Vascular Physiology

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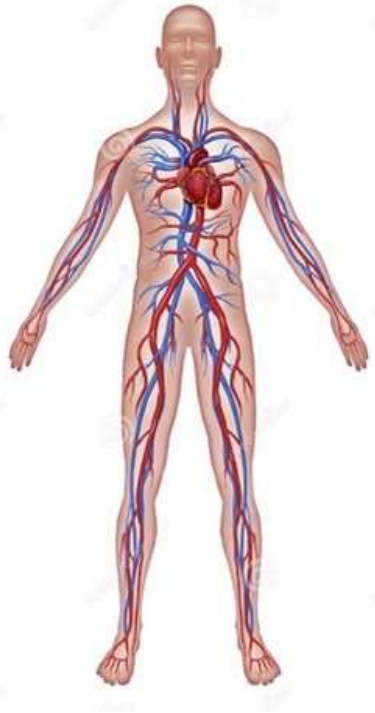
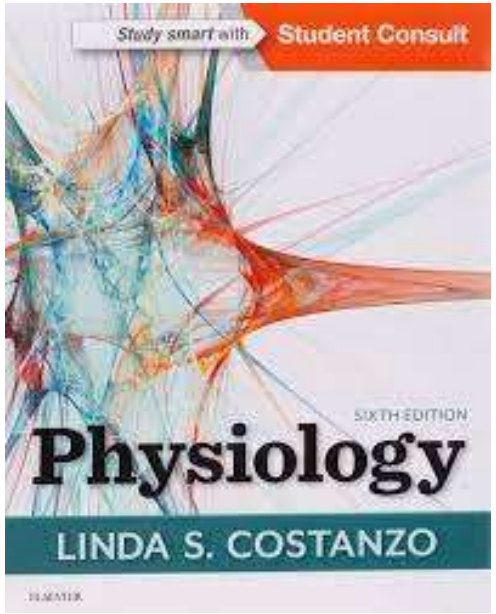


References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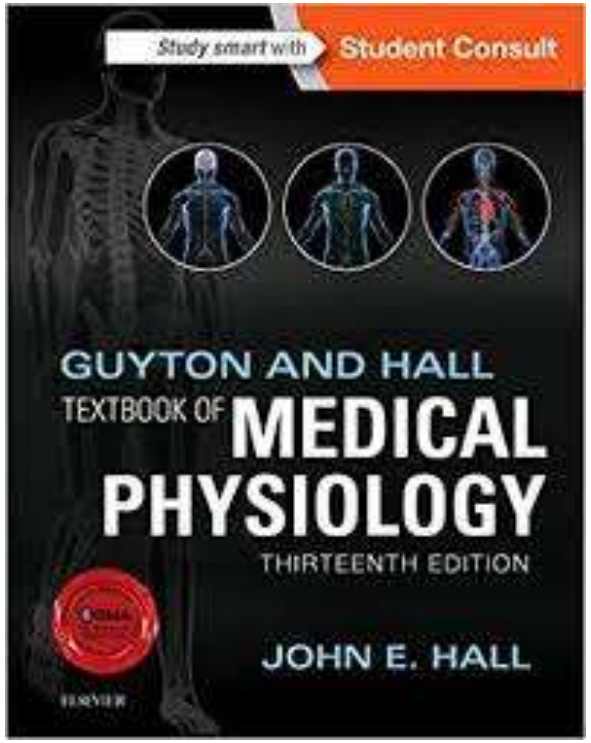
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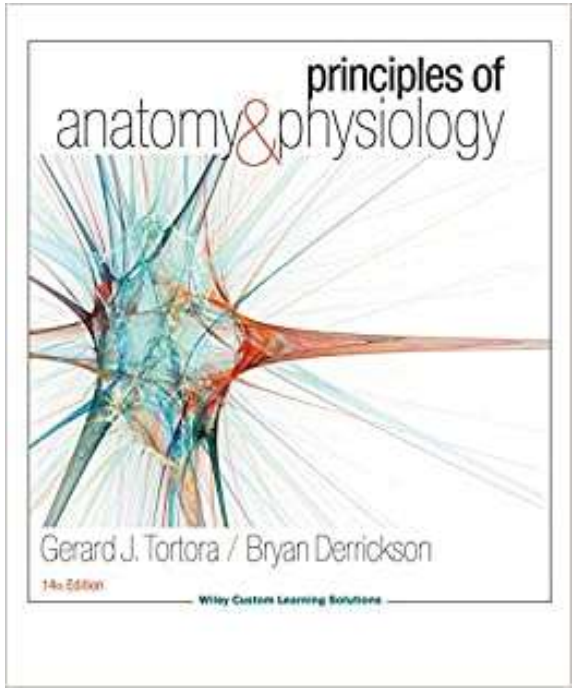
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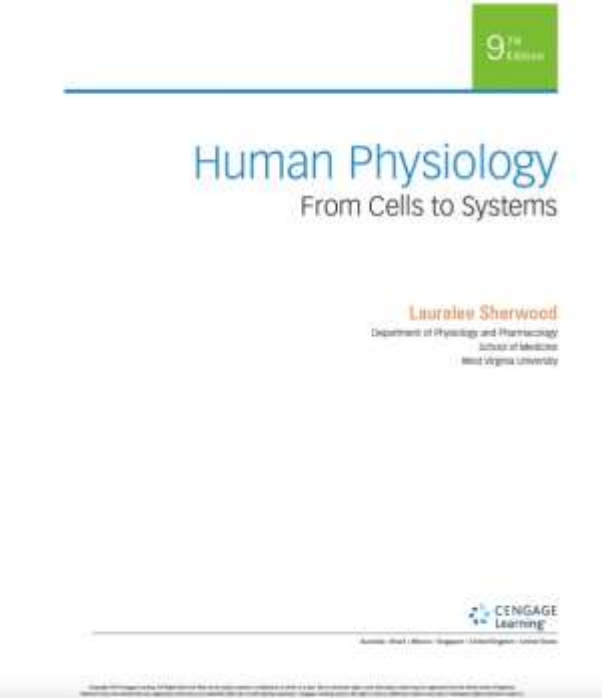

Human Physiology
From Cells to Systems
Lauralee Sherwood
CENGAGE Learning

Blood pressure control

Clinical implications

- Postural hypotension
- Spinal anesthesia
- Vasovagal syncope

Short term control

- Short-term (within seconds) adjustments are made by alterations in CO and TPR, mediated by means of autonomic nervous system influences on the heart, veins, and arterioles.

Long term control

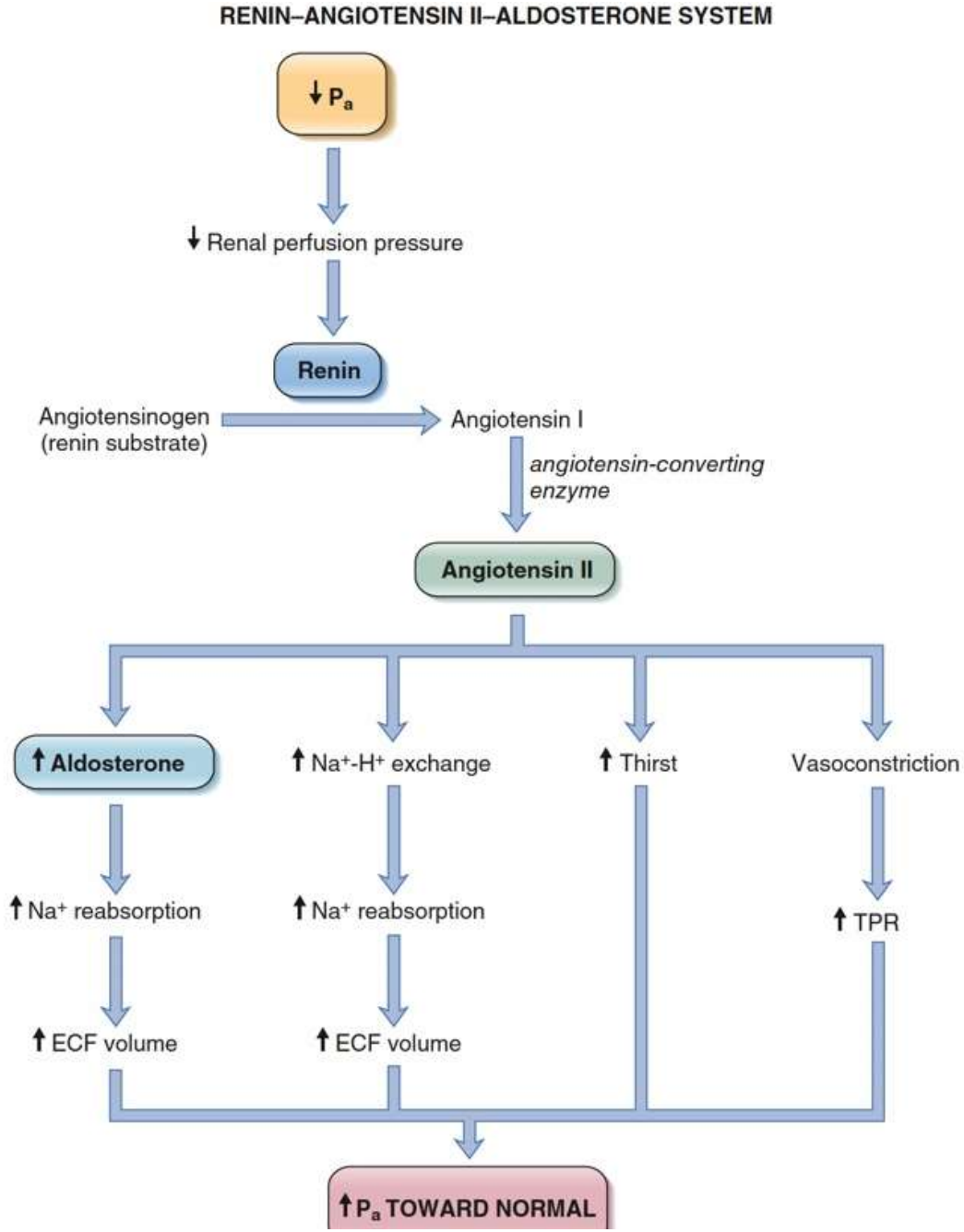
- Long-term (requiring minutes to days) control involves adjusting total blood volume by restoring normal salt and water balance through mechanisms that regulate urine output and thirst.
- The size of the total blood volume, in turn, has a profound effect on CO and thereby, MAP.

Capillary control

- Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments

Renal control of BP

- The kidneys regulate blood pressure long-term by adjusting sodium and water excretion.
- When BP rises, they excrete more sodium and water (pressure–natriuresis).
- When BP falls, they activate RAAS, increase sympathetic tone, and enhance water/Na^+ reabsorption—all of which raise blood volume, cardiac output, and vascular tone, restoring BP.
- Hormones such as ADH and ANP further fine-tune these processes.


RENIN–ANGIOTENSIN II–ALDOSTERONE SYSTEM
↓ P_a
↓ Renal perfusion pressure
Renin
Angiotensinogen (renin substrate)
Angiotensin I
angiotensin-converting enzyme
Angiotensin II
↑ Aldosterone
↑ Na^+-H^+ exchange
↑ Thirst
Vasoconstriction
↑ Na^+ reabsorption
↑ Na^+ reabsorption
↑ TPR
↑ ECF volume
↑ ECF volume
↑ P_a TOWARD NORMAL

RAAS

- A decrease in P causes a decrease in renal perfusion pressure, which is sensed by mechanoreceptors in afferent arterioles of the kidney.
- The decrease in P causes prorenin to be converted to renin in the juxtaglomerular cells.
- Renin secretion by the juxtaglomerular cells is also increased by stimulation of renal sympathetic nerves and by β1 agonists.

RAAS

- Renin is an enzyme. In plasma, renin catalyzes the conversion of angiotensinogen to angiotensin I.
- Angiotensin I has little biologic activity.
- In the lungs and kidneys, angiotensin I is converted to angiotensin II, catalyzed by angiotensin-converting enzyme (ACE).

RAAS

- Angiotensin II has the following biologic actions in the adrenal cortex, vascular smooth muscle, kidneys, and brain, where it activates type 1 G protein–coupled angiotensin II receptors (AT1 receptors).

RAAS

- Angiotensin II acts on the zona glomerulosa cells of the adrenal cortex to stimulate the synthesis and secretion of aldosterone.
- Aldosterone then acts on the principal cells of the renal distal tubule and collecting duct to increase Na+ reabsorption and, thereby, to increase ECF volume and blood volume.
- The actions of aldosterone require gene transcription and new protein synthesis in the kidney. These processes require hours to days to occur and account for the slow response time of the renin–angiotensin II–aldosterone system.

RAAS

- Angiotensin II also has its own direct action on the kidney, independent of its actions through aldosterone.
- Angiotensin II stimulates Na+-H+ exchange in the renal proximal tubule and increases the reabsorption of Na+ and HCO3−.

RAAS

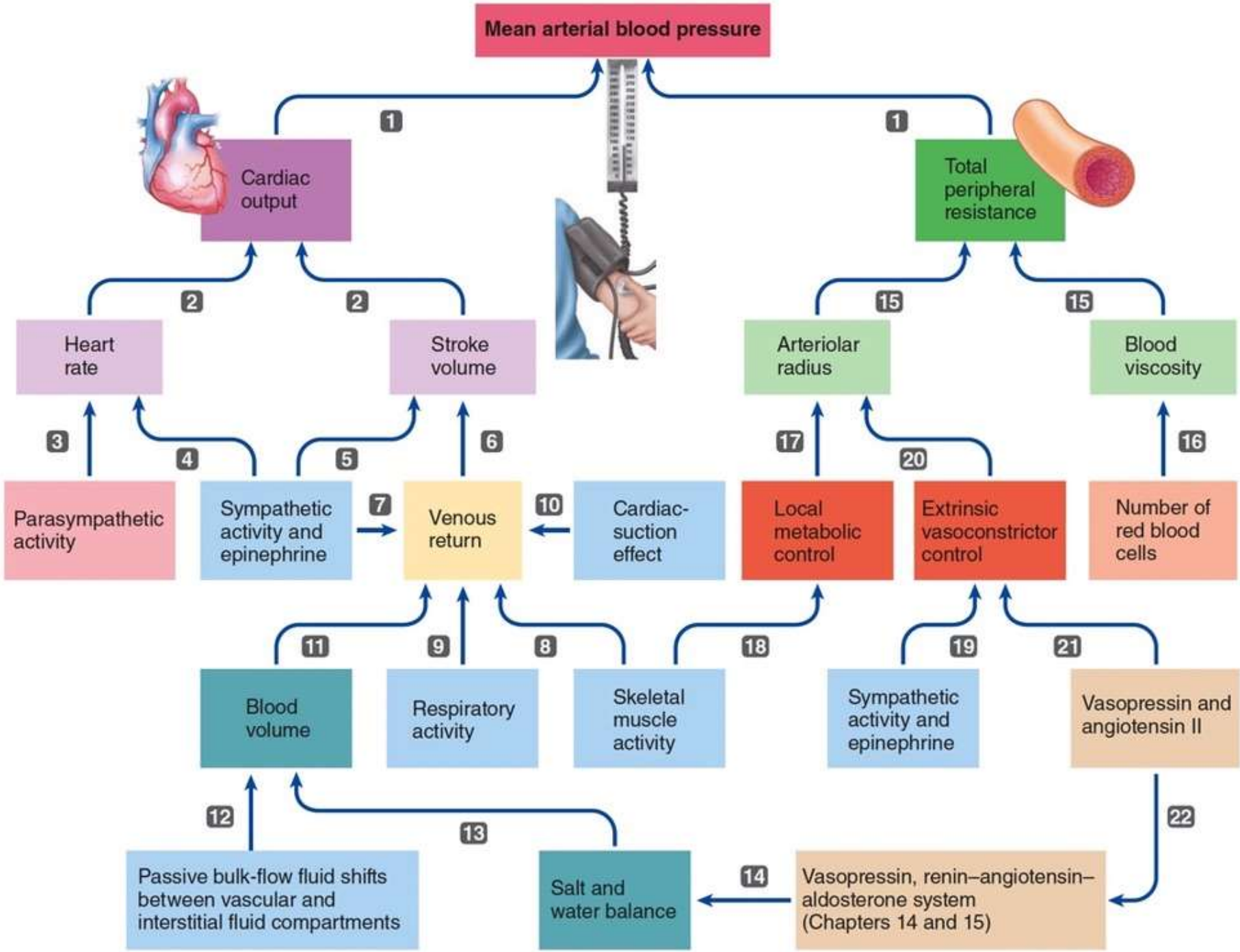
- Angiotensin II acts on the hypothalamus to increase thirst and water intake.
- It also stimulates secretion of antidiuretic hormone (ADH), which increases water reabsorption in collecting ducts.
- By increasing total body water, these effects complement the increases in Na+ reabsorption (caused by aldosterone and Na+-H+ exchange), thereby increasing ECF volume, blood volume, and blood pressure.

RAAS

- Angiotensin II also acts directly on the arterioles by binding to G protein–coupled AT1 receptors and activating an inositol 1,4,5-triphosphate (IP3)/Ca2+ second messenger system to cause vasoconstriction.
- The resulting increase in TPR leads to an increase in P.

ADH

- ADH, a hormone secreted by the posterior lobe of the pituitary gland, regulates body fluid osmolarity and participates in the regulation of arterial blood pressure.
- There are two types of receptors for ADH: V1 receptors, which are present in vascular smooth muscle, and V2 receptors, which are present in principal cells of the renal collecting ducts.
- When activated, the V1 receptors cause vasoconstriction of arterioles and increased TPR.
- The V2 receptors are involved in water reabsorption in the collecting ducts and the maintenance of body fluid osmolarity.
- ADH secretion from the posterior pituitary is increased by two types of stimuli: by increases in serum osmolarity and by decreases in blood volume and blood pressure.


Mean arterial blood pressure
Cardiac output
Total peripheral resistance
Heart rate
Stroke volume
Arteriolar radius
Blood viscosity
Parasympathetic activity
Sympathetic activity and epinephrine
Venous return
Cardiac-suction effect
Local metabolic control
Extrinsic vasoconstrictor control
Number of red blood cells
Blood volume
Respiratory activity
Skeletal muscle activity
Sympathetic activity and epinephrine
Vasopressin and angiotensin II
Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments
Salt and water balance
Vasopressin, renin–angiotensin–aldosterone system (Chapters 14 and 15)
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High blood pressure (Hypertension)

- Definition
- Stages
- Types
- Prevalence
- Complications

High blood pressure (Hypertension)

- Until complications occur, hypertension is asymptomatic because the tissues are adequately supplied with blood.

Complications of Hypertension

- The **heart** has an increased workload because it is pumping blood out against an increased TPR, and the high internal pressure may **damage blood vessels**, particularly when the vessel wall is weakened by the degenerative process of atherosclerosis.

Complications of Hypertension

- **left ventricular hypertrophy** in the early stages as the heart muscle thickens to pump a normal stroke volume against an elevated blood pressure, followed in later stages by systolic **heart failure** as the heart weakens and becomes unable to pump continuously against a sustained elevation in arterial pressure

Complications of Hypertension

- Strokes
- heart attacks
- Nephropathy
- Retinopathy

American Heart Association.

HOME BLOOD PRESSURE MEASUREMENT INSTRUCTIONS

Before You Measure

- No smoking, caffeinated beverages, alcohol or exercise 30 minutes prior.
- Use a validated device with the correct cuff size. (Visit Validate BP to find a device you can trust.)
- Empty your bladder.
- Sit quietly for more than 5 minutes and do not talk.

Proper Positioning

- Sit upright with back supported, feet on floor and legs uncrossed.
- Rest your arm comfortably on a flat surface at heart level.
- Wrap the cuff on your bare skin above the bend of the elbow, not over clothing.

During Measurement

- Stay relaxed and do not talk.
- Take at least two readings, 1 minute apart.
- Record all results once measurement is completed and share them with your health care professional to help confirm your office blood pressure category.

BLOOD PRESSURE HIGHER THAN 180/120 MM HG

MAY BE A HYPERTENSIVE EMERGENCY*

* Wait a few minutes and take blood pressure again.

American Heart Association recommended office blood pressure categories

Thank you