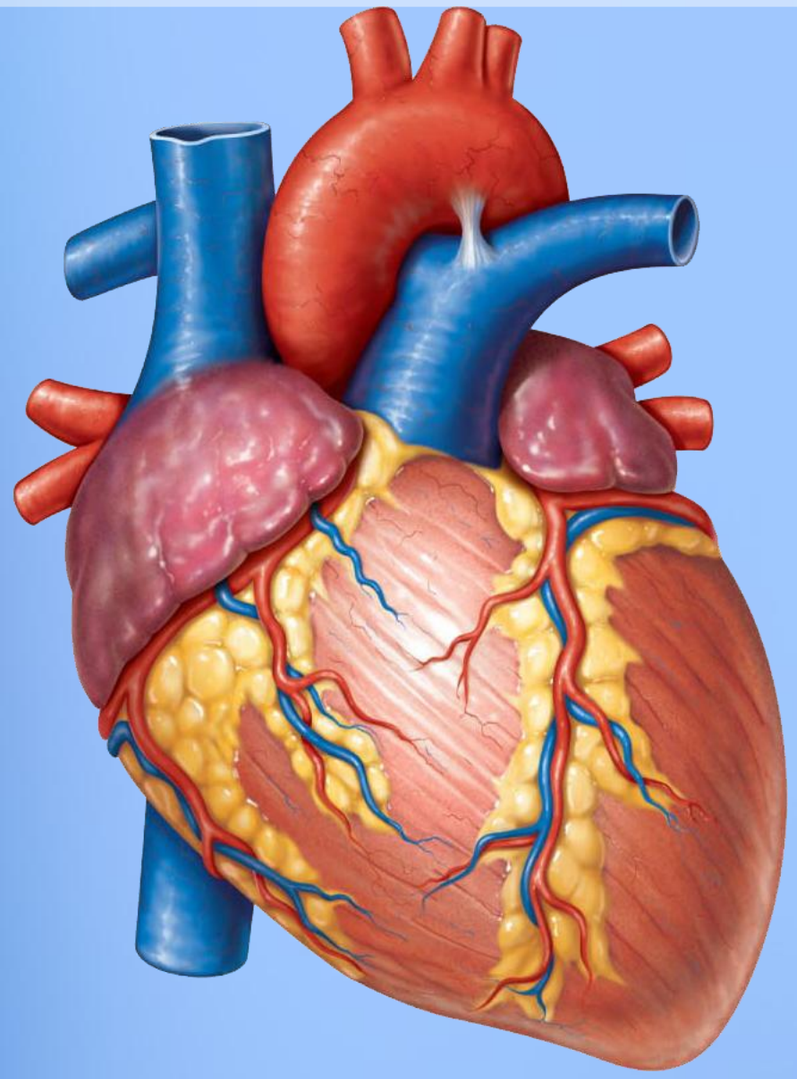
REGULATION OF STROKE VOLUME (PRELOAD, CONTRACTILITY &AFTERLOAD) &VENOUS RETURN & CARDIAC OUTPUT& STROKE VOLUME



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Cardiovascular Block

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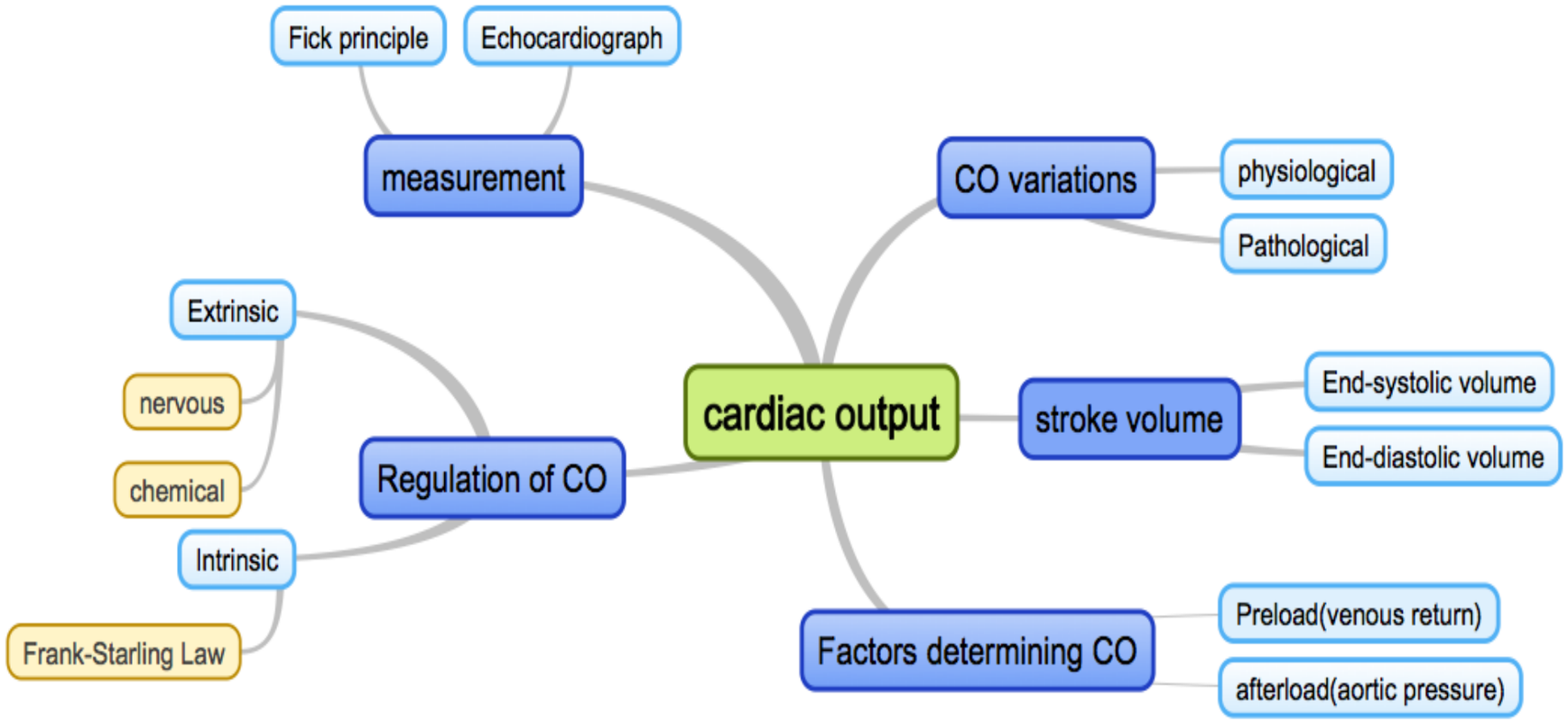


ABBREVIATION

CO : cardiac output
HR : heart rate
SV: Stroke volume
VR : venous return
HF: heart failure
BF : blood flow
AP: action potential
RV : right ventricle
LV : left ventricle
BSA: body surface area

Definitions

Cardiac output

- **Amount of blood pumped by each ventricle per minute.**
- Amount of blood pumped by RV = LV
- CO = 5 L/min ~ 4900ml/min

Cardiac Index(CI)

- The index is usually calculated using the following formula:
  - CI= CO/ BSA= (SV x HR)/BSA.
  - determine whether CO is enough for a person BSA

Stroke volume

- Volume of blood ejected by each ventricle per beat.
- **SV x HR = CO ex: 70ml/beat X 70beat/min = 4900 ml/min during rest heart**
- **SV = EDV-ESV ex: 140ml – 70ml = 70 ml/beat**

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**

NOTE:
*Structural properties are branched, gap junction …etc.
Physiological properties: excitability , conductivity, Contractility: ability to contract in response to stimulus.
*cardiac impulse: AP of the S-A node.
↑Force of contraction↑amount of blood pumped .
*Importance of blood remaining by the end of systole: supply the heart & make pressure difference

Physiological variations

Cardiac Output is increased by	Cardiac Output is decreased by
Exercise (up to 700%)	**Sitting or standing from lying position (20-30%)**
Eating (30%) The GIT active so need more blood	
High environmental temperature (increase in heat lead to vasodilatation blood vessel to evolve heat away)	
Pregnancy There is 2 circulation (mother & baby)	NOTE for the Pathological variations: Complete heart block so the impulses not go to the S-A node to the ventricle Then the ventricle will work by it's rate 25-40
Anxiety (50-100%) (sympathetic activity)	
Sympathomimitics, epinephrine	

Pathological variations

Fever Increase HR & force of contraction	**Hypothermia , Hypothyroidism**
Hyperthyroidism Excessive secretion of thyroxin ↑ HR& force of contraction	**Myocardial diseases** e.g. infarction, failure…
Anemia ↓ Oxygen carrying capacity ↑ blood flow, HR ,CO	**Rapid arrhythmias** ↑ HR ↓ filling of blood , ejection

Factors determining CO

1- Preload (Venous Return)

the initial stretching of the cardiac myocytes prior to contraction.
applied to either the ventricles or atria. Regardless of the chamber, the preload is related to the chamber volume just prior to contraction.

2- Afterload (Aortic pressure)

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

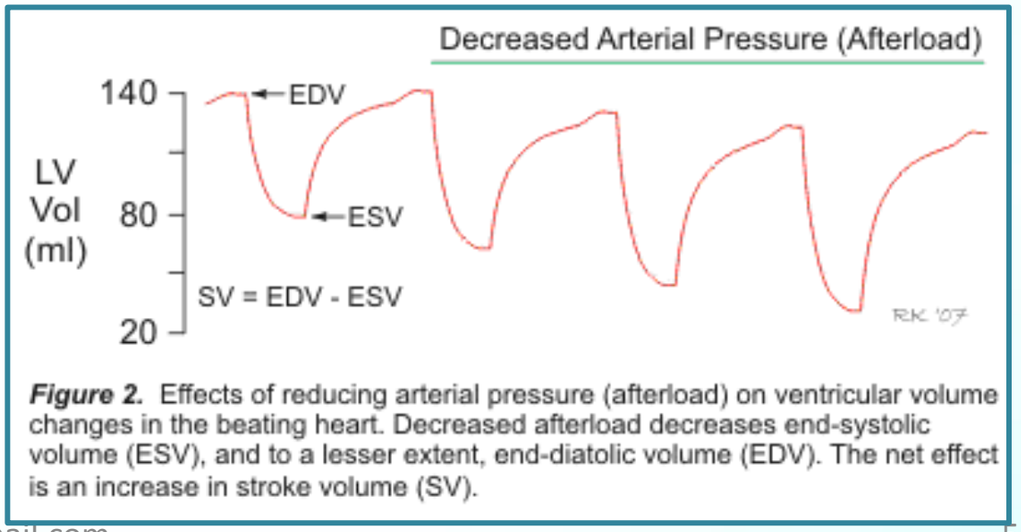
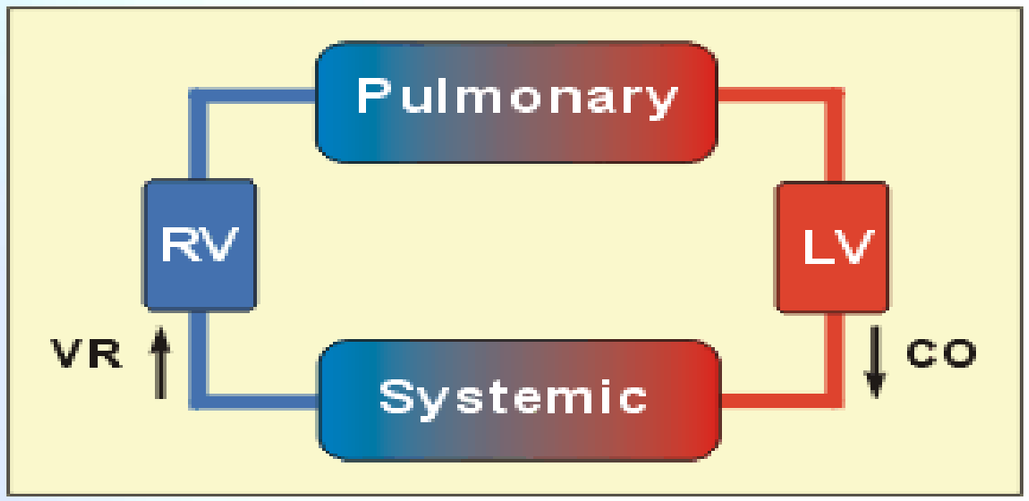


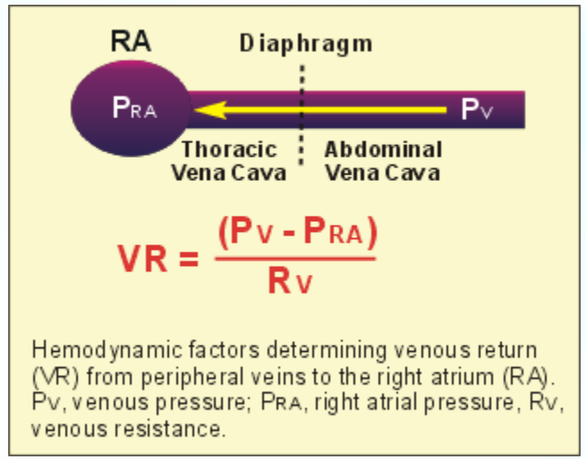
Figure 2. Effects of reducing arterial pressure (afterload) on ventricular volume changes in the beating heart. Decreased afterload decreases end-systolic volume (ESV), and to a lesser extent, end-diatolic volume (EDV). The net effect is an increase in stroke volume (SV).

1- venous return (preload)

- 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 (ex: RV defect but LV work effectively).

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).





Hemodynamic factors determining venous return (VR) from peripheral veins to the right atrium (RA). PV, venous pressure; PRA, right atrial pressure, RV, venous resistance.

*If RV not pumping amount of blood coming to it so it will stay in the circulation (stasis in systemic circulation) (systemic edema)
*If LV unable to pump blood so will accumulate and back to pulmonary (pulmonary edema)

*Venous sympathetic vasoconstriction tone make the different in pressure between right atrium and systemic circulation .
*Acute loss of sympathetic vasoconstriction tone → Vasodilation → no different in pressure → ↓VR → ↓CO → Shock

Frank-Starling Law
(Intrinsic regulation of CO)

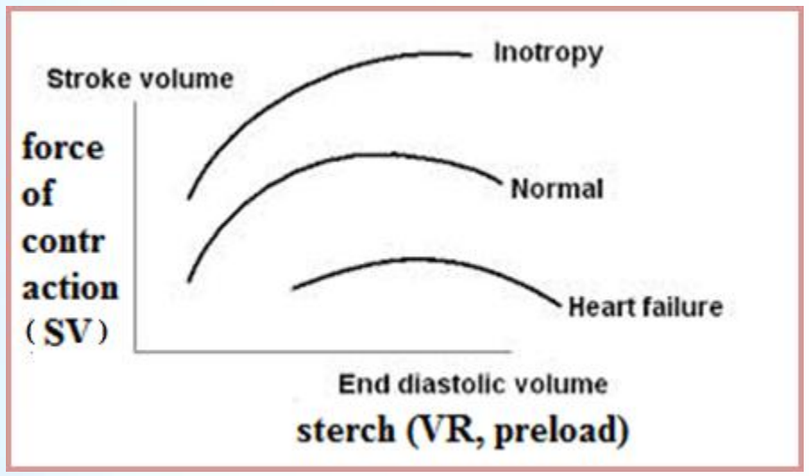
- Ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return is called the **Frank-Starling mechanism.**
- Frank-Starling law **states that the heart can pump all blood coming to it without allowing systemic venous stasis (within physiological limit).**

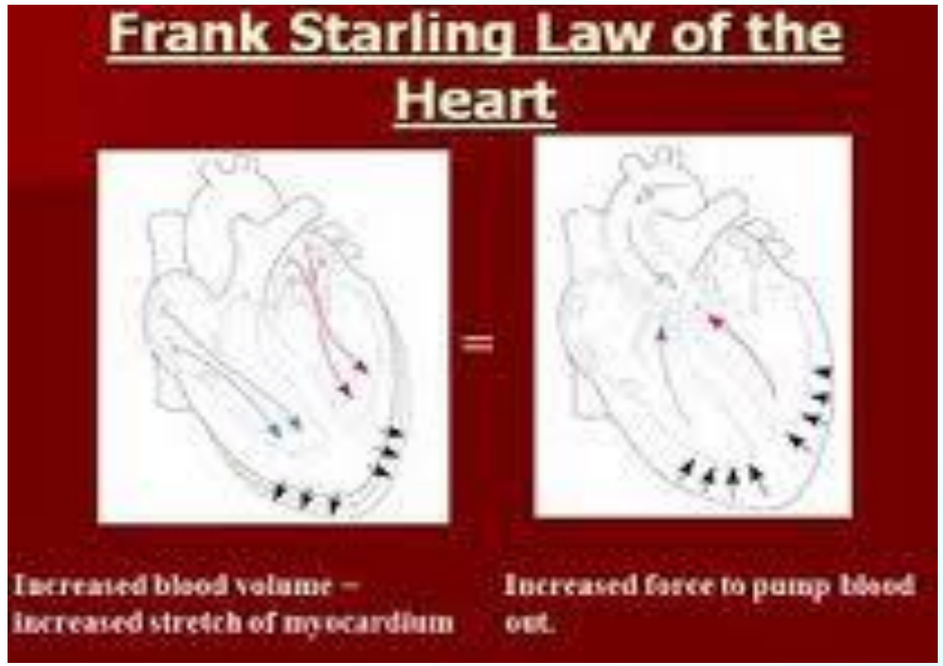
Also, the force of cardiac contraction is directly proportional to the initial length of cardiac muscle (EDV), within physiological limits.

*Heart work effectively so no stasis .

* Frank-Starling law: explain the how VR regulate CO &SV

↑ venous return ↑ the ventricular filling (end-diastolic volume → 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 stroke volume↑





Factors affecting VR (Preload) & CO

Volume:
ex: bleeding ↓blood volume↓CO
Ex: sever diarrhea→lose fluid ↓BV,VR,SV,CO

Gravity :
venous return **decreases** when changing from supine to erect posture..

Muscle pump.:
Rhythmical contraction of limb muscles as during normal locomotion (walking, running, swimming) **promotes** venous return by the muscle pump mechanism (push blood).
(muscle action squeezes veins & forces blood back to the heart)
20% of muscle contract during rest

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

Sympathetic vasoconstrictor tone:
Sympathetic activation of veins **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.

Tissue Metabolism:
An increase in tissue metabolism as after meals, or in hyperthyroidism causes an **increase** in venous return.
Ex; during exercise muscle contract so need more blood↑ VR, CO
hyperthyroidism→vasodilator substances→↑BF,VR

Respiratory activity.:
During inspiration, the venous return **increases** because of a **decrease** in **right atrial pressure**.
inspiration thoracic cavity **negative** ↑ suction force ↑VR
Expiration thoracic cavity **positive** ↓VR

Note : In Valsalva maneuvre (forceful expiration against a closed glottis) the intrapleural pressure become positive which is transmitted to the large veins in the chest → decrease venous return

FACTORS AFFECTING EDV SIMLAR TO VR

↑ EDV ↑ degree of stretch ↑ VR
Amount of blood remain in the heart depend on VR

-**Stronger atrial contraction.**

-**Increased total blood volume**

-**Increased venous tone.**

-**Increased skeletal muscle pump.**

-**Increased negative intrathoracic pressure.**

-**Standing**

-**Increased intrapericardial pressure.**

-**Decreased ventricular compliance**

Extrinsic Regulation of CO
Also regulation cardiac contractility:

1. Nervous:

A- Sympathetic: ↑ HR & SV & force of cont.
* Supply all the heart

B- Parasympathetic: ↓ HR
* Supply S-A & A-V node

2. Chemical:

A- Ions: -↑Potassium k ↓force of cont. ↓ SV,CO
But important in REPOLARZATION

-↑Calcium Ca ↑
force of cont. SV,CO

B- **Hormones**: **-Thyroxin↑HR & SV &CO**

-↑Catecholamine eg. Adrenaline
HR & SV &CO

Factors affecting CO
Venous return
Heart rate
Stroke volume
Blood volume
Arterial blood pressure (ABP)
Neural factors

Arrhythmia

Tachyarrhythmia:

Sever increase heart rate ↓ SV&CO

*Because the time of filling ventricle is shorted .

Ex: 350beat/ml x 10ml/beat = 3500ml/min

Bradyarrhythmia : (heart block)

Sever decrease heart rate ↑ SV ↓ CO

*longer time for ventricle filling but not enough to give normal CO .

Ex: 25beat/min x 100ml/beat =2500 ml/min

*normal CO = 4900 ml/beat ~ 5L/min

Measurement of CO

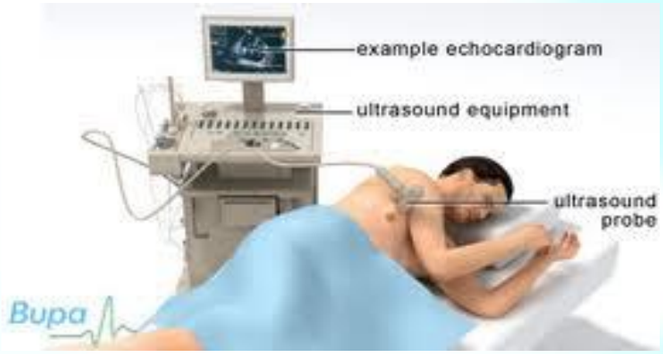
1- Echocardiographic :

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**.

SV x HR = CO

Ejection fraction= SV/EDV X 100 *↑EF so good heart

***↓EF so heart failure because ↓ SV & ↑ EDV**



2- Fick Principle :

An old technique used to **compute cardiac output (CO)** indirectly from whole **body oxygen consumption (VO2) and the mixed venous (O2ven) and arterial oxygen contents (O2art);** however, this technique is seldom used in clinical practice these days.

The CO is calculated as follows:

$$CO = VO2/(O2art - O2ven)$$

From any artery from R atrium

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 O2/ml blood, and the VO2 is expressed in units of ml O2/min.

If O2art and O2ven contents are 0.2 ml and 0.15 ml O2/ml blood, respectively, and VO2 is 250 ml O2/minute, then CO = 5000 ml/min, or 5 L/min. Ventricular stroke volume would simply be the cardiac output divided by the heart rate.

Stroke Volume

Systolic function of the heart is governed by:

- Contractile state of the myocardium.
- Preload of the ventricle.
- Afterload applied to the ventricle.
- Heart Rate.

*The contractility of the myocardium influence on SV.
*Changes in heart rate and rhythm affect myocardial contractility.
* Contractility is increased in response to sympathetic stimulation and this is reflected by shifting the pressure volume-loop upward and to the left (positive inotropic effect).

Remember That :

Preload of the ventricle

- increase in venous return to the heart, increases the filled volume (EDV) of the ventricle

Afterload applied to the ventricle.

(Aortic pressure)

- related to the pressure that the ventricle generate to eject blood into the aorta.

during rest :

CO = 5 L/min ~4900ml/min

HR = 70 beat/min

SV =70 ml/beat

ESV = 120-140 ml

EDV = 50-70 m

Question

When a person moves quickly from a lying to a standing position , which one of following decrease?

A. Venous return
B. Cardiac output
C. all of them

What is the name of the volume contained in the left ventricle immediately before it contract ?

A. end-diastolic volume
B. End-systolic volume

Which one of following cause an increase in stroke volume from the left ventricle?

A. Increased contractility ➡ EDV is preload and aortic pressure is afterload
B. Decreased in end-diastolic volume
C. Increased in aortic pressure

VR Must be

A. equal CO
B. Less than CO
C. More than CO

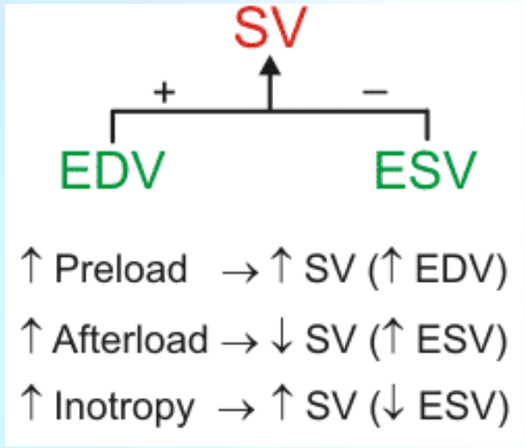
VR decrease During

A. Expiration
B. Inspiration
C. both

Watch the videos then click "quiz" ☺

http://education-portal.com/academy/lesson/heart-rate-cardiac-output-stroke-volume.html#lesson
http://education-portal.com/academy/lesson/heart-rate-and-blood-flow-regulation.html#lesson

SUMMARY



Cardiac output

Preload(VR)

The blood enter the heart either by pulmonary veins to the left atrium or superior & inferior vena cava to the right atrium

↑Preload↑ force of contraction↑ CO

Important to determine the degree of ventricle stretch before systole

Afterload

(aortic pressure)

resistance against which the heart has to overcome before systole

↑Aortic pressure

↑resistance to the ventricle to eject the blood

So the ↑ ventricle the contraction pressure to open the aortic valve

Regulation of CO

Extrinsic

↑Increase
*Sympathetic
*CA& thyroxin
*Catecholamine

↓Decrease
*Parasympathetic
*Potassium

Intrinsic

frank starling law.
TO prevents venous stasis.

* The blood enters the heart as **venous return** and leaves it as **cardiac output** meaning both amounts has to be **equal**.
* VR regulate CO through **frank starling law mechanism** when VR increases it will lead to a degree of stretch of ventricle so this will increase the force of contraction therefore CO increase .

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Good luck