الفسلجة
مرحلة ثانية

الأستاذ المساعد الدكتور
خالد سعود صالح

Cardoivascular System
CARDIAC OUTPUT

Heart Rate

Cardiac rate is controlled primarily by the autonomic nerves, with:

sympathetic stimulation increasing the rate.

Stroke volume is also determined in part by neural input, with:

sympathetic stimuli making the myocardial muscle fibers contract with greater strength at a given length.

When the strength of contraction increases without an increase in fiber length:

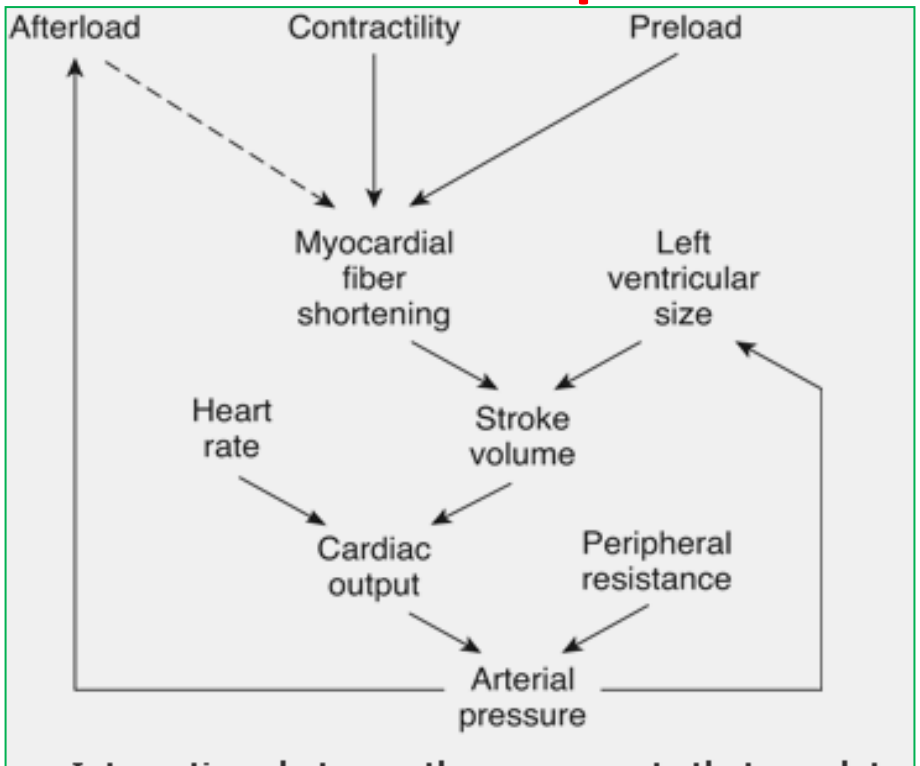
More blood is expelled from the ventricles ; that is, the ejection fraction increases.

parasympathetic stimuli having the opposite effect.

parasympathetic stimulation decreasing it.

The cardiac accelerator action of the catecholamines liberated by sympathetic stimulation is referred to as their **chronotropic action.**

whereas their effect on the strength of cardiac contraction is called their **inotropic action**.



Interactions between the components that regulate cardiac output and arterial pressure.

Regulation of Stroke Volume

1. The end-diastolic volume (EDV), which is the volume of blood in the ventricles at the end of diastole;
2. the total peripheral resistance, which is the frictional
resistance, or impedance to blood flow, in the arteries; and
3. the contractility, or strength, of ventricular contraction.

The end-diastolic volume

is the amount of blood in the ventricles immediately before they begin to contract. This is a workload imposed on the ventricles prior to contraction, and thus is sometimes called a preload.

The stroke volume is directly proportional to the preload; an increase in EDV results in an increase in stroke volume. (This relationship is known as the *Frank-Starling law of the heart,* discussed shortly.) The stroke volume is also directly proportional to contractility; when the ventricles contract more forcefully, they pump more blood.

The pressure in the arterial system before the ventricle contracts is, in turn, a function of the total peripheral resistance.
the higher the peripheral resistance, the higher the pressure. As blood begins to be ejected from the ventricle, the added volume of blood in the arteries causes a rise in mean arterial pressure against the “bottleneck” presented by the peripheral resistance; ejection of blood stops shortly after the aortic pressure becomes equal to the intraventricular pressure. The total peripheral resistance thus presents an impedance to the ejection of blood from the ventricle, or an afterload imposed on the ventricle after contraction has begun.

the stroke volume is inversely proportional to the total peripheral resistance; the greater the peripheral resistance, the lower the stroke volume.

The proportion of the end-diastolic volume that is ejected against a given afterload depends on the strength of ventricular contraction. Normally, contraction strength is sufficient to eject 70 to 80 ml of blood out of a total end diastolic volume of 110 to 130 ml.

The **ejection fraction** is thus about 60%. More blood is pumped per beat as the EDV increases, and thus the ejection fraction remains relatively constant over a range of end-diastolic volumes. In order for this to be true, the strength of ventricular contraction must increase as the end-diastolic volume increases.

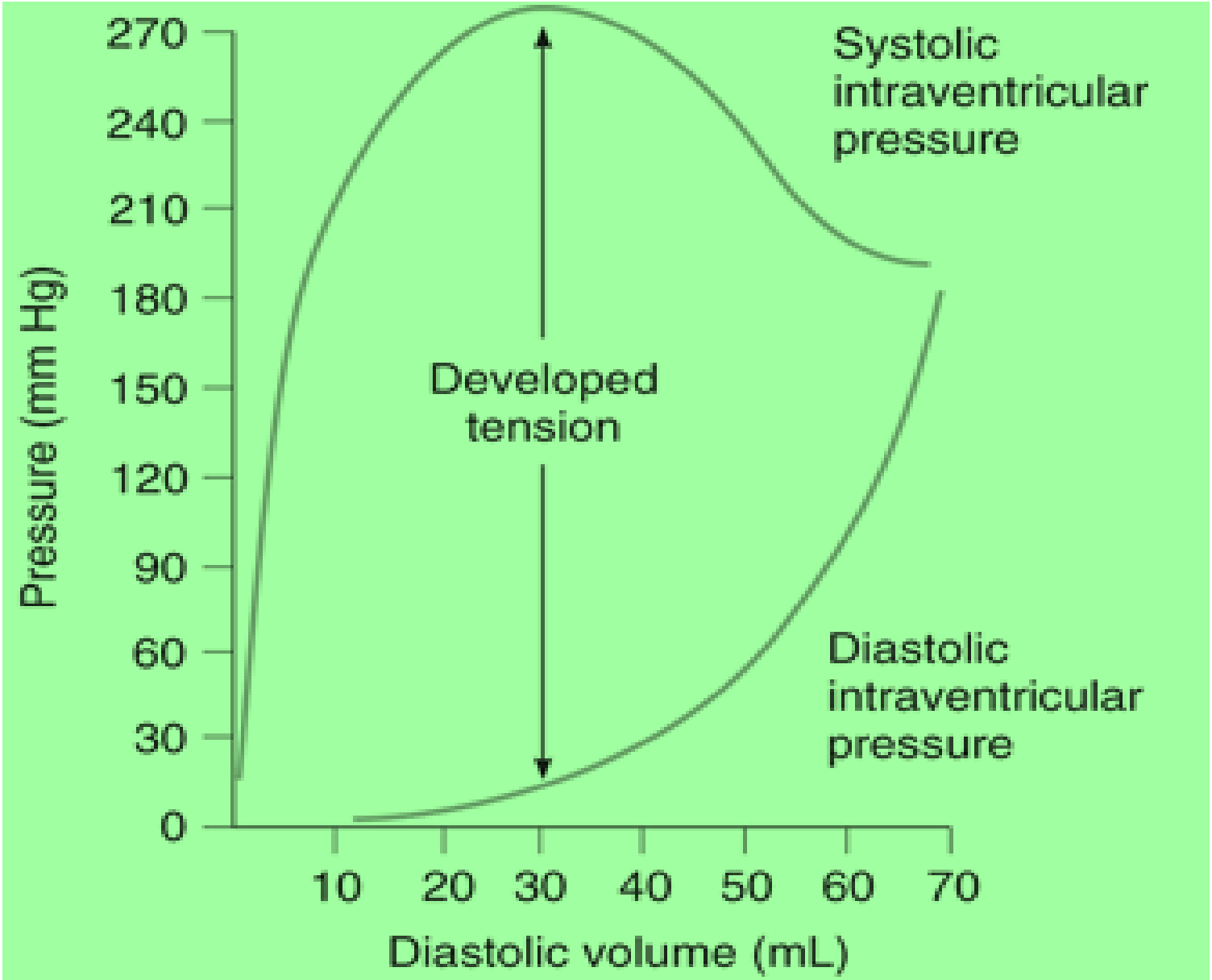
Relation of Tension to Length in Cardiac Muscle: (the Frank–Starling law)

The length–tension relationship is similar to that in skeletal muscle;

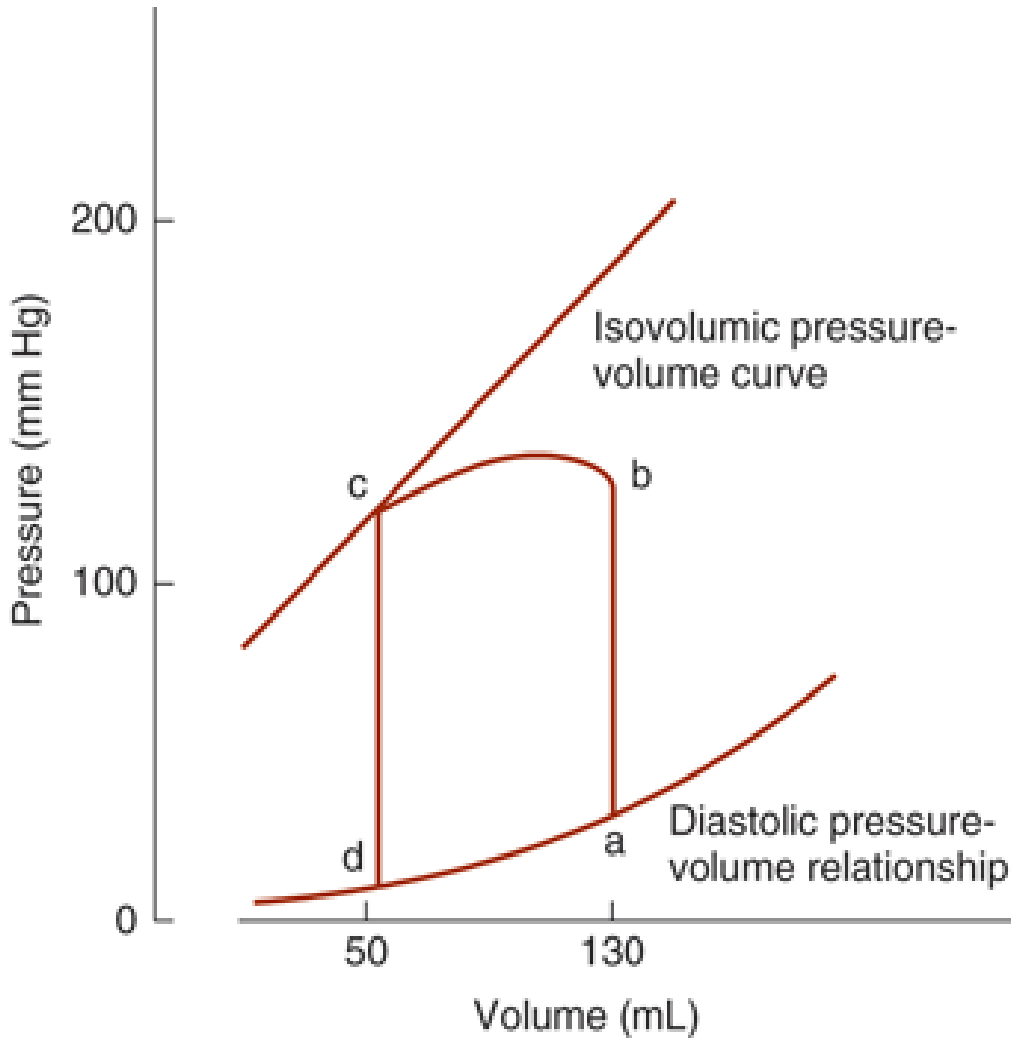
as the muscle is stretched, the developed tension increases to a maximum and then declines as stretch becomes more extreme.

Starling pointed this out stating that the "energy of contraction is proportional to the initial length of the cardiac muscle fiber " (*Starling's law of the heart*) or (*Frank–Starling law*).

The strength of ventricular contraction varies directly with the end-diastolic volume. Even in experiments where the heart is removed from the body (and is thus not subject to neural or hormonal regulation) and where the still-beating heart is filled with blood flowing from a reservoir, an increase in EDV within the physiological range results in increased contraction strength and, therefore, in increased stroke volume. This relationship between EDV, contraction strength, and stroke volume is thus a built in, or *intrinsic,* property of heart muscle, and is known as the **Frank-Starling law of the heart.**



Length–tension relationship for cardiac muscle.

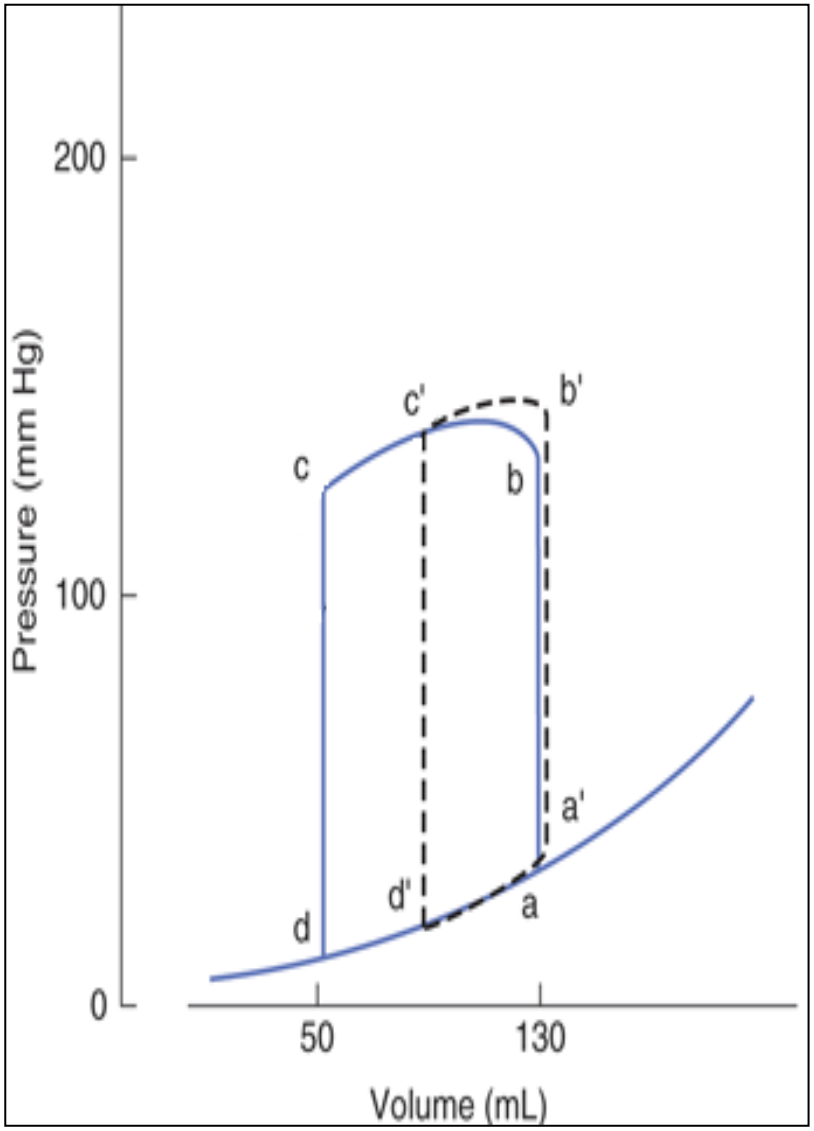

Pressure (mm Hg)
200
100
0
Isovolumic pressure-volume curve
c
b
d
a
Diastolic pressure-volume relationship
50
130
Volume (mL)

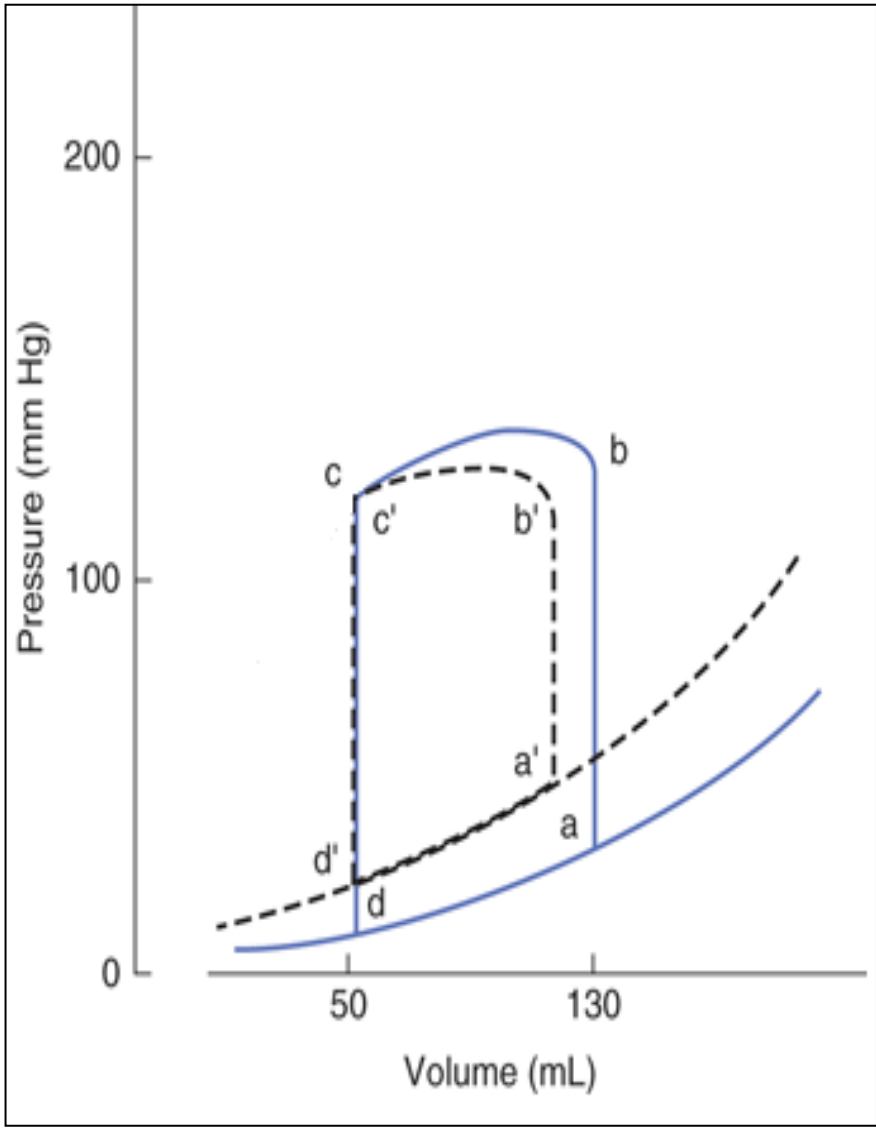
Factors Affecting End-Diastolic Volume

1. increase in intrapericardial pressure limits the extent to which the ventricle can fill (eg, as a result of infection or pressure from a tumor), as does a decrease in ventricular compliance; that is, an increase in ventricular stiffness produced by myocardial infarction, infiltrative disease, and other abnormalities.
2. Atrial contractions aid ventricular filling.

3. Factors affecting the amount of blood returning to the heart likewise influence the degree of cardiac filling during diastole.

a. An increase in total blood volume increases venous return
b. Constriction of the veins reduces the size of the venous reservoirs, decreasing venous pooling and thus increasing venous return.
c. An increase in the normal negative intrathoracic pressure increases the pressure gradient along which blood flows to the heart, whereas a decrease impedes venous return. Standing decreases venous return.
d. muscular activity increases it as a result of the pumping action of skeletal muscle.


200
100
0
Pressure (mm Hg)
c
c'
b'
b
a'
d
d'
a
50
130
Volume (mL)


200
100
0
Pressure (mm Hg)
c
c'
b'
b
a'
a
d'
d
50
130
Volume (mL)