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

or

- **BP = CO x TPR**
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

Blood flow (F) depends on:

1. Pressure Gradient ($\Delta P$) - heart
2. Resistance (R) - blood vessels
   - viscosity
   - vessel length
   - vessel diameter

Vessel diameter is the main determinant of vascular resistance.
Arteries:

- Low resistance, rapid transit passageways
- Muscle & elastic connective tissue in walls
  - elastic recoil

Arteries act as pressure reservoirs because the elastic walls collapse inward during ventricular diastole (when there is less blood in the arteries):
• Resistance – opposition to flow
  ◦ Measure of the amount of friction blood encounters
  ◦ Generally encountered in the systemic circulation
  ◦ Referred to as peripheral resistance (PR)
• The three important sources of resistance are blood viscosity, total blood vessel length, and blood vessel diameter
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
• Changes in vessel diameter are frequent and significantly alter peripheral resistance
• Resistance varies inversely with the fourth power of vessel radius
  ◦ For example, if the radius is doubled, the resistance is 1/16 as much

Resistance Factors: Blood Vessel Diameter
• Small-diameter arterioles are the major determinants of peripheral resistance
• Fatty plaques from atherosclerosis:
  ◦ Cause turbulent blood flow
  ◦ Dramatically increase resistance due to turbulence
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)**
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
Normal blood pressures are said to range from 100/60 mmHg to 150/90 mmHg.

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

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Systolic pressure (mmHg)</th>
<th>Diastolic pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>New-born</td>
<td>80</td>
<td>46</td>
</tr>
<tr>
<td>10</td>
<td>103</td>
<td>70</td>
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<td>20</td>
<td>120</td>
<td>80</td>
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<tr>
<td>40</td>
<td>126</td>
<td>84</td>
</tr>
<tr>
<td>60</td>
<td>135</td>
<td>89</td>
</tr>
</tbody>
</table>
The pumping action of the heart generates blood flow through the vessels along a pressure gradient, always moving from higher- to lower-pressure areas.

Pressure results when flow is opposed by resistance.

**Systemic Blood Pressure**
• **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**
• Arterial BP reflects two factors of the arteries close to the heart
  ◦ Their elasticity (compliance or distensibility)
  ◦ The amount of blood forced into them at any given time

• Blood pressure in elastic arteries near the heart is pulsatile (BP rises and falls)
• **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**

---

**Arterial Blood Pressure**
• 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
• 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
Venous BP alone is too low to promote adequate blood return and is aided by the:
- Respiratory “pump” – pressure changes created during breathing suck blood toward the heart by squeezing local veins
- Muscular “pump” – contraction of skeletal muscles “milk” blood toward the heart
- Valves prevent backflow during venous return

Factors Aiding Venous Return
Factors Aiding Venous Return

![Diagram showing factors aiding venous return.
- Valve (open)
- Contracted skeletal muscle
- Valve (closed)
- Vein
- Direction of blood flow]
Maintaining blood pressure requires:

- Cooperation of the heart, blood vessels, and kidneys
- Supervision of the brain
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
- Cardiac output is determined by venous return and neural and hormonal controls.
- Resting heart rate is controlled by the cardioinhibitory center via the vagus nerves.
  - Stroke volume is controlled by venous return (end diastolic volume, or EDV).
- Under stress, the cardioacceleratory center increases heart rate and stroke volume.
  - The end systolic volume (ESV) decreases and MAP increases.
Cardiac Output (CO)

Figure 19.7

- BP activates cardiac centers in medulla
- Parasympathetic activity
- Sympathetic activity
  - Activity of respiratory pump (ventral body cavity pressure)
  - Activity of muscular pump (skeletal muscles)
  - Sympathetic vеноconstriction
  - Epinephrine in blood

- Venous return
  - ↑ EDV
- Contractility of cardiac muscle
  - ↓ ESV

- Stroke volume (SV)
- Heart rate (HR)

Key:
- ↑ Increased
- ↓ Decreased

- Initial stimulus
- Physiological response
- Result

Cardiac output (CO = SV x HR)
• Short-term controls:
  ◦ Are mediated by the nervous system and bloodborne chemicals
  ◦ Counteract moment-to-moment fluctuations in blood pressure by altering peripheral resistance

• Long-term controls regulate blood volume

Controls of Blood Pressure
Neural controls of peripheral resistance:
- Alter blood distribution in response to demands
- Maintain MAP by altering blood vessel diameter

Neural controls operate via reflex arcs involving:
- Baroreceptors
- Vasomotor centers and vasomotor fibers
- Vascular smooth muscle

Short-Term Mechanisms: Neural Controls
**Vasomotor center** – a cluster of sympathetic neurons in the medulla that oversees changes in blood vessel diameter

- Maintains blood vessel tone by innervating smooth muscles of blood vessels, especially arterioles

**Cardiovascular center** – vasomotor center plus the cardiac centers that integrate blood pressure control by altering cardiac output and blood vessel diameter

**Short-Term Mechanisms:**
**Vasomotor Center**
**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**
Increased blood pressure stimulates the **cardioinhibitory center to:**
- Increase vessel diameter
- Decrease heart rate, cardiac output, peripheral resistance, and blood pressure

**Short-Term Mechanisms:**
**Baroreceptor-Initiated Reflexes**
• Declining blood pressure stimulates the cardioacceleratory center to:
  ◦ Increase cardiac output and peripheral resistance
• Low blood pressure also stimulates the vasomotor center to constrict blood vessels

Short-Term Mechanisms: Baroreceptor-Initiated Reflexes
Impulse traveling along afferent nerves from baroreceptors:
- Stimulate cardio-inhibitory center (and inhibit cardio-acceleratory center)

Baroreceptors in carotid sinuses and aortic arch stimulated

Arterial blood pressure rises above normal range

Stimulus: Rising blood pressure

Sympathetic impulses to heart (HR and contractility)

Rate of vasomotor impulses allows vasodilation (vessel diameter)

Impulse from baroreceptors: Stimulate cardio-acceleratory center (and inhibit cardio-inhibitory center)

Homeostasis: Blood pressure in normal range

Arterial blood pressure falls below normal range

Stimulus: Declining blood pressure

CO and R return blood pressure to homeostatic range
Impulse traveling along afferent nerves from baroreceptors: Stimulate cardio-inhibitory center (and inhibit cardio-acceleratory center).

Baroreceptors in carotid sinuses and aortic arch stimulated.

Arterial blood pressure rises above normal range.

Stimulus: Rising blood pressure.

Inhibit vasomotor center.

Rate of vasomotor impulses allows vasodilation (vessel diameter).

↓CO and ↓R return blood pressure to homeostatic range.

↓Sympathetic impulses to heart (↓HR and ↓contractility).

Homeostasis: Blood pressure in normal range.
Homeostasis: Blood pressure in normal range

Stimulus: Declining blood pressure

Impulses from baroreceptors:
- Stimulate cardio-acceleratory center
- (and inhibit cardio-inhibitory center)

Arterial blood pressure falls below normal range
- Baroreceptors in carotid sinuses and aortic arch inhibited

Vasomotor fibers stimulate vasoconstriction

↑CO and ↑R return blood pressure to homeostatic range

↑Peripheral resistance (R)

↑Cardiac output (CO)

Sympathetic impulses to heart
- (HR and contractility)

Stimulate vasomotor center
Impulse traveling along afferent nerves from baroreceptors:
Stimulate cardio-inhibitory center (and inhibit cardio-acceleratory center)

Baroreceptors in carotid sinuses and aortic arch stimulated
Arterial blood pressure rises above normal range

Stimulus: Rising blood pressure

Homeostasis: Blood pressure in normal range

Impulses from baroreceptors: Stimulate cardio-acceleratory center (and inhibit cardio-inhibitory center)

Sympathetic impulses to heart (HR and contractility)

Rate of vasomotor impulses allows vasodilation (vessel diameter)

CO and R return blood pressure to homeostatic range

Cardiac output (CO)
Peripheral resistance (R)
Vasomotor fibers stimulate vasoconstriction

Figure 19.8
Blood pressure is regulated by chemoreceptor reflexes sensitive to oxygen and carbon dioxide
- Prominent chemoreceptors are the carotid and aortic bodies
- Reflexes that regulate BP are integrated in the medulla
- Higher brain centers (cortex and hypothalamus) can modify BP via relays to medullary centers

Short-Term Mechanisms: Chemical Controls
• **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**
Kidneys act directly and indirectly to maintain long-term blood pressure

- Direct renal mechanism alters blood volume
- Indirect renal mechanism involves the renin-angiotensin mechanism

Kidney Action and Blood Pressure
• Declining BP causes the release of renin, which triggers the release of angiotensin II
• Angiotensin II is a potent vasoconstrictor that stimulates aldosterone secretion
• Aldosterone enhances renal reabsorption and stimulates ADH release
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-creatine 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)
• Efficiency of the circulation can be assessed by taking pulse and blood pressure measurements
• Vital signs – pulse and blood pressure, along with respiratory rate and body temperature
• Pulse – pressure wave caused by the expansion and recoil of elastic arteries
  ◦ Radial pulse (taken on the radial artery at the wrist) is routinely used
  ◦ Varies with health, body position, and activity

Monitoring Circulatory Efficiency
Palpated Pulse

- Temporal artery
- Facial artery
- Common carotid artery
- Brachial artery
- Radial artery
- Femoral artery
- Popliteal artery
- Posterior tibial artery
- Dorsalis pedis artery
Systemic arterial BP is measured indirectly with the auscultatory method
- A sphygmomanometer is placed on the arm superior to the elbow
- Pressure is increased in the cuff until it is greater than systolic pressure in the brachial artery
- Pressure is released slowly and the examiner listens with a stethoscope

Measuring Blood Pressure
The first sound heard is recorded as the systolic pressure.
The pressure when sound disappears is recorded as the diastolic pressure.
• Blood pressure cycles over a 24-hour period
• BP peaks in the morning due to waxing and waning levels of retinoic acid
• Extrinsic factors such as age, sex, weight, race, mood, posture, socioeconomic status, and physical activity may also cause BP to vary
- Hypotension – low BP in which systolic pressure is below 100 mm Hg
- Hypertension – condition of sustained elevated arterial pressure of 140/90 or higher
  - Transient elevations are normal and can be caused by fever, physical exertion, and emotional upset
  - Chronic elevation is a major cause of heart failure, vascular disease, renal failure, and stroke
- Orthostatic hypotension – temporary low BP and dizziness when suddenly rising from a sitting or reclining position
- Chronic hypotension – hint of poor nutrition and warning sign for Addison’s disease
- Acute hypotension – important sign of circulatory shock
  - Threat to patients undergoing surgery and those in intensive care units
Hypertension may be transient or persistent.

Primary or essential hypertension – risk factors in primary hypertension include diet, obesity, age, race, heredity, stress, and smoking.

Secondary hypertension – due to identifiable disorders, including excessive renin secretion, arteriosclerosis, and endocrine disorders.
Blood flow, or tissue perfusion, is involved in:

- Delivery of oxygen and nutrients to, and removal of wastes from, tissue cells
- Gas exchange in the lungs
- Absorption of nutrients from the digestive tract
- Urine formation by the kidneys

Blood flow is precisely the right amount to provide proper tissue function.
• Blood velocity:
  ◦ Changes as it travels through the systemic circulation
  ◦ Is inversely proportional to the cross-sectional area
• Slow capillary flow allows adequate time for exchange between blood and tissues
Relative cross-sectional area of different vessels of the vascular bed

<table>
<thead>
<tr>
<th>Total area (cm²) of the vascular bed</th>
<th>5000</th>
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<th>3000</th>
<th>2000</th>
<th>1000</th>
<th>0</th>
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<td>0</td>
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<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Velocity of blood flow (cm/s)

Figure 19.13
- Autoregulation – automatic adjustment of blood flow to each tissue in proportion to its requirements at any given point in time.
- Blood flow through an individual organ is intrinsically controlled by modifying the diameter of local arterioles feeding its capillaries.
- MAP remains constant, while local demands regulate the amount of blood delivered to various areas according to need.
• Declining tissue nutrient and oxygen levels are stimuli for autoregulation
• Hemoglobin delivers nitric oxide (NO) as well as oxygen to tissues
• Nitric oxide induces vasodilation at the capillaries to help get oxygen to tissue cells
• Other autoregulatory substances include: potassium and hydrogen ions, adenosine, lactic acid, histamines, kinins, and prostaglandins

Metabolic 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
• Is evoked when short-term autoregulation cannot meet tissue nutrient requirements
• May evolve over weeks or months to enrich local blood flow
Angiogenesis takes place:
  ◦ As the number of vessels to a region increases
  ◦ When existing vessels enlarge
  ◦ When a heart vessel becomes partly occluded
  ◦ Routinely in people in high altitudes, where oxygen content of the air is low

Long-Term Autoregulation
- Resting muscle blood flow is regulated by myogenic and general neural mechanisms in response to oxygen and carbon dioxide levels.
- When muscles become active, hyperemia is directly proportional to greater metabolic activity of the muscle (active or exercise hyperemia).
- Arterioles in muscles have cholinergic, and alpha (α) and beta (β) adrenergic receptors.
- α and β adrenergic receptors bind to epinephrine.

Blood Flow: Skeletal Muscles
• Muscle blood flow can increase tenfold or more during physical activity as vasodilation occurs
  ◦ Low levels of epinephrine bind to $\beta$ receptors
  ◦ Cholinergic receptors are occupied

Blood Flow: Skeletal Muscle Regulation
• Intense exercise or sympathetic nervous system activation results in high levels of epinephrine
  ◦ High levels of epinephrine bind to $\alpha$ receptors and cause vasoconstriction
    • This is a protective response to prevent muscle oxygen demands from exceeding cardiac pumping ability

**Blood Flow: Skeletal Muscle Regulation**
Blood flow to the brain is constant, as neurons are intolerant of ischemia.

Metabolic controls – brain tissue is extremely sensitive to declines in pH, and increased carbon dioxide causes marked vasodilation.

Myogenic controls protect the brain from damaging changes in blood pressure:
- Decreases in MAP cause cerebral vessels to dilate to ensure adequate perfusion.
- Increases in MAP cause cerebral vessels to constrict.

**Blood Flow: Brain**
The brain can regulate its own blood flow in certain circumstances, such as ischemia caused by a tumor.

The brain is vulnerable under extreme systemic pressure changes:
- MAP below 60mm Hg can cause syncope (fainting).
- MAP above 160 can result in cerebral edema.

**Blood Flow: Brain**
Blood flow through the skin:
- Supplies nutrients to cells in response to oxygen need
- Helps maintain body temperature
- Provides a blood reservoir
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
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
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
The autoregulatory mechanism is exactly opposite of that in most tissues

- Low oxygen levels cause vasoconstriction; high levels promote vasodilation
- This allows for proper oxygen loading in the lungs
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
• Under resting conditions, blood flow through the heart may be controlled by a myogenic mechanism
• During strenuous exercise:
  ◦ Coronary vessels dilate in response to local accumulation of carbon dioxide
  ◦ Blood flow may increase three to four times
  ◦ Blood flow remains constant despite wide variation in coronary perfusion pressure

Blood Flow: Heart
Oxygen, carbon dioxide, nutrients, and metabolic wastes diffuse between the blood and interstitial fluid along concentration gradients

- Oxygen and nutrients pass from the blood to tissues
- Carbon dioxide and metabolic wastes pass from tissues to the blood
- Water-soluble solutes pass through clefts and fenestrations
- Lipid-soluble molecules diffuse directly through endothelial membranes

**Capillary Exchange of Respiratory Gases and Nutrients**
Capillary Exchange of Respiratory Gases and Nutrients

Figure 19.15.1
Capillary Exchange of Respiratory Gases and Nutrients
• 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)
- At the venous end of a bed, osmotic forces dominate (fluids flow in)
- More fluids enter the tissue beds than return blood, and the excess fluid is returned to the blood via the lymphatic system

**Net Filtration Pressure (NFP)**
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
Circulatory shock – any condition in which blood vessels are inadequately filled and blood cannot circulate normally
Results in inadequate blood flow to meet tissue needs
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
Figure 19.17

Acute bleeding (or other events leading to blood volume loss)

leads to

1. Inadequate tissue perfusion → ↓ O₂ and nutrients to cells
2. Cells begin to metabolize anaerobically (without O₂) → lactic acid accumulates
3. Interstitial fluid moves into blood → tissues dehydrate

Compensatory mechanisms activated

- Chemoreceptors activated (by ↓ in blood pH)
  - major effect
  - Activation of respiratory centers
    - ↓ Rate and depth of breathing
      - CO₂ blown off; blood pH rises
    - Tachycardia, weak, thready pulse
      - Skin becomes cold, clammy, and cyanotic
  - minor effect
    - Heart rate

- Baroreceptor firing reduced (by ↓ in blood volume and blood pressure)
  - Cardioacceleratory and vasomotor centers activated
  - Sympathetic nervous system activated
    - Intense vasoconstriction (only heart and brain spared)
    - ↓ Renal blood flow
      - Renin released
      - Angiotensin II produced in the blood
      - Aldosterone released
        - Kidneys retain salt and water
          - ↓ Urine output
          - ↑ Blood volume
    - Skin becomes cold, clammy, and cyanotic

- Hypothalamus activated (by ↓ pH and ↓ blood volume)
  - Blood pressure maintained
    - Thirst
    - Neurons depressed by ↓ pH
      - Restlessness (early sign)
      - Central nervous system depressed
        - Coma (late sign)

- ADH released
  - Targets
  - Kidney
    - Water retention
      - ↓ Urine output

*If fluid volume continues to decrease, blood pressure ultimately drops. ↓ Blood pressure is a late sign.*
The vascular system has two distinct circulations

- Pulmonary circulation – short loop that runs from the heart to the lungs and back to the heart
- Systemic circulation – routes blood through a long loop to all parts of the body and returns to the heart
The endothelial lining of blood vessels arises from mesodermal cells, which collect in blood islands.
- Blood islands form rudimentary vascular tubes through which the heart pumps blood by the fourth week of development.

- Fetal shunts (foramen ovale and ductus arteriosus) bypass nonfunctional lungs.
- The ductus venosus bypasses the liver.
- The umbilical vein and arteries circulate blood to and from the placenta.

**Developmental Aspects**
Blood vessels are trouble-free during youth

Vessel formation occurs:
- As needed to support body growth
- For wound healing
- To rebuild vessels lost during menstrual cycles

With aging, varicose veins, atherosclerosis, and increased blood pressure may arise