VESSELS PHYSIOLOGY

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Vascular Endothelium

Vasodilators	Vasoconstrictors
Nitric Oxide **Prostacyclin** **Endothelium-derived hyperpolarizing factor** **Bradykinin**	**Endothelin-1** **Angiotensin II**

Wilson SH, Lerman A.
Heart Physiology and Pathophysiology, Academic Press (edited by Sperelakis N.) 473-480

- Vasodilator
- Inhibitor of vascular smooth muscle cell proliferation
- Inhibitor of platelet adherence/aggregation
- Inhibitor of leukocyte/endothelial interactions

Nitric Oxide (NO) Function

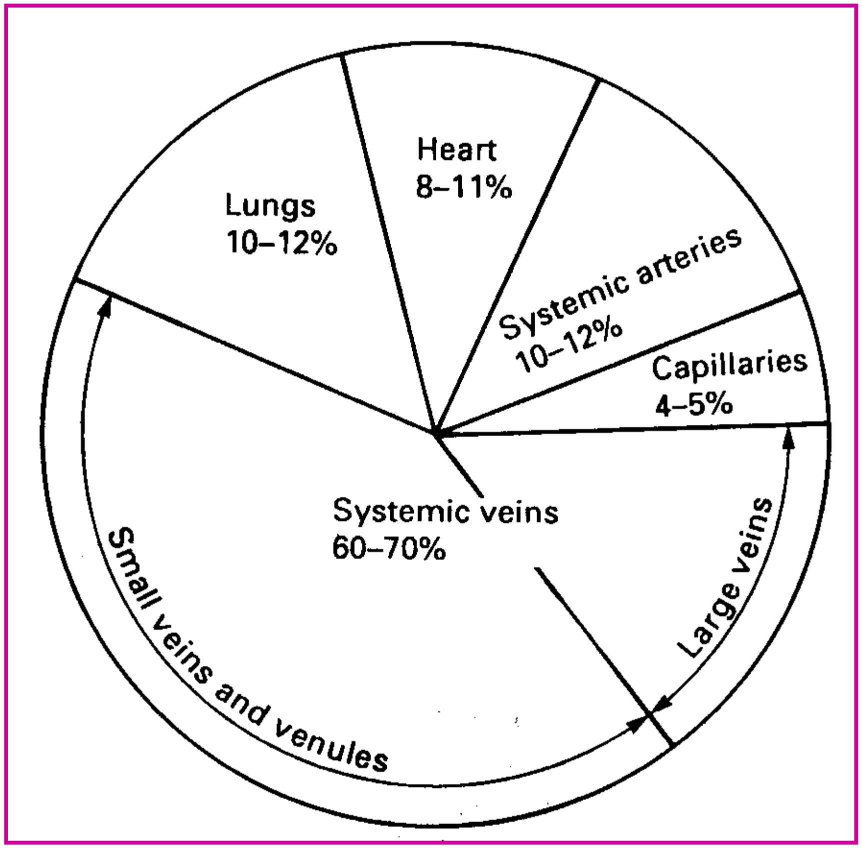
- Imbalance of endothelium-derived relaxing and contracting factors

Endothelial Dysfunction

Atherosclerotic risk factors

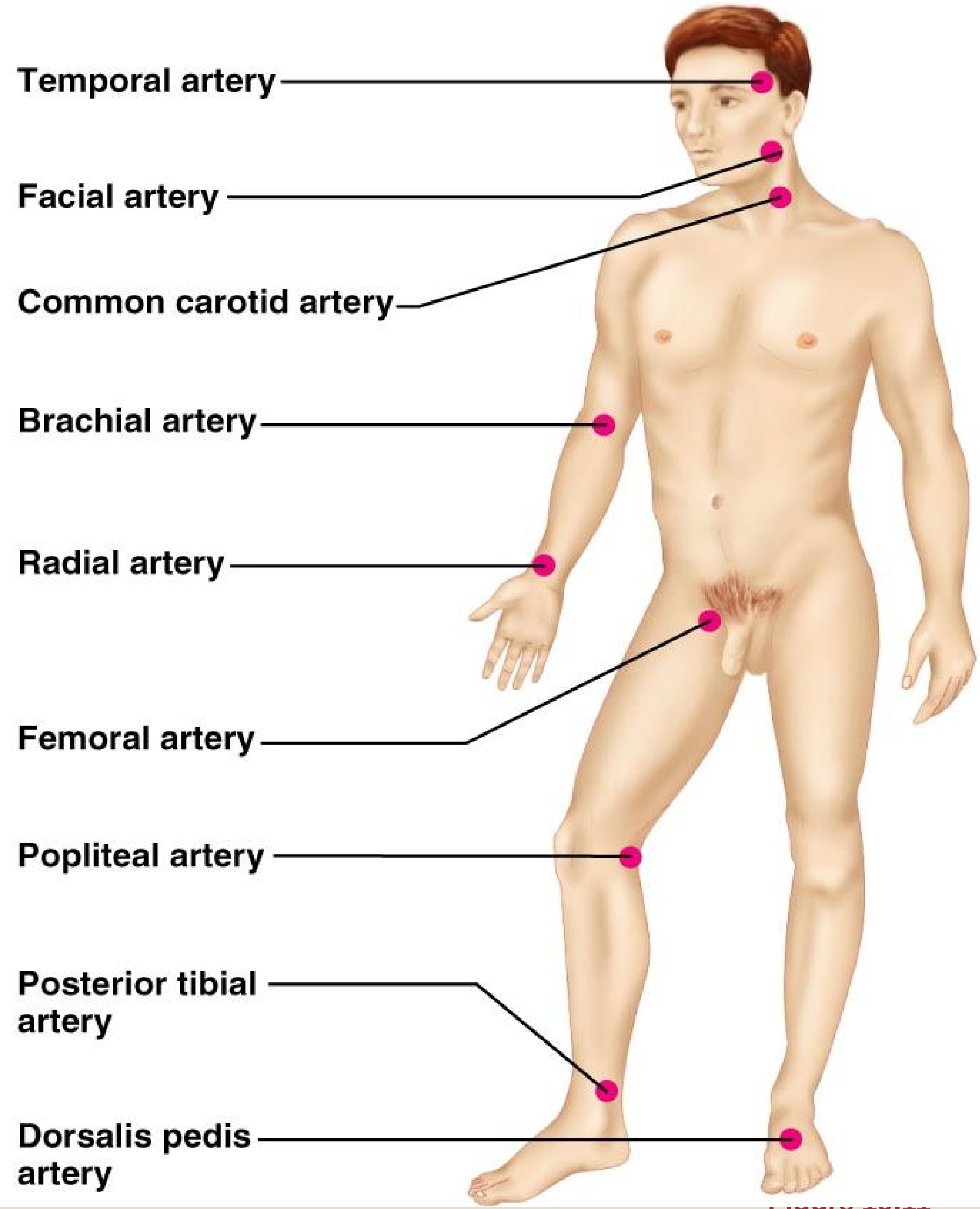
Decreased NO bioavailability
Increased levels of ET-1

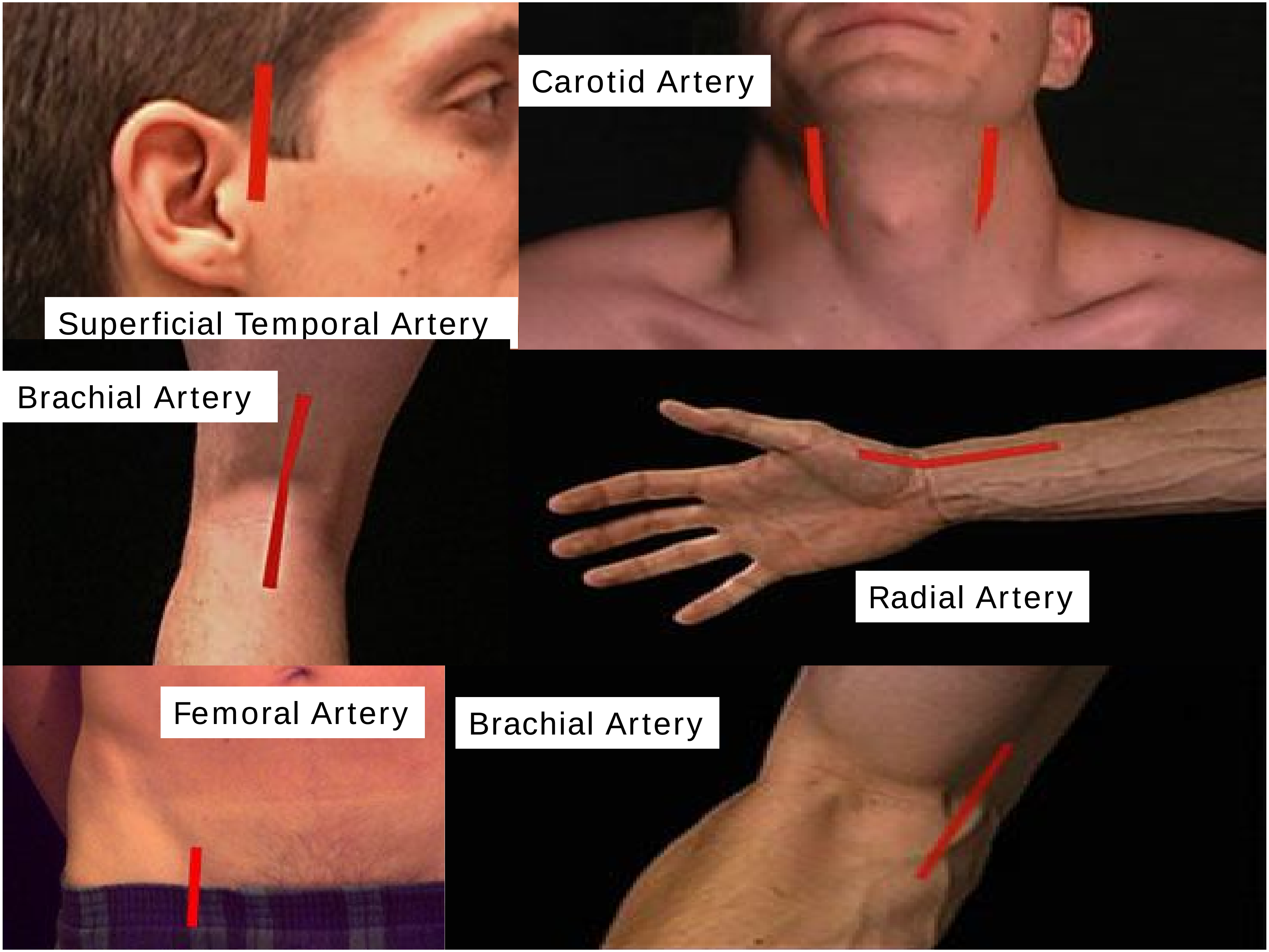
Distribution of blood volume in a resting man (5.5 litres)

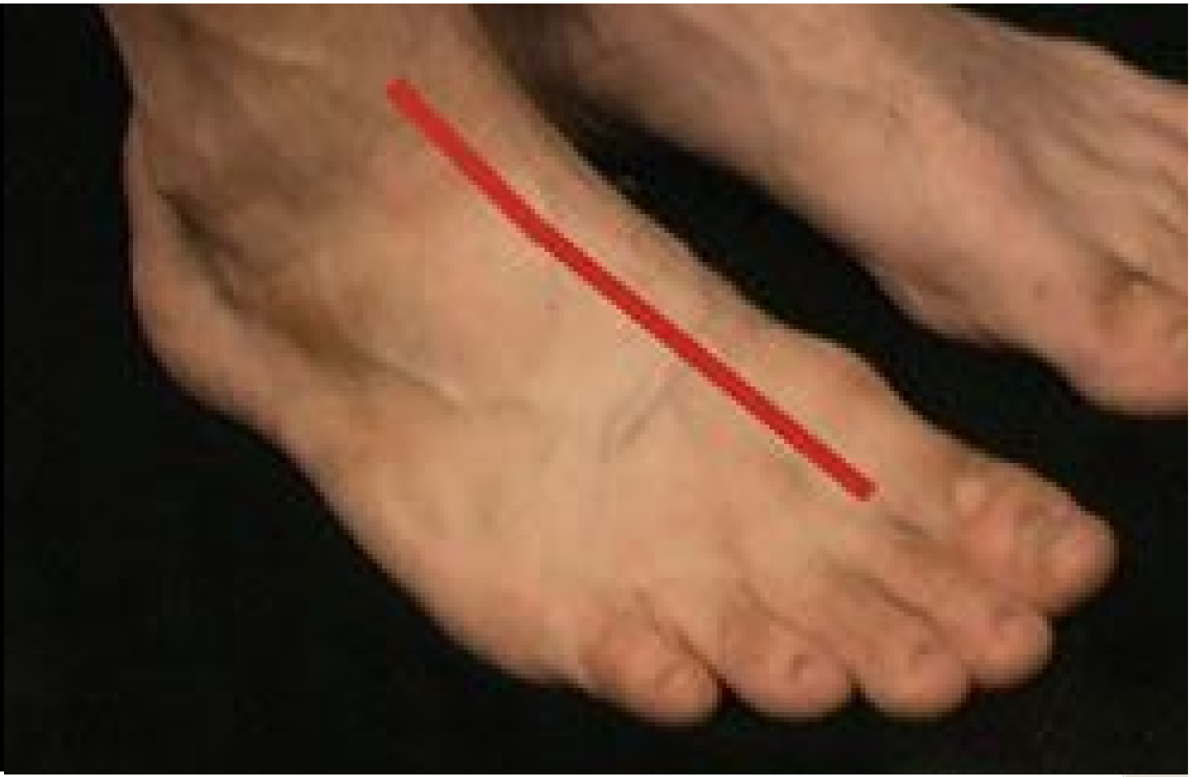


Folkow B, Neil E. 1971,
Oxford University Press, London

Palpated Pulse



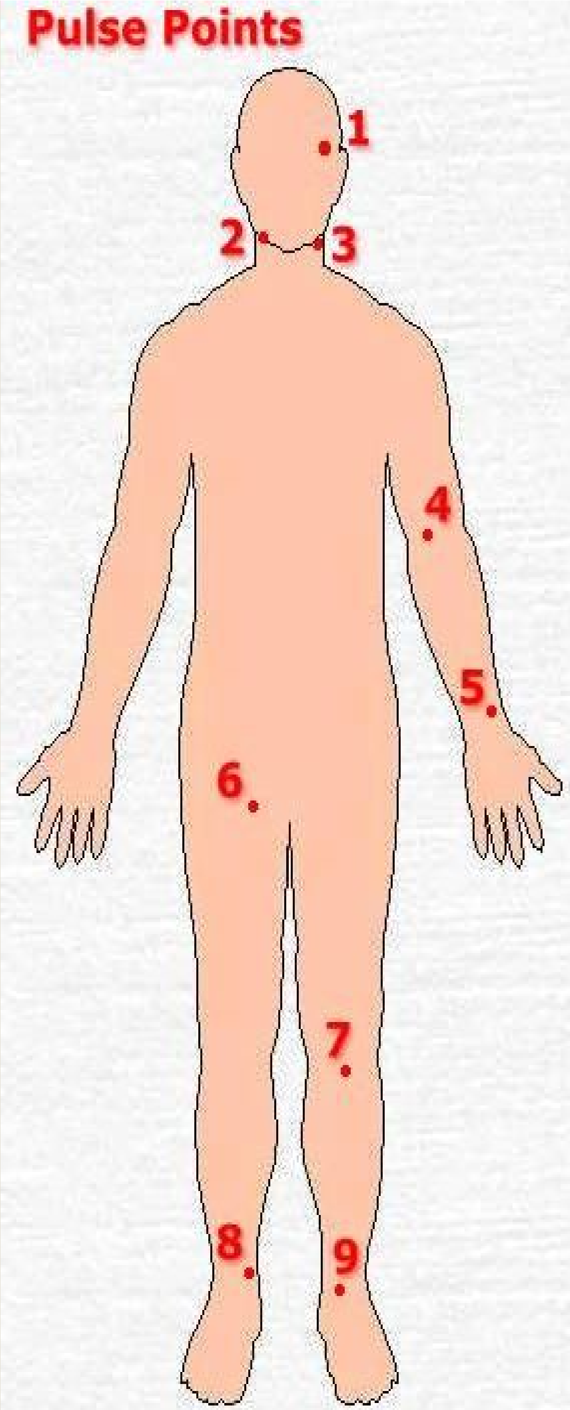

Carotid Artery
Superficial Temporal Artery
Brachial Artery
Radial Artery
Femoral Artery
Brachial Artery



Posterior Tibial Artery

Dorsalis Pedis Artery

http://www.med.umich.edu/lrc/coursepages/M1/anatomy/html/surface/pulses/pulses.html



shows the common sites where the pulse is felt.

1. Temporal artery at the temple above and to the outer side of the eye
2. External maxillary (facial) artery at the point of crossing the mandible (lower jaw)
3. Carotid artery on the side of the neck
4. Brachial artery on the inner side of the biceps
5. Radial artery on the radial bone side of the wrist
6. Femoral artery in the groin
7. Popliteal artery behind the knee
8. Posterior tibial pulse behind the inner ankle
9. Dorsalis pedis artery on the upper front part (anteriosuperior aspect) of the foot

- Your pulse is the rate at which your heart beats.
- Your pulse is usually called your heart rate, which is the number of times your heart beats each minute (bpm).

•Example :
•Given the following data:
Mean Blood Pressure = 100 mmHg
Diastolic Blood Pressure = 90 mmHg
Pulse rate [PR] = 25 pulses/15seconds

PR = (25 pulses /15 seconds) x (60seconds / 1 minute) = 100 pulses / minute
HR = pulse rate = 100 b/min

Pulse rate

- At normal resting heart rates *MAP* can be approximated using the more easily measured systolic and diastolic pressures, *SP* and *DP:*

$$MAP \simeq DP + \frac{1}{3}(SP - DP)$$

- or equivalently

$$MAP \simeq \frac{(2 \times DP) + SP}{3}$$

- or equivalently

$$MAP \simeq DP + \frac{1}{3}PP$$

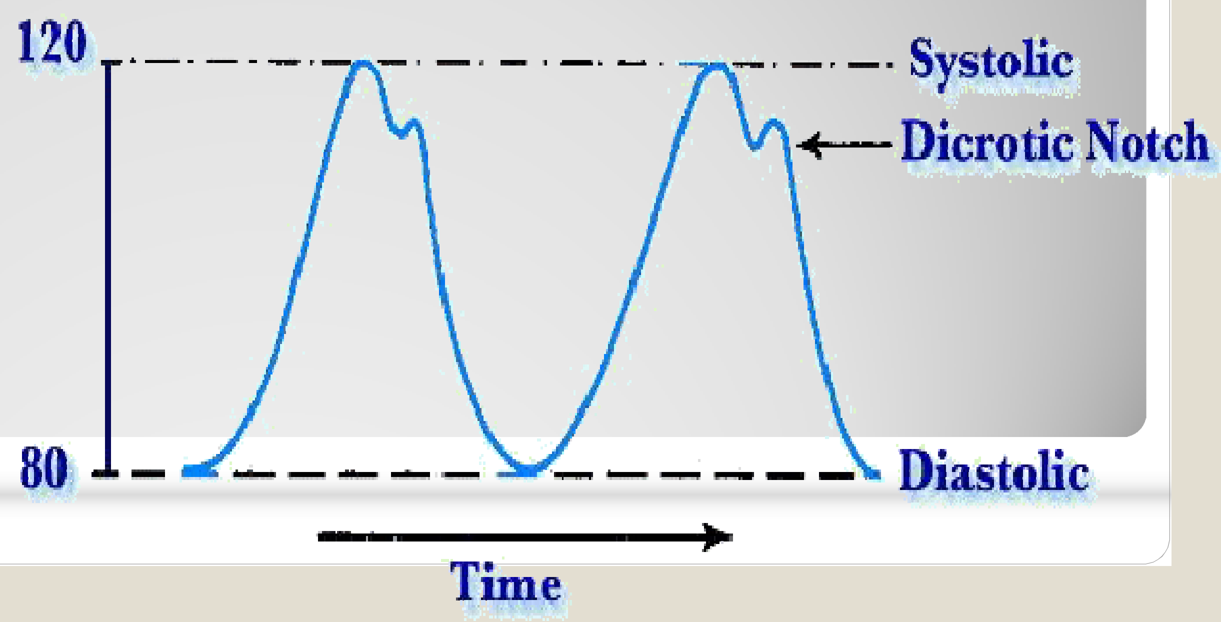
- where *PP* is the pulse pressure, *SP* − *DP*

Mean Blood Pressure

- **Blood Pressure = Cardiac Output x Total Peripheral Resistance**

or

- **BP = CO x TPR**



Maintaining Blood Pressure

- **The main factors influencing blood pressure are**:
  - ***Cardiac output (CO)***
  - ***Peripheral resistance (PR)***
  - ***Blood volume***
- **Blood pressure** = CO x PR
- Blood pressure varies directly with CO, PR, and blood volume

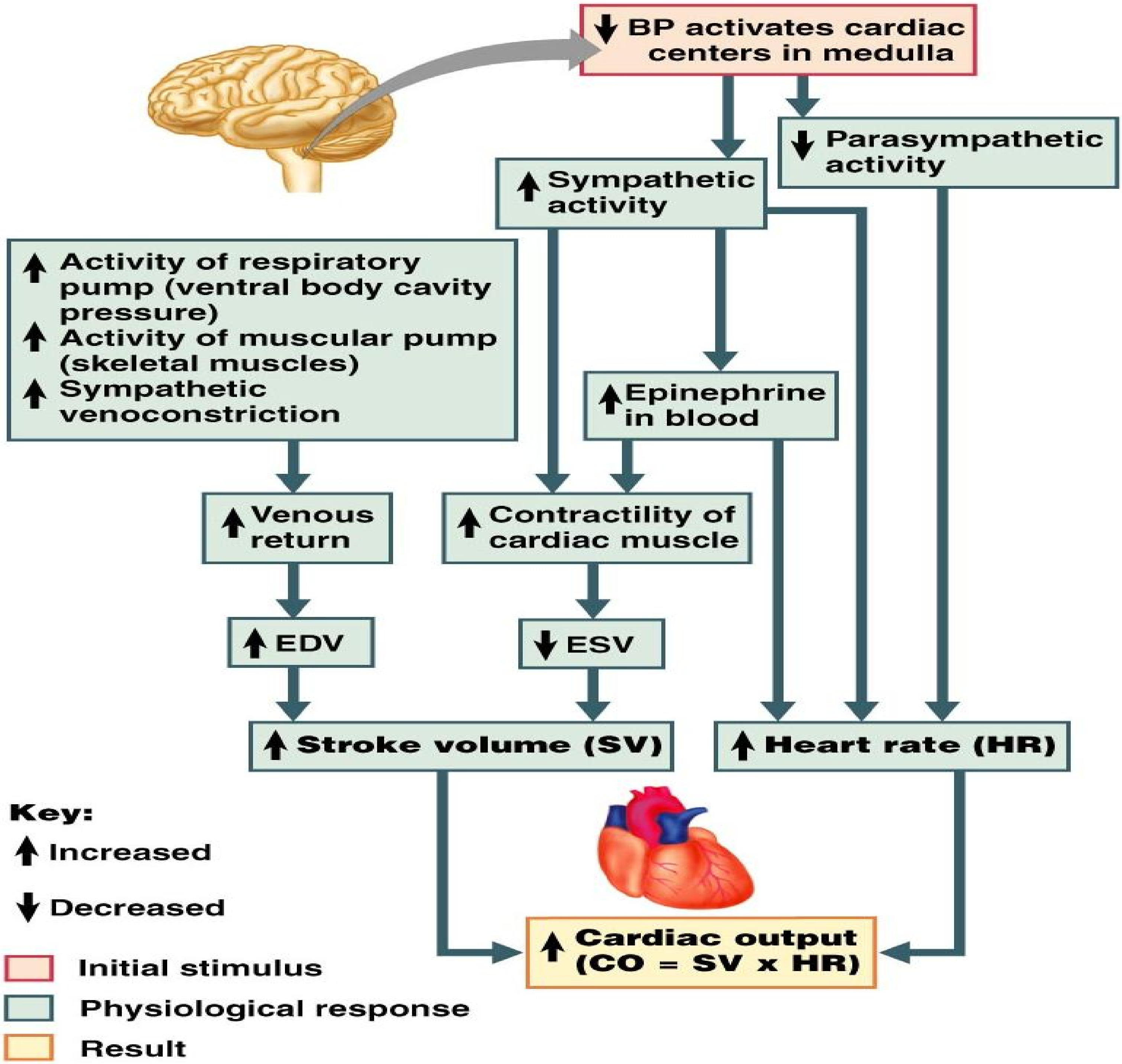
- **Stroke Volume (SV)** = EDV – ESV
- **Ejection Fraction (EF)** = (SV / EDV) × 100%
- **Cardiac Output (Q)** = SV × HR
- **Cardiac Index (CI)** = Q / Body Surface Area (BSA) = SV × HR/BSA
- HR is Heart Rate, expressed as BPM (Beats Per Minute) BSA is Body Surface Area in square metres.

- Flow = Pressure/Resistance
- **Q [co]= (MAP – RAP) / TPR**

Where MAP = Mean Aortic (or Arterial) Blood Pressure in mmHg,

- RAP = Mean Right Atrial Pressure in mmHg and TPR = Total Peripheral Resistance in dynes-sec-cm-5.
- **Q ≈ (HR x SV) ≈ MAP / TPR**

Cardiac Output (CO)



Blood Flow

- Actual volume of blood flowing through a vessel, an organ, or the entire circulation in a given period:
  - Is measured in ml per min.
  - Is **equivalent to cardiac output (CO), considering the entire vascular system**
  - Is relatively constant when at rest
  - Varies widely through individual organs

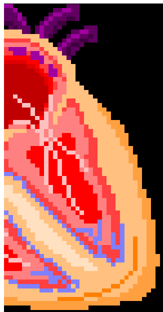
- **Blood flow (F) is directly proportional to the difference in blood pressure (ΔP) between two points in the circulation**
  - If ΔP increases, blood flow speeds up; if ΔP decreases, blood flow declines
- **Blood flow is inversely proportional to resistance (R)**
  - If R increases, blood flow decreases
- **R is more important than ΔP in influencing local blood pressure**

Blood Flow, Blood Pressure, and Resistance

Flow = Difference in pressure / resistance

Flow rate through blood vessels

- directly proportional to the pressure gradient
- inversely proportional to vascular resistance



Blood Flow

$$F = \frac{\Delta P}{R}$$

Blood flow (F) depends on:

① Pressure Gradient (ΔP) - *heart*

② Resistance (R) - *blood vessels*

- viscosity
- vessel length
- vessel diameter

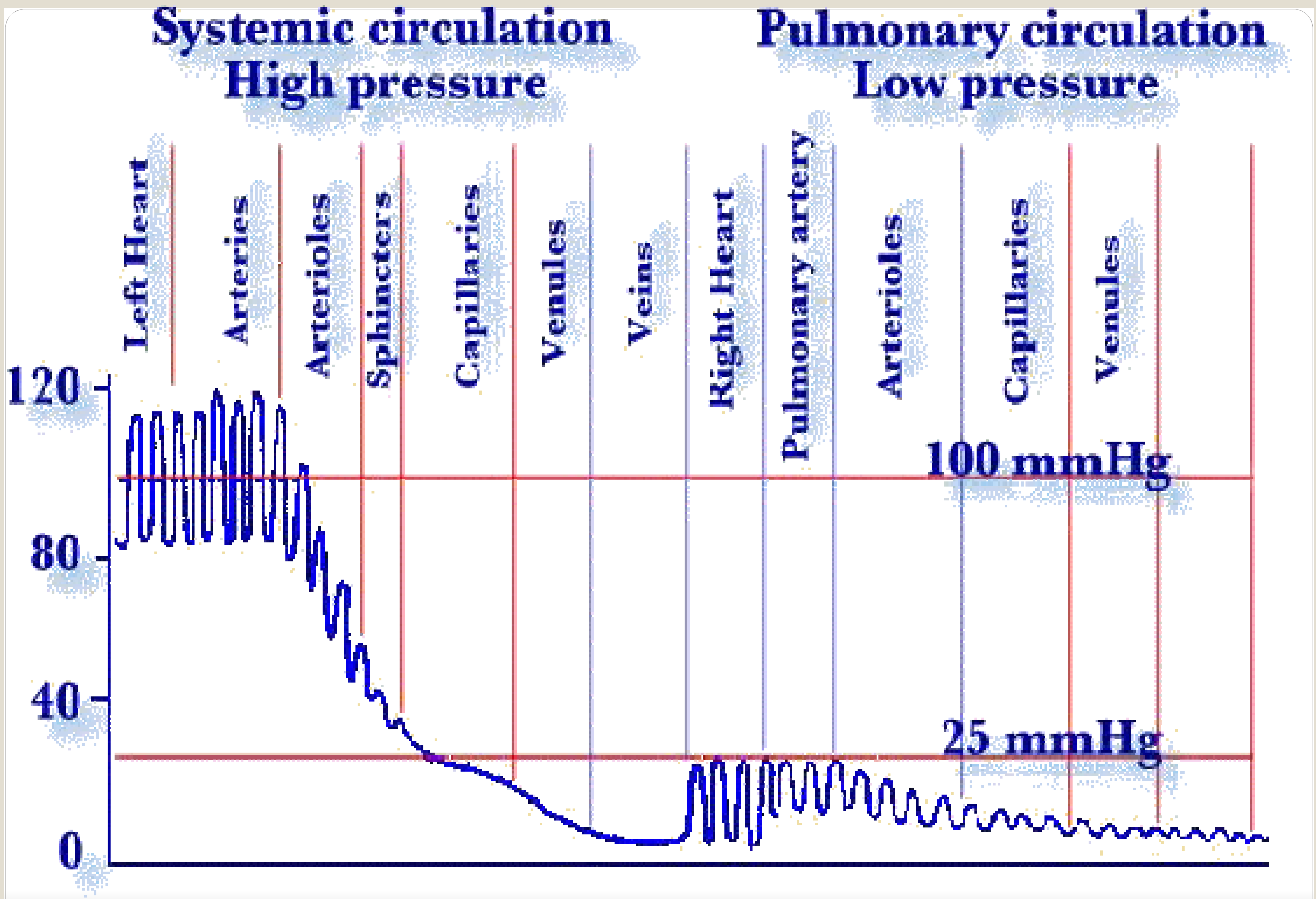
Vessel diameter is the main determinant of vascular resistance.

- Resistance factors that remain relatively constant are:
  - **Blood viscosity** – "stickiness" of the blood
  - **Blood vessel length** – the longer the vessel, the greater the resistance encountered

Resistance Factors: Viscosity and Vessel Length

- Force per unit area exerted on the wall of a blood vessel by its contained blood
  - Expressed in millimeters of mercury (mm Hg)
  - **Measured in reference to systemic arterial BP in large arteries near the heart**
- **The differences in BP within the vascular system provide the driving force that keeps blood moving from higher to lower pressure areas**

Blood Pressure (BP)



Systemic circulation
High pressure
Pulmonary circulation
Low pressure
Left Heart
Arteries
Arterioles
Sphincters
Capillaries
Venules
Veins
Right Heart
Pulmonary artery
Arterioles
Capillaries
Venules
120
80
40
0
100 mmHg
25 mmHg

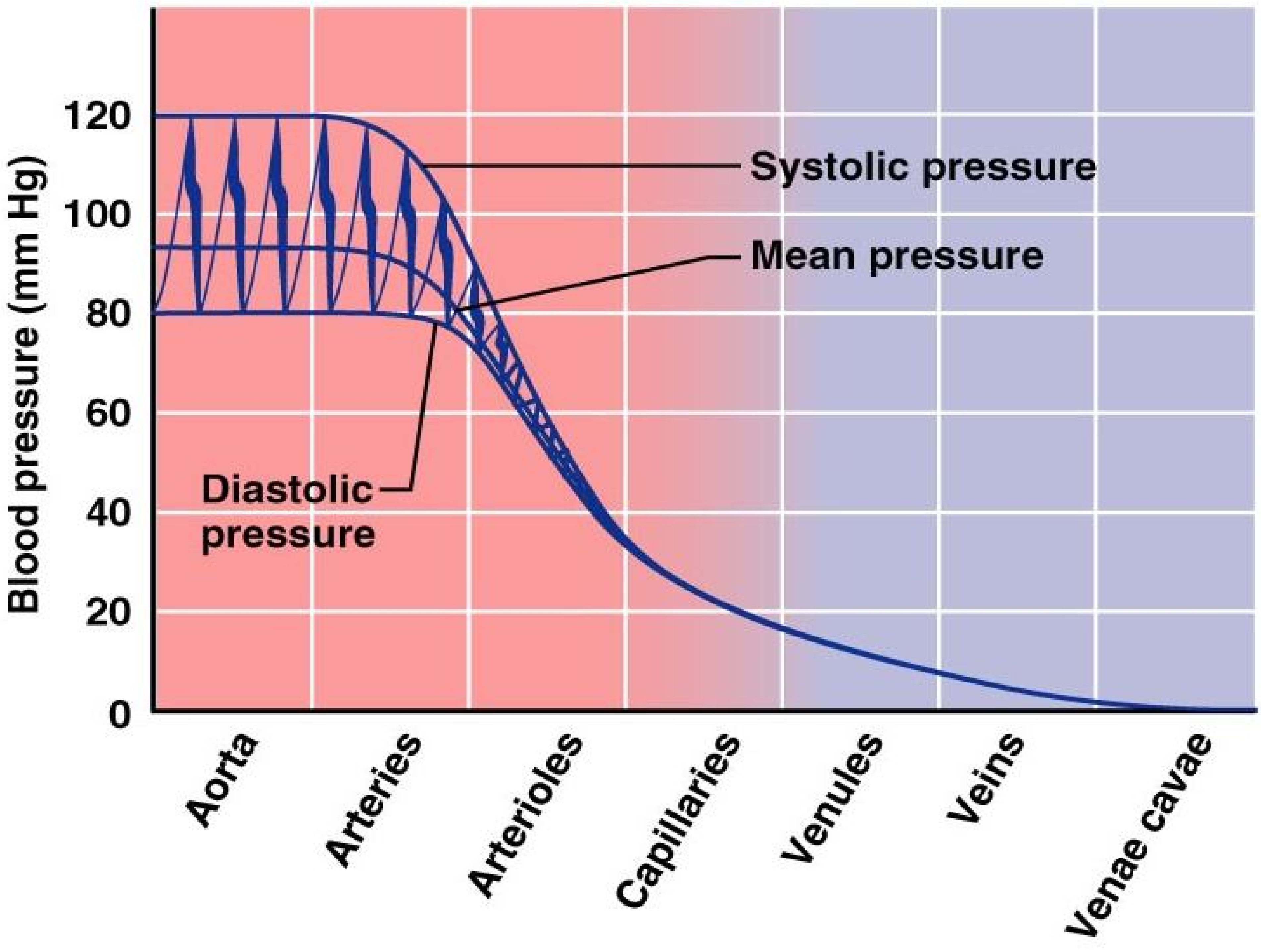
Normal blood pressures are said to range from 100/60 mmHg to 150/90 mmHg.

Table 1. Some 'average' blood pressures relating to age

Age (years)	Systolic pressure (mmHg)	Diastolic pressure (mmHg)
New-born	80	46
10	103	70
20	120	80
40	126	84
60	135	89

- **Systemic pressure:**
  - Is highest in the aorta
  - Declines throughout the length of the pathway
  - **Is 0 mm Hg in the right atrium**
- **The steepest change in blood pressure occurs in the arterioles**

Systemic Blood Pressure


Blood pressure (mm Hg)
120
100
80
60
40
20
0
Systolic pressure
Mean pressure
Diastolic pressure
Aorta
Arteries
Arterioles
Capillaries
Venules
Veins
Venae cavae

Arterial Blood Pressure

- Systolic pressure – pressure exerted on arterial walls during ventricular contraction
-
- Diastolic pressure – lowest level of arterial pressure during a ventricular cycle
- Pulse pressure – the difference between systolic and diastolic pressure
- Mean arterial pressure (MAP) – pressure that propels the blood to the tissues
- MAP = diastolic pressure + 1/3 pulse pressure[systolic]

- **Capillary BP ranges from 20 to 40 mm Hg**
- Low capillary pressure is desirable because high BP would rupture fragile, thin-walled capillaries
- Low BP is sufficient to force filtrate out into interstitial space and distribute nutrients, gases, and hormones between blood and tissues

Capillary Blood Pressure

Venous Blood Pressure

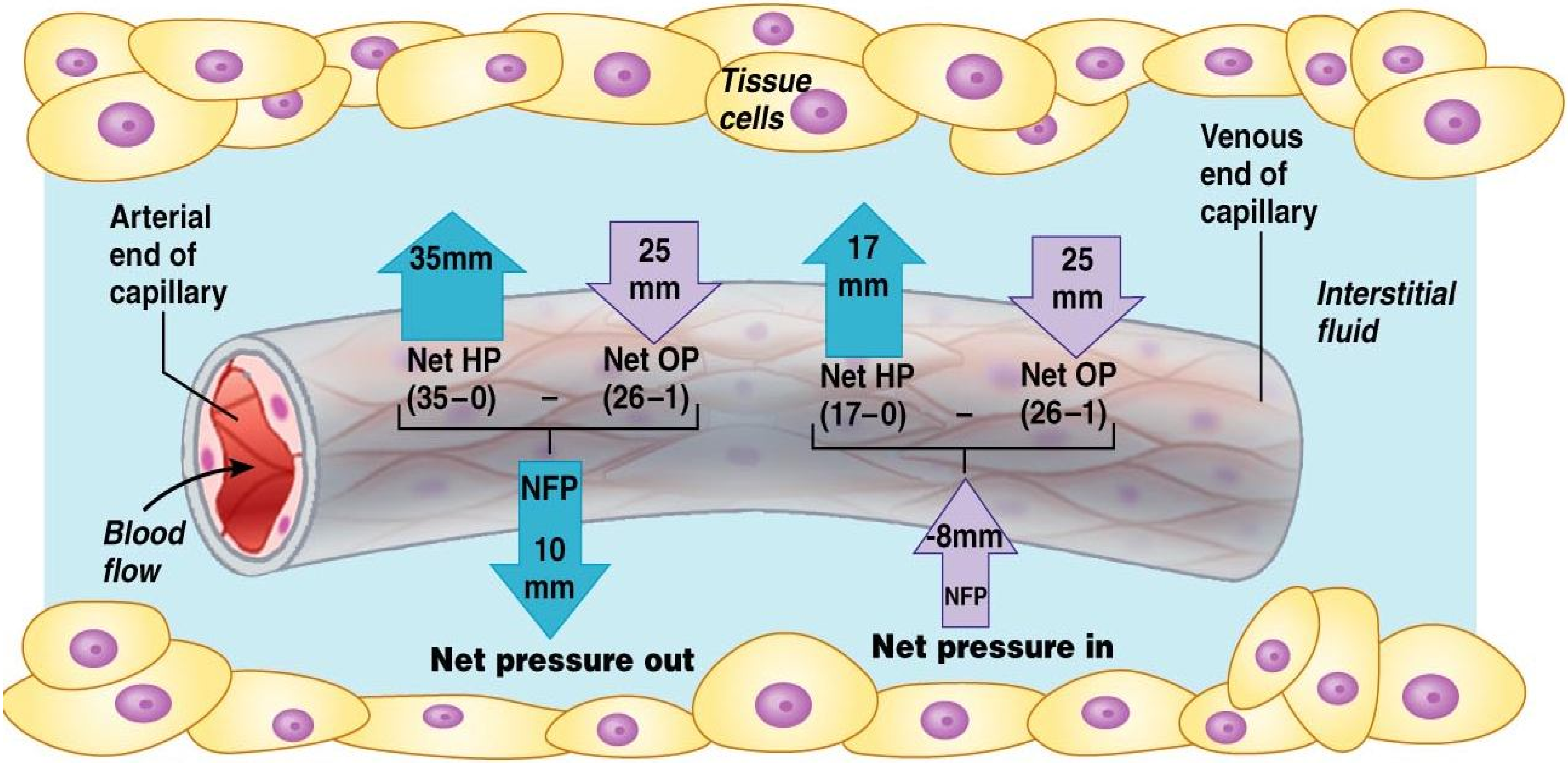
- Venous BP is steady and changes little during the cardiac cycle
- **The pressure gradient in the venous system is only about 20 mm Hg**
- A cut vein has even blood flow; a lacerated artery flows in spurts

- Direction and amount of fluid flow depends upon the difference between:
  - Capillary hydrostatic pressure (HP_c)
  - Capillary colloid osmotic pressure (OP_c)
- HP_c – pressure of blood against the capillary walls:
  - Tends to force fluids through the capillary walls
  - Is greater at the arterial end of a bed than at the venule end
- OP_c– created by nondiffusible plasma proteins, which draw water toward themselves

Capillary Exchange: Fluid Movements

- NFP – all the forces acting on a capillary bed
- NFP = $(HP_c - HP_{if}) - (OP_c - OP_{if})$
- At the arterial end of a bed, hydrostatic forces dominate (fluids flow out)

Net Filtration Pressure (NFP)



Key to pressure values:

HP_c at arterial end = 35 mm Hg HP_{if} = 0 mm Hg OP_{if} = 1 mm Hg

HP_c at venous end = 17 mm Hg OP_c = 26 mm Hg

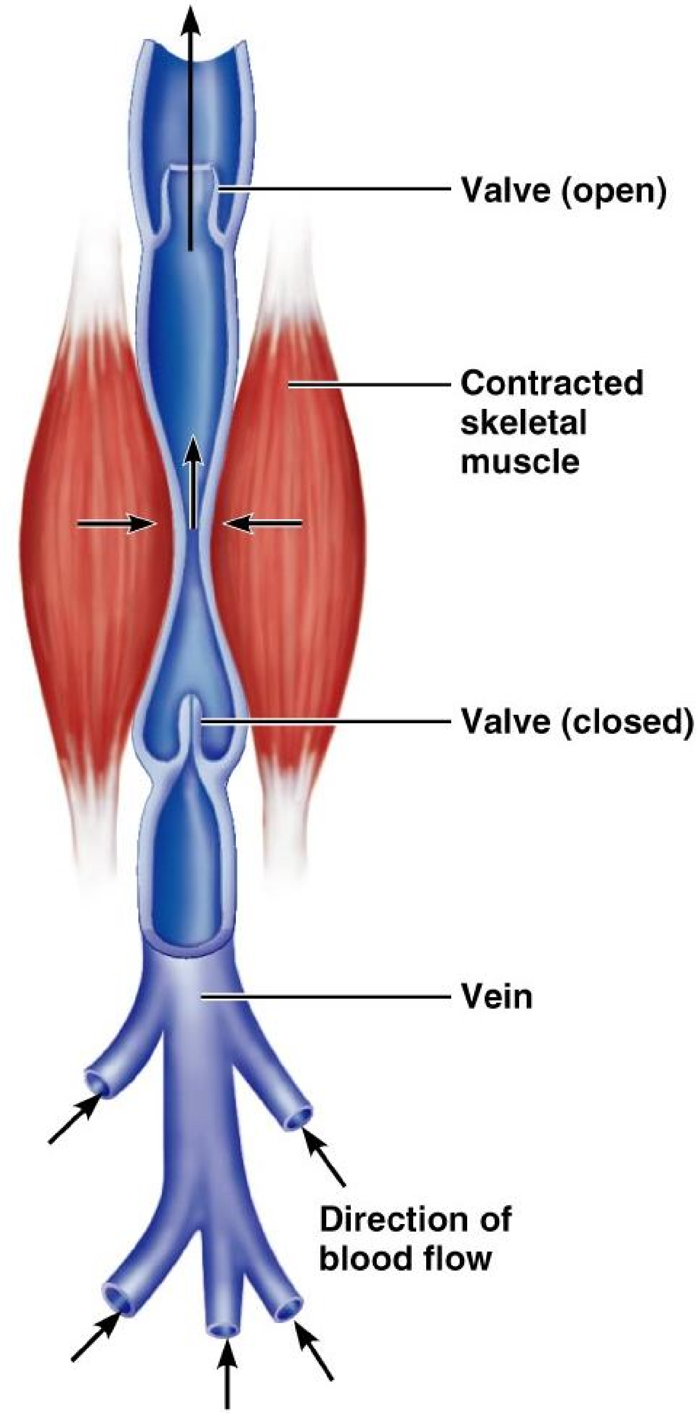
Net Filtration Pressure (NFP)

Figure 19.16

- Small vessel coronary circulation is influenced by:
  - Aortic pressure
  - The pumping activity of the ventricles
- During ventricular systole:
  - Coronary vessels compress
  - Myocardial blood flow ceases
  - Stored myoglobin supplies sufficient oxygen
- During ventricular diastole, oxygen and nutrients are carried to the heart

Blood Flow: Heart

Factors Aiding Venous Return



Myogenic Controls

- Inadequate blood perfusion or excessively high arterial pressure:
  - Are autoregulatory
  - Provoke myogenic responses – stimulation of vascular smooth muscle
- Vascular muscle responds directly to:
  - Increased vascular pressure with increased tone, which causes vasoconstriction
  - Reduced stretch with vasodilation, which promotes increased blood flow to the tissue

- **Sympathetic activity causes:**
  - Vasoconstriction and a rise in BP if increased
  - BP to decline to basal levels if decreased
- **Vasomotor activity is modified by:**
  - Baroreceptors (pressure-sensitive), chemoreceptors (O_2, CO_2, and H^+ sensitive), higher brain centers, bloodborne chemicals, and hormones

Short-Term Mechanisms: Vasomotor Activity

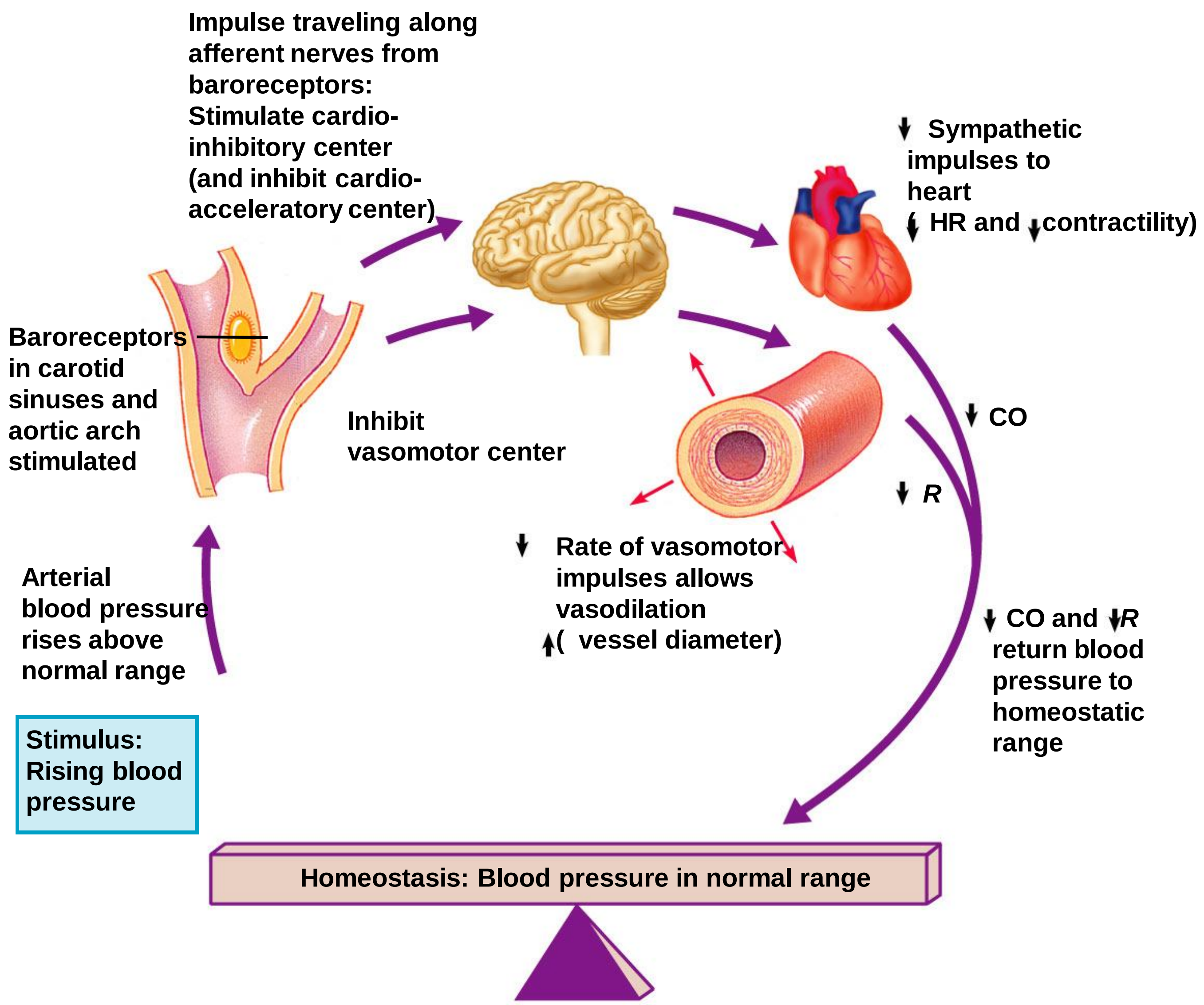

Impulse traveling along afferent nerves from baroreceptors: Stimulate cardio-inhibitory center (and inhibit cardio-acceleratory center)
Sympathetic impulses to heart
(HR and contractility)
Baroreceptors in carotid sinuses and aortic arch stimulated
Inhibit vasomotor center
CO
R
Rate of vasomotor impulses allows vasodilation
(vessel diameter)
Arterial blood pressure rises above normal range
CO and R return blood pressure to homeostatic range
Stimulus: Rising blood pressure
Homeostasis: Blood pressure in normal range

Figure 19.8

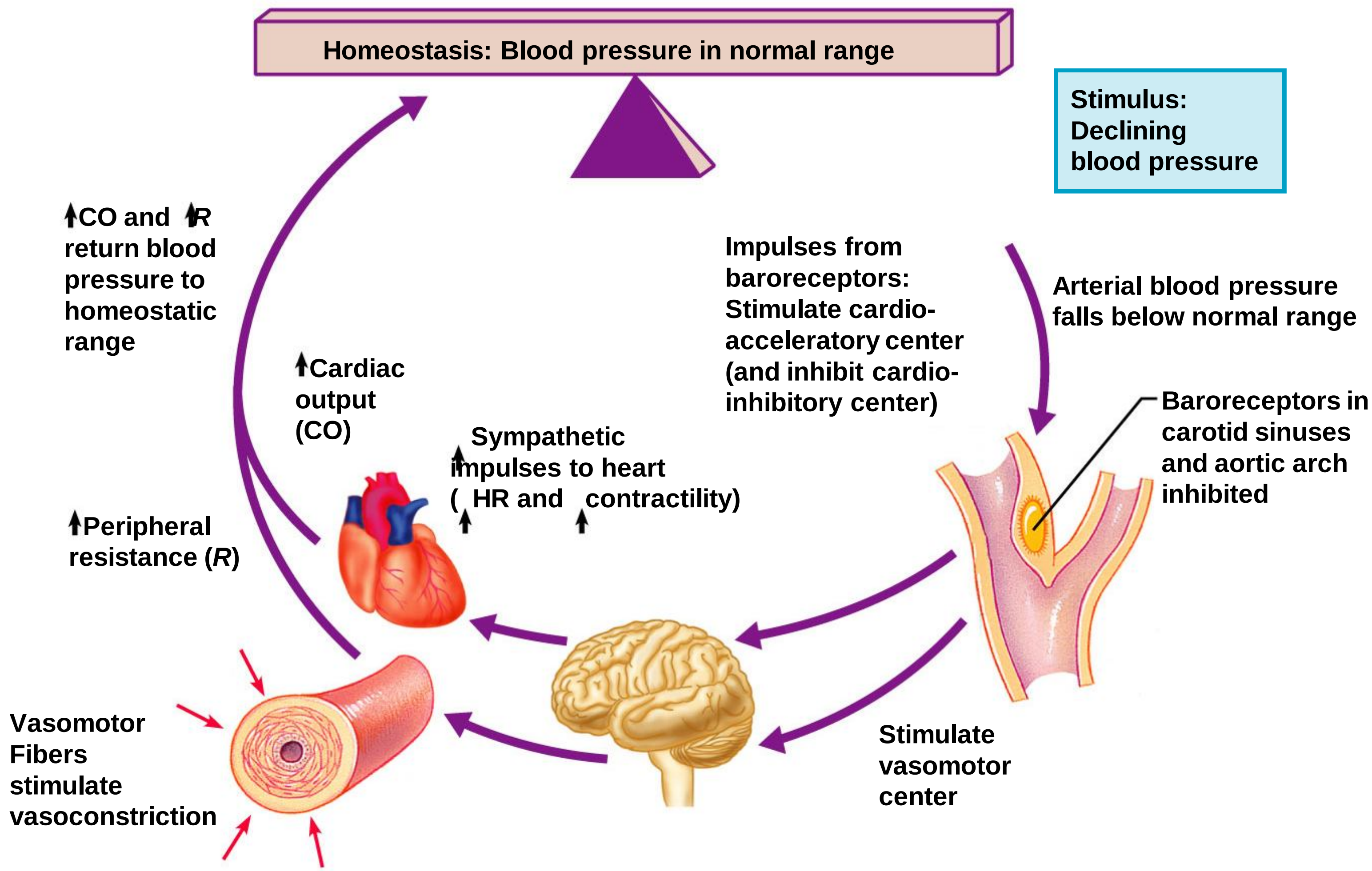

Homeostasis: Blood pressure in normal range
Stimulus: Declining blood pressure
Arterial blood pressure falls below normal range
Baroreceptors in carotid sinuses and aortic arch inhibited
Impulses from baroreceptors: Stimulate cardio-acceleratory center (and inhibit cardio-inhibitory center)
Stimulate vasomotor center
Sympathetic impulses to heart (↑HR and ↑contractility)
↑Cardiac output (CO)
Vasomotor Fibers stimulate vasoconstriction
↑Peripheral resistance (R)
↑CO and ↑R return blood pressure to homeostatic range

Figure 19.8

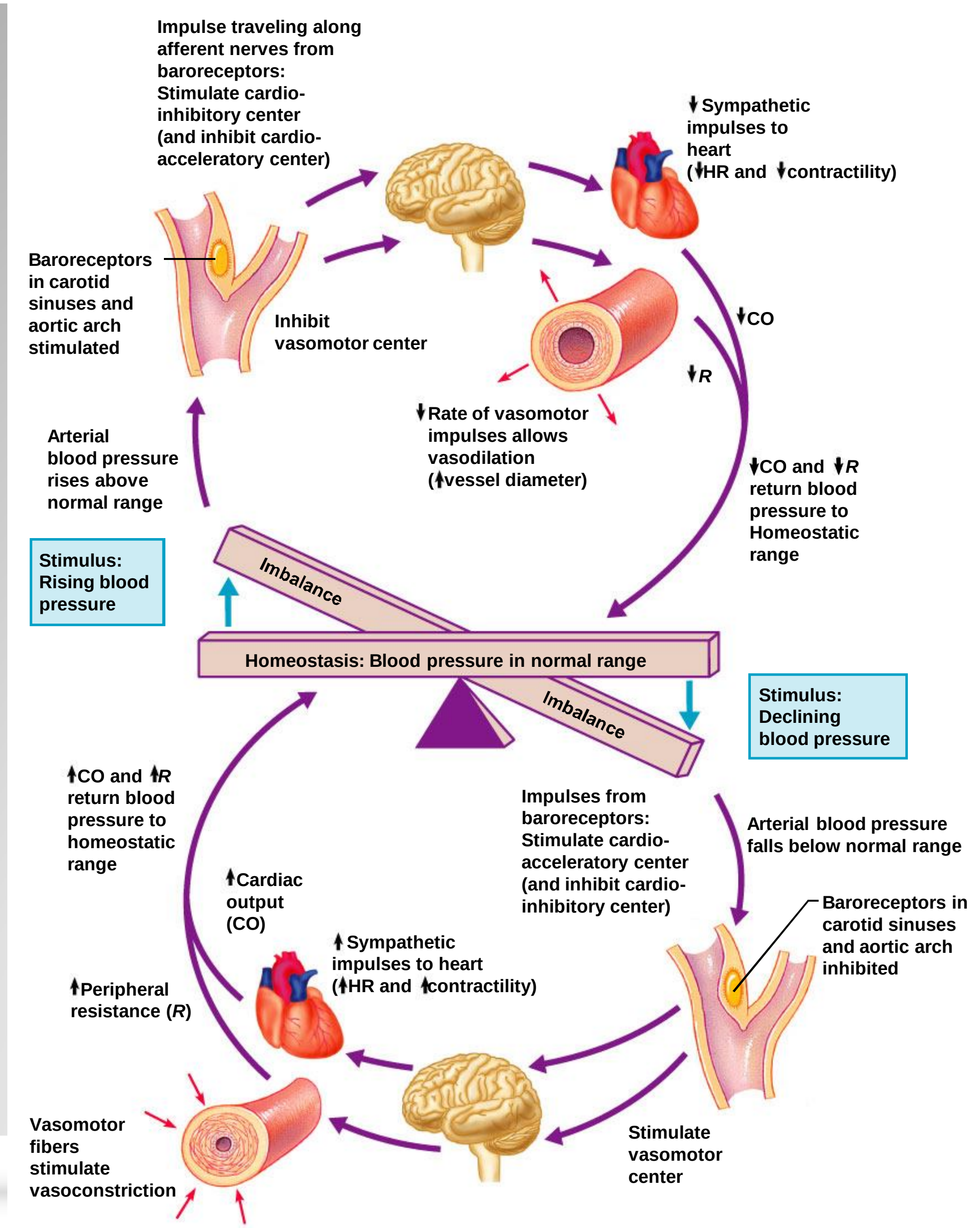

Impulse traveling along afferent nerves from baroreceptors: Stimulate cardio-inhibitory center (and inhibit cardio-acceleratory center)
↓Sympathetic impulses to heart (↓HR and ↓contractility)
Baroreceptors in carotid sinuses and aortic arch stimulated
Inhibit vasomotor center
↓CO
↓R
↓Rate of vasomotor impulses allows vasodilation (↑vessel diameter)
Arterial blood pressure rises above normal range
↓CO and ↓R return blood pressure to Homeostatic range
Stimulus: Rising blood pressure
Imbalance
Homeostasis: Blood pressure in normal range
Imbalance
Stimulus: Declining blood pressure
↑CO and ↑R return blood pressure to homeostatic range
Impulses from baroreceptors: Stimulate cardio-acceleratory center (and inhibit cardio-inhibitory center)
Arterial blood pressure falls below normal range
↑Cardiac output (CO)
↑Sympathetic impulses to heart (↑HR and ↑contractility)
Baroreceptors in carotid sinuses and aortic arch inhibited
↑Peripheral resistance (R)
Vasomotor fibers stimulate vasoconstriction
Stimulate vasomotor center

Figure 19.8

- **Adrenal medulla hormones** – norepinephrine and epinephrine increase blood pressure
- **Antidiuretic hormone (ADH)** – causes intense vasoconstriction in cases of extremely low BP
- **Angiotensin II** – kidney release of renin generates angiotensin II, which causes vasoconstriction
- **Endothelium-derived factors** – endothelin and prostaglandin-derived growth factor (PDGF) are both vasoconstrictors

Chemicals that Increase Blood Pressure

- **Atrial natriuretic peptide (ANP)** – causes blood volume and pressure to decline
- **Nitric oxide (NO)** – is a brief but potent vasodilator
- **Inflammatory chemicals** – histamine, prostacyclin, and kinins are potent vasodilators
- **Alcohol** – causes BP to drop by inhibiting ADH

Chemicals that Decrease Blood Pressure

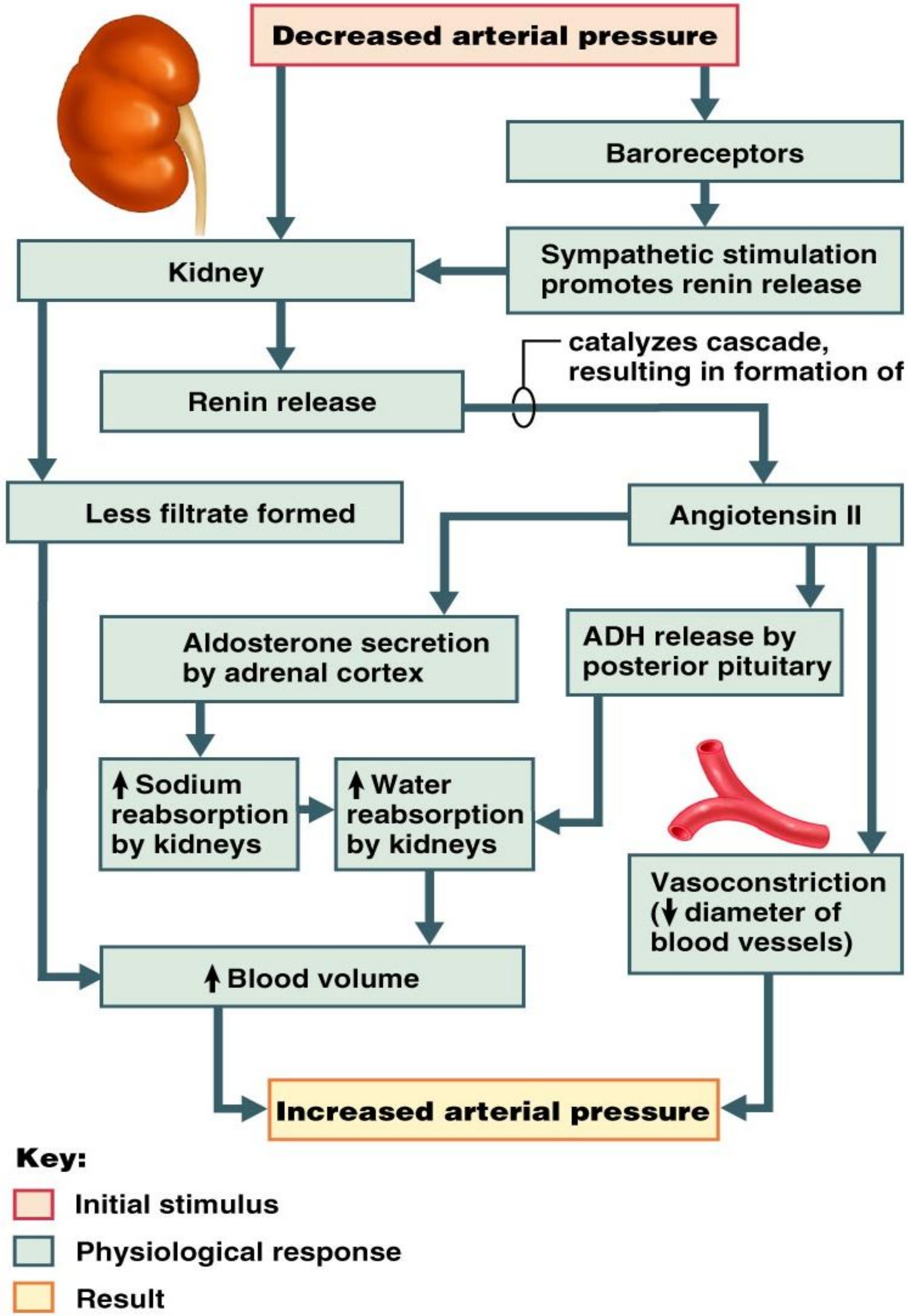
- Long-term mechanisms control BP by altering blood volume
- **Baroreceptors adapt to chronic high or low BP**
  - Increased BP stimulates the kidneys to eliminate water, thus reducing BP
  - Decreased BP stimulates the kidneys to increase blood volume and BP

Long-Term Mechanisms: Renal Regulation

Kidney Action and Blood Pressure

Table 138. Clinical Evaluation of Patients at Increased Risk of Chronic Kidney Disease

All Patients
Measurement of blood pressure
Serum creatinine to estimate GFR
Protein-to-creatinine ratio or albumin-to-creatinine ratio in a first-morning or random untimed "spot" urine specimen
Examination of the urine sediment or dipstick for red blood cells and white blood cells
Selected Patients, Depending on Risk Factors
Ultrasound imaging (for example, in patients with symptoms of urinary tract obstruction, infection or stone, or family history of polycystic kidney disease)
Serum electrolytes (sodium, potassium, chloride and bicarbonate)
Urinary concentration or dilution (specific gravity or osmolality)
Urinary acidification (pH)



- Blood flow to venous plexuses below the skin surface:
  - Varies from 50 ml/min to 2500 ml/min, depending on body temperature
  - Is controlled by sympathetic nervous system reflexes initiated by temperature receptors and the central nervous system

Blood Flow: Skin

- As temperature rises (e.g., heat exposure, fever, vigorous exercise):
  - Hypothalamic signals reduce vasomotor stimulation of the skin vessels
  - Heat radiates from the skin
- Sweat also causes vasodilation via bradykinin in perspiration
  - Bradykinin stimulates the release of NO
- As temperature decreases, blood is shunted to deeper, more vital organs

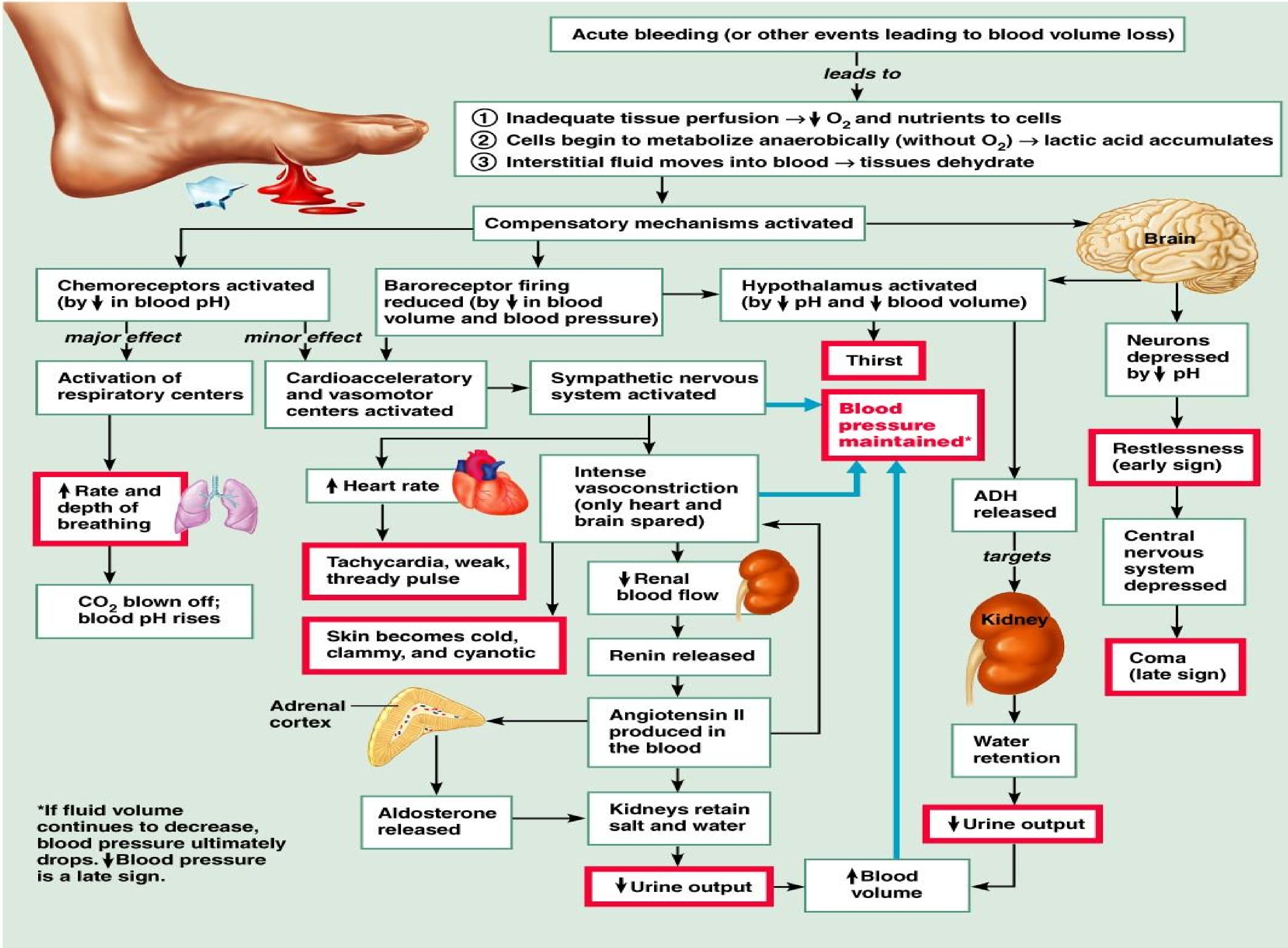
Temperature Regulation

- Blood flow in the pulmonary circulation is unusual in that:
  - The pathway is short
  - Arteries/arterioles are more like veins/venules (thin-walled, with large lumens)
  - They have a much lower arterial pressure (24/8 mm Hg versus 120/80 mm Hg)

Blood Flow: Lungs

- **Three types include:**
  - Hypovolemic shock – results from large-scale blood loss
  - Vascular shock – poor circulation resulting from extreme vasodilation
  - Cardiogenic shock – the heart cannot sustain adequate circulation

Circulatory Shock


Acute bleeding (or other events leading to blood volume loss)
leads to
① Inadequate tissue perfusion → ↓ O_2 and nutrients to cells
② Cells begin to metabolize anaerobically (without O_2) → lactic acid accumulates
③ Interstitial fluid moves into blood → tissues dehydrate
Compensatory mechanisms activated
Brain
Chemoreceptors activated (by ↓ in blood pH)
major effect
minor effect
Baroreceptor firing reduced (by ↓ in blood volume and blood pressure)
Hypothalamus activated (by ↓ pH and ↓ blood volume)
Neurons depressed by ↓ pH
Thirst
Activation of respiratory centers
Cardioacceleratory and vasomotor centers activated
Sympathetic nervous system activated
Blood pressure maintained*
Restlessness (early sign)
↑ Rate and depth of breathing
↑ Heart rate
Intense vasoconstriction (only heart and brain spared)
ADH released
targets
Kidney
Central nervous system depressed
Tachycardia, weak, thready pulse
↓ Renal blood flow
CO₂ blown off; blood pH rises
Skin becomes cold, clammy, and cyanotic
Renin released
Coma (late sign)
Adrenal cortex
Angiotensin II produced in the blood
Water retention
Aldosterone released
Kidneys retain salt and water
↓ Urine output
↓ Urine output
↑ Blood volume
*If fluid volume continues to decrease, blood pressure ultimately drops. ↓ Blood pressure is a late sign.