HEMODYNAMICS

Heart Rate, Stroke Volume, Cardiac Output and Cardiovascular Control

Dr I.O Mustafa

Department of Physiology
Federal University Oye-Ekiti

Definitions and Values

	Definition	At Rest
Heart Rate	The number of beats of the heart per minute	60-100
Stroke Volume	The volume of blood ejected from the left ventricle **per beat.** It is the **EDV-ESV**	70mL/ beat
Cardiac Output	The volume of blood ejected from the left ventricle **per minute**	5L/min

Note:

In a trained athlete, at rest, the heart rate could be as low as 50 beats per minute; and the stroke volume up to 100mL per heart beat. The cardiac output stays relatively the same at 5L/min

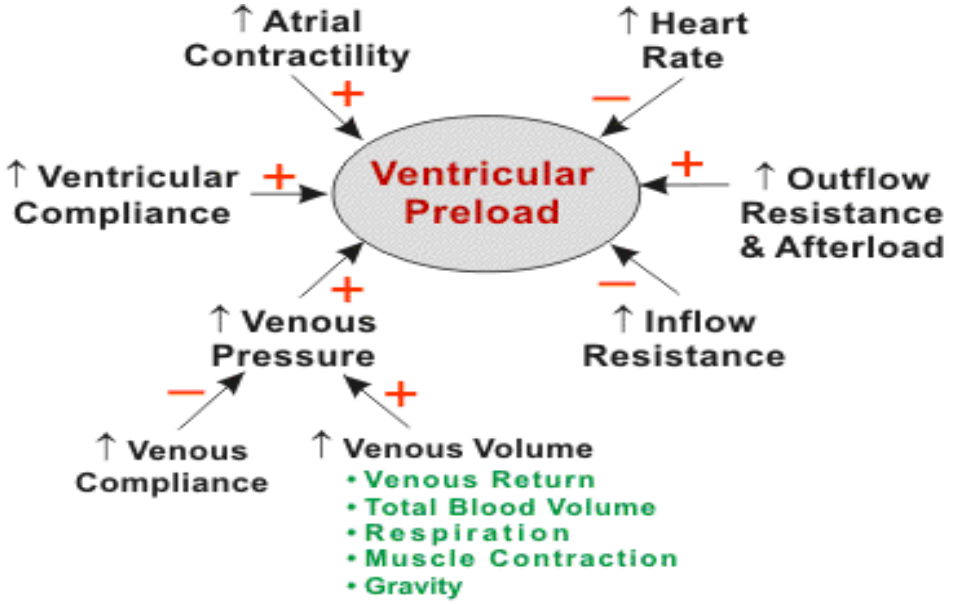
Cardiac output

- Volume of blood ejected from left ventricle in one minute
- It is the determinant of oxygen transport from the heart to the body
- It reflects the efficiency of cardiovascular system
- There no absolute value for cardiac output measurement

Cardiac Output Influencing Factors

Ventricular Preload

- Volume of blood in the ventricle at the end of diastole
- Any changes in the ventricular preload will affect the ventricular stroke volume



Factors determining ventricular preload. A "+" sign indicates that an increase in this particular variable increases ventricular end-diastolic volume, and therefore preload, while the "-" indicates that the variable decreases preload.

Cardiac Output Influencing Factors

Contractility

- Frank-Starling law states that the force or tension developed in a muscle fiber depends on the extent to which the fiber is stretched
- Increased contractility (inotropy), will shift Starling's curve to the left
- Decreased contractility will shift Starling's curve to the right

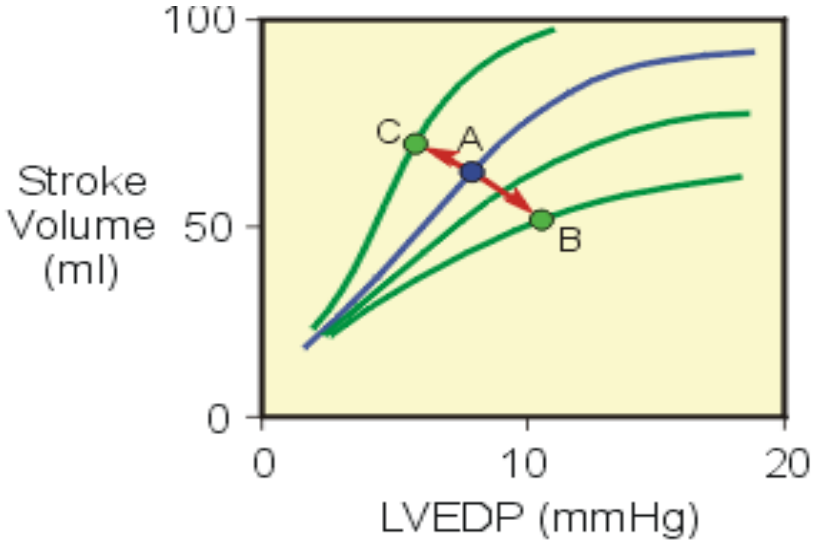


Figure 1. Effects of changes in inotropy on Frank-Starling curves. A shift from A to B occurs with decreased inotropy, and from A to C with increased inotropy.

Cardiac Output Influencing Factors

Afterload

- Related to ventricular wall stress in accordance with **Laplace's law**

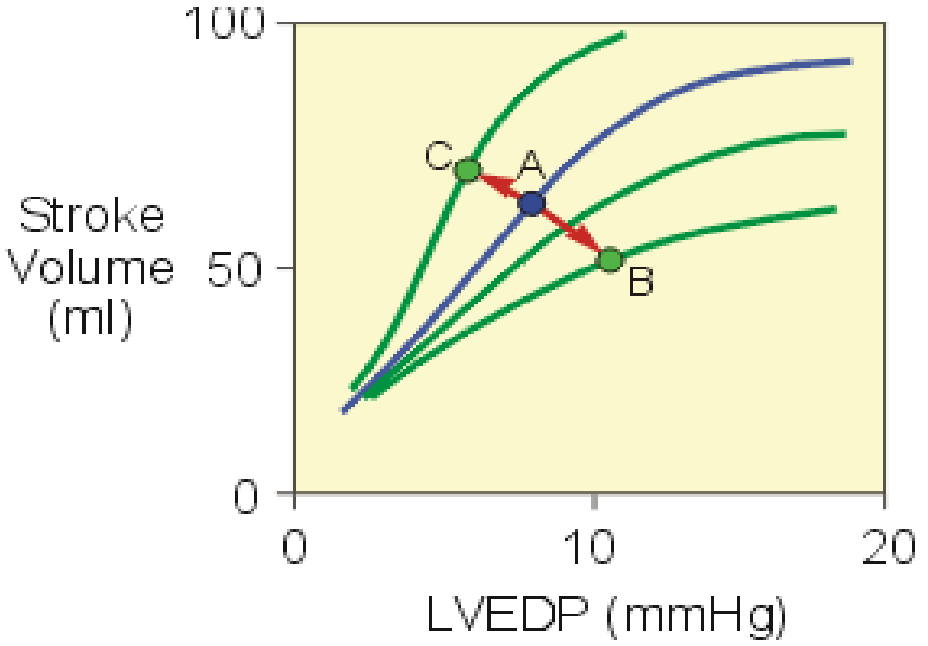


Figure 1. Effects of changes in afterload on Frank-Starling curves. A shift from A to B occurs with increased afterload, and from A to C with decreased afterload.

Cardiac Output Influencing Factors

Ventricular Compliance (distensibility of the relaxed ventricle, defined in terms of its diastolic pressure-volume relationship)

$$\text{Compliance} = \delta V / \delta P$$

- Determined by the structural properties of the cardiac muscle e.g. muscle fibers and their orientation, connective tissue….
- Given a change in the pressure causes a change in the volume

Cardiac Output Influencing Factors

Ejection Fraction

- Is a fraction of blood ejected by the ventricle relative to its end-diastolic volume

$$EF = SV / EDV. 100$$

- Ejection fraction is used as a clinical index to evaluate the inotropic status of the heart

Methods of Calculating and Measuring Cardiac Output

Simple Method:

- CO = SV x HR
- SV = 2ml x pulse pressure
- CO = [2ml x pulse pressure] x HR

Pulse Pressure = Systolic Blood Pressure – Diastolic Blood Pressure

Methods of calculating and measuring cardiac output

Fick Principle: “gold standard”

$$CO = VO_2 / O_{2\,art} - O_{2\,ven}$$

Arterial O_2 = Hb x 1.34 x O_2 sat.

Venous O_2 = Mixed venous blood

VO_2 = Oxygen consumption

- **Fick Principle relies on the total uptake of a substances by peripheral tissue is equal to the product of blood flow to the peripheral tissue and arterial–venous concentration difference of the substances**

- Fick cardiac outputs are infrequently used because difficulties in collecting and analyzing exhaled gas conc. In critically ill patients because may not have normal VO_2 value

Heart rate regulation – Neural Control

SYMPATHETIC NERVOUS SYSTEM

The body systems must adapt to the environment around it to allow them to perform as efficiently as possible. When exercise is undertaken, the muscles require greater volumes of oxygen to help in the breakdown of fats and glucose to produce ATP for muscular contractions. The sympathetic nervous system responds by stimulating the SA node to increase heart rate.

PARASYMPATHETIC NERVOUS SYSTEM

Once exercise has finished and the body begins to recover the parasympathetic nervous system will act to reduce stimulation of the SA node reducing heart rate. This will eventually return to resting levels.

Sympathetic nervous system

SYMPATHETIC NERVOUS SYSTEM

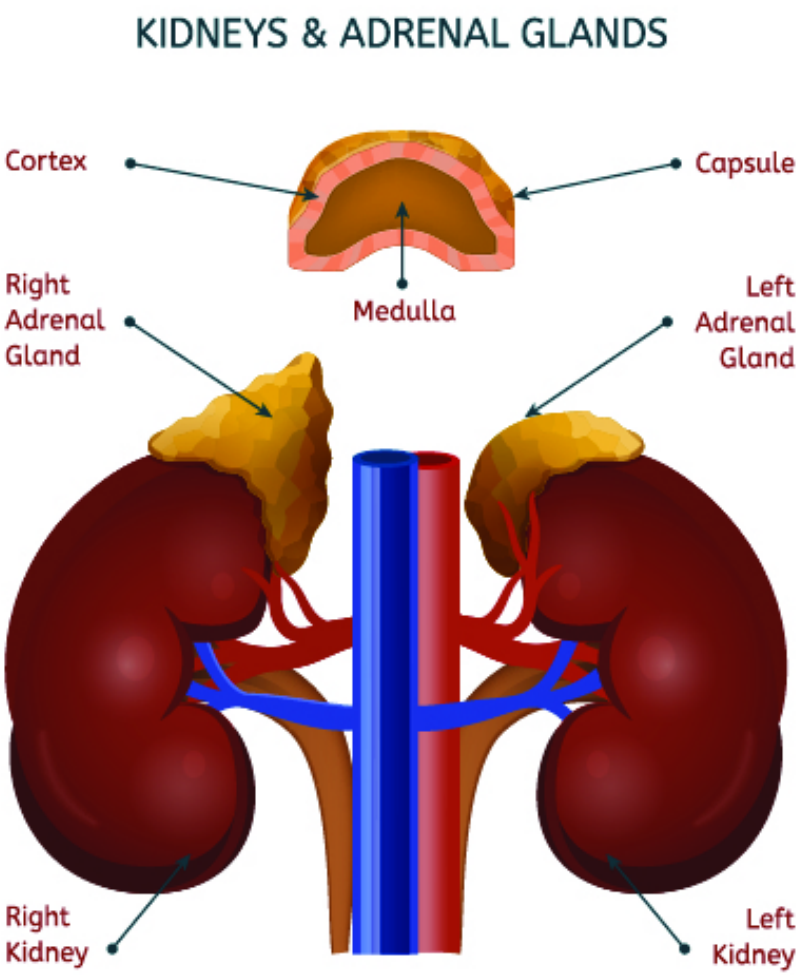
The sympathetic nervous system is responsible for increasing heart rate. This is controlled by the **CARDIAC CONTROL CENTRE** situated in the **MEDULLA OBLONGATA** of the brain.

The Cardiac Control Centre receives information from receptors concerning various changes in the body as a result of exercise being undertaken.

The CCC then sends impulses down the **ACCELERATOR NERVE** to increase the firing rate of the SA Node, thus increasing heart rate meaning more oxygen is delivered to the working muscles.

Heart Rate Regulation – Hormonal Control

In response to exercise the **ADRENALIN** and **NOR-ADRENALIN** are released from the **ADRENAL GLAND.** These hormones have a direct effect on the force of contraction of the heart muscle, thus increasing Stroke Volume and the firing rate of the SA node, thus increasing Heart Rate. The combined effect will increase Cardiac Output and delivery of oxygenated blood to the working muscles.



Venous return mechanisms

POCKET VALVES – one way valve located in the veins which prevent the backflow of blood.

MUSCULAR PUMP – the contraction of skeletal muscle during exercise which compresses the veins forcing blood back towards the heart.

RESPIRATORY PUMP – During inspiration and expiration a pressure difference between the thoracic and abdominal cavities is created which squeezes blood back towards the heart.

SMOOTH MUSCLE – The layer of smooth muscle in the walls of the veins venoconstricts to create **VENOMOTOR TONE** maintaining pressure in the vein and thus helping the transport of blood back to the heart.

GRAVITY – Blood from above the heart returns towards the heart with the help of gravity.

Redistribution of Cardiac Output

Cardiac output at rest is approximately **5 litres per minute**. During maximal exercise this can increase to **25-40 litres per minute** depending on fitness levels.

To further aid performance during exercise the increased cardiac output is redistributed to areas of the body which need it most, namely the working muscles. This is done by the **VASCULAR SHUNT MECHANISM**.

The redistribution of blood is controlled by the **VASOMOTOR CONTROL CENTRE** in the medulla oblongata.

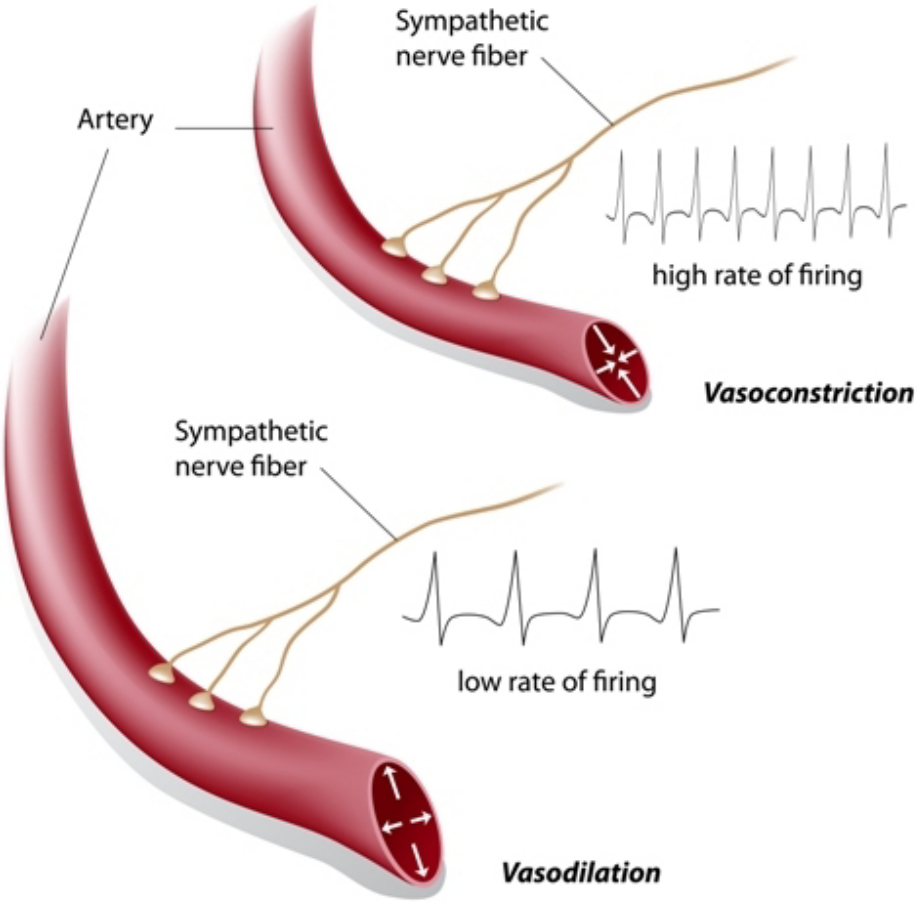
The VCC receives information from **CHEMORECEPTORS** about increases in blood acidity and **BARORECEPTORS** regarding pressure changes on arterial walls.

This causes the VCC to alter stimulation of arterioles in different areas of the body via the vasomotor nerves.

Vasomotor control

VASOCONSTRICTION is where increased stimulation from the vasomotor nerve causes the smooth muscle layer to contract reducing lumen diameter and therefore blood flow.

VASODILATION is where decreased stimulation from the vasomotor nerve causes the smooth muscle layer to relax increasing lumen diameter and therefore blood flow.



Vascular shunt mechanism

Distribution	At rest		During exercise	
To organs	Arterioles	Pre-Capillary Sphincters	Arterioles	Pre-Capillary Sphincters
	Vasodilate	Relaxed	Vasoconstrict	Contract
To working muscles	Arterioles	Pre-Capillary Sphincters	Arterioles	Pre-Capillary Sphincters
	Vasoconstrict	Contract	Vasodilate	Relaxed
Effect	More blood travels to organs than muscles		More blood travels to muscles than organs	