Cardiac Output and Its Regulation

① Cardiac output is the amount of blood pumped by each ventricle per minute.

Stroke Volume is the amount of blood pumped by each ventricle per beat. It is about 70 ml.

Heart Rate is the number of heart beats per minute.

Cardiac Output (CO) = Stroke Volume (SV) × Heart Rate (HR)

= 70 ml × 72

≈ 5 Litres/min.

Note:-

Cardiac Index = Cardiac Output/per minute/per square meter of body surface

= 3.2 L/min/m^2

Cardiac Reserve is the difference between a person's maximum cardiac output and cardiac output at rest.

② Since cardiac output equals the stroke volume multiplied by Heart rate Thus variations in cardiac output (CO) can be produced by changes in heart rate (HR) or Stroke Volume (SV)

Regulation of Cardiac Output (CO)	=	Stroke Volume (SV) (Conditions that affect SV will effect CO)	×	Heart Rate (HR) (Conditions that affect HR will effect CO)

③ Conditions that affect SV / Regulation of SV.

Stroke volume (SV) is determined by neural input, sympathetic stimuli which makes the cardiac muscle fibres contract with greater strength and parasympathetic stimuli which makes the cardiac muscle fibres contract with lesser strength.

Three factors regulate Stroke Volume

ⓐ Preload (degree of stretch on heart before contraction)

ⓑ Contractility (strength of contraction at preload)

ⓒ Afterload (pressure to be overcome before semilunar valve can open)

(a) Preload : Effect of Stretching

→ The force of contraction of the cardiac muscle fibres increases if there is a greater preload (stretch) on the cardiac muscle fibres.

→ The more the heart fills with blood during diastole, the greater the force of contraction during systole (Frank - Starling law of the heart)

→ The preload depends on the end-diastolic volume (EDV) which is the volume of blood in the ventricles at the end of diastole.

→ EDV in turn depends on
(i) Duration of ventricular diastole
(ii) Volume of blood returning to the right ventricle. (Venous Return)

→ When EDV is less then the preload is lower and the stroke volume declines

→ When EDV is increased then preload is larger and the stroke volume increases

(b) Contractility.

→ The cardiac acceletor action of the Catecholamines (adrenaline or noradrenalin) liberated by sympathetic stimulation is called Chronotropic Action

→ The effect of catecholamines on the strength of the cardiac contraction is called Inotropic Action

→ Substances that increase contractility are called Positive inotropic agents (eg ⊕ sympathetic part of autonomic nervous system, epinephrine, norepinephrine, increased Ca^{2+} level and the drug digitalis)

→ Substances that decrease contractility are called Negative inotropic agents (eg ⊖ sympathetic part of autonomic nervous system, acidosis, anoxia and increased K^+ level)

→ At a constant preload the stroke volume increases when a positive inotropic substance is present whereas, the stroke volume decreases when a negative inotropic substance is present.

Note:- ⊕ denotes stimulation; ⊖ denotes inhibition

(c) Afterload.

→ Blood is ejected from the heart when pressure in right ventricle exceeds pressure in pulmonary trunk (20mmHg), and when pressure in left ventricle exceeds pressure in the aorta (80mmHg).

→ The higher pressure in the ventricles causes blood to push the semilunar valves open. The pressure that must be overcome before a semilunar valve opens is called afterload.

→ An increase in afterload causes stroke volume to decrease.

→ Conditions like hypertension, atherosclerosis can increase afterload.

(4) Conditions that affect HR/Regulation of HR

The heart rate is mainly controlled by cardiac innervation, sympathetic stimulation which increases the heart rate and parasympathetic stimulation which decreases the heart rate.

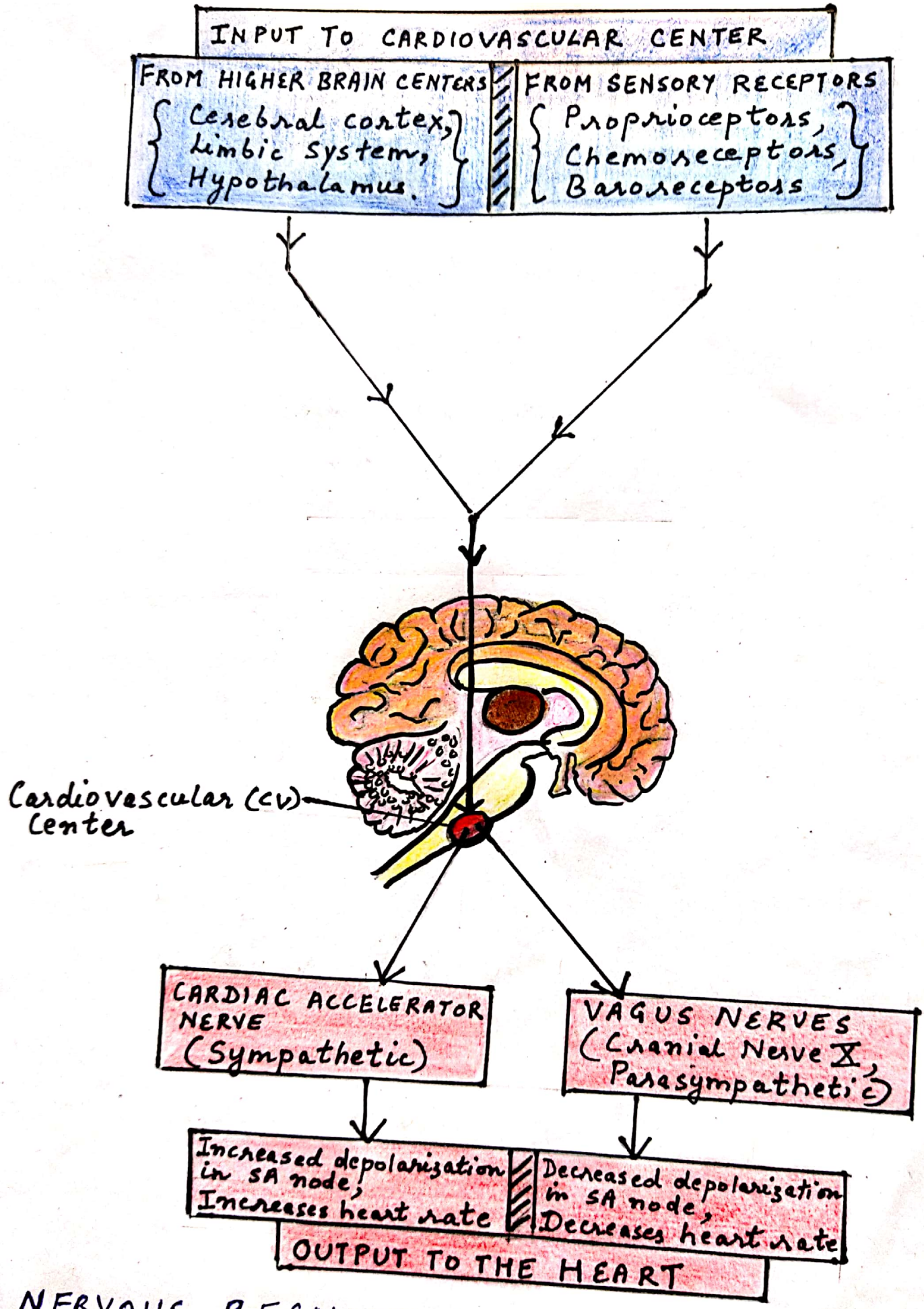
Many factors regulate the heart rate, the most important are as follows:

(a) Nervous regulation of the Heart Rate

→ Cardiovascular center in the medulla oblongata is responsible for nervous system regulation of the heart rate.

→ The cardiovascular center recieves inputs from higher brain centers (cerebral cortex, limbic system and hypothalamus) and from a variety of sensory receptors (proprioceptors, chemoreceptors and baroreceptors).

→ The cardiovascular centre then sends output to the heart via the
(i) Cardiac accelerator nerves (sympathetic) which increases depolarization in SA node and increases heart rate
(ii) Vagus nerves (cranial nerve X, parasympathetic) which decreases depolarization in SA node and decreases heart rate.



NERVOUS REGULATION OF THE HEART RATE

(b) Chemical Regulation of the Heart Rate

Various chemical substances affect the heart rate

(i) Oxygen

→ Low oxygen (hypoxia) stimulates heart rate
→ Very low oxygen inhibits heart rate
→ Prolonged low oxygen causes heart failure

(ii) Carbon dioxide

→ High CO_2 causes inhibition of heart rate (Hypercapnia)
→ Low blood CO_2 causes excitation of heart rate (Hypocapnia)

(iii) Transmitter substances and Hormones

→ Acetylcholine causes inhibition of heart rate
→ Norepinephrine and Epinephrine causes excitation of heart rate
→ Thyroxine increases heart rate

(iv) Inorganic ions.

→ K^+ causes inhibition of heart rate
→ Na^+ causes inhibition of heart rate
→ Ca^{2+} causes increase of heart rate
→ Excess Ca^{2+} stops heart in contracted state called "Calcium Rigour"

(V) Drugs.

→ Digitalis increases heart rate

→ Atropin increases heart rate

→ Muscarin inhibits heart rate

→ Nicotine inhibits initially and then increases the heart rate

(VI) Acidosis

→ Low pH decreases heart rate

(VII) Alkalosis

→ High pH decreases heart rate.

Note :-

Tachycardia : An abnormally rapid resting heart beat or elevated resting heart rate (over 100 beats per minute)

Bradycardia : A slow resting heart rate (under 50 beats per minute)

(c) Other factors regulating heart rate

(i) Age

Newborn baby has a high resting heart rate of over 120 beats/minute.

(ii) Gender

Adult females usually have a higher resting heart rate than adult males.

(iii) Physical fitness

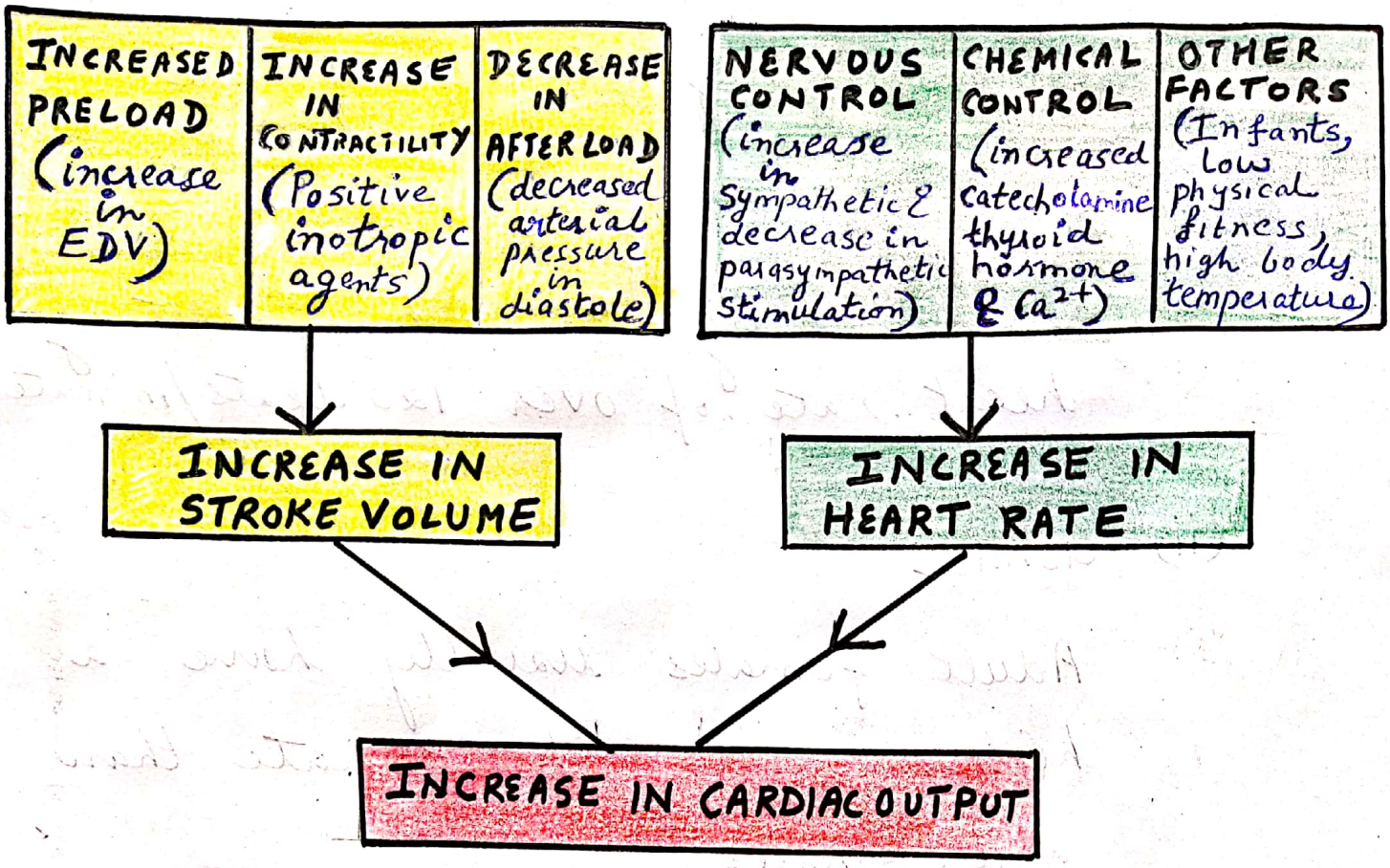
Exercise tends to bring down heart rate in both the sexes

(iv) Body temperature

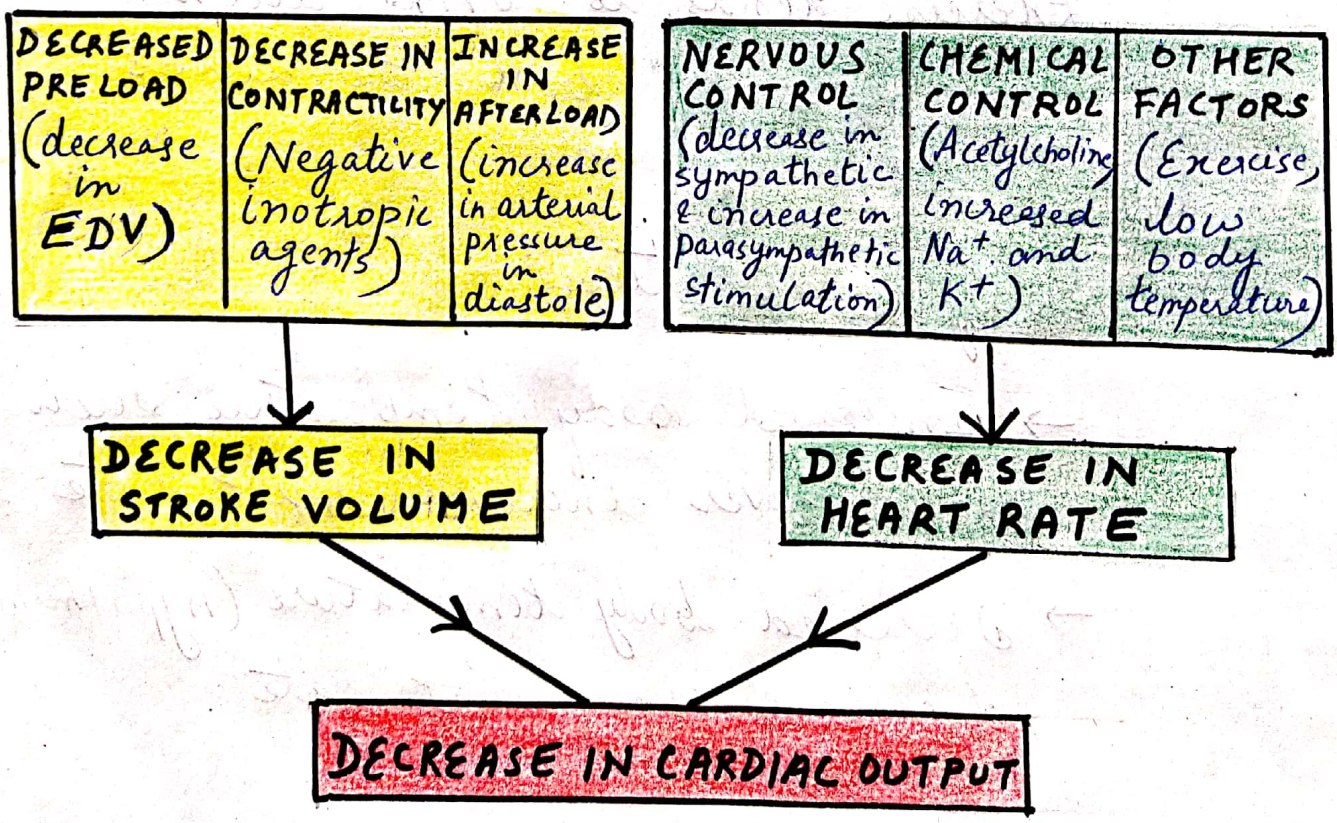
→ Increased body temperature such as in fever increases heart rate

→ Decreased body temperature (hypothermia) decreases the heart rate.

SUMMARY OF FACTORS THAT INCREASE CARDIAC OUTPUT



SUMMARY OF FACTORS THAT DECREASE CARDIAC OUTPUT



Note :-

→ Regulation of cardiac output as a result of changes in cardiac muscle fibre length is called heterometric regulation.

→ Regulation of cardiac output as a result of changes in contractility independent of length is called homometric regulation

References

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3. Vander A, Sherman J, and Luciano D (2014) Vander's Human Physiology : The mechanism of Body function. XIII Edition, Mc Graw Hills