Cardio Vascular Physiology

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CARDIOVASCULAR PHYSIOLOGY

CORONARY CIRCULATION

Outline:

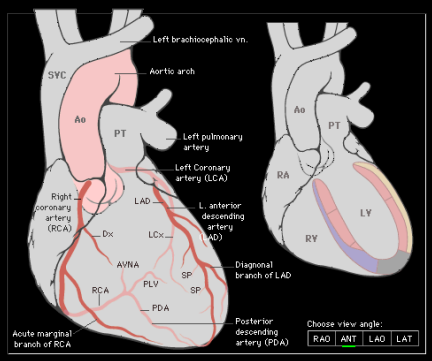
- **Blood supply of the heart (arterial supply & venous drainage)**
- **Characteristics of the coronary circulation**
- **Coronary blood flow (CBF)**
- **Factors affecting CBF (coronary circulation)**
- **Coronary Heart Disease (CHD)**
- **Angina pectoris and coronary thrombosis**

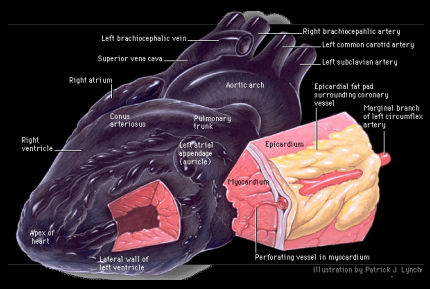
CORONARY CIRCULATION

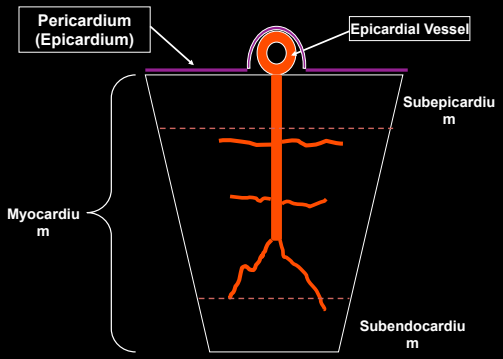
BLOOD SUPPLY OF THE HEART:

a) Arterial supply:

- **The cardiac muscle is supplied by the first two branches of the aorta i.e. right & left coronary arteries.**
- **The coronary arteries branch freely to form a rich capillary network. There is about one capillary for each cardiac muscle fiber.**
- **Coronary arteries are considered as functional end arteries. There are small anostomatic connections between the small branches of the two coronary arteries and between the coronary arterioles and extra cardiac arterioles.**







These anatomizes are not sufficient to supply the cardiac muscle with blood if one of the coronary arteries is occluded.

Thus, occlusion of a large branch of the coronary artery e.g. by coronary thrombosis → necrosis (=death) of the muscle supplied by that branch.

b) Venous Drainage: Coronary venous drainage occurs through two systems:
   1) Superficial system: which drains the left ventricle. It is formed of coronary sinus and anterior cardiac veins that open into the right atrium.
   2) deep system: which drains the rest of the heart. It is formed of the basian veins and arterio-sinusoidal vessels that open directly into the heart chamber.

CHARACTERISTICS OF THE CORONARY CIRCULATION

1) It is very short and very rapid (so it is essential to the heart).
2) The blood flow in this circulation occurs mainly during cardiac diastole
3) There is no efficient anastomoses between the coronary vessels.
4) It is a rich circulation (5% of the CO while the heart weight is 300gm).
5) Its regulation is mainly by metabolites and not neural
6) The capillary permeability is high (the cardiac lymph is rich in protein)

7) The coronary vessels are susceptible to degeneration and atherosclerosis.
8) There is evident regional distribution: The subendocardial myocardial layer in the left ventricle receives less blood, due to more myocardial compression (but this is normally compensated during diastoles by V.D). However, this renders this area more liable to ischemia and infarction.

CORONARY BLOOD FLOW

- Under resting conditions coronary blood flow (CBF) in the human heart is about 250 ml/ minute (=5% of the cardiac output).

  In severe muscular exercise, the work of the heart increased and the CBF may be increased up to 2 liters/ minute.

- Coronary Inflow (arterial) occurs mainly during diastole, because during systole the coronary arteries are mechanically compressed by the contracting myocardium, i.e.
  - Systole of the heart → ↓ coronary inflow
  - Diastole of the heart → ↑ coronary inflow
- Coronary Outflow (venous) occurs mainly during systolic due to compression of the coronary veins by the contracting myocardium. During diastole coronary outflow ↓ and veins are filled.
- Normal *diastolic blood pressure* is important for coronary filling because filling of coronary arteries occurs mainly during ventricular *diastolic*.

CARDIOVASCULAR PHYSIOLOGY

PULMONARY CIRCULATION

Outline:

Pulmonary Circulation:
- Functions and Characteristics of pulmonary circulation
- Pulmonary blood pressure and factors affecting it.
- Pulmonary edema.

PULMONARY CIRCULATION

Pulmonary Circulation is the circulation between right ventricle and left atrium. It has the following functions:

1. Carriage of blood from right to left side of the heart.
2. Exchange of gases between blood and alveolar air. The venous blood becomes oxygenated and some CO_2 is removed.
3. Acts as a blood reservoir.

The pulmonary circulation is *shorter* than systemic circulation, as the pulmonary circulation time is about 5 sec. only.

PULMONARY CIRCULATION

With blood returning FROM the Body TO the Heart…(via the Vena Cava):

Right Atrium → Tricuspid Valve → Right Ventricle → Pulmonary Semilunar Valve→ Pulmonary Trunk → Pulmonary Arteries (2) → Alveoli in the Lungs → → Capillaries in the Alveoli

ONCE BLOOD REACHES THE CAPILLARIES – GAS & NUTRIENT EXCHANGE OCCURS!

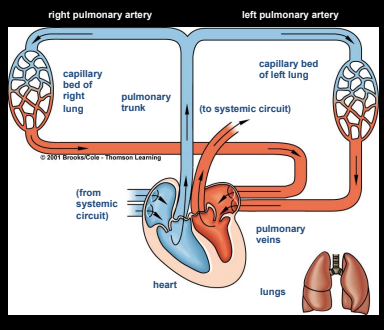
In Pulmonary Circulation, this means that Carbon Dioxide is released to the alveoli in the lungs and Oxygen is picked up by the bloodstream.

→ Pulmonary Veins (4) → Left Atrium → Back to Systemic Circulation

REMEMBER…the Left & Right sides of the heart pump blood SIMULTANEOSLY!

Pulmonary Circuit

This loop oxygenates blood



PULMONARY BLOOD PRESSURE (PBP)

- **The blood pressure is 25 mm Hg *systolic* & 10 mm Hg *diastolic* in pulmonary arteries, 10 mm Hg in pulmonary capillaries & 6 mm Hg in pulmonary veins.**
- **The *mean pulmonary blood pressure* is 16 of the aortic pressure as the pulmonary peripheral resistance is low because of:**

  a) Little amount of smooth muscles in pulmonary arterioles.

  b) Short pulmonary capillaries and veins which are easily distensible

CHARACTERISTICS of the PULMONARY CIRCULATION

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6. **The blood flow in the pulmonary capillaries is rapid 0.75 second at rest.**
7. **Both the capillary surface area and capillary permeability are great.**
8. **The *regional pulmonary blood flow* is controlled by *gravity* (it is greater in the bases of the lungs) and O_2 tension (it is reduced in hypoxic areas).**
9. ***The alveoli are normally kept dry*. This prevents pulmonary edema, and is due to rich lymph drainage and –ve pressure in the lung interstitial spaces.**
10. **It has special reactions to gas changes. *Hypoxia*, hypercapnia and rise of H' produce *V.C.* (and not V.D as they produce in other tissues).**

The large *distensibility* of the pulmonary vessels renders the pulmonary peripheral resistance to be considerably *low* (about 1/6 that of the systemic circulation). Accordingly, the pulmonary arterial *B.P.* is normally low (25/10 mm Hg).

SYSTEMIC CIRCULATION

With blood RETURNING from the Lungs TO the Heart…(via the Pulmonary Veins)

Left Atrium → Bicuspid Valve → Left Ventricle → Aortic Semilunar Valve → Aorta → Arteries → Arterioles → Capillaries in Tissues, Muscles, and Organs

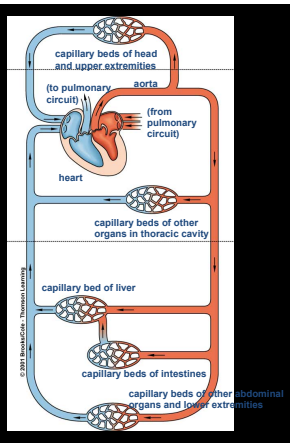
ONCE BLOOD REACHES THE CAPILLARIES – GAS & NUTRIENT EXCHANGE OCCURS!

In Systemic Circulation, this means that Oxygen and other Nutrients (like Glucose) are delivered to the tissues, muscles, & organs, and Carbon Dioxide and other wastes are transferred into the bloodstream for removal.

→ Venules → Veins → Superior OR Inferior Vena Cava → Right Atrium

Systemic Circuit

Longer loop carries blood to and from body tissues



ANGINA PECTORIS & CORONARY THROMBOSIS

- The coronary arteries are liable to degenerative changes leading to their narrowing or occlusion by blood thrombosis. This is a common complication of the atherosclerosis and hypertension.
- Angina Pectoris is to narrowing of the coronary arteries → ischemia of the cardiac muscle → pain which is retrosternal (behind the sternum). The anginal pain may radiate to left shoulder, left arm or forearm, or abdomen.
- Coronary Thrombosis → occlusion of one of the coronary arteries or its branches → necrosis of the area supplied by the occluded artery → myocardial infarction.

CORONARY HEART DISEASE (CHD)

ANGINA PECTORIS

- Angina Pectoris means severe chest pain (usually retrosternal i.e. behind the sternum) due to ischemia of the cardiac muscle.

  The anginal pain may radiate to the left shoulder, left arm or forearm (=referred pain).
- Angina pectoris is usually due to narrowing of the coronary arteries → ischemia.
- Anginal pain may be relieved by rest & coronary VD drugs.

MYOCARDIAL INFARCTION

- Myocardial Infarction means necrosis of a part of the myocardium due to

  a) Severe & prolonged ischemia due to narrowing of the coronary arteries.

  b) Occlusion of one of the coronary arteries or its branches by coronary thrombosis → severe ischemia.
- Myocardial Infarction produces also chest pain which is more severe than that of angina and it cannot be relieved by rest or coronary VD drugs.
- It is frequently complicated by ventricular fibrillation → death.

HAEMORRHAGE

Haemorrage means loss of blood (bleeding) from the cardiovascular system.

It may be:

- External haemorrhage: in which the blood is shed outside the body or
- Internal haemorrhage: in which the blood passes from vascular system to tissue spaces or to the body cavities (e.g. chest and abdomen)

Also, haemorrhage may be:

- Small repeated haemorrhage (as in piles) or
- Big sudden haemorrhage.

EFFECTS OF HAEMORRHAGE

- These depend upon two factors:

  1. Volume of blood lost.
  2. The rate at which this loss occurs.

- So, loss of small amounts of blood every day over many months, does not disturb the circulation though it may produce anaemia. The body can compensate for it.
- But when more than 30% of the blood volume is lost, the body can not compensate for it and unless blood transfusion is done death results.
- If the lost blood volume exceeds 30%: the replacement of the lost blood volume by I.V. administration of fluid becomes life saving: fluids which can be given are: blood, saline, plasma or plasma substitutes.

EFFECTS OF HAEMORRHAGE

- The following effects are produced by haemorrhage:

1. **Hypotension:** because the loss of blood → ↓ blood volume → ↓ C.O.P. → ↓ A.B.P.
2. Rapid and weak pulse: and in severe haemorrhage, the pulse is hardly felt.
3. ↑ Respiration in rate and depth.
4. Pale and cold skin: the skin is pale due constriction of skin capillaries and it is cold due to constriction of skin arterioles → ↓ blood volume passing through the skin.
5. ↓ Urine Formation: due to ↓ renal blood flow and ↑ secretion of antidiuretic hormone.
6. Fainting (=loss of consciousness) and death may occur in severe haemorrhage due to brain ischaemia.

EFFECTS OF HAEMORRHAGE

- Hypotension (↓ ABP) → inadequate perfusion
- Cerebral hypoxia (ischaemia) → depression of brain (cortex and centers) → coma.

BODY REACTION TO HAEMORRHAGE

Compensatory reactions in acute haemorrhage

Immediate compensatory reactions

(A) Immediate reactions — aim at rapid elevation of the arterial B.P.

1. ***Reactions that correct the hypovolaemia***: (a) Capillary fluid shift from the tissue spaces to the bloodstream (b) Mobilization of the labile tissue protein into the bloodstream (c) Splenic contraction (which adds the stored blood in the spleen to the circulating blood).

2. ***Reactions that increase the cardiac output (CO) and peripheral resistance (PR):*** These are produced due to *stimulation of the VCC* by (a) signals from the ischaemic peripheral chemoreceptors (b) its release from the inhibitory effect of the arterial and atrial baroreceptors (c) the CNS ischaemic response. The VCC leads to generalized sympathetic stimulation and secretion of catecholamine, and both produce the following effects:
   a) Tachycardia and increase of the stroke volume (both increase the CO)
   b) Generalized V.C. (which increases the PR)
   c) Generalized venoconstriction (which increases the VR, CVP and CO)

   Respiration is also accelerated (which helps increasing the VR and consequently the CO) and ACTH is secreted (this hormone stimulates release of glucocorticoids which increase the vascular reactivity to catecholamine)

Delayed compensatory reactions

(B) Delayed reactions — aim at keeping the arterial B.P. close to its normal level

1. ***Reactions that maintain a high PR***: These include mainly (a) Secretion of ADH (= vasopressin, which causes V.C) (b) Formation of angiotensin II (which is also V.C) as a result of rennin secretion by the ischaemic kidneys.
2. ***Reactions that maintain a normal blood volume***: These include:
   a- ***Restoration of the plasma volume*** by (a) ADH (helps water retension in the body) (b) Secretion of aldosterone (by effect of angiotensin II) which increases Na^+ and water retension in the body (c) Drinking water (as a result of the increased thirst sensation) (d) Inhibition of secretion of the atrial natriuretic peptide (ANP).
   b- ***Restoration of the plasma proteins*** (by increased synthesis from the tissue reserve proteins as well as the diet proteins)
   c- ***Restoration of the red blood cells*** (by increased formation in the bone marrow under effect of the *erythropoietin hormone*, which is released by the kidneys as a result of O_2 lack).

Summary

BODY REACTION TO HAEMORRHAGE

Immediate Compensatory Reactions	Delayed Compensatory Reactions
⊙ ↑ heart rate → ↑ COP	⊙ Secretion of ADH & aldosterone → retention of water → ↑ plasma volume.
⊙ Vasoconstriction of arterioles (↑PR)	⊙ Mobilization of labile and reserve proteins from the tissues to the → ↑ plasma proteins.
⊙ Venoconstriction → ↑ VR	
⊙ Contraction of spleen.	
⊙ ↑ secretion of adrenaline & Noradrenaline	

- ⊙ Activation of renin-agiotensin sys
- ⊙ ↑ secretion of vaso-pressin (ADH).
- ⊙ Capillary fluid shift
- ⊙ ↓ urine formation.
- ⊙ ↑ formation of erythropoietin → ↑ production of RBCs

These reactions restore blood pressure and blood volume in mild or moderate haemorrhage.

SHOCK

- Shock is a clinical syndrome characterized by inadequate tissue perfusion due to decreased cardiac output and decreased ABP (hypotension).

- It is generally classified into 4 types:
  1. Hypovolaemic shock
  2. Low-resistance shock
  3. Cardiogenic shock
  4. Obstructive shock

HYPOVOLAEMIC SHOCK

- *It occurs a result of excessive loss of blood or plasma, e.g.*
  - Haemorrhagic shock
  - Traumatic shock
  - Surgical shock
  - Burn shock
- *It is also called cold shock.*

CARDIOGENIC SHOCK

- *It occurs as a result of decreased pumping action of the left ventricle e.g. due to:*
  - Myocardial infarction.
  - Severe ventricular tachycardia

LOW-RESISTANCE SHOCK

- *It occurs as a result of massive vasodilatation → ↑ circulatory capacity and → venous return → ↓ COP → ↓ ABP e.g. neurogenic shock, septic shock anaphylactic shock (histamine shock).*
- *It is also called warm shock.*

OBSTRUCTIVE SHOCK

- *This occurs as a result of obstruction of blood flow in the lungs or Heart e.g. due to a large pneumotorax. Cardiac tamponade or massive pulmonary embolism.*

HEART FAILURE

Heart failure (HF) means decreased ability of the heart to perform its proper pumping action (due to decreased force of contraction of the ventricles). HF may be left-sided HF or right sided HF or both (congestive HF).

Left-sided heart failure	Right-sided heart failure
Causes: ▪ Systemic hypertension (chronic or untreated). ▪ Coronary heart disease → myocardial infarction in the LV. ▪ Aortic stenosis or incompetence (valvular disease).	*Causes:* ▪ Pulmonary hypertension ▪ Mitral stenosis pulmonary hypertension. ▪ Left sided heart failure
Left sided heart failure → ▪ Forward failure i.e. failure to maintain an adequate cardiac output from the left ventricle. ▪ Backward failure i.e. failure of the left ventricle to pump the blood it receives from the pulmonary veins.	**Right sided heart failure →** ▪ Forward failure i.e. failure to maintain an adequate cardiac output from the right ventricle. ▪ Backward failure i.e. failure of the right ventricle to pump the venous blood it receives from the systemic veins.

Forward failure →	Forward failure →
▪ Easy fatigability and weakness. ▪ Skin pallor and cold extremities.	▪ Same manifestations as those produced by left-sided HF
Backward failure →	**Backward failure →**
▪ Pulmonary congestion → dyspnea, orthopnea and pulmonary edema. ▪ Pulmonary hypertension and right-sided HF.	▪ Systemic venous congestion → edema in dependent parts of the body + congested neck veins + enlargement of the limbs + transudation of fluid in the pleural sac.