



CARDIAC OUTPUT

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Definitions

- **Cardiac output (CO):**

Amount of blood pumped by each ventricle per minute.

- **Stroke volume (SV):**

Volume of blood ejected by each ventricle per beat

$$SV \times HR = CO$$

- Stroke volume is expressed in ml/beat and heart rate in beats/minute. Therefore, cardiac output is in ml/minute. Cardiac output may also be expressed in liters/minute.

$$SV = EDV - ESV$$

- **Cardiac Index (CI):**

The index is usually calculated using the following formula:

$$CI = CO / BSA = (SV \times HR) / BSA.$$

Definitions

End-diastolic volume (EDV):

Amount of blood remaining in the heart by the end of diastole.

140 mL

End-systolic volume (ESV):

Amount of blood remaining in the heart by the end of systole.

=70 mL

$SV = EDV - ESV$

Physiological variations

CO is increased by:

Exercise (up to 700%)

Eating (30%)

High environmental temperature

Pregnancy

Anxiety (50-100%)

Sympathomimetics, epinephrine

CO is decreased by:

Sitting or standing from lying position (20-30%)

Pathological variations

Increasing COP:

- Fever
- Hyperthyroidism
- Anemia

Decreasing COP:

- Hypothermia.
- Hypothyroidism
- Myocardial diseases e.g. infarction, failure...
- Rapid arrhythmias

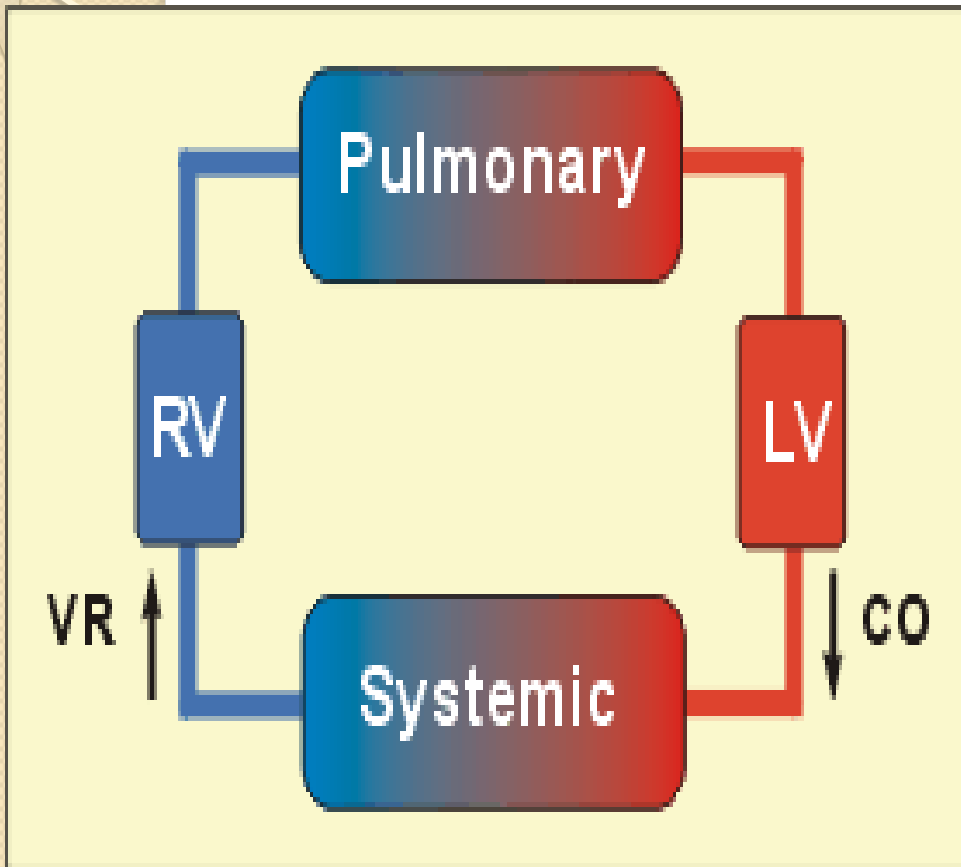
Factors determining CO

- Preload (VR)
- Afterload (Aortic pressure)

Definition,.....

Preload can be defined as the initial stretching of the cardiac myocytes prior to contraction.

I. Venous Return (Preload)



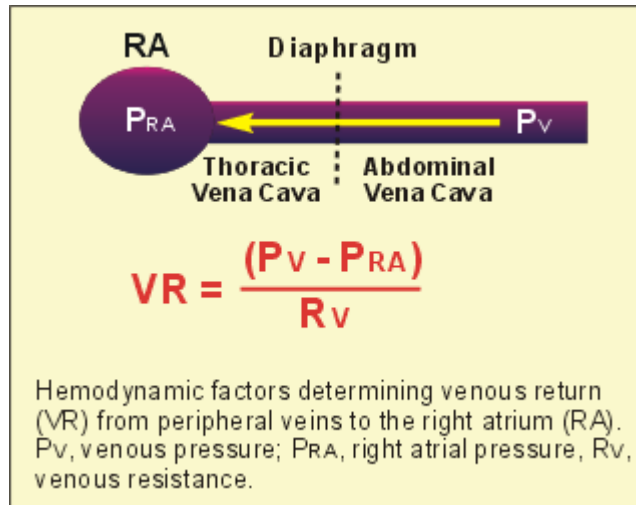
- Venous return (VR) is the flow of blood back to the heart. Under steady-state conditions, venous return must equal cardiac output (CO) when averaged over time because the cardiovascular system is essentially a closed loop. Otherwise, blood would accumulate in either the systemic or pulmonary circulations. Although cardiac output and venous return are interdependent, each can be independently regulated .

Venous return (Preload), continues,.....

The concept of preload can be applied to either the ventricles or atria. Regardless of the chamber, the preload is related to the chamber volume just prior to contraction.

Venous return (Preload), continues,.....

- Venous return (VR) to the heart from the venous vascular beds is determined by a pressure gradient (venous pressure), PV ‘minus right atrial pressure, (PRA).



Definition of Frank-Starling Law (Intrinsic regulation of CO)

- Ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return is called the **Frank-Starling mechanism**

Frank-Starling law states that the heart can pump all blood coming to it without allowing systemic venous stasis (within physiological limit).

Also, the force of cardiac contraction is directly proportional to the initial length of cardiac muscle (EDV), within physiological limits.

Starling Law,

- Increased venous return increases the ventricular filling) end-diastolic volume and therefore preload which is the initial stretching of the cardiac myocytes prior to contraction. This mechanism enables the heart to eject the additional venous return, thereby increasing stroke volume.

Factors affecting VR (Preload)

- **Muscle pump.:**

Rhythmical contraction of limb muscles as during normal locomotion (walking, running, swimming) promotes venous return by the muscle pump mechanism.

- **Sympathetic vasoconstrictor tone:**

Sympathetic activation of veins decreases increases central venous pressure and promotes venous return and augmenting cardiac output through the Frank-Starling mechanism, which increases the total blood flow through the circulatory system.

- **Respiratory activity.:**

During inspiration, the venous return increases because of a decrease in right atrial pressure.

Factors affecting VR (Preload)

- **Vena cava compression:**

when the thoracic vena cava becomes compressed during a Valsalva maneuver or during late pregnancy, decreases venous return.

Tissue Metabolism:

An increase in tissue metabolism as after meals, or in hyperthyroidism causes an increase in venous return.

- **Gravity :**

venous return decreases when changing from supine to erect posture..

Factors increasing PRELOAD:

- **Ventricular filling and therefore preload is increased by:**
- Increased central venous pressure that can result from sympathetic venoconstriction or increased thoracic blood volume . e.g., head-down tilt
- Increased ventricular volume which results in a greater expansion of the chamber during filling at a given filling pressure.
- Increased atrial force of contraction resulting from sympathetic stimulation of the atria or from increased filling of the atria and therefore increased atrial contractile force through **the Frank-Starling mechanism**.
- Reduced heart rate, which increases ventricular filling time
- Increased aortic pressure, which increases the **afterload** on the ventricle, reduces stroke volume by increasing **end-systolic volume** and leads to a secondary increase in preload.
- Pathological conditions such as **ventricular systolic failure** and valve defects such as **aortic stenosis** and aortic regurgitation, pulmonary valve stenosis and regurgitation have similar effects on right ventricular preload.

Factors decreasing preload:

- **Ventricular preload is decreased by:**
- Decreased venous blood pressure (most commonly resulting from reduced blood volume e.g., hemorrhage) or gravity causing blood to pool in the lower limbs when standing upright.
- Impaired atrial contraction that can result from atrial arrhythmias such as atrial fibrillation.
- Increased heart rate (e.g. atrial tachycardia (which reduces ventricular filling time).
- Decreased ventricular afterload, which enhances forward flow (i.e., ejection) thereby reducing end-systolic volume and end-diastolic volume secondarily.
- Ventricular diastolic failure (decreased ventricular compliance) caused, for example, by ventricular hypertrophy or impaired relaxation.
- Inflow (mitral and tricuspid)valve stenosis which reduces ventricular filling.

What are the factors affecting EDV:

Increase:

- Stronger atrial contraction.
- Increased total blood volume
- Increased venous tone.
- Increased skeletal muscle pump.
- Increased negative intrathoracic pressure.

Decrease:

Standing

Increased intrapericardial pressure.

Decreased ventricular compliance

Afterload

- Afterload can be thought of as the "load" that the heart must eject blood against. In simple terms, the afterload is closely related to the aortic pressure.

Afterload

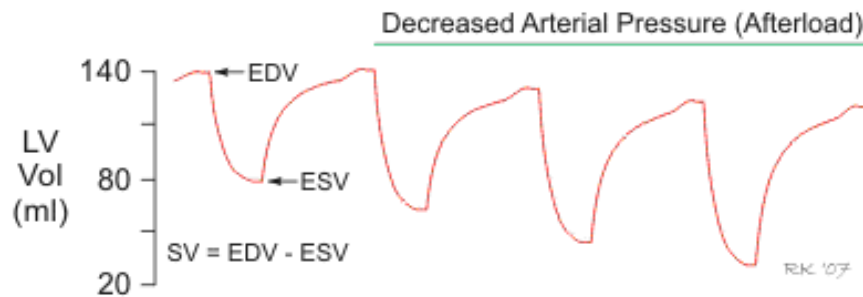


Figure 2. Effects of reducing arterial pressure (afterload) on ventricular volume changes in the beating heart. Decreased afterload decreases end-systolic volume (ESV), and to a lesser extent, end-diastolic volume (EDV). The net effect is an increase in stroke volume (SV).

- When arterial pressure is reduced, the ventricle can eject blood more rapidly, which increases the stroke volume and thereby decreases the end-systolic volume. Because less blood remains in the ventricle after systole, the ventricle will not fill to the same end-diastolic volume found before the afterload reduction. Therefore, in a sense, the end-diastolic volume (preload) is "pulled along" and reduced as end-systolic volume decreases. Stroke volume increases overall because the reduction in end-diastolic volume is less than the reduction in end-systolic volume .

Extrinsic Regulation of CO

1. Nervous:

- Sympathetic: ↑ HR & SV.
- Parasympathetic: ↓ HR

2. Chemical

- Potassium
- Calcium.
- Thyroxin.
- Catecholamine.

Factors affecting CO

- Venous return
- Heart rate
- Stroke volume
- Blood volume.
- ABP
- Neural factors

Measurement of CO

- **Echocardiographic** techniques and radionuclide imaging techniques can be used to estimate real-time changes in ventricular dimensions, thus computing stroke volume, which when multiplied by heart rate, gives cardiac output.
- **Fick Principle** :An old technique based on the can be used to compute cardiac output (CO) indirectly from whole body oxygen consumption (VO_2) and the mixed venous (O_{2ven}) and arterial oxygen contents (O_{2art}); however, this technique is seldom used. The CO is calculated as follows:
 - **$CO = VO_2 / (O_{2art} - O_{2ven})$**
 - To calculate CO, the oxygen contents of arterial and venous blood samples are measured, and at the same time, whole body oxygen consumption is measured by analyzing expired air. The blood contents of oxygen are expressed as ml O_2 /ml blood, and the VO_2 is expressed in units of ml O_2 /min. If O_{2art} and O_{2ven} contents are 0.2 ml and 0.15 ml O_2 /ml blood, respectively, and VO_2 is 250 ml O_2 /minute, then $CO = 5000$ ml/min, or 5 L/min. Ventricular stroke volume would simply be the cardiac output divided by the heart rate.