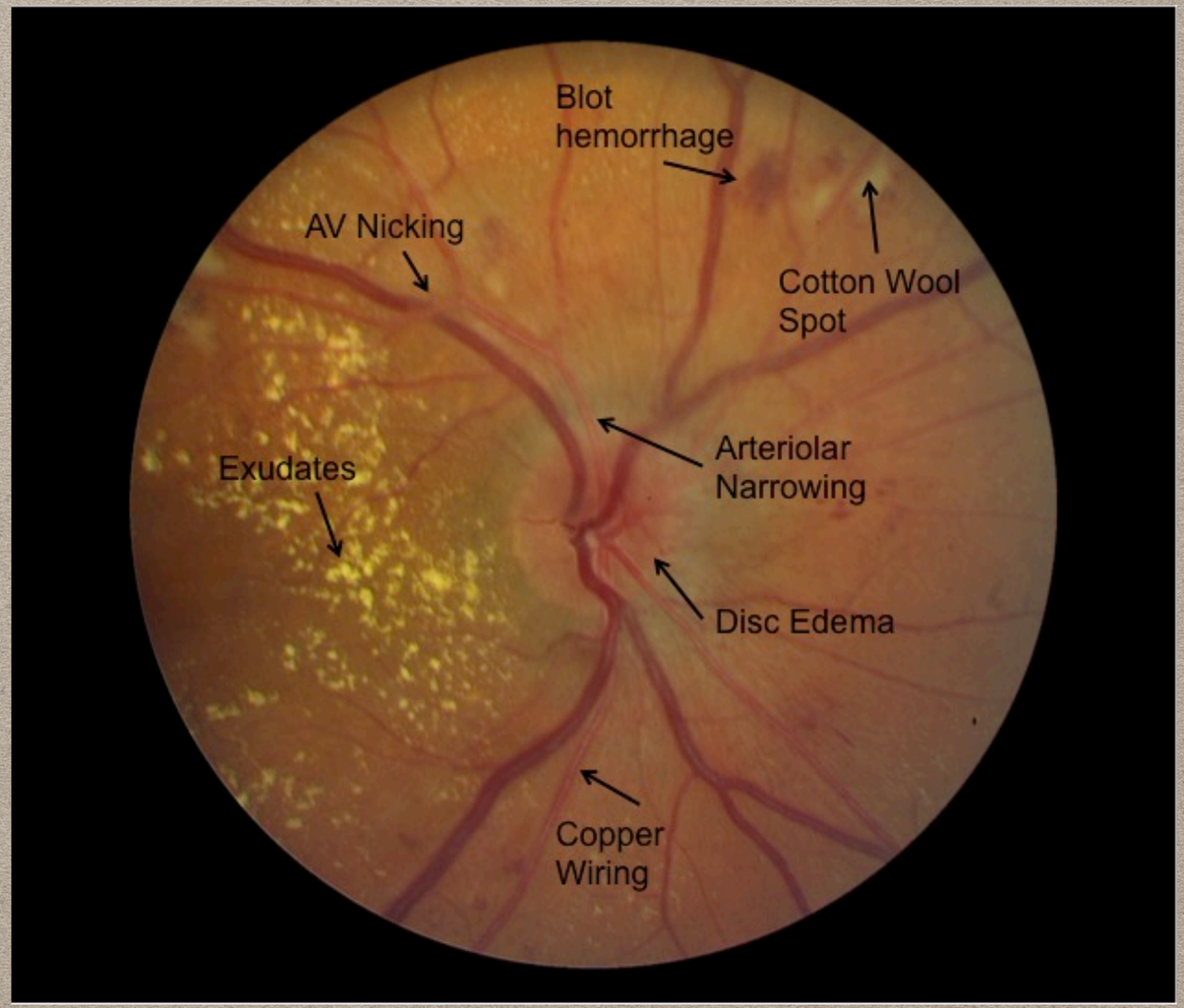
HYPERTENSION-WIDENING OF THE PULSE PRESSURE.
TYPICAL END ORGAN DAMAGE

- Cerebrovascular damage.
  - Acute hypertensive encephalopathy.
  - Acute stoke.
  - Intracerebral hemorrhage.
  - Lacunar infarction.
  - Vascular dementia.
  - Retinopathy.



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HYPERTENSION-WIDENING OF THE PULSE PRESSURE.
TYPICAL END ORGAN DAMAGE

- Heart disease.
  - Left ventricular hypertrophy.
  - Atrial fibrillation.
  - Coronary microangiopathy.
  - Coronary heart disease.
  - Myocardial infarction.
  - Heart failure.

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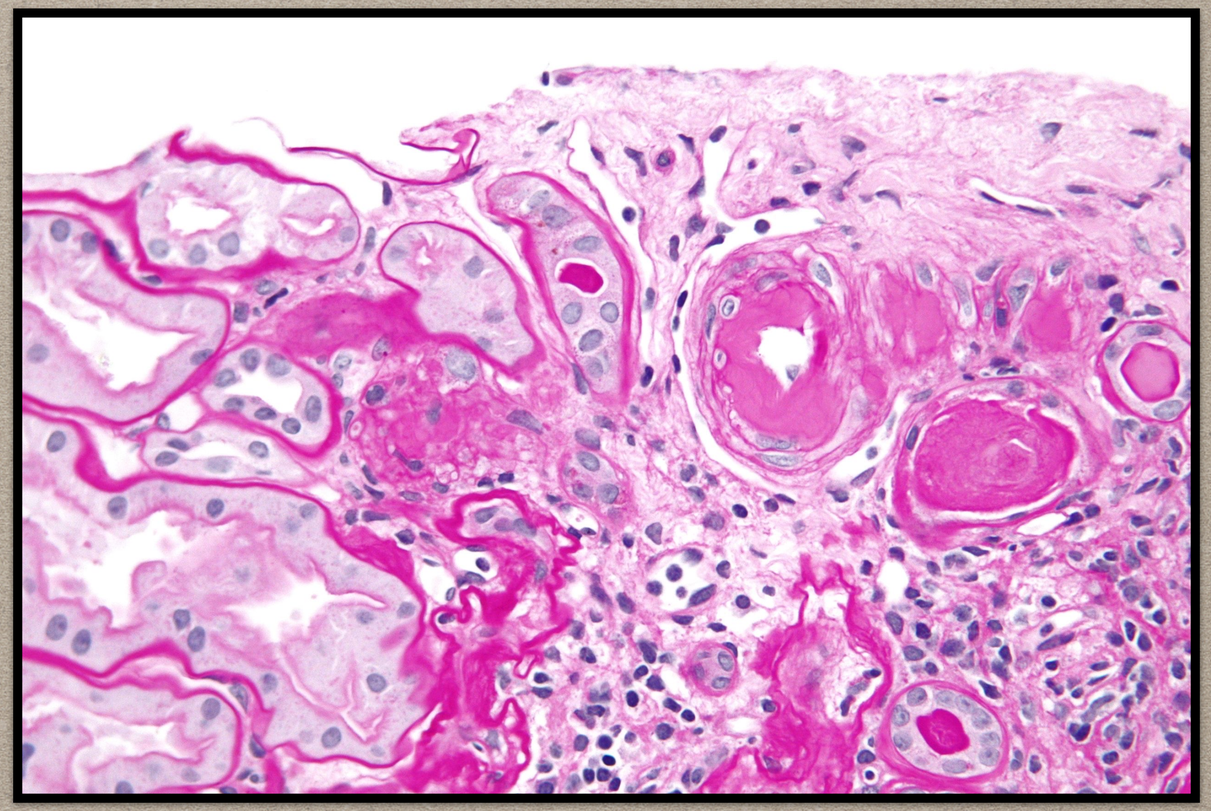
HYPERTENSION-WIDENING OF THE PULSE PRESSURE.
TYPICAL END ORGAN DAMAGE

- Kidney disease (hypertension is the second leading cause of kidney disease)
  - Albuminuria.
  - Proteinuria.
  - Chronic kidney disease (renal insufficiency).
  - Kidney failure.



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HYPERTENSION
CEREBRAL PERFUSION PRESSURE

- CPP is the net pressure gradient causing cerebral blood flow to the brain (brain perfusion).
- It must be maintained within narrow limits because too little pressure could cause brain tissue to become ischemic and too much pressure could raise intracranial pressure.
- Normal pressure is 50-70 mm/Hg

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HYPERTENSION
CEREBRAL PERFUSION PRESSURE

- CPP=MAP-ICP (if ICP is higher than JVP).
- CPP=MAP-JVP (if JVP is higher than ICP).

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HYPERTENSION
CEREBRAL PERFUSION AUTO REGULATION.

- Cerebral auto-regulation is the ability of the cerebral vasculature to maintain stable blood flow in the brain despite wide changes in systemic blood pressure.
- Normal CPP is maintained between 50-70 mm/Hg.

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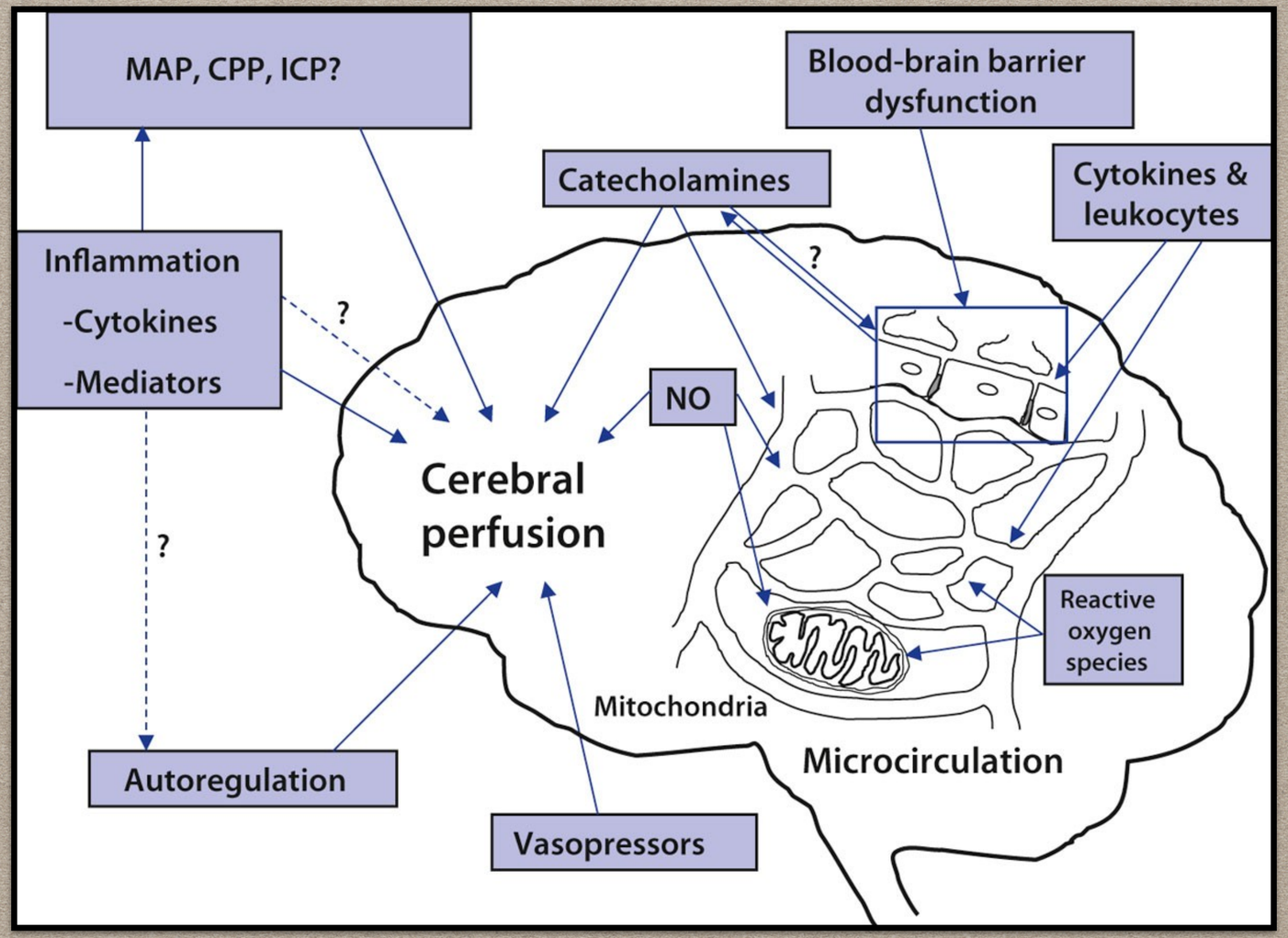
HYPERTENSION
CEREBRAL PERFUSION AUTO REGULATION.



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HYPERTENSION
CEREBRAL PERFUSION PRESSURE

- Four mechanisms for auto regulation.
  - Myogenic.
  - Neurogenic.
  - Metabolic.
  - Endothelial.

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HYPERTENSION
CEREBRAL PERFUSION PRESSURE

- After many years of uncontrolled hypertension, this cerebral perfusion pressure setpoint of auto regulation can be reset to a higher value such that the brain expects a higher perfusion pressure.
- If you try to bring a patients blood pressure down too quickly, you may underperfuse the brain leading to symptoms.
- I try to reduce a patients blood pressure in brackets to avoid this.
    - Example: drop them from average SBP of 180 to 170 for a few weeks then drop to 160 for a few weeks, etc.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Remember the underlying physiology.
  - The blood vessels are very stiff and non compliant.
    - Therefore, medications that function as vasodilators by relaxing blood vessels tend to not work as well.
- The best treatment is with beta blockers and vascular volume management and calcium channel blockers.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Beta blockers
  - Negative inotropic-reduce the force of contraction of the heart. Will help reduce systolic pressure more than diastolic pressure.
  - Negative chronotrope-reduces the pulse rate.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Diuretics-removes fluid volume from the circulation.
  - Loop diuretics-forces increased sodium loss via the kidneys. Sodium is osmotically active and pulls water with it. You obtain water and sodium loss.
  - Thiazides-same as loop diuretics but work in the distal nephron. Commonly used with loop diuretics.
  - Aldosterone antagonists-block the sodium retaining effect of aldosterone. Causes potassium retention (spironolactone, eprelenone, Finerenone)
  - Ethacrynic acid-mild diuretic. Works in proximal nephron.
  - Vaptans-blocks aquaporin receptors-causes water loss without solute loss (aquaresis).
  - SGLT-2 inhibitors-block glucose reabsorption by the kidneys. This traps glucose in the urine. Glucose is osmotically active and pulls water into the urine.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Angiotensin converting enzyme inhibitors.
  - Angiotensin is a very powerful vasoconstrictor. By reducing angiotensin production, relaxation of the blood vessels occurs.
  - Renin levels are not raised in wide pulse pressure hypertension.
  - Science has shown that people with high blood pressure and normal renin do better than people with lower blood pressure and high renin.
  - High renin causes vasculopathy.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Angiotensin converting enzyme inhibitors.
  - High renin with normal aldosterone suggests sensitivity to sodium.
  - Low renin and high aldosterone suggest adrenal gland dysfunction.
  - High renin and high aldosterone is consistent with kidney disease.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Angiotensin receptor blockers.
  - Block the effect of angiotensin at the receptor causing relaxation of the vessels.
- Clonidine-centrally acting alpha-2 adrenergic agonist. Decreases pulse rate and causes vasodilation. Also used to treat ADHD, anxiety, severe menstrual cramps, Tourette's syndrome and heroine addiction and withdraw. Watch for rebound hypertension when the drug wears off (duration of effect is 3-5 hours. Half life is 12-16 hours).
  - Clonidine is highly abused on the street.

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Hydralazine-blocks vasoconstriction in the arteries. Has a lot of side effects.
- Calcium channel blockers-prevent calcium from entering cardiac myocytes thus reducing the force of contraction of the heart and prevents calcium from entering the muscle cells of the arteries reducing vasoconstriction.
  - Dihydropyridines (nifedipine and amlodipine).
  - Non-Dihydropyridines (diltiazem and verapamil).

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HYPERTENSION-WIDENING OF THE PULSE PRESSURE. TREATMENT OPTIONS

- Alpha blockers-block the peripheral alpha receptor from binding Adrenalin causing decreased vasoconstriction.
- Aldosterone receptor blockers-block the effect of aldosterone at the receptor. Aldosterone causes sodium retention and therefore water retention.
- Direct renin inhibitors-Aliskiren (tekturna).

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THE BEST WAY TO TREAT DISEASE IS TO PREVENT DISEASE.™

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