Hypertension

by Dr Swathi Swaroopa .B

Introduction

- ***Hypertension is a common disease that is simply defined as persistently elevated arterial blood pressure (BP).***

- ***The percentage of men with high BP is higher than that of women before the age of 45 years, but After age 45 years, the percentage is slightly higher with women.***

- ***BP values increase with age, and hypertension (persistently elevated BP values) is very common in the elderly.***

Hypertension doubles the risk of

- ***Cardiovascular diseases, including***
- ***Coronary heart disease (CHD),***
- ***Congestive heart failure (CHF),***
- ***Ischemic and hemorrhagic stroke,***
- ***Renal failure, and***
- ***Peripheral arterial disease.***

Types of hypertension

- ***Essential or primary hypertension***

No identifiable cause for their disorder. This form of hypertension cannot be cured, but it can be controlled.

90% of individuals with high BP have essential hypertension

- ***Secondary hypertension***

Have a specific identified cause for elevated BP . If the cause can be identified, hypertension in these patients has the potential to be cured.

Fewer than 10% of patients have secondary hypertension

Pseudo hypertension

An artificially and falsely elevated blood pressure obtained due to arteriosclerotic, calcified blood vessels which do not physiologically compress with pressure, results in high blood pressure reading than it truly ought to be.

White-Coat Hypertension

White-coat hypertension describes patients who have consistently elevated BP values measured in a clinical environment in the presence of a health care professional (e.g., physician's office), yet when measured elsewhere or with 24-hour ambulatory monitoring, BP is not elevated.

Etiology

Essential/primary hypertension

- ***No identifiable cause***
- ***Genetic factors***
- ***Genetic mutations***

Secondary hypertension etiology

Disease	***Drugs and food***
Primary aldosteronism *Renovascular disease* *Thyroid disease* *Parathyroid disease* *Pheochromocytoma* *Coarctation of the aorta* *Obstructive sleep apnea* *Cushing's syndrome* *Chronic kidney disease*	*Prednisone , cyclosporine and tracolimus* *Nonsteroidal antiinflammatory drugs, cyclooxygenase-2 inhibitors* *β-blocker or centrally acting α-agonists (when abruptly discontinued) Cocaine* *Nicotine withdrawal* *Ephedra alkaloids* *Sodium* *Ethanol* *Licorice*

Disease	Drugs and Other Products Associated with Hypertension[a]
Chronic kidney disease Cushing's syndrome Coarctation of the aorta Obstructive sleep apnea Parathyroid disease Pheochromocytoma Primary aldosteronism Renovascular disease Thyroid disease	**Prescription drugs** • Amphetamines (amphetamine, dexmethylphenidate, dextroamphetamine, lisdexamfetamine, methylphenidate, phendimetrazine, phentermine) and anorexiants (sibutramine) • Antivascular endothelin growth factor agents (bevacizumab, sorafenib, sunitinib) • Corticosteroids (cortisone, dexamethasone, fludrocortisone, hydrocortisone, methylprednisolone, prednisolone, prednisone, triamcinolone

Disease	Drugs and Other Products Associated with Hypertension[a]
	• Calcineurin inhibitors (cyclosporine, tacrolimus) • Decongestants (pseudoephedrine, phenylephrine) • Ergot alkaloids (ergonovine, methysergide) • Erythropoiesis-stimulating agents (erythropoietin, darbepoetin) • Estrogen-containing oral contraceptives • Nonsteroidal antiinflammatory drugs—cyclooxygenase-2 selective (celecoxib) and nonselective (aspirin, choline magnesium trisalicylate, diclofenac, diflunisal, etodolac, fenoprofen, flurbiprofen, ibuprofen, indomethacin, ketoprofen, ketorolac, meclofenamate, mefenamic acid, meloxicam, nabumetone, naproxen, naproxen sodium, oxaprozin, piroxicam, salsalate, sulindac, tolmetin)

Disease	Drugs and Other Products Associated with Hypertension[a]
	• Others: desvenlafaxine, venlafaxine, bupropion Situations: β-blocker or centrally acting α-agonists (when abruptly discontinued); β-blocker without α-blocker first when treating pheochromocytoma; use of a monoamine oxidase inhibitor (isocarboxazid, phenelzine, tranylcypromine) with tryamine-containing foods or certain drugs **Street drugs and other products** Cocaine and cocaine withdrawal Ephedra alkaloids (e.g., Ma huang), "herbal ecstasy," other analogues Nicotine and withdrawal, anabolic steroids, narcotic withdrawal, ergot-containing herbal products, St. John's wort **Food substances** Sodium Ethanol Licorice

Classification of hypertension for adults

Blood Pressure Classification	SBP mmHg	DBP mmHg
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage 1 Hypertension	140–159	or 90–99
Stage 2 Hypertension	≥160	or ≥100

Hypertensive crisis

- ***Hypertensive crises are clinical situations where BP values are very elevated, typically greater than 180/120 mm Hg.***

- ***They are categorized as either***
  - ***Hypertensive emergency***
  - ***Hypertensive urgency.***
- ***Hypertensive emergencies are extreme elevations in BP that are accompanied by acute or progressing target-organ damage.***
- ***Hypertensive urgencies are high elevations in BP without acute or progressing target-organ injury.***
- ***Isolated systolic hypertension Patients with DBP values less than 90 mm Hg and SBP values ≥140 mm.***

PATHOPHYSIOLOGY

1)*HUMORAL MECHANISMS*

RAAS(Renin–Angiotensin–Aldosterone System)

Natriuretic hormones,

Insulin resistance and hyperinsulinemia

2)NEURONAL REGULATION

Autonomic nervous system

Baroreceptors

Central nervous system

3)PERIPHERAL AUTOREGULATORY COMPONENTS

4)VASCULAR ENDOTHELIAL MECHANISMS

5)ELECTROLYTES AND OTHER CHEMICALS

Renin–Angiotensin–Aldosterone System

- *RAAS is primarily governed by the kidney*
- *RAAS regulates sodium, potassium, and fluid balance.*
- ***Renin** is an enzyme that is stored in the juxtaglomerular cells, which are located in the afferent arterioles of the kidney.*
- *Juxtaglomerular cells function as a **baroreceptor-sensing** device.*

Sodium and chloride delivered to the distal tubule

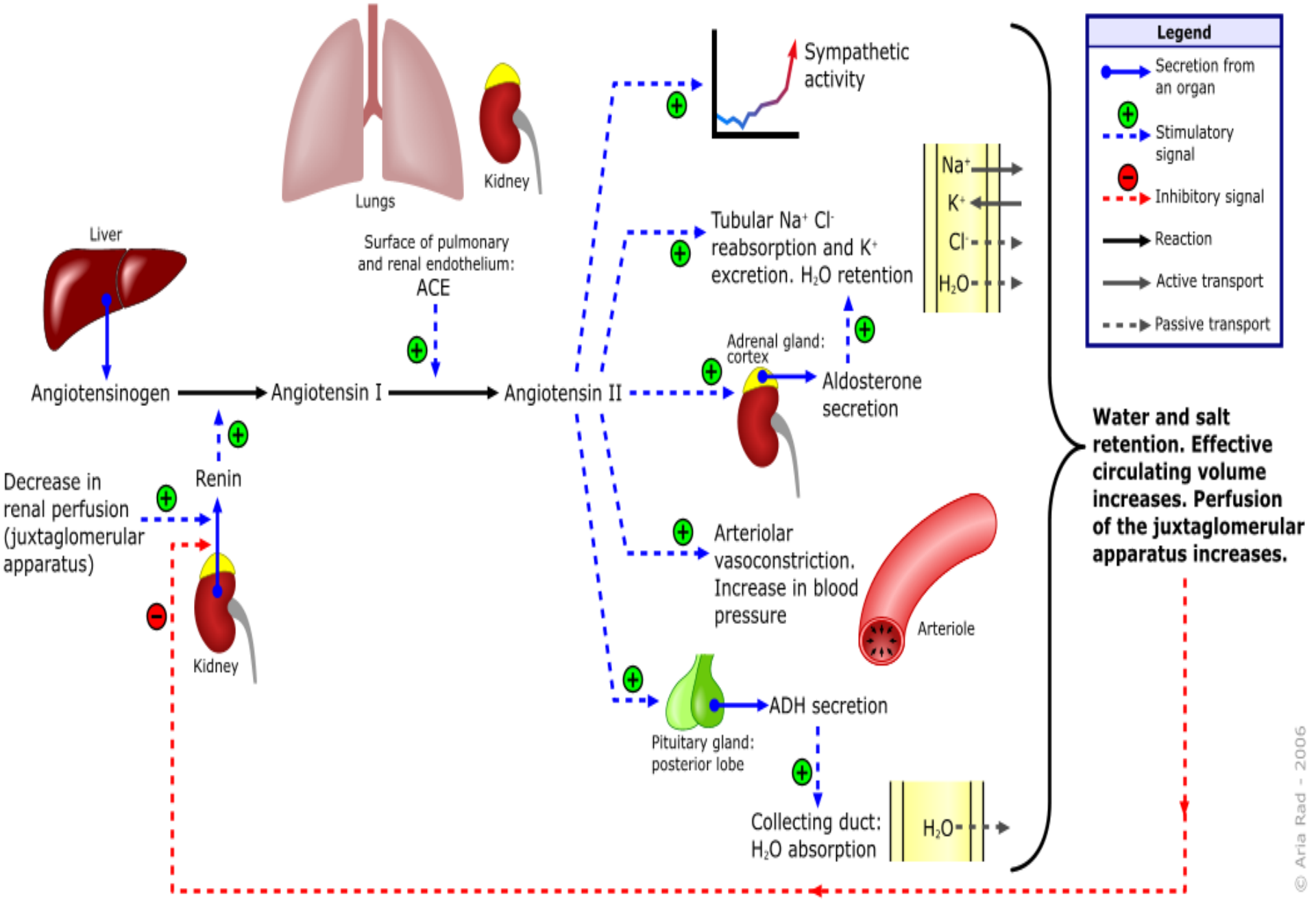
Sympathetic tone

Serum potassium and/or intracellular calcium

BP

All these 4factorsactivate the juxtaglomerular cells resulting in renin secretion.

Renin-angiotensin-aldosterone system



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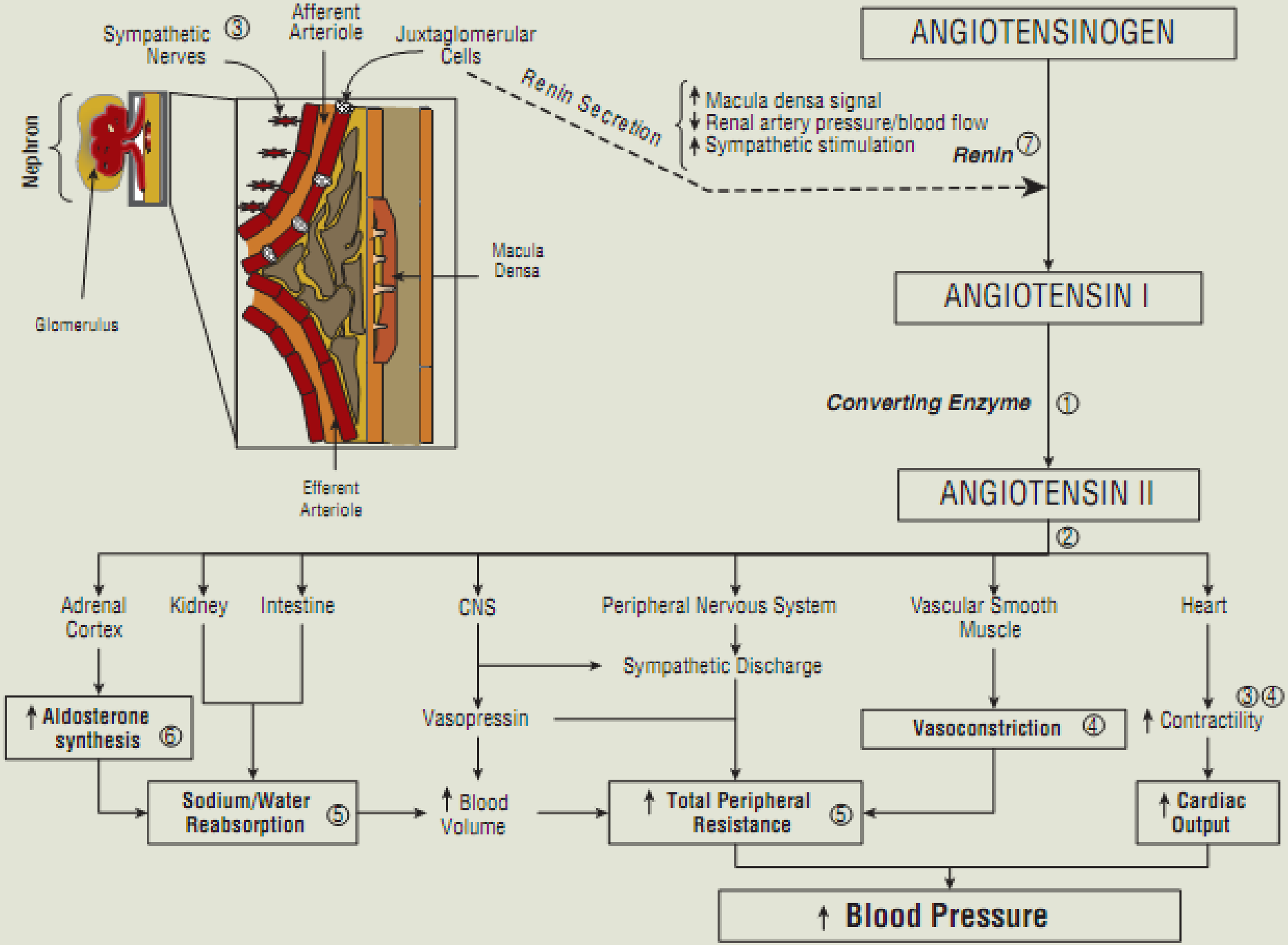
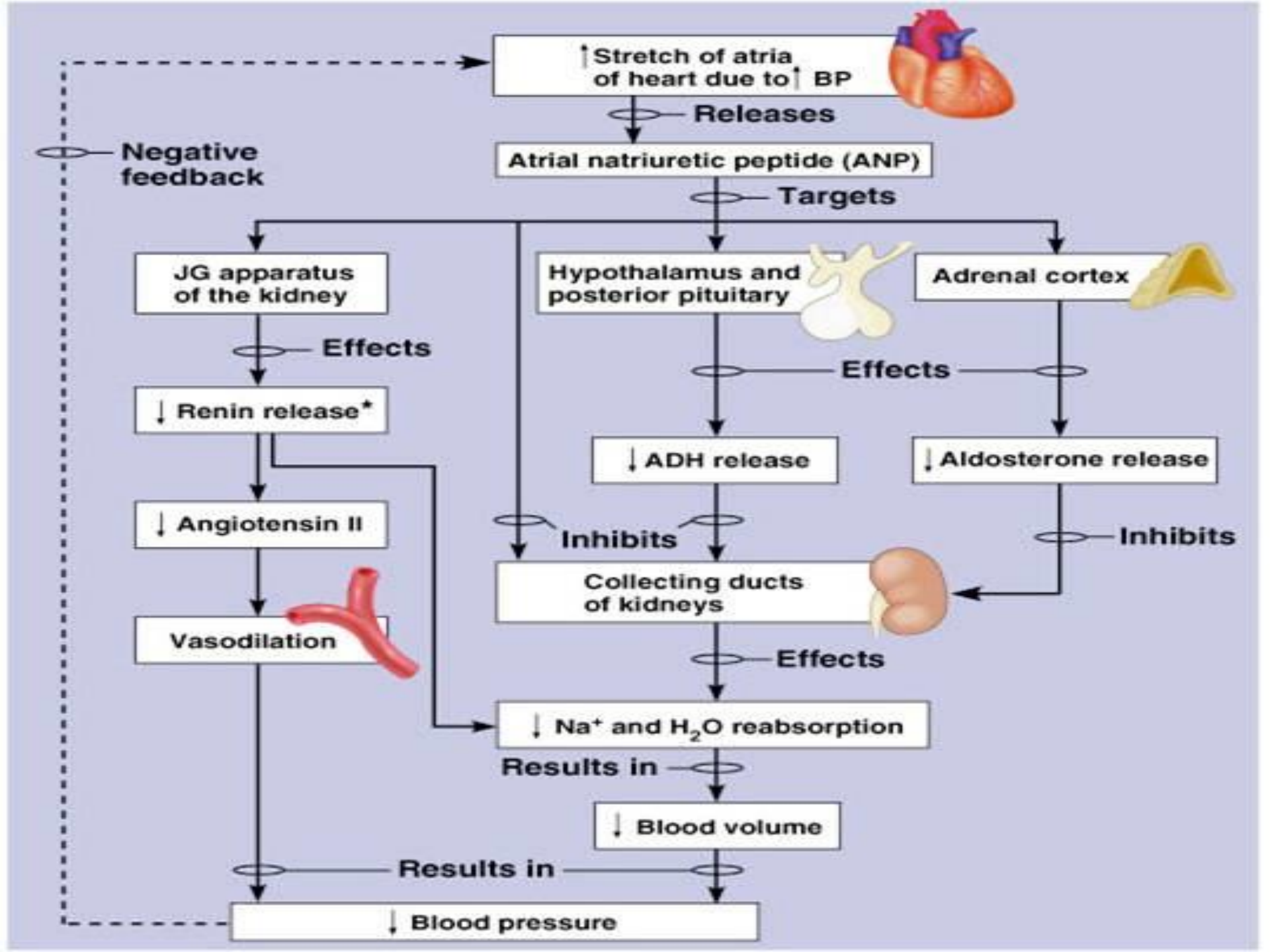

Sympathetic Nerves ③
Afferent Arteriole
Juxtaglomerular Cells
Nephron
Glomerulus
Macula Densa
Efferent Arteriole
Renin Secretion
↑ Macula densa signal
↓ Renal artery pressure/blood flow
↑ Sympathetic stimulation
ANGIOTENSINOGEN
Renin ⑦
ANGIOTENSIN I
Converting Enzyme ①
ANGIOTENSIN II
②
Adrenal Cortex
Kidney
Intestine
CNS
Peripheral Nervous System
Vascular Smooth Muscle
Heart
↑ Aldosterone synthesis ⑥
Sympathetic Discharge
Vasopressin
Vasoconstriction ④
↑ Contractility ③④
Sodium/Water Reabsorption ⑤
↑ Blood Volume
↑ Total Peripheral Resistance ⑤
↑ Cardiac Output
↑ Blood Pressure

- *Circulating angiotensin II can elevate BP through pressor and volume effects*
- *Pressor effects include direct*
  - *Vasoconstriction,*
  - *Stimulation of catecholamine release from the adrenal medulla,*
  - *Centrally mediated increases in sympathetic nervous system activity.*
- *Volume effect-*
  - *Aldosterone synthesis from the adrenal cortex. This leads to sodium and water reabsorption that increases plasma volume, total peripheral resistance, and ultimately BP.*

Atrial natriuretic peptide/ harmone

Atrial natriuretic peptide (ANP)is a powerful vasodilator, and a protein (polypeptide) hormone secreted by heart muscle cells.

- ***ANP is secreted in response to:***
  - ***Increased intravascular volume***
  - ***Atrial distention***


↑Stretch of atria of heart due to↑ BP
Releases
Atrial natriuretic peptide (ANP)
Negative feedback
Targets
JG apparatus of the kidney
Hypothalamus and posterior pituitary
Adrenal cortex
Effects
Effects
↓ Renin release*
↓ ADH release
↓ Aldosterone release
↓ Angiotensin II
Inhibits
Inhibits
Collecting ducts of kidneys
Vasodilation
Effects
↓ Na+ and H2O reabsorption
Results in
↓ Blood volume
Results in
↓ Blood pressure

Natriuretic hormone inhibits sodium and potassium-ATPase and thus interferes with sodium transport across cell membranes

Inherited defects in the kidney's ability to eliminate sodium can cause increased blood volume.

A compensatory increase in the concentration of circulating natriuretic hormone theoretically could increase urinary excretion of sodium and water.

However, this hormone might block the active transport of sodium out of arteriolar smooth muscle cells.

The increased intracellular sodium concentration ultimately would increase vascular tone and BP.

Insulin Resistance and Hyperinsulinemia

- *The development of hypertension and associated metabolic abnormalities is referred to as the **metabolic syndrome.***

- *Increased insulin concentrations may **increase renal sodium retention** and **enhanced sympathetic nervous system activity***

- *Insulin has growth hormone–like actions that can induce **hypertrophy of vascular smooth muscle cells***

- *Also may elevate BP by **increasing intracellular calcium**, which leads to increased vascular resistance.*

2)NEURONAL REGULATION

Autonomic nervous system

- *Autonomic nervous system is involved in the regulation of arterial BP.*

- *The α and β presynaptic receptors play a role in negative and positive feedback to the norepinephrine-containing vesicles located near the neuronal ending.*

- *Stimulation of presynaptic α-receptors (α2) exerts a negative inhibition on norepinephrine release.*

Autonomic nervous system

- ***Stimulation of presynaptic βreceptors facilitates norepinephrine release.***

- ***Stimulation of postsynaptic α1 receptors in smooth muscles elicit vasoconstriction.***

- ***Stimulation of β1-receptors in the heart results in an increase in heart rate and contractility.***

- ***Stimulation of β2-receptors in the arterioles and venules causes vasodilation.***

Baroreceptors

- *The baroreceptor reflex system is the major negative-feedback mechanism that controls sympathetic activity.*

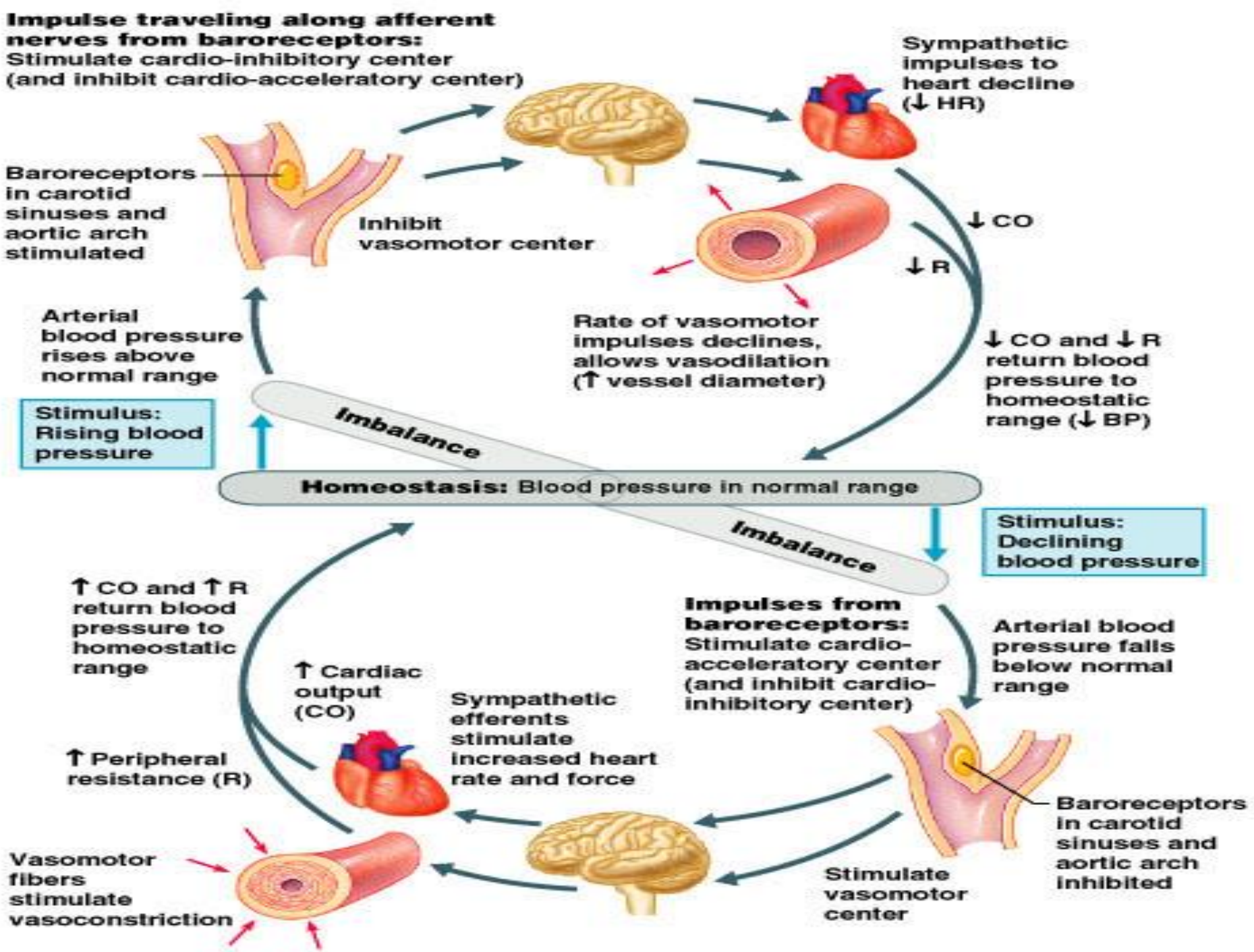
- *Baroreceptors are nerve endings lying in the walls of large arteries, especially in the carotid arteries and aortic arch.*

- *Changes in arterial pressure rapidly activate baroreceptors that then transmit impulses to the brainstem through the ninth cranial nerve and vagus nerves.*

Baroreceptors

- ***In this reflex system, a decrease in arterial BP stimulates baroreceptors, causing reflex vasoconstriction and increased heart rate and force of cardiac contraction.***

- ***These baroreceptor reflex mechanisms may be blunted (less responsive to changes in BP) in the elderly and those with diabetes.***



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Central nervous system

- ***Stimulation of certain areas within the central nervous system (nucleus tractus solitarius, vagal nuclei, vasomotor center, and the area postrema) can either increase or decrease BP.***

- ***For example,***

Adrenergic stimulation within the central nervous system decreases BP through an inhibitory effect on the vasomotor center.

Angiotensin II increases sympathetic outflow from the vasomotor center, which increases BP.

- ***The purpose of these neuronal mechanisms is to regulate BP and maintain homeostasis.***

- ***Pathologic disturbances in any of the four major components could conceivably lead to chronically elevated BP.***
- ***Autonomic nerve fibers,***
- ***Adrenergic receptors,***
- ***Baroreceptors, or***
- ***Central nervous system.***

3)PERIPHERAL AUTOREGULATORY COMPONENTS

- ***Abnormalities in renal or tissue autoregulatory systems could cause hypertension.***

- ***When BP drops, the kidneys respond by increasing retention of sodium and water.***

- ***These changes lead to plasma volume expansion that increases BP and vice versa when BP increases.***

3)PERIPHERAL AUTOREGULATORY COMPONENTS

- ***Tissue autoregulatory processes maintain adequate tissue oxygenation.***

- ***When tissue oxygen demand is normal to low, the local arteriolar bed remains relatively vasoconstricted.***

- ***However, increases in metabolic demand trigger arteriolar vasodilation that lowers peripheral vascular resistance and increases blood flow and oxygen delivery through autoregulation.***

- *Intrinsic defects in these renal adaptive mechanisms could lead to plasma volume expansion and increased blood flow to peripheral tissues, even when BP is normal.*

- *Local tissue autoregulatory processes that vasoconstrict would then be activated to offset the increased blood flow.*

- *This effect would result in increased peripheral vascular resistance, and if sustained, would also result in thickening of the arteriolar walls.*

4) VASCULAR ENDOTHELIAL MECHANISMS

- ***Vascular endothelium and smooth muscle play important roles in regulating blood vessel tone and BP mediated by vasoactive substances that are synthesized by endothelial cells.***

- ***Nitric oxide is produced in the endothelium, relaxes the vascular epithelium, and is a very potent vasodilator.***

- ***The nitric oxide system is an important regulator of arterial BP.***

- *Patients with hypertension may have an intrinsic **deficiency in nitric oxide**, resulting in inadequate vasodilation*

- *It has been postulated that a **deficiency** in the local synthesis of vasodilating substances (prostacyclin and bradykinin)*

or

- ***Excess** vasoconstricting substances (angiotensin II and endothelin I) contribute to essential hypertension, atherosclerosis, and other CV diseases*

5)ELECTROLYTES AND OTHER CHEMICALS

Sodium

- *Excess sodium intake will lead to hypertension*

- *The exact mechanisms by which excess sodium leads to hypertension are unknown.*

- *When NaCl intake exceeds the capacity of the kidney to excrete sodium, vascular volume initially expands and cardiac output increases.*

Sodium

- *Atrial natriuretic hormone is thought to block the active transport of sodium out of arteriolar smooth muscle cells.*

- *The increased intracellular sodium concentration ultimately would increase vascular tone and BP.*

Calcium

- ***A lack of dietary calcium hypothetically can disturb the balance between intracellular and extracellular calcium, resulting in an increased intracellular calcium concentration.***

- ***This imbalance can alter vascular smooth muscle function by increasing peripheral vascular resistance.***

Potassium

- *The role of potassium fluctuations is also inadequately understood.*

- *Potassium depletion may increase peripheral vascular resistance, but the clinical significance of small serum potassium concentration changes is unclear.*

Symptoms

- ***Most patients with hypertension are asymptomatic***

- ***A "hypertensive headache" observed in patients with severe hypertension which occurs in the morning and is localized to the occipital region.***

Signs: Previous BP values in either the prehypertension or the hypertension category

Other nonspecific symptoms

- ***Dizziness ,***
- ***Palpitations ,***
- ***Easy fatigability.***

Complications

- Damage to the heart and coronary arteries,
- Stroke
- CKD
- Vision loss
- Ischeic heart disease
- Heart failure
- Myocardial infarction
- Erectile dysfunction
- Cognitive changes
- Fluid in the lungs
- Hepatomegaly
- Cor-pulmanale
- Angina
- Peripheral artery disease

Thank you