THE HEART AS A PUMP

REGULATION OF CARDIAC OUTPUT •
Heart Rate via sympathetic & parasympathetic nerves
Stroke Volume –
Frank-Starling “Law of the Heart” •
Changes in Contractility •
MYOCARDIAL CELLS (FIBERS) •
Regulation of Contractility –
Length-Tension and Volume-Pressure Curves –
The Cardiac Function Curve –
CARDIAC OUTPUT = STROKE VOLUME \times HEART RATE

Autoregulation
(Frank-Starling “Law of the Heart”)

Contractility

Sympathetic Nervous System

Parasympathetic Nervous System
CAR DI A C  F U N C T I O N  C U R V E

THE  FRANK-STARLING  "LAW  OF  THE  HEART"

CARDIAC OUTPUT (L/min)

RAP mmHg

 Increased Contractility
WORK OUTPUT OF THE HEART

1. VOLUME PRESSURE CURVE
2. REGULATION OF HEART PUMPING
   A. FRANK-STARLING MECHANISM
   B. AUTONOMIC NERVOUS SYSTEM
   C. CALCIUM AND POTASSIUM
   D. EFFECT OF TEMPERATURE
**Fig. 23-16** Pressure-volume loop of the left ventricle for a single cardiac cycle (ABCDEF).
Pressure Volume Loop

**Fig. 23-16** Pressure-volume loop of the left ventricle for a single cardiac cycle (ABCDEF).
Pressure Volume Loop

**Fig. 23-16** Pressure-volume loop of the left ventricle for a single cardiac cycle (ABCDEF).

Early Diastole
Pressure Volume Loop

![Diagram of a pressure-volume loop with labeled points A, B, C, D, E, F and a black arrow pointing to the late diastole phase.]

**Fig. 23-16** Pressure-volume loop of the left ventricle for a single cardiac cycle (ABCDEF).
Pressure Volume Loop

**Fig. 23-16** Pressure-volume loop of the left ventricle for a single cardiac cycle (ABCDEF).
Therefore, in Figure 9-9 the curve labeled "III," or "period of ejection," traces the changes in volume and systolic pressure during this period of ejection.

Phase IV: Period of isovolumetric relaxation. At the end of the period of ejection (point D: Figure 9-10), the aortic valve closes and the ventricular pressure falls back to the diastolic pressure level. The line labeled "IV" (Figure 9-9) traces this decrease in intraventricular pressure without any change in volume. Thus, the ventricle returns to its starting point, with about 50 milliliters of blood left in the ventricle and at an atrial pressure of 2 to 3 mm Hg.

The area subtended by this functional volume-pressure diagram (the shaded area, labeled "EW") represents the net external work output of the ventricle during its contraction cycle. In experimental studies of cardiac contraction, this diagram is used for calculating cardiac work output.

When the heart pumps large quantities of blood, the area of the work diagram becomes much larger. That is, it extends far to the right because the ventricle fills with more blood during diastole, it rises much higher because the ventricle contracts with greater pressure, and contracts to a smaller volume.

For cardiac contraction to be complete, the ventricle must have become filled.

The afterload or the load imposed on the ventricle by the aorta leading from the heart is described by the systolic curves of the ventricle's work cycle rather than its afterload. The afterload is loosely that which the ventricle is required to pump the blood through.

The importance of the afterload is that in many instances, the heart or circulation system's work load is not constant. For example, the ventricle must provide work for heart muscle, like the other muscles of the body, to fuel the day's activities and provide the work of the heart.

Chemical Energy and Oxygen Utilization

Heart muscle, like the other muscles of the body, requires energy to function. However, compared to the other muscles, heart muscle has a higher metabolic rate. This is because the heart must pump blood through the body to fuel the body's chemical processes. The heart muscle must have a steady supply of oxygen to perform its work. Oxygen is taken up by the heart during diastole and is used during systole. The oxygen uptake by the heart is a major factor in the heart's energy production.

In summary, the heart is a muscular organ that pumps blood through the body to supply oxygen and nutrients.
Figure 9-13. Cardiac sympathetic and parasympathetic nerves. (The vagus nerves to the heart are parasympathetic nerves.) A-V, atrioventricular; S-A, sinoatrial.

The two curves of this figure represent function of the two ventricles of the human heart based on data extrapolated from experimental animal studies. As the right and left atrial pressures increase, the respective ventricular volume outputs per minute also increase.

Thus, ventricular function curves are another way of expressing the Frank-Starling principle.
Figure 9-14. Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.
Figure 9-15. Constancy of cardiac output up to a pressure level of 200 mm Hg. Only when the arterial pressure rises above this normal range does the increasing pressure load cause the cardiac output to decrease significantly.

Effect of Calcium Ions. Excess calcium ions cause effects almost exactly opposite to those of potassium ions. They cause the heart to move toward spastic contraction. This effect is caused by a direct effect of calcium ions on the muscle to initiate the cardiac contractile process, as explained earlier in this chapter.

Conversely, deficiency of calcium ions causes cardiac weakness, similar to the effect of high potassium. Fortunately, calcium ion levels in the blood normally are regulated within a very narrow range. Therefore, calcium effects of abnormal calcium concentrations are seldom a clinical concern.

EFFECT OF TEMPERATURE ON HEART FUNCTION