

The cardiovascular system

Cardiac output (cont'd)

Relation of tension to length in cardiac muscle

When the muscle is stretched, the developed tension increases to a maximum and then declines as stretch becomes more extreme. Starling's law of the heart or Frank-Starling's law stated that the "energy of contraction is proportional to the initial length of the cardiac muscle fiber". For the heart, the length of the muscle fibers (i.e., the extent of the preload) is proportional to the end-diastolic volume. The relation between ventricular stroke volume and end-diastolic volume is called the Frank-Starling curve.

Basically, this law states that when increased quantities of blood flow into the heart, the increased blood stretches the walls of the heart chambers. As a result of the stretch, the cardiac muscle contracts with increased force, and this action empties the extra blood that has entered from the systemic circulation. Therefore, the blood that flows into the heart is automatically pumped without delay into the aorta and flows again through the circulation.

Another important factor is that stretching the heart causes the heart to pump faster, resulting in an increased heart rate. That is, stretch of the sinus node in the wall of the right atrium has a direct effect on the rhythmicity of the node to increase the heart rate as much as 10-15%.

Factors affecting end-diastolic volume

Alterations in systolic and diastolic function have different effects on the heart. When systolic contractions are reduced, there is a primary reduction in stroke volume, which in turn reduces COP. Diastolic function also affects stroke volume in the following ways:

Factors decreasing diastolic ventricular filling

1. Increased intra-pericardial pressure due to infection or tumor.
2. Increased ventricular stiffness produced by myocardial infarction, infiltrative disease, and other abnormalities.
3. Decreased normal negative intra-thoracic pressure will decrease venous return to the heart.
4. Standing also decreases venous return.

Factors increasing diastolic ventricular filling

1. Increased total blood volume increases venous return.
2. Constriction of the veins reduces the size of the venous reservoirs, decreasing venous pooling and thus increasing venous return.
3. Increased normal negative intra-thoracic pressure will increase venous return to the heart.
4. Increased skeletal muscular activity will increase venous return.

Myocardial contractility

It is the intrinsic ability of the cardiac muscle to develop force at a given muscle length, also known as inotropism. It exerts a major influence on stroke volume. Factors affecting contractility are as follows:

Factors increasing contractility (positive inotropism)

1. Sympathetic nervous stimulation to the heart via β_1 adrenergic receptors.
2. Circulating catecholamine.
3. Ventricular extra-systoles make the myocardium contract in such a way that the next succeeding contraction is stronger than the preceding normal contraction. This post-extra-systolic potentiation is independent of ventricular filling.
4. Heart rate: myocardial contractility increases as the heart rate increases, although this effect is relatively small.
5. Xanthines such as caffeine and theophylline.
6. Digoxin, a drug used for heart failure and other medical conditions.

Factors decreasing contractility (negative inotropism)

1. Parasympathetic stimulation (to a lesser extent).
2. Heart failure.
3. Hypercapnia, hypoxia and acidosis.
4. Drugs such as quinidine, procainamide and barbiturates.

Cardiac rate

It is defined as the number of ventricular contractions per minute. Normal heart rate in a young adult averages about 60-100 BPM. Cardiac pacemaker or the SA node can generate action potential with a rate of about 70-80 pulses/ minute. When SA node can't generate an

action potential, AV node does at a rate of about 40-60 pulses/ minute, and Purkinje fibers can do so at a rate of 15-40 pulses/ minute.

- **Normocardia:** It is the normal heart rate, about 60-100 BPM.
- **Tachycardia:** Fast heart rate, more than 100 BPM.
- **Bradycardia:** Slow heart rate, less than 60 BPM.

Factors increasing the heart rate (positive chronotropism)

1. Sympathetic stimulation.
2. Circulating catecholamine.
3. Decreased activity of baroreceptors.
4. Inspiration.
5. Excitement.
6. Anger.
7. Painful stimuli.
8. Hypoxia.
9. Exercise.
10. Thyroid hormones.
11. Fever.

Factors decreasing the heart rate (negative chronotropism)

1. Parasympathetic stimulation.
2. Increased activity of baroreceptors.
3. Expiration.
4. Fear.
5. Grief.
6. Increased intracranial pressure.

Athlete's heart

Athletic heart syndrome (AHS), also known as athlete's heart, athletic bradycardia, or exercise-induced cardiomegaly is a non-pathological condition commonly seen in sports medicine, in which the human heart is enlarged, and the resting heart rate is lower than normal. A larger heart results in higher cardiac output, which also allows it to beat more slowly, as more blood is pumped out with each beat. Sinus bradycardia in athletes is due to the heart adapting to the physical stresses that it is put under by the athlete's physical activity. This causes the heart to become more efficient producing a greater stroke volume, which in return allows the heart to circulate the same amount of blood with fewer contractions.