

# **CARDIAC OUTPUT, VENOUS RETURN AND THEIR REGULATION.**

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- **OBJECTIVES**
- Define Stroke volume, Cardiac output Venous return ,& identify their normal values.
- Describe control (intrinsic & extrinsic) of Cardiac output & Venous return.
- Enumerate the causes of high & low cardiac output states.
- Cardiac Output

- **Def:** Volume of blood pumped out by each ventricle per minute ( i.e minute output/ventricle)
- **Normal value:** 5L / min. (i.e RV pumps 5L/min to lungs & same time LV pumps 5L/min to aorta )
- **Cardiac Index:** CO/Sq.mt.body surface area/min

Normal value: 3.5L/Sq.mt/min.

Significance of CI: It is a better parameter for comparing CO of the people of same age and sex.

- CO
- Distribution: at rest

Brain = 750 ml / min.

Heart = 250 ml / min.

Liver = 1500 ml / min.

5L /min

Kidneys =

1200ml/min



- Normal Values=70ml to 90ml.
- Stroke Volume =End Diastolic Volume-End Stroke volume.
- Venous Return.
- Def:- Volume of blood returning from systemic circulation to heart.
- Any factor/condition increases venous return increases End diastolic volume.
- Factors determining
- **Venous return**

- Ventricular compliance
- Duration of ventricular diastole
- Atrial systole.

**Factors determining venous return:**

- Blood volume
- Gravity
- Pressure gradient
- Negative intrapleural pressure
- Valves of veins
- Muscular pump

Increased venous return

EDV increases

Force of Contraction increases

Stroke volume increases

Cardiac output increases.

# • Factors determining Stroke Volume.

- Starling's law of heart
- Def: Force of contraction of myocardium is directly proportional to the initial length of muscle fiber within physiological limits.
- In heart : When EDV increases  
Stroke volume increases  
(EDV stretching of vent. Initial length FC )
- Starling's law

- Molecular basis
  
- When initial length of muscle is increased. The sarcomere length increases. Increased number of cross linkages are formed. force of contraction
- Importance of Starling's law in heart
  1. An increase in venous return to the heart increases ventricular EDV (preload) and the force of ventricular contraction, which enables the heart to eject the

additional blood that was returned to it. Conversely, a decrease in venous return leads to a decrease in SV by this mechanism.

- **Importance of Starling's law in heart**

2. To match the stroke volumes of two ventricles so exactly that neither a stagnation ( pulmonary edema ) nor total emptying of the pulmonary circulation can occur. As both of them are fatal

**Force – frequency relation:**

When frequency = HR increases, for initial few beats, the force of contraction increases due to increased  $Ca^{2+}$  availability ( Beneficial effect )



- HEART RATE

The rate at which heart beats / min.

Normal range: 60 - 80 beats/min.

< 60 bpm =

Bradycardia

> 100 bpm =

Tachycardia.

**Method of study:**

- 1.Examination of arterial pulse(eg Radial)
- 2.Auscultation of heart sounds by stethoscope.
- 3.ECG

- Factors determining HR
- Age :New borns, Old age increases.

- Sex :Slightly more in females than males.
- Body temperature, Posture, Emotions, Respiration it increases.
- Hormonal influences: - Adrenaline, NA, Thyroxine, Vasopressin.

- Factors determining HR
- Neuronal influences:- Central nervous system & Peripheral Nervous System
- Cardio Vascular reflexes:
  - - Baro receptor reflex

- - Chemoreceptor reflex
- - Bain-Bridge reflex
- - Cushing's reflex.
- -Bezold-Jarisch reflex.
- Influences from higher centers: -  
VMC, CIC,
- -  
Hypothalamus,  
- Limbic system
  
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- Heart Rate.
- Age Vs HR: Infants: 140 – 150 bpm.  
Adults: 60 – 80 bpm.

Decline in HR during aging is due to increase in vagal tone.

- BSA Vs HR:  $HR \propto 1/\sqrt{BSA}$ .
- Sex Vs HR: Females have higher HR than males of same age, due to: sympathetic tone / vagal tone / BSA / BP.
- Emotion & HR: Anger, Anxiety: HR ( Sympathetic tone)  
Shock, Grief: HR ( Vagal. Tone)
- BT & HR: for every rise in 1°C: HR by 20bpm.  
1°F: HR by 10 bpm.
- HR

- **Posture & HR:** Lying down to standing: HR due to Mary's law.
- **Respiration & HR:** HR increases during inspiration & HR decreases during expiration = **Sinus arrhythmia.** discharge of inspiratory center spill over vagal nucleus and cause inhibition vagal tone HR
- **Barometric pressure & HR:** Increase barometric pr. HR, High altitude: barometric pressure HR
- Factors affecting/modifying CO
- Age



- **Why Measure Cardiac Output?**
- The reasons for determining cardiac output are many, including: checking for arterial blockage, determining efficiency of the heart pump, determining mean vascular pressure, and diagnoses of various inlet impedance problems, including microstenosis, venous obstruction, atrial fibrillation, cardiac tamponade, ventricular non-



compliance, and  
extremely rapid heartrate.

- Methods of measurement CO
- 1. Fick's Principle.
- 2. Dilution technique.
  - - a. Dye dilution – T<sub>1824</sub>,  
Indocyanin green
  - - b. Thermo dilution – cold  
saline
  - - c. Isotope dilution – P<sub>32</sub>,  
I<sub>131</sub>
- 3. Ballistocardiography.
- 4. Echocardiography with dopler.
- 5. X ray cardiometry.
- 6. Electromagnetic flow meters ( in  
animals )



## Fick Principle

Def: Amount of sub ( x ) taken up or given out by an organ is equal to arterio-venous difference of that substance times blood flow.

$$Q_x = ( A_x - V_x ) \times \text{Blood flow.}$$

This can be modified as:

$$\text{Blood flow} = \frac{Q_x}{A_x - V_x} = \text{ml / min.}$$

- CO
- **BF of Rt.Vent. = BF of Lt.Vent. = CO**
- Hence Pulmonary blood flow is determined as follows:
  - Sub. = O<sub>2</sub>, Organ = Lungs.
  - Q<sub>x</sub> = 250 ml / min.
  - A<sub>x</sub> = 21ml / min ( Arterial O<sub>2</sub>%)

- $V_x = 16 \text{ ml / min}$  ( Pul.artery's blood sample is collected by cardiac catheterization )
- Now BF ( CO ) =  $250 / 21-16 \times 100 = 5\text{L / min.}$
- **Disadvantage:** As cardiac catheterization can not be done during exercise, CO during exercise can not be measured.
- Applied aspect of CO
- Low CO is seen in:
  - \* Myocardial Infarction
  - \* Congestive cardiac failure
  - \* Complete heart block
  - \* Atrial fibrillation
  - \* Pericardial effusion
- High CO is seen in:
  - \* Thyrotoxicosis
  - \* Cushing's syndrome
  - \* Anaemia.
- Objectives /summary

*At the end of this session, the student should be able to :*

- Define Cardiac Output, Stroke volume, Venous return with its normal values.
  - Describe the mechanism of regulation of Cardiac output.
  - Name the diseases in which Cardiac output increases & decreases.
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- References: Guyton & Hall, Text book of Physiology, 12<sup>th</sup> edition. Page 229-234.
  - Ganong, 24<sup>th</sup> edition.
  - Internet.

