12- Special Circulation

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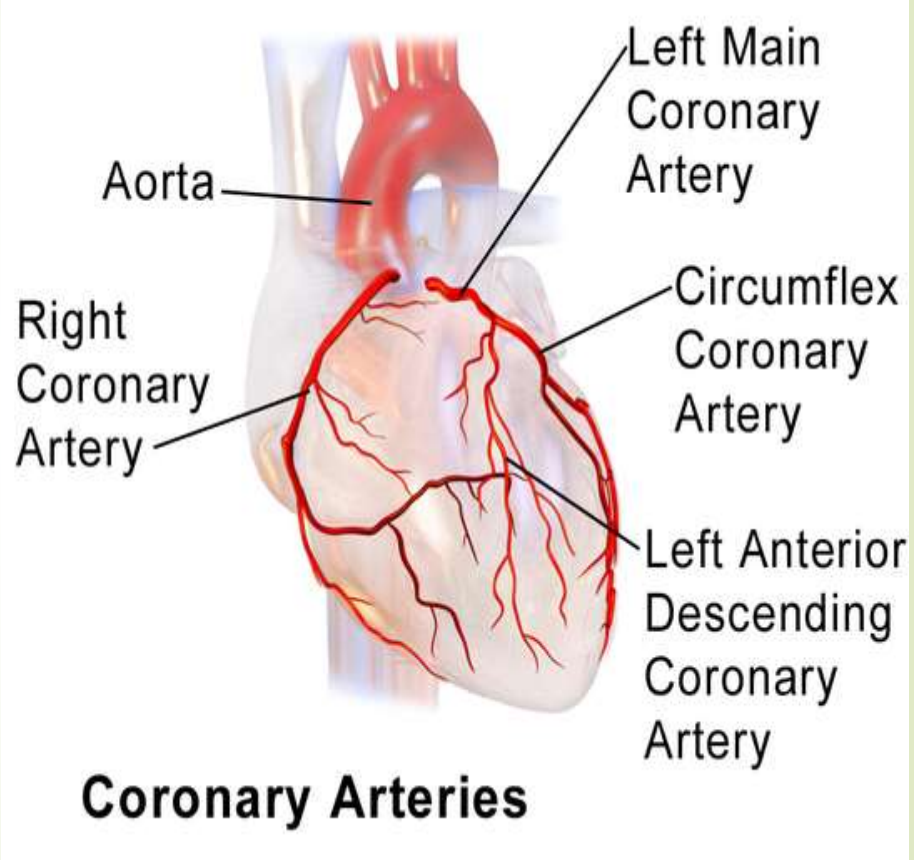
Coronary circulation

A-Coronary arteries: The heart is supplied by two coronary arteries which arise just behind the cusps of the aortic valve.

The left coronary artery supplies the anterolateral wall of the left ventricle and anterior part of the septum

The right coronary artery supplies the right ventricle, posterior part of the left ventricle and posterior part of the septum.

-There are small anastomosis between the small arteries (but there are no anastomosis between large arteries) which are insufficient to compensate for acute obstruction of large arteries. Therefore , the coronary arteries are considered to be **end arteries**.



B-Coronary capillaries:

There are about one capillary for each cardiac muscle fiber and this ratio remain constant through life. When the heart becomes hypertrophied the number of capillaries does not increase , but their diameter will be increased up to certain degree giving limiting factor for hypertrophy of cardiac muscle and heart failure finally occurs.

C-Venous drainage

1- Superficial system

Which opens into the **right atrium** directly drain the left ventricle represent about **60%** of venous drainage.

2-Deep system

Drain the rest of the heart and **open directly into the cardiac chambers**

- ***Coronary blood flow***

***1-During rest*:** it is about **80ml /100gm** of the heart **/min**. It is about **250 ml/min** which equal to **5%** of cardiac output.

N.B the right ventricle receives about 2/3 the blood flow to the left ventricle

***2-During exercise*:** It is increased about **3-5 folds** that of normal resting level and in athletic person may reach up to **600 ml /100 gm** heart weight.

Cardiac oxygen extraction:

During rest it is about 70-80% (75%) O_2 in each unite of blood. The **arterial blood** contain **19ml O_2%** and the **coronary venous blood** contain **5ml O_2%** i.e. (14 ml O_2 are extracted from every 100 ml blood, however in other tissues ,the venous blood contain 14 ml O_2%i.e only 5 ml O_2 are extracted.

So, the **cardiac O_2 consumption** during exercise can be increased significantly only by increasing the **coronary blood flow**

Regulation of coronary blood flow

1-*Chemical factors (auto-regulation) (Intrinsic mechanism):*

- Observed in the denervated heart (transplanted heart) and **independent on neural control**.

- As the cardiac work increase e.g. during muscular exercise, there is **hypoxia** ,**increase CO_2 , H^+, K^+, lactate** and other metabolites.

- The most effective vasodilator is **hypoxia** of the cardiac muscle which produces coronary vasodilatation through: 1- Direct effect on the coronary blood vessels

2- Release of chemical substances most probably **adenosine** (the greatest vasodilator substance)

-When the cardiac work is **decrease**, there is **coronary vasoconstriction**

2- *Mechanical factors*

The coronary blood flow is markedly affected by the mechanical events **during the cardiac cycle**

A- In left coronary artery:

The blood flow follow the aortic pressure:

1-In the isometric contraction phase, it is markedly **decreased** due to: a-Compression of the coronary vessels b- The low aortic pressure

2-In the maximum ejection phase, it is **gradually increased** due to rise of the aortic pressure which pushes some blood in the coronary artery in spite of the compressing effect of the contraction muscle.

3-In reduced ejection phase, it is **decreased** again due to decrease of aortic pressure.

4-In the isometric relaxation phase **maximum coronary blood flow occurs** when:

a- The aortic pressure is relatively still high. b- The cardiac muscle fibers relax

-Then the coronary blood flow gradually decrease as the aortic pressure is gradually decreased

B- In the right coronary :

- There are similar changes also occur , but they are **milder** because the force of contraction is much weaker in the thinner right ventricle and the blood flow in the right coronary artery increases towards the end of systole, but **maximum inflow is reached also in early diastole**.

- Blood flow in the coronary arteries occur mainly during **diastole**, so any pathological condition which **increase the heart rate** markedly, **shorten the diastolic period** of the ventricles and **decreased the coronary flow** .

- The coronary blood flow is controlled by the mean arterial pressure in aorta so decrease in aortic pressure lead to decrease in the coronary blood flow (e.g. shock , aortic stenosis)

3 -*Neural factor*:

A-Effect of sympathetic nervous system:

Indirect effect: an increase in heart rate, force of contractions and marked increase in myocardial work lead to accumulation of metabolites, that causes marked vasodilatation on the coronary blood vessels.

Direct effect as the coronary arteries contain **α- adrenergic** receptors , which mediate V.C and **β-adrenergic** receptors which mediate V.D. The **α-** receptors predominant in the epicardial vessels, this direct effect alone causes VC rather than V.D.

N.B: The indirect effect of sympathetic stimulation is much more effective than the direct effect.

B- Effect of parasympathetic (vagal) stimulation:

Indirect effect: It causes a decrease in heart rate, force of contraction and reduction of coronary blood flow, which is secondary to decrease in the cardiac work and metabolism.

Direct effect Acetylcholine (released at the vagal nerve ending), produces slight VD on the coronary vessels.

4- *Chemicals, hormones, and drugs:*

Chemicals cause VC	Chemicals cause VD
ADH (vasopressin).	Thyroxin.
Angiotensin II.	Adrenaline.
Endothellins.	EDRF.
Thromboxan A2.	Alcohol & ADP & Caffeine & Nitrites.

Cerebral Circulation

-Arterial supply: the brain is supplied by 2 internal carotid arteries and 2 vertebral arteries unite to form basilar arteries. The 4 arteries form the **Circle of Willis** at the base of the brain below the hypothalamus. From this circle 6 branches arise; 2 anterior, 2 middle, 2 posterior cerebral arteries.

-There are anastomosis between the cerebral arterioles but they are insufficient to maintain circulation and prevent cerebral infarction if large artery is occluded so the cerebral arteries are considered as **end arteries**.

-Venous drainage: it occurs by **deep veins** and **Dural sinuses**, then into the **internal jugular vein.**

-Cerebral capillaries: they have a very **low permeability** because they are:

-Non fenestrated with relatively strong wall and tight junction between their endothelial cells (which limits edema formation and passage of substances through this junction).

-Surrounded by end feet of the glial cells (astrocytes) supporting its wall and sharing in the formation of the blood brain barrier.

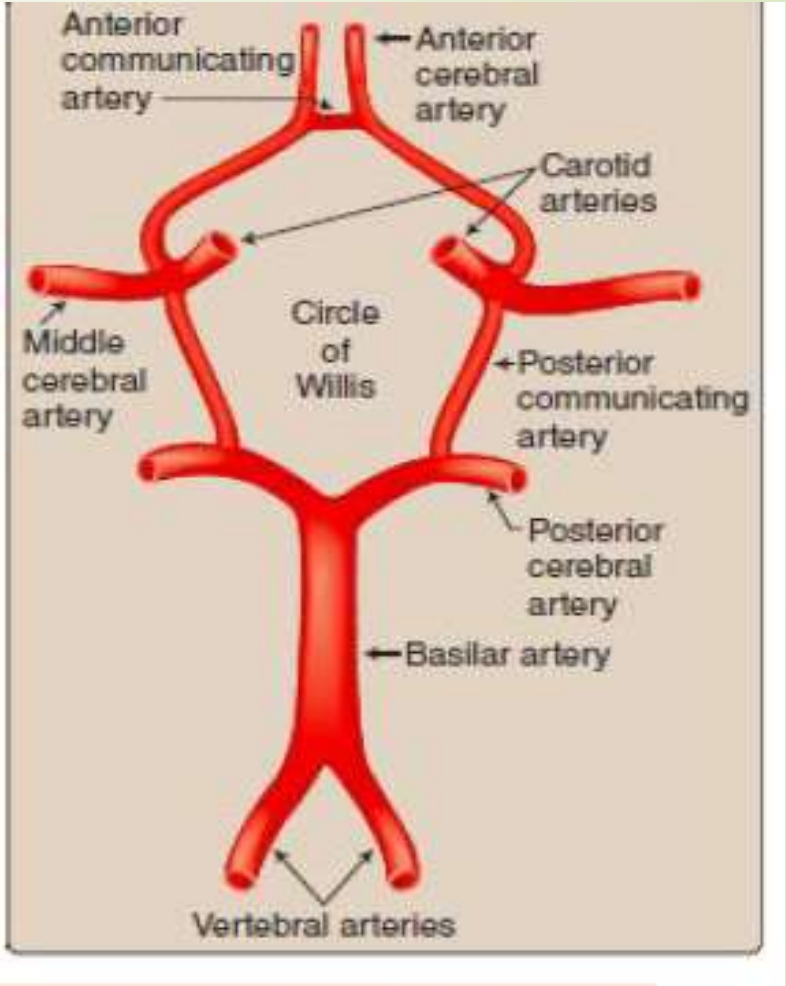
Blood Brain Barrier (B.B.B)

It separates the **brain** tissues from the **systemic circulation**, limiting the passage of many substances and drugs (proteins, bile pigments, H ions and dopamine) from blood to the brain. However some substances (e.g. H_2O, O_2, CO_2, lipid soluble substances as **anesthetics** and **alcohol** and some drugs can cross B.B.B. easily.

-Tight junction between endothelium of cerebral capillaries.

-The feet end of the glial cells (astrocytes).

-High metabolic activity in the cerebrovascular endothelial cells. (contain numerous enzymes that degrade blood born substances and prevent it from entering the brain)



Cerebral blood flow

-Normal cerebral blood flow is **54 ml /100gm** brain tissue / min. the weight of adult brain is 1400 gm so cerebral blood flow is **750 ml/min**. for the whole brain (**15% of Cardiac output**).

Regulation of cerebral blood flow (C.B.F.):

C.B.F. is regulated to **maintain constant** blood flow to the brain under various conditions (it is not increased during exercise or by mental activity and it is not decreased during sleep) however, **the regional blood flow** can be altered (it increases in active areas and decreases in inactive areas).

Factors affecting the total cerebral blood flow:

1-Autoregulation: It maintains the cerebral blood flow almost constant if the arterial blood pressure varies from **60** to **140** mmHg.

Mechanisms:

The metabolic theory: a fall of B.P →↓ C.B.F. → ↓ blood perfusion to the brain leading to accumulation of metabolites as hypoxia, ↑CO_2 and ↑ H^+ causing vasodilatation which increases cerebral flow to normal levels. Also if arterial blood pressure is increased it increases cerebral blood flow → wash of metabolites→ cerebral vasoconstriction → ↓ blood flow to normal.(**CO_2 excess** is the most potent cerebral vasodilator).

The myogenic theory is also included (i.e. stretch of the vascular wall causing vasoconstriction preventing further increase in the C.B.F.).

2-**Circulating Vasoactive substances:** e.g. **Angiotensin II** and **endothelin** cause vasoconstriction and **E.D.R.F**. causes vasodilatation.

3-**Arterial blood pressure and cardiac output:** marked ↓ in C.O.P. and A.B.P (as in hemorrhage) → ↓ C.B.F. which may cause serious brain damage. Also sudden stoppage in C.B.F. for **10 seconds** → loss of consciousness while impairment of C.B.F. for more than **3 minutes** may cause irreversible brain damage.

4-<u>Effective perfusion pressure</u>: The arterial and venous pressures at the brain level: **C.B.F** is **directly proportionate** to the difference between these pressures.

5-<u>Venous obstruction</u>: as in jugular vein thrombosis or in valsalva maneuver (as in straining) → ↓ C.B.F. temporary

6-<u>Blood Viscosity</u>: the **C.B.F**. varies **inversely** with blood **viscosity**.

7-<u>Effect of intracranial pressure</u>: in adults, the brain C.S.F. and the cerebral vessels are enclosed within a solid structure (the skull). Brain and C.S.F. are incompressible, so the volume of the blood, C.S.F. and brain in the cranium must be relatively constant at any time .

Rise in the intracranial pressure compresses the cerebral vessels and decreases the cerebral blood flow.

N.B. the intracranial pressure follow the mean cerebral venous pressure, so, **a rise in venous pressure → ↓ C.B.F**. by: **- decreasing the effective perfusion pressure.**

- increasing the intracranial pressure (which compresses the cerebral vessels).

←

8-Nervous factors affecting C.B.F.:

The **sympathetic** stimulation causes **V.C**. of the cerebral B.V. **but the C.B.F**. is compensated passively **by the increase in arterial blood pressure**.

9-Brain activity:

During mental activity there is **no increase** in the **total C.B.F.** but there is shift of blood to the active areas of the brain (during active movements of the muscles, more blood is shifted to the motor area and so on.).

N.B. sleep doesn't decrease C.B.F.

10-Age: In newly born and children the C.B.F. is **100 ml/100gm** brain weight **(double adult flow)** but after puberty it decreases to normal levels (sex hormones may be responsible for this effect).

-The brain is surrounded by **cerebrospinal fluid** with pressure of **13 mmHg** and the rigid skull, these factors limit the expansion of cerebral blood vessels and make the blood flow depends on the blood pressure.

E. Skeletal muscle

Is controlled by the **extrinsic sympathetic innervation** of blood vessels in skeletal muscle and by **local metabolic factors.**

1. Sympathetic innervation

- is the primary regulator of blood flow to the skeletal muscle**.**
- The arterioles of skeletal muscle are densely innervated by **sympathetic fibers**. The veins also are innervated, but less densely.
- There are both α1 and β2 receptors on the blood vessels of skeletal muscle.
- Stimulation of **α1 receptors** causes **vasoconstriction.**
- Stimulation of **β2 receptors** causes **vasodilation.**
- Stimulation of **Sympathetic cholinergic post ganglionic N.F** causes **vasodilation** during **Exercise.**
- The state of constriction of skeletal muscle arterioles is a major contributor to the **TPR** (because of the large mass of skeletal muscle).

2. Local metabolic control

- Blood flow in skeletal muscle exhibits autoregulation and active and reactive hyperemia.
- Demand for O2 in skeletal muscle varies with metabolic activity level, and blood flow is regulated to meet demand.
- During **exercise,** when demand is high, these local metabolic mechanisms are dominant.
- The local vasodilator substances are **lactate, adenosine, and K+**.
- Mechanical effects during exercise temporarily compress the arteries and decrease blood flow. During the post-occlusion period, reactive hyperemia increases blood flow.

table 3.3 Summary of Control of Special Circulations

Circulation* (% of Resting Cardiac Output)	Local Metabolic Control	Vasoactive Metabolites	Sympathetic Control	Mechanical Effects
Coronary (5%)	Most important mechanism	Hypoxia Adenosine	Least important mechanism	Mechanical compression during systole
Cerebral (15%)	Most important mechanism	CO_2 H^+	Least important mechanism	Increases in intracranial pressure decrease cerebral blood flow
Muscle (20%)	Most important mechanism during exercise	Lactate K^+ Adenosine	Most important mechanism at rest (α_1 receptor causes vasoconstriction; β_2 receptor causes vasodilation)	Muscular activity causes temporary decrease in blood flow
Skin (5%)	Least important mechanism		Most important mechanism (temperature regulation)	
Pulmonary† (100%)	Most important mechanism	Hypoxia vasoconstricts	Least important mechanism	Lung inflation

*Renal blood flow (25% of resting cardiac output) is discussed in Chapter 5.
†Pulmonary blood flow is discussed in Chapter 4.

Thank You