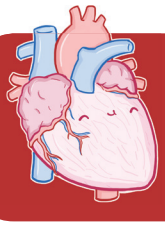

NOTES
NORMAL VARIATIONS

- Physiological adaptations within cardiovascular system in response to changes such as hemorrhage, exercise, postural changes

CARDIOVASCULAR CHANGES DURING EXERCISE

osms.it/cardiovascular-changes-exercise

- Involves central nervous system (CNS), local mechanisms
  - *CNS responses:* changes in autonomic nervous system (ANS) due to inputs from cerebral motor cortex
  - *Local responses:* exercise causes ↑ blood flow, O_2 delivery to skeletal muscles
- Exercise results in ↑ sympathetic (ß1 receptors), ↓ parasympathetic activity to heart → ↑ cardiac output due to ↑ heart rate + ↑ stroke volume
- Muscle changes also occur
  - ↑ metabolites (lactate, potassium, adenosine) are produced → metabolites stimulate local vasodilation → ↑ blood flow → ↓ overall total peripheral resistance (TPR)

OVERALL RESPONSE TO EXERCISE

- *Central command:* ↑ cardiac output (CO), vasoconstriction in some vascular beds (excludes exercising skeletal muscle, cerebral, coronary circulations)
  - ↑ CO → ↑ heart rate, contractility
  - ↑ contractility → ↑ stroke volume → ↑ pulse pressure
  - ↑ CO due to ↑ venous return (sympathetic vein constriction, squeezing action of skeletal muscle on veins)

CV RESPONSES TO EXERCISE OVERVIEW

	RESPONSE
HEART RATE	↑↑
STROKE VOLUME	↑
PULSE PRESSURE	↑ (increased stroke volume)
CARDIAC OUTPUT	↑↑
VENOUS RETURN	↑
MEAN ARTERIAL PRESSURE	↑ (slight)
TPR	↓↓
ARTERIOVENOUS O_2 DIFFERENCE	↑↑ (increased tissue O_2 composition)

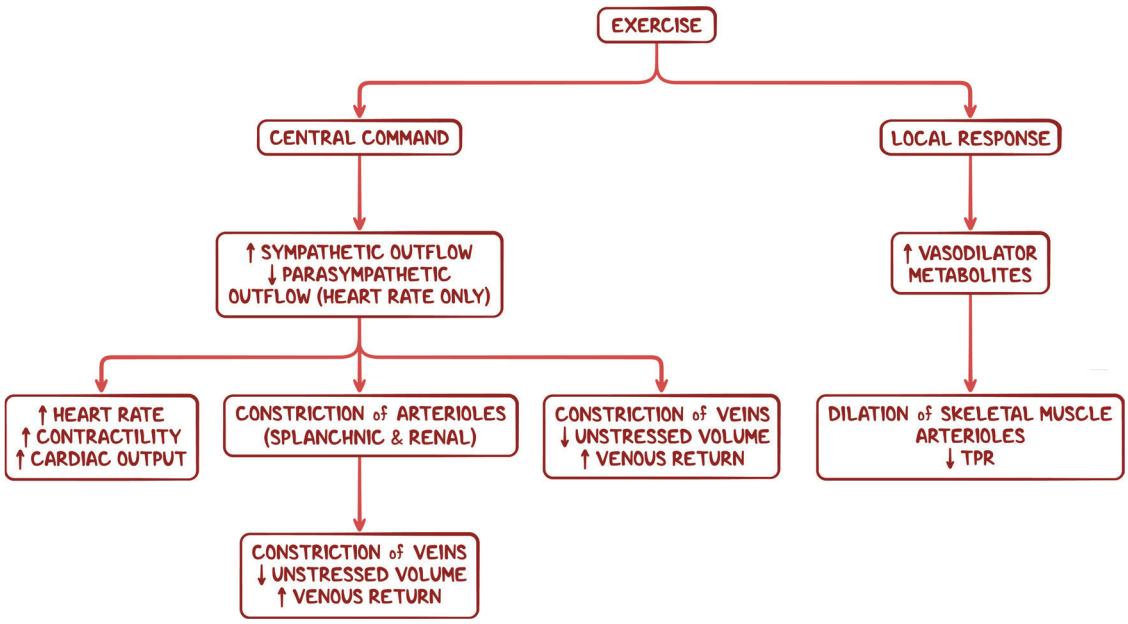

Figure 20.1 Flowchart showing cardiovascular response to exercise.

CARDIOVASCULAR CHANGES DURING HEMORRHAGE

osms.it/cardiovascular-changes-hemorrhage

- Blood loss → ↓ arterial pressure → compensatory responses to restore arterial pressure
  - Response mediated by baroreceptor reflex, renin-angiotensin-aldosterone system (RAAS), vascular actions

Decrease in arterial pressure

- Hemorrhage → ↓ total blood volume → ↓ venous return to heart, ↓ right atrial pressure → ↓ cardiac output → ↓ P_a as a product of cardiac output, TPR

Return of arterial pressure

- Baroreceptors in carotid sinus detect ↓ P_a → relay information to medulla via carotid sinus nerve → ↑ sympathetic outflow to heart, blood vessels; ↓ parasympathetic outflow to heart → ↑ heart rate, ↑ contractility, ↑ TPR, constriction of veins
- ↓ mean arterial pressure → ↓ perfusion to kidney → response via RAAS
  - Kidney secretes renin from renal juxtaglomerular cells → ↑ angiotensin I production → converted to angiotensin II (causes arteriolar vasoconstriction, stimulates aldosterone secretion)
- Capillary changes favor fluid reabsorption
  - ↑ sympathetic outflow to blood vessels, angiotensin II → arteriolar vasoconstriction → ↓ capillary hydrostatic pressure (P_c) → restricts filtration out of capillaries, favors absorption

OTHER RESPONSES IN HEMORRHAGE

- *Hypoxemia (↓ arterial P_{O2}):* carotid, aortic bodies chemoreceptors sense ↓ P_{O2} → ↑ sympathetic outflow to blood vessels → ↑ vasoconstriction, TPR, P_a
- *Cerebral ischemia:* local ↑ P_{CO2}

- ↓ blood volume → ↓ return of blood to heart → detection by atria volume receptors → ADH secretion to maintain adequate blood pressure → water reabsorption by renal collecting ducts → arteriolar vasoconstriction

CV RESPONSES TO HEMORRHAGE OVERVIEW

	RESPONSE
CAROTID SINUS NERVE FINDING RATE	↓
HEART RATE	↑
CONTRACTILITY	↑
CARDIAC OUTPUT	↑
UNSTRESSED VOLUME	↓ (produces increased venous return)
TPR	↑
RENIN	↑
ANGIOTENSIN II	↑
ALDOSTERONE	↑
CIRCULATING EPINEPHRINE	↑ (secreted from adrenal medulla)
ANTIDIURETIC HORMONE (ADH)	↑ (stimulated by decreased blood volume)

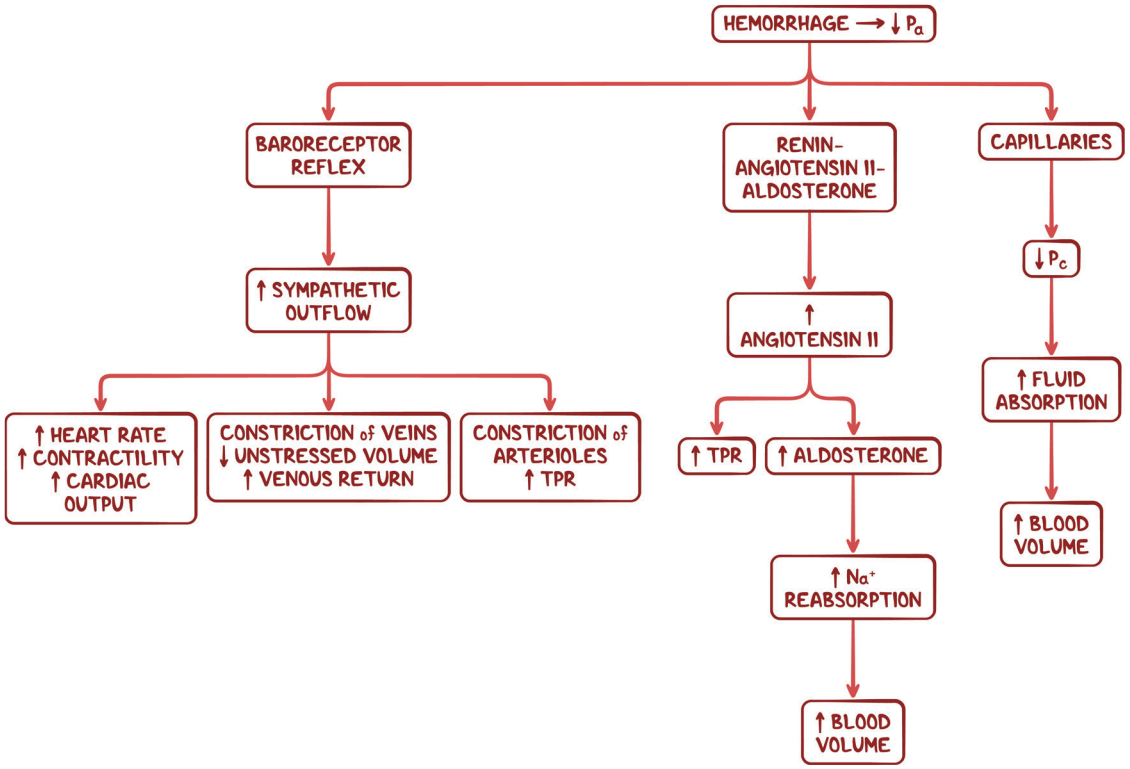

Figure 20.2 Flowchart showing cardiovascular responses to hemorrhage.

CARDIOVASCULAR CHANGES DURING POSTURAL CHANGE

osms.it/cardiovascular-changes-postural

- Standing up quickly → lightheadedness, sometimes fainting (due to delayed constriction of lower extremity blood vessels → orthostatic hypotension)
  - ↓ in systolic blood pressure > 20mmHg/ diastolic blood pressure > 10mmHg within three minutes of standing
- *Initiating event:* pooling of blood in extremities
  - *Moving from supine to standing position:* blood pools in veins of lower extremities → ↓ venous return to heart, ↓ cardiac output → ↓ mean arterial pressure
  - Venous pooling → ↑ hydrostatic pressure in leg veins → ↑ fluid filtration into interstitial fluid, ↓ intravascular volume
  - Severe ↓ blood pressure → syncope

Response of baroreceptor reflex

- Responsible for homeostatic blood pressure maintenance
- Carotid sinus baroreceptors detect ↓ P_a → sends information to medullary vasomotor center → inactivates medulla vagal neurons, activates sympathetic neurons → ↑ sympathetic outflow to heart, blood vessels, ↓ parasympathetic outflow to heart to normalize P_a
- ↑ systemic vascular resistance, cardiac output act in negative feedback mechanism to maintain P_a

CV RESPONSE TO STANDING

	INITIAL RESPONSE	COMPENSATORY RESPONSE
MEAN ARTERIAL PRESSURE	↓	↑ (toward normal)
HEART RATE	–	↑
STROKE VOLUME	↓ (decreased venous return)	↑ (toward normal)
CARDIAC OUTPUT	↓ (decreased stroke volume)	↑ (toward normal)
TPR	–	↑
CENTRAL VENOUS PRESSURE	↓ (pooling of blood in lower extremities)	↑ (toward normal)

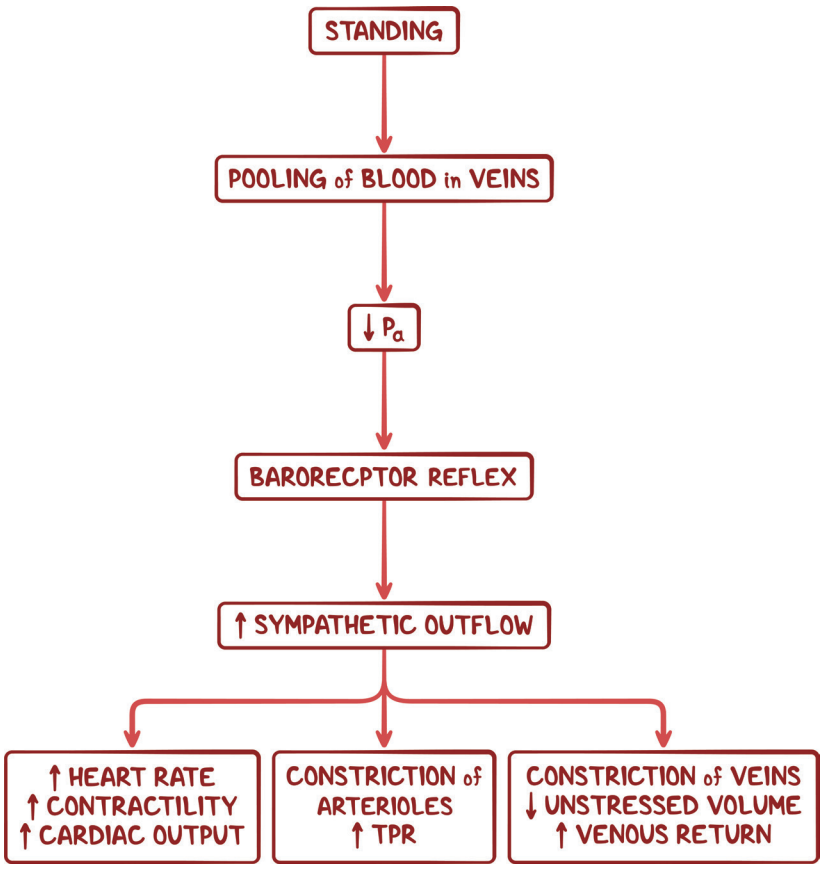

Figure 20.3 Flowchart showing cardiovascular response to postural change.