

BIO 2402 - Human Anatomy & Physiology

Week 7

Hi everyone! I hope your first exam went well! This week we are going to be wrapping up chapter twenty four on the heart. We will be looking at factors that affect heart rate and blood pressure. If you have any questions please let me know!

Remember that the Tutoring Center offers free individual and group tutoring for this class. Our Group Tutoring sessions will be every Wednesday from 6:00-7:00 PM CST. You can reserve a spot at <https://baylor.edu/tutoring>.

KEY TERMS: Chronotropic, Inotropic, Stroke Volume, EDV, Preload, Contractility, Afterload

We have talked about the mechanical workings of cardiac muscle as well as the EKG so far, now we are going to take a look at what the blood in the heart is doing during contraction and relaxation.

Cardiac Output: ml/min of blood, OR $CO = HR \times SV$

Venous Return: volume of blood returning to the atria

Chronotropic factors are factors that affect the rate at which the cardiac muscle fibers contract.

Positive chronotropic effect: increases HR

Negative chronotropic effect: decreases HR

Effects of autonomic stimulation

- Cardiac nerves release NE that excites nodes and contractile cells, resulting in faster and stronger heart beats; *positive*
- Vagus nerves release ACh that inhibits nodes, resulting in slower heart beat; responsible for vagal tone; *negative*

Effects of Hormones

- Epinephrine (E), norepinephrine (NE), and thyroid hormone (T_3); *positive*

Effect of Venous return: *positive*

Inotropic factors are factors that affect the amount of force that the cardiac fibers contract with.

Positive inotropic factors (ino-, fiber; tropic-, turning)

Increases SV (decreases ESV -- end-systolic volume)

Negative inotropic factors decreases SV (increases ESV)

Stroke Volume: the amount of blood the heart ejects in one cycle

$$EDV - ESV = SV$$

Factors Affecting Stroke Volume (SV)			
	Preload	Contractility	Afterload
Raised due to:	<ul style="list-style-type: none"> fast filling time increased venous return <p>Increases end diastolic volume, Increases stroke volume</p>	<ul style="list-style-type: none"> sympathetic stimulation epinephrine and norepinephrine high intracellular calcium ions high blood calcium level thyroid hormones glucagon <p>Decreases end systolic volume, Increases stroke volume</p>	<ul style="list-style-type: none"> increased vascular resistance semilunar valve damage <p>Increases end systolic volume, Decreases stroke volume</p>
Lowered due to:	<ul style="list-style-type: none"> decreased thyroid hormones decreased calcium ions high or low potassium ions high or low sodium low body temperature hypoxia abnormal pH balance drugs (i.e., calcium channel blockers) <p>Decreases end diastolic volume, Decreases stroke volume</p>	<ul style="list-style-type: none"> parasympathetic stimulation acetylcholine hypoxia hyperkalemia <p>Increases end systolic volume, Decreases stroke volume</p>	<ul style="list-style-type: none"> decreased vascular resistance <p>Decreases end systolic volume, Increases stroke volume</p>

https://upload.wikimedia.org/wikipedia/commons/thumb/c/c0/2035_Factors_in_Stroke_Volume.jpg/

SV is positively correlated to:

- **EDV** is positively correlated with filling time (duration of ventricular diastole)

- Filling time is negatively correlated with heart rate

- **Preload** (amount of ventricle stretch prior to systole)

- Preload is positively correlated to EDV

- Frank-Starling

principle states that the greater the stretch of the cardiac muscle tissue, the stronger the contraction of the muscle fibers. This phenomenon, pictured above, is due to the fact that the more stretch a fiber experiences the better the overlap between thick and thin myofilaments.

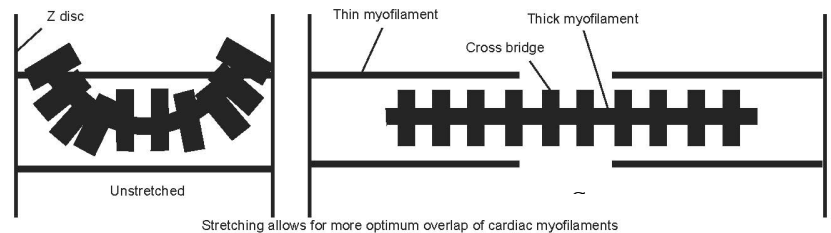


Figure 4-10. The Frank-Starling principle

- **Contractility**: amount of force generated during contraction

- *Positive inotropic factors* increase contractility;
- *negative inotropic factors* decrease contractility

- **Afterload**: amount of tension the ventricle must exert to open a semilunar valve

- The greater the afterload, the longer the period of isovolumetric contraction, the shorter the duration of ventricle contraction, and the greater the ESV

The graph below shows the relationship between the different factors that can effect CO from the heart.

1. As venous return increases, preload increases
2. As preload increases, contractility increases
3. As contractility increases, SV increases
4. As SV increases, ESV decreases
5. As ESV decreases, CO increases

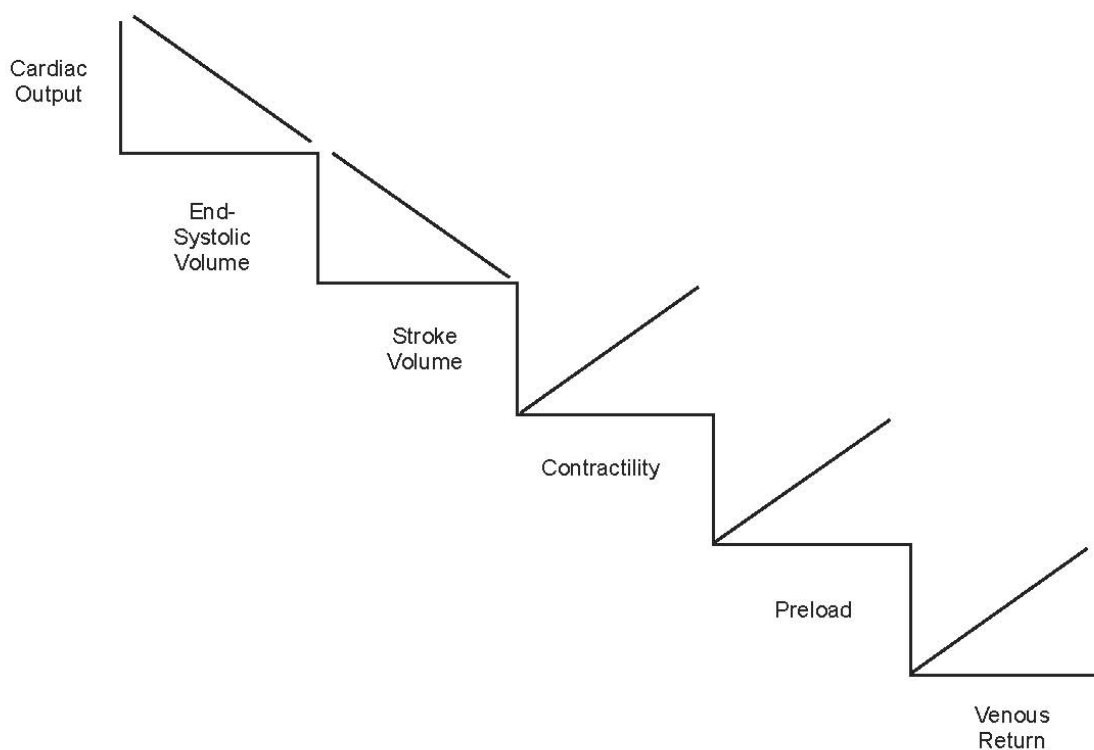


Figure 4-10. Relational graphs for selected heart variables