
CIRCULATION

PULSE PRESSURE, VENOUS PRESSURE,
REGULATION OF THE PERIPHERAL
CIRCULATION

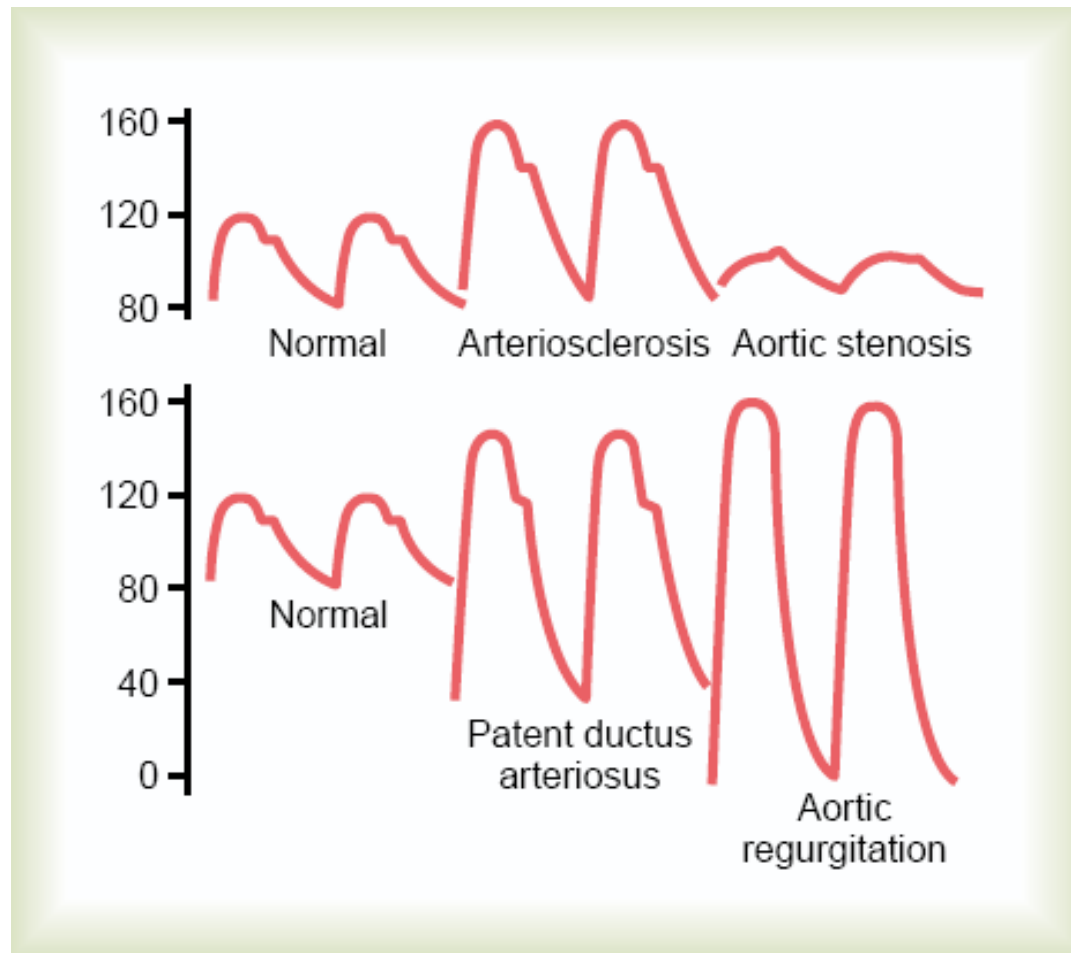
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PULSE PRESSURE

- represents the rise of pressure produced by the degree of distention of the arterial tree which is produced by the entrance of one SV of blood into the aorta during one systole.
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It is affected by 2 major factors

- SV output
- Compliance
- Character of ejection from the heart during systole
- The greater the increased of SV output, the greater the amount of blood that must be accommodated in the arterial tree with each heartbeat , therefore the greater is the **P** rise and fall during systole and diastole thereby increasing **PP**.
- The lesser the compliance of the arterial system, the higher the rise in **P** for a given SV of blood pumped into the arteries.



Pulse Pressure

■ Effect of change of Cardiac Output

Effect of change only of SV

↑ SV without change of HR → ↑ PP & ABP →
greater increase of systolic P than of diastolic P

Effect of change only of HR

↑ of HR without change of SV will ↑ P. During the rise of P the diastolic PP will be less than systolic PP, but when the new steady state is established, the PP will be the same as control and systolic and diastolic pressure will have risen to the same extent as P.

Pulse Pressure

- Effects of change of TPR

Increase of TPR, without change of SV, HR, or arterial distensibility, increases P but does not change PP. Systolic P and diastolic P rise equally. Corresponding effects occur with decrease of TPR.

- Effects of a change of arterial distensibility and capacity

A decrease of arterial distensibility will increase PP. Alterations of capacity without concomitant change of distensibility will be without effect on PP.

Vascular Distensibility

- All blood vessels are distensible
 - Increase in pressure → dilatation, decreased resistance, increased blood flow
 - Arteries: accommodate the pulsatile output of the heart and to average out the pressure pulsations
 - Veins are the most distensible
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VASCULAR DISTENSIBILITY- fractional increase in volume for each mm of Hg rise in P.

$$\frac{\uparrow \text{ in volume}}{\uparrow \text{ Pressure} \times \text{original volume}}$$

That is if 1 mm Hg causes a vessel that originally contain 10 ml of blood to increase its volume by 1 ml, the distensibility would be 0.1 per mm Hg, or 10% per mm Hg

VASCULAR COMPLIANCE- total quantity of blood that can be stored in a given portion of the circulation for each mm of Hg **P** rise.

$$\frac{\uparrow \text{volume}}{\uparrow \text{Pressure}}$$

- A highly distensible vessel that has a slight volume may have far less compliance than a much less distensible vessel that has a large volume because compliance = distensibility x volume
 - Compliance of a vein is 24x that of its corresponding artery because it is about 8x distensible and has volume 3x as great
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VENOUS PRESSURE

Right Atrial Pressure is regulated by a balance between:

1. The ability of the heart to pump blood out of the **RA**.
2. The tendency for blood to flow from the peripheral vessels back into the **RA**.

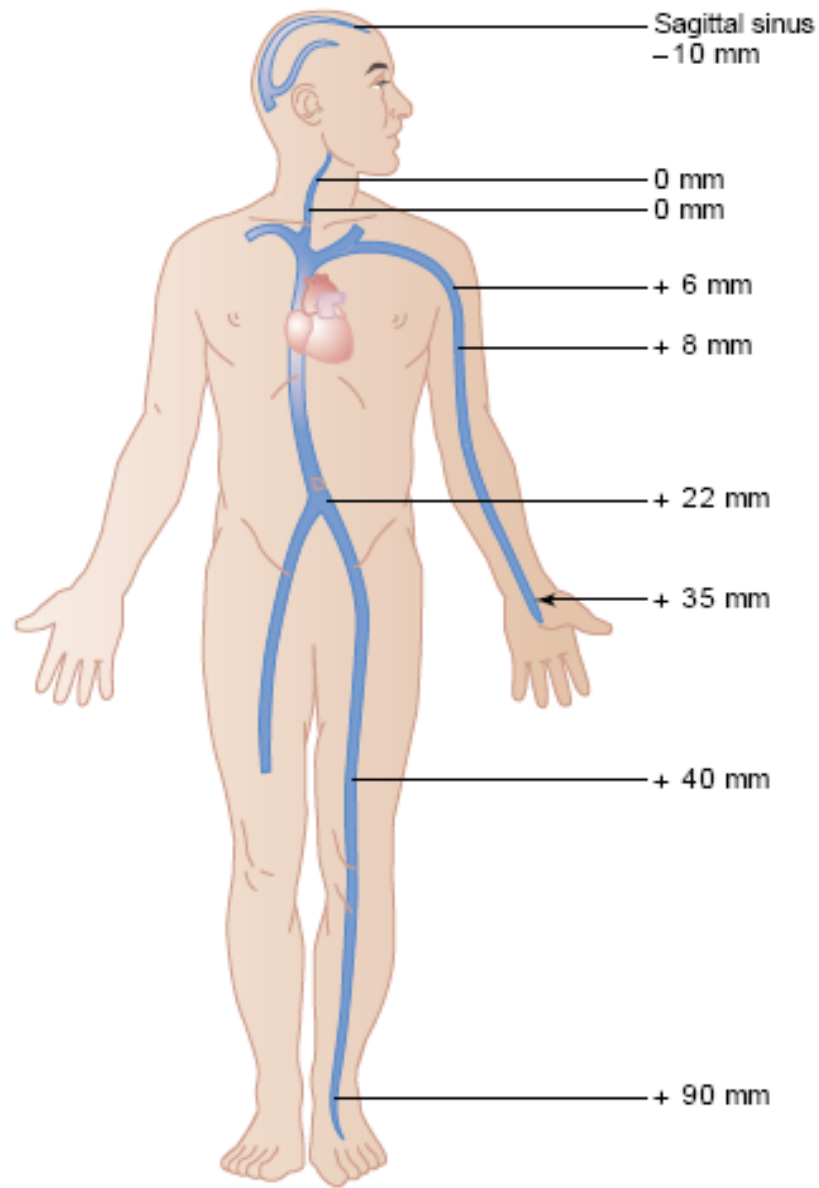
Some of the factors that increase VR are:

1. Increase blood volume
 2. Increase large vessel tone throughout the body with resultant increase peripheral venous pressure.
 3. Dilatation of the arterioles, which decrease peripheral resistance and allows rapid flow of blood from the arteries to the veins.
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Venous Pressure

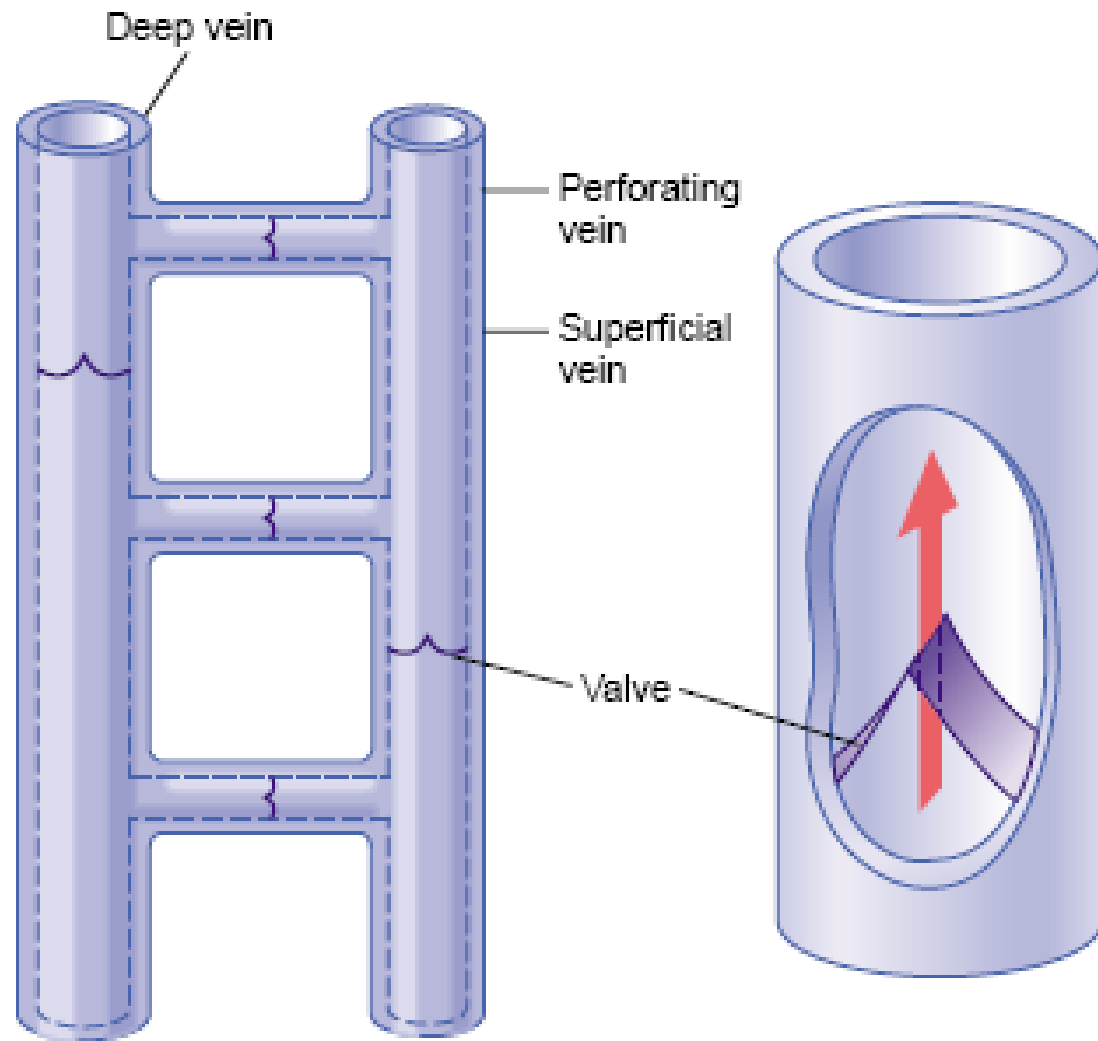
- Normal RAP = 0 mmHg
- RAP = 20-30 mmHg: serious heart failure, after massive transfusion
- RAP = -3 to -5 mmHg: when the heart pumps with exceptional vigor, when the flow of blood into the heart from the peripheral vessels is greatly depressed (after severe hemorrhage).
- Effect of high RAP on peripheral VP: increase RAP (4-6 mmHg) → blood begins to back up in the large veins and to open them up. The P in the peripheral veins do not increase until all the collapsed points between the peripheral veins and large central veins have opened up.

As the RAP increases further, additional increase in P is reflected by a corresponding increase in peripheral venous resistance



Venous Pressure

- Effect of Abdominal Pressure on venous pressure of the leg:
N = 6mmHg
AP = 15-30 mmHg: pregnancy, large tumors, or ascites
- Effect of HP on VP:
Hydrostatic Pressure occurs in the vascular system because of the weight of the blood in the vessels
VP feet = 90 mmHg
VP arm = 6 mmHg at the level of the 1st rib (HP down the length of arm is determined by the distance below the level of this rib)
- Effect of venous valves and venous pump on venous pressure: → VP maintained < 25 mmHg
standing still → VP ↑ to hydrostatic value of 90 mmHg in 30 sec → ↑P in capillaries → fluid leaks into tissue spaces → swelling of legs, ↓ blood volume from the circulatory system within 15 minutes of standing still



- Effect of change of force of ventricular contraction and/or HR on VR

↑ vigor &/or ↑ ventricular contraction → ↑ rate of transfer of blood from the central venous reservoir to the arterial tree → ↑ gradient pressure, ↑ rate of flow into capillaries, simultaneously ↓ CVP → ↑ VR

- Effect of change of precapillary resistance:

↑ precapillary resistance → ↑ pressure gradient from arteries to veins → ↓ volume of blood and pressure in the arterial tree and ↓ volume of blood and pressure in the capillaries and veins → this shifts the SFP point away from the central venous reservoir and ↓ rate of VR
→ ↓ CVP ↓ CO

Regulation of Blood Pressure

The cardiovascular center: helps regulate heart rate and stroke volume. It also controls neural, hormonal, and local negative feedback systems that regulate blood pressure and blood flow to specific tissues.

The Cardiovascular Centers found in the medulla are:

1. CAC has for its efferent sympathetic nerves to the heart
 2. VCC sends sympathetic fibers to the arterioles and capacitance vessels
 3. CIC sends impulses via the vagus to the heart
 4. VDC
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Cardiovascular Center

- \uparrow ABP \rightarrow distention of artery \rightarrow distortion of pressoreceptors \rightarrow \uparrow rate of firing by pressoreceptors \rightarrow \uparrow impulse activity in afferent fibers \rightarrow \uparrow (-) CAC and \uparrow (-) VCC \rightarrow \uparrow activation of CIC \rightarrow \uparrow vagal activity to the heart \rightarrow \downarrow HR \rightarrow \downarrow ABP
- \uparrow (-) CAC \rightarrow \downarrow sympathetic activity to the SA node \rightarrow \downarrow HR \downarrow sympathetic activity to the myocardium \rightarrow \downarrow force and magnitude of contraction \rightarrow \uparrow ESV \downarrow SV
- \uparrow (-) VCC \rightarrow \downarrow sympathetic constrictor activity to the arterioles \rightarrow \downarrow tone of arteriolar smooth muscle \rightarrow arteriolar dilatation \rightarrow \downarrow TPR
- \downarrow CO \downarrow TPR \rightarrow \downarrow ABP

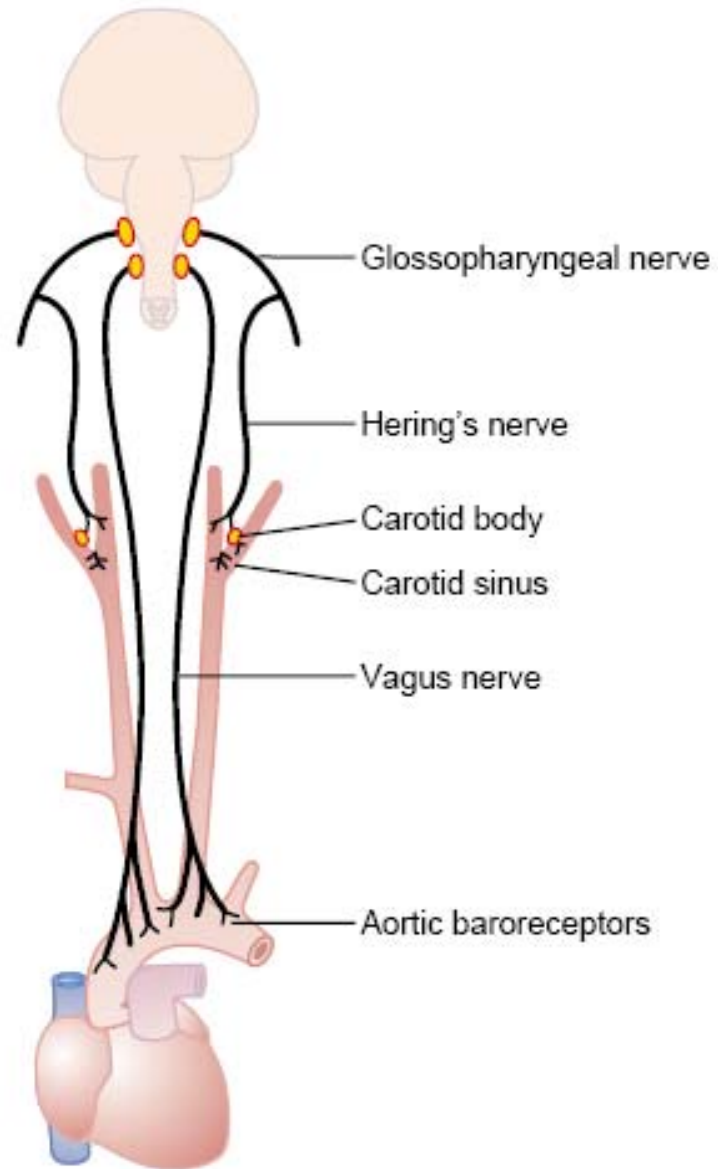
REGULATION OF ABP

3 Basic Controlling Mechanisms:

- Nervous (Baroreceptor reflexes / Chemoreceptor reflexes)
- Capillary fluid shift and the vascular stress relaxation mechanisms
- Renal- fluid volume mechanism

Nervous Regulation of the ABP

- most rapid of all mechanisms for P control
 - Rapidly acting but not long acting
 - pressoreceptors adapt readily
 - central chemoreceptors although initially stimulated appear to be eventually depressed by a sustained high and increased carbon dioxide
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I Baroreceptors – spray type nerve endings that lie in the walls of arteries.

They are stimulated when stretched.

They are abundant in:

- carotid sinus
 - wall of aortic arch
 - Carotid sinus → Hering's nerve → glossopharyngeal nerve → tractus solitarius in the medullary area of the brain stem
 - Arch of aorta → vagus nerve → same area of medulla
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II. Chemoreceptor Reflex- the chemoreceptors are chemosensitive cells which respond to O₂ lack, CO₂ excess, or H⁺ excess.

- located in the bifurcation of the carotid A (carotid bodies) and in the arch of the aorta (aortic bodies).
- Each body is supplied by a nutrient artery which enables the receptors to monitor PCO₂ level.

↓ ABP (below critical level, < 80 mmHg)



↓ BF to the bodies



↓ O₂, ↑ CO₂ and H⁺



Stimulate vasomotor center



↑ ABP

CNS Ischemic Response (Central Chemoreceptor Reflex)

There are chemoreceptors in the medulla which are sensitive to changes in CO₂ level.

An increased PCO₂ around the receptors causes them to stimulate not only the inspiratory center but also the CAC and VCC.

The CO₂ level is determined by the rate of blood flow to the area.

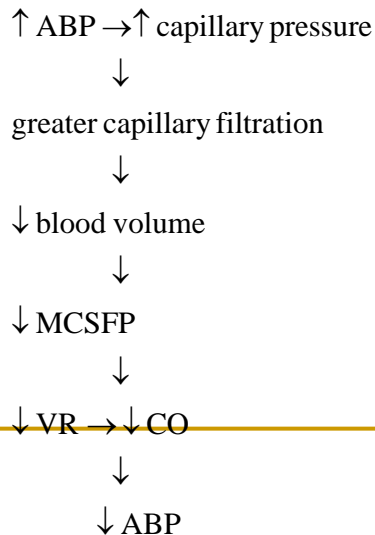
↓ ABP to 40 mmHg or lower → ↑ CO
→ intense CAC and VCC activity →
↑ ABP

Capillary Fluid Shift and Vascular Stress Relaxation Mechanisms.

- Intermediately acting pressure regulators
- Proceeds slowly until a new state of equilibrium is reached only after a few hours.

Capillary fluid shift.

Anytime capillary pressure falls too low, fluid is absorbed by osmosis from the tissue into the circulation.



Stress Relaxation

↓ ABP → lesser transmural or distending

Pressure allowing the walls of vessels to recoil →

→ ↓ VC, ↑ MCSFP, VR improves → CO improves →

→ ↑ ABP

- if ABP suddenly ↑, the ↑ venous pressure causes veins to progressively distend (stress relaxation).

When the pressure in the blood vessel becomes too high, they become stretched and keep on stretching more and more for minutes or hours, as a result the pressure in the vessels falls toward normal.

The Renal Fluid Volume Mechanism

- Long term regulation of ABP
- Long acting but slowly reacting
- Mediated via homeostasis of blood volume

1. Alterations in the GFR

↓ ABP → ↓ renal artery bld. flow → ↓ glom. cap. pressure
→ ↓ effective filtration pressure → ↓ filtration → ↓ urine formed
and retention of sodium → preservation of blood volume.
- if fluid intake is not D/C → ↑ Bld. vol. → ↑ MCSFP → ↑ VR
→ ↑ CO → improved ABP.

2. Renin- Angiotensin- Aldosterone- ADH Mechanism.

↓ ABP → ↓ renal artery P → ↓ renal artery BF
→ release of renin by the JGA of the kidney
→ conversion of Angiotensinogen in the blood
to A_I by renin → A_{II} → stimulates release of
aldosterone, ↑ reabsorption of Na in distal tubules
of the kidney → ↑ osmolality of the blood → stimulate
of release of ADH from posterior pituitary →
↑ permeability of distal tubules and collecting ducts
to water → ↑ water reabsorption → ↑ Bld. volume
→ return of ABP to normal.

Mechanisms of BF Control

- Acute control – achieved by rapid changes in local VD or VC of blood vessels occurring within seconds to minutes
 - Long-term control – slow, controlled changes in flow over a period of days, weeks, or even months
 - Better control of flow in proportion to the needs of the tissues
 - The changes come about as a result of inc or dec in the physical sizes and numbers of actual blood vessels
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REGULATION OF BLOOD FLOW

- A. Acute Control of Local BF
 - A. Effect of tissue metabolism
 - B. Effect of changes in oxygen availability
 - C. Autoregulation
 - B. Long-term BF regulation
 - 1. Change in tissue vascularity
 - 2. Utilization of collateral circulation
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Basic Theories

■ Vasodilator Theory

- The greater the rate of metabolism or the less the availability of oxygen or some other nutrients to a tissue, the greater the rate of formation of vasodilator substances in the tissue cells
 - Adenosine, carbon dioxide, adenosine phosphate compounds, histamine, potassium ions, hydrogen ions
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Oxygen Lack Theory

- Nutrient lack theory
 - Increased utilization of oxygen in the tissues would cause local VD
 - The strength of contraction of the sphincters would increase with an increase in oxygen concentration
 - The sphincters would open when oxygen concentration falls low
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Autoregulation

- The ability of a tissue to automatically adjust its blood flow to match its metabolic demands
 - achieved by rapid changes in local constriction of the arterioles, metarterioles, and pre capillary sphincters
 - occur within seconds to minutes to provide a rapid means for maintaining appropriate local tissue blood flow
 - has been observed in the brain, heart, kidneys, skeletal muscles, intestines, and liver
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Autoregulation

- An acute increase in arterial pressure causes immediate rise in BF
 - Within less than a minute, BF returns almost to normal even though AP is kept elevated
 - Metabolic
 - Myogenic
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Metabolic theory

- When AP becomes too great, the excess flow provides too much oxygen and other nutrients to the tissues → VC to return BF to normal despite increased AP
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Myogenic Theory

- Sudden stretch of small blood vessels (inc AP) causes the smooth muscle of the vessel wall to contract for a few seconds → reduce BF nearly back to normal
 - At low pressures, less degree of stretch → smooth muscle relaxes, increased flow
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Long-term blood flow regulation

- slow changes in flow over a period of days, weeks, or even months
 - provide far better control, far more complete regulation
 - Important when the long-term metabolic demands of a tissue change
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Long-term BF Regulation

- Change in tissue vascularity
 - Increased metabolism of a tissue for a prolonged period, vascularity increases
 - Vascularity increases with hypoxia, decreases with hyperoxia
- ↓BF → ↓oxygen → ↑size and diameter of blood vessel
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Long-term blood flow regulation

- Utilization of collateral circulation
 - Blockage of a blood vessel → a new vascular channel develops around the blockage and allows partial resupply of blood to affected tissue
 - Dilation of small vascular loops, further opening within the ensuing hours
 - Occlusion of the main arteries create pressure gradient between vessels in main vascular bed →
↑ collateral flow

Humoral Control of the Circulation

- Control by substances secreted or absorbed into the body fluids – hormones and ions
 - Humoral factors:
 - Vasoconstrictor Agents
 - Vasodilator Agents
 - Ions/ Chemical factors:
 - Calcium, potassium, magnesium, hydrogen, carbon dioxide, anions (acetate and citrate)
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Vasoconstrictor Agents

- Norepinephrine and Epinephrine
 - Angiotensin II
 - Vasopressin
 - Endothelin
-

Vasodilator Agents

- Bradykinin
- Histamine

