# The Cardiac Output And Its Regulation I & II

Dr. Waleed R. Ezzat

# Lecture Objectives:

1. Define the cardiac output and cardiac index.
2. Describe the relationship between cardiac output, stroke volume and the heart rate.
3. Describe how stimulation of the sympathetic and parasympathetic affect the cardiac output.
4. Explain the relationship between EDV and ventricular performance (Frank-Starling curve).
5. Describe factors affecting EDV and define cardiac reserve.
6. Describe the effect of autonomic nervous system, and ions on the pumping activity of the heart.
7. Describe the energy expended and $O_2$ utilization by the heart.
8. Understand methods of determination of cardiac output.

# *The Cardiac Output (CO)*

- (def.) The CO is the volume of blood pumped by each ventricle per minute. It is ***not*** the total amount of blood pumped by the heart.
- The CO is about 5-6 L/min at rest. As blood volume is about 5-6 L, hence the whole blood volume circulates in ***one*** minute.
- During exercise the CO can increase to 20-25 L/min and even to as high as 40 L/min in well-trained athletes.
- The CO is ***equal*** to aortic blood flow, venous return, or pulmonary blood flow.
- **CO = SV X HR**

- **The Stroke Volume (SV):** the amount of blood pumped out of each ventricle per beat. It is about 70 ml in a resting man in supine position

  **SV = EDV-ESV**

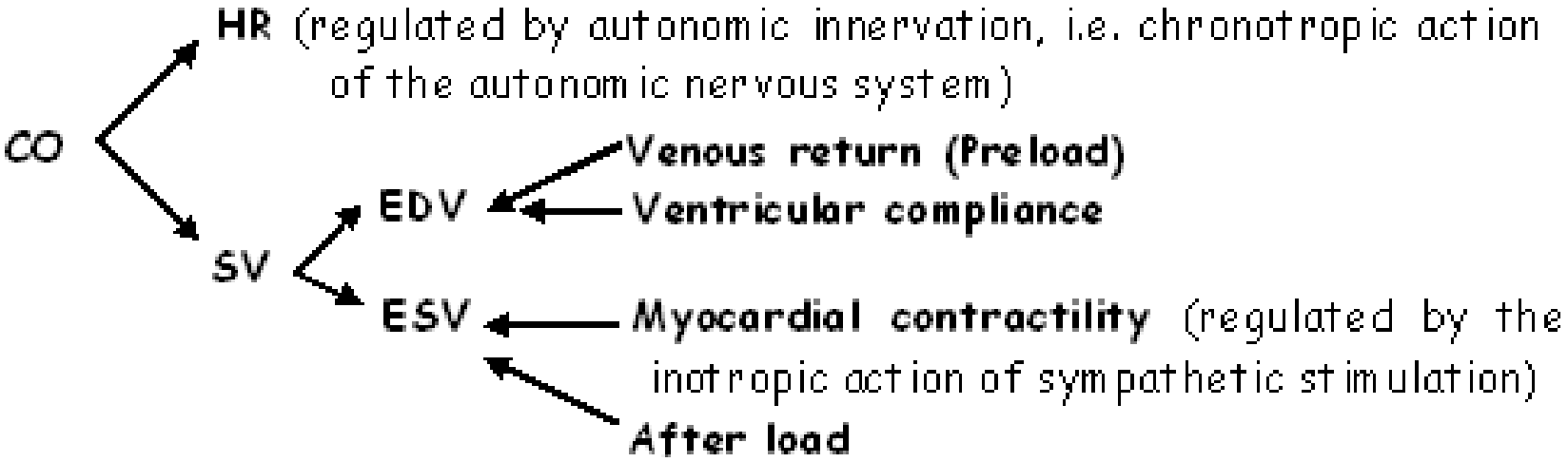
- **The Cardiac Reserve:** the difference between the cardiac output at rest and the maximal volume of blood the heart is capable of pumping per minute

- **The Cardiac Index (CI):** It is the output per minute per square meter of body surface area. CI is about 3.2 L/min.$m^2$

- **The Ejection Fraction % (EF %):** the percent of the end diastolic volume that is ejected with each stroke (**EF = SV/EDV %**), it is about 65%. EF is an index for the resting myocardial contractility.

# *Factors controlling Cardiac Output*

The two determinants of CO are the ***Heart rate (HR)*** and the ***Stroke Volume (SV)***. However, no rise or fall in CO can take place unless it is preceded by an increase or a decrease in venous return respectively



# *Definitions*

1. The Preload – is the degree to which the myocardium is stretched before it contracts

2. The after load – is the resistance against which blood is expelled from the ventricle

3. The myocardial contractility – is the strength (or power) of contraction at any given end diastolic volume

# *The Heart rate*

- The normal range is 60-90 beats/min. The effective range in changing CO is between (40) - (180-200) beats/min.
- Heart rate > 110 = Tachycardia
- Heart rate < 50 = Bradycardia
- The rate is determined primarily by autonomic influences on the SA node.
- The parasympathetic and sympathetic effects on heart rate are antagonistic.
- The inherent rate of the SA node's spontaneous discharge is about 110 beats/min, however, because of vagal tone the average rate is close to 70 beats/min.
- The heart rate can be regulated by other factors such as body temperature, circulating hormones (such as adrenaline and noradrenaline, thyroxin, glucagon, etc.), and stretching of the right atrium (Bainbridge reflex).
- If heart rate increases without an increase in venous return, **SV will decrease in a paradoxical fashion** so that the CO remains constant.

# *The Preload (EDV)*

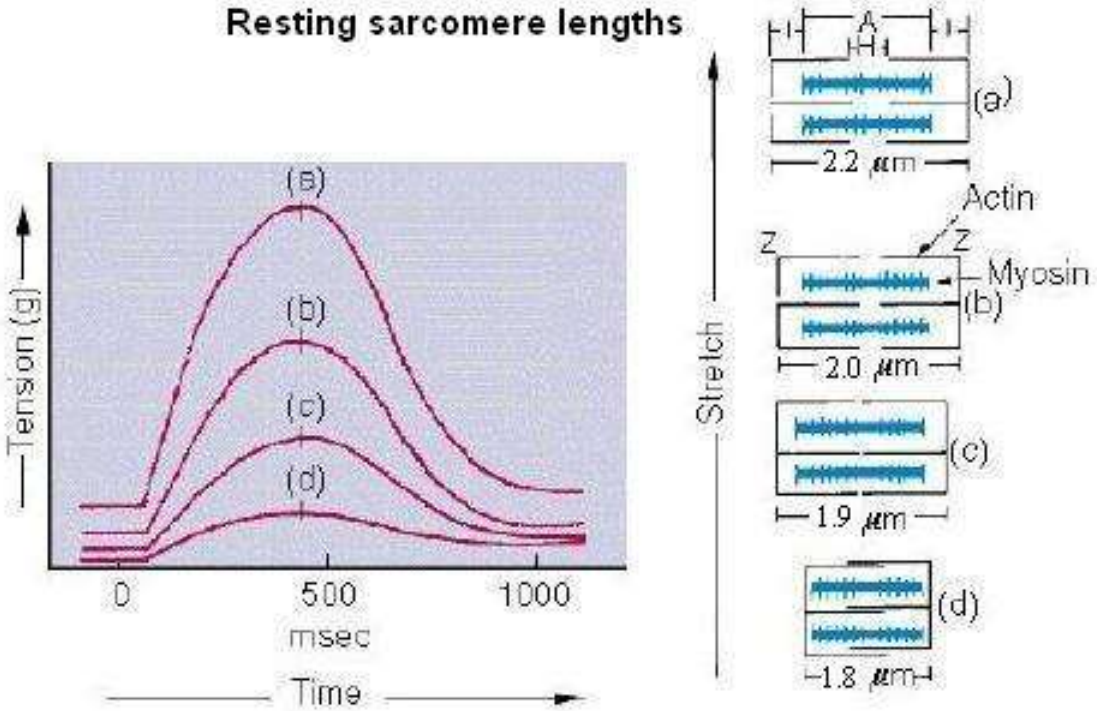
- If heart rate remains stable and more blood returns to the heart (i.e. more venous return) the EDV will increase. This results in a greater force of contraction, and consequently, a greater stroke volume and a greater CO (**Frank-Starling law of the heart**). This fact is applied to both sides of heart.
- Venous return increases if the gradient between the heart and the peripheral veins increases. This happens in response to;
  1. Increased blood volume (↑ mean systemic filling pressure).
  2. Venoconstriction (i.e. ↑ sympathetic tone).
  3. Dilation of arterioles.
  4. Skeletal muscle pump.
  5. Respiratory pump.
  6. Gravitational effect.
  7. Reduced right atrial pressure (i.e. ↑ contractility)

# *Intrinsic Regulation of Heart Pumping - The Frank-Starling Law and Mechanism*

- The amount of blood pumped by the heart each minute is determined almost entirely by the **venous return**.
- This intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the ***Frank-Starling mechanism of the heart.***
- (def.) The energy of contraction is proportional to the initial length of the cardiac muscle fiber (i.e. the preload) which is proportionate to the EDV.
- This means that (within physiological limits, the heart pumps all the blood that returns to it by way of the veins). In other words ***the heart pumps what it receives.***
- Mechanism: stretching of ventricles brings actin and myosin filaments to a more optimal degree of interdigitation for force generation. This law describes the length-tension relationship of the myocardial fiber.
- In addition, Stretch of the right atrial wall directly increases the heart rate by 10 to 20 percent, which also helps increase the amount of blood pumped each minute.

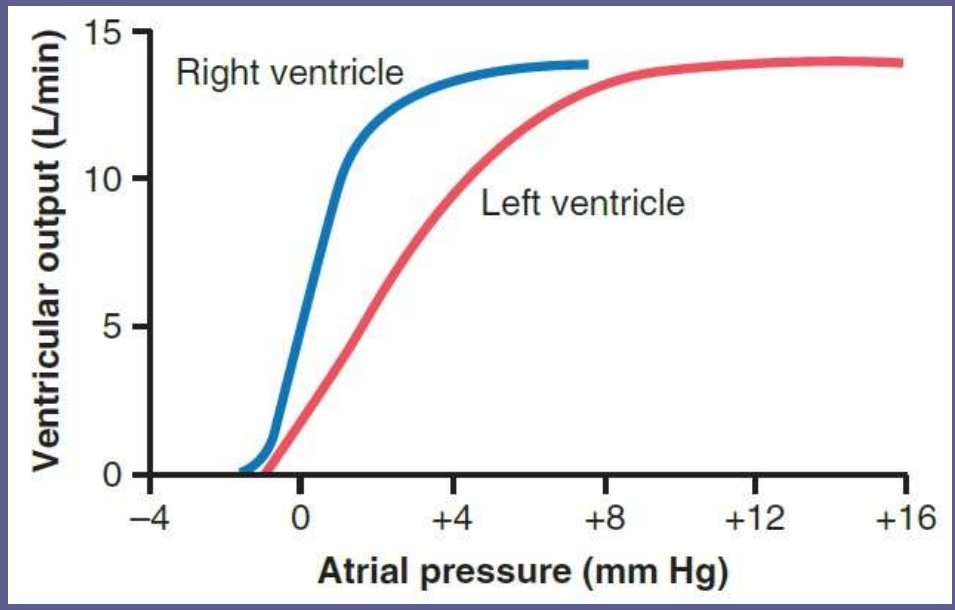
# Frank-Starling Law

Increases in end-diastolic ventricular volume produce increases in stroke volume (Compare to ascending limb of length-tension plot). The force of heart muscle varies with wall fibre tension, which is a function of length.



# Ventricular Function Curve

The two curves of this figure represent function of the two ventricles of the human heart. As the right and left atrial pressures increase, each ventricular volume and strength of cardiac muscle contraction increase, causing the heart to pump increased quantities of blood into the arteries per minute.



# Effect of autonomic nervous system on cardiac pumping

- Sympathetic stimulation can increase pumping (output) by 100%. By contrast, the output can be decreased to as zero by vagal (parasympathetic) stimulation.
- Sympathetic stimulation can increase cardiac pumping by (1) increasing the heart rate from a mean of 70 to 180-200 beats/min. Also, sympathetic stimulation (2) increases myocardial contractility, therefore increasing the pumping rate and the ejection pressure.
- Conversely, inhibition of the sympathetic nerves to the heart eliminates the sympathetic tone. This inhibition can decrease cardiac pumping to a moderate extent (about 30% below normal).

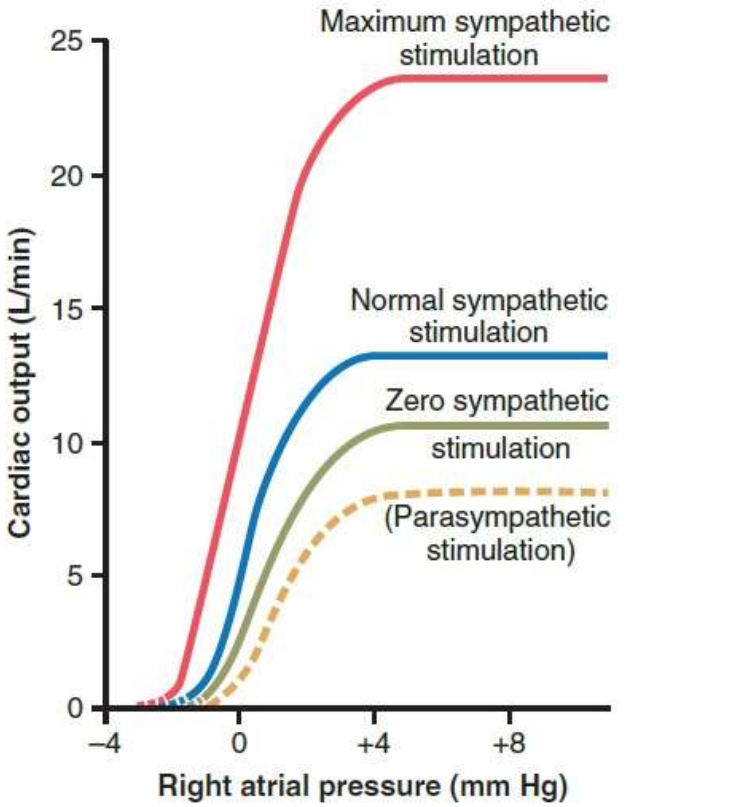



**Figure** Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.

# Effect of autonomic nervous system on cardiac pumping (cont.)

- Parasympathetic (vagal) stimulation, if continuous, can stop heartbeat for few seconds, then heart "escapes" and start beating at 20-40 beats/min. This will reduce the pumping rate greatly.
- The effect of vagal stimulation is mainly to decrease the heart rate rather than to decrease greatly the myocardial contractility. Nevertheless, the great decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping by 50 percent or more.

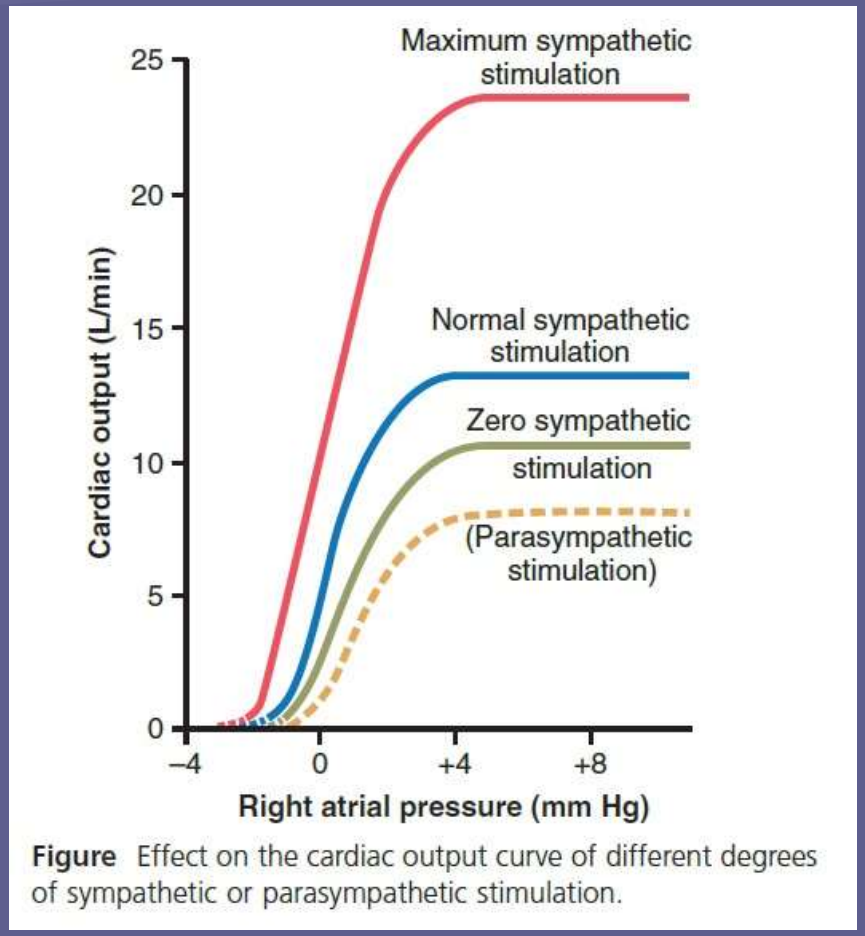
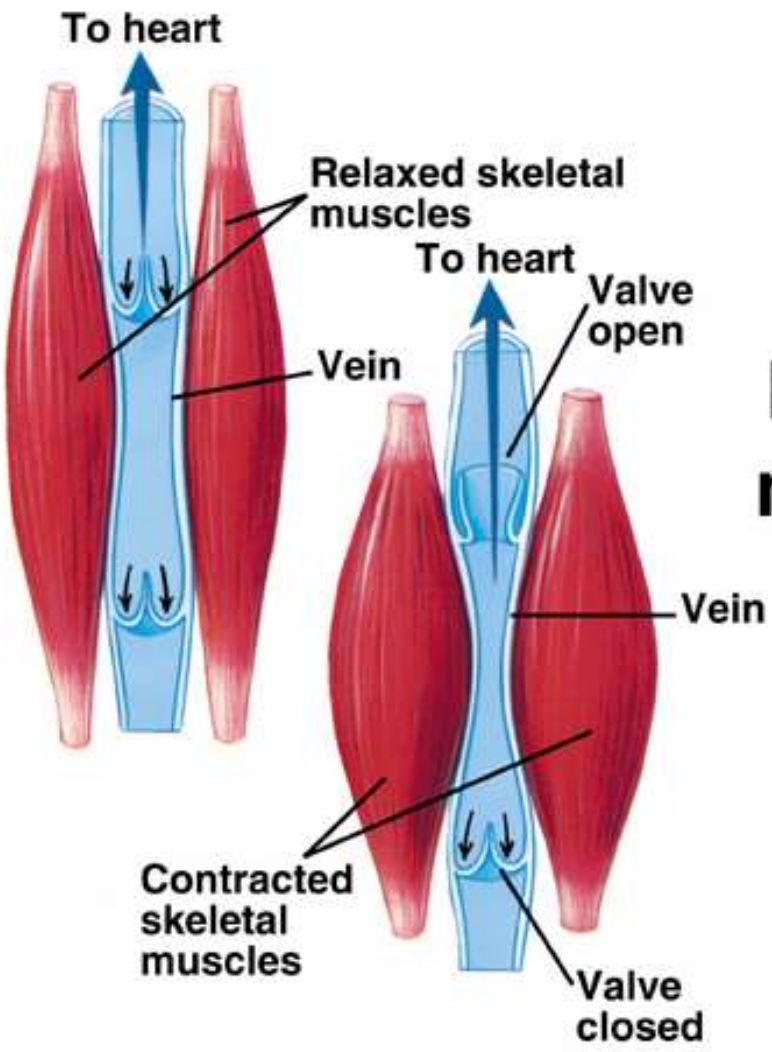


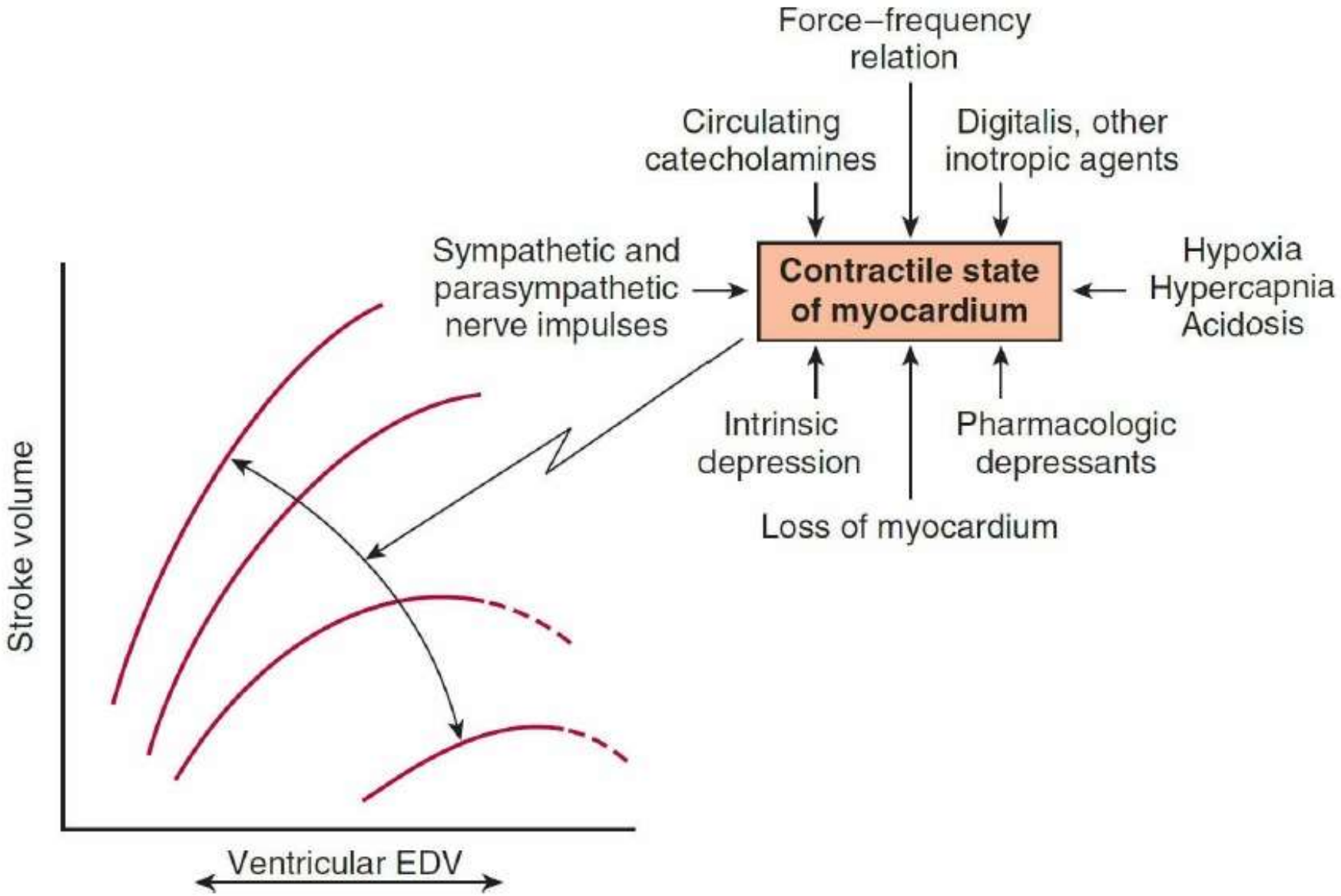

**Figure** Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.




# Exercise and muscle pump

# *The Myocardial Contractility*

- Sympathetic stimulation and epinephrine enhances the contractility and squeezing out a greater percentage of the blood out of the ventricle leading to more complete ejection (**i.e. less ESV**).
- Heart failure can affect one or both ventricles. It is the inability of the cardiac output to keep pace with body's demands for supplies and removal of wastes. It is due to reduced cardiac contractile power despite the sympathetic compensatory reflex. The most ***two*** common reasons are;
  1. Damaged myocardium due to impaired coronary circulation
  2. Prolonged pumping against chronic elevated resistance (i.e. elevated after load)




**Effect of changes in myocardial contractility on the Frank–Starling curve**

# *The after load*

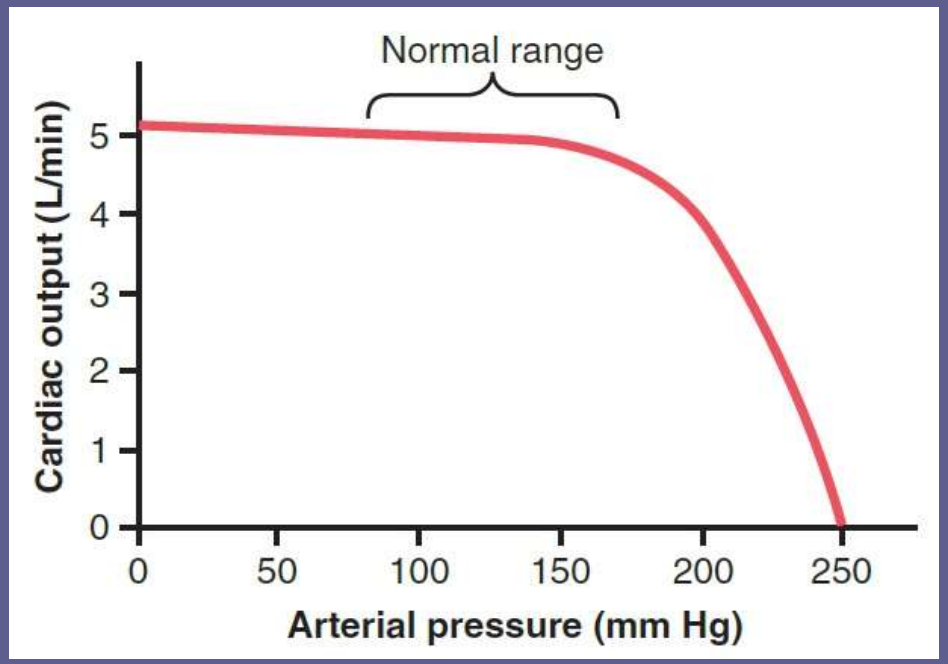
The total peripheral resistance the heart is facing is mainly due to;

1. Arteriolar resistance.
2. Major arterial compliance.
3. Blood volume within the arterial tree at the moment of ventricular ejection.

Note: The higher the after load the higher the ESV, the lower is the SV and the CO. However, the CO will be re-adjusted by the increase in the preload (Frank-Starling law of the heart). Therefore, hypertensive patients have relatively normal CO.

# Effect of Increasing the Arterial Pressure Load (i.e. Afterload) on Cardiac Pumping

The increase in the arterial pressure in the aorta does not decrease the cardiac output until the mean arterial pressure rises above about **160 mm Hg**.



# Energy and $O_2$ utilization by the heart

1. Like skeletal muscle, the energy is derived mainly (70-90%) from oxidative metabolism of fatty acids; other nutrients are lactate and glucose.
2. $O_2$ consumption is taken as a measure of the chemical energy liberated during cardiac work.
3. $O_2$ consumption is determined by the intra-myocardial tension (resting tension), the contractile state of the myocardium, and the heart rate. An increase in aortic pressure (i.e. increased afterload) is associated with a greater increase in $O_2$ consumption than an increase in preload (***LaPlace law***).

# Energy and $O_2$ utilization by the heart (cont.)

4. The high energy cost of increasing afterload reflects the large amount of energy expended to do internal work during isovolumic contraction (increases the amount of energy that must be expended to stretch internal elasticities).
5. The product of heart rate and aortic pressure correlates closely with cardiac oxygen consumption.

**Energy expenditure $\alpha$ HR × P**

6. An increase in cardiac output due to an increase in heart rate consumes less $O_2$ than that due to an increase in preload.
7. Cardiac efficiency is not more than 25%. In persons with heart failure, this efficiency can decrease to as low as 5 to 10 percent.

# *Methods for measuring Cardiac output*

- *Direct:* by the use of flow-meter, usually used in experimental animals.
- *Indirect:* Such methods can be applied to humans. These methods are;
  1. Oxygen Fick's method.

$$CO = \frac{\text{Oxygen utilization by body (ml } O_2\text{/min)}}{\text{Arterial } O_2 \text{ content (ml } O_2\text{/L bl.)} - \text{Mixed venous } O_2 \text{ content (ml } O_2\text{/L bl.)}}$$

  2. Indicator dilution method.
  3. Ultra Sound (echo).

# Test:

**Q. Which of the following will promote an increase in the stroke volume of the heart?**

A. A reduction in venous tone
B. A pneumothorax
C. Dehydration
D. General anesthetics
E. Skeletal muscle contraction

# Test:

**Q. The factor common to most changes in cardiac muscle contractility is the:**

A. Amplitude of the action potential
B. Availability of cellular ATP
C. Cytoplasmic calcium concentration
D. Rate of neural stimulation
E. The magnitude of the EDV