

Frank-Starling mechanism

The larger the stretch of the cardiac muscle is the larger the force with which the muscle contracts becomes. This is due to the muscle fibers being in a more optimal position to allow for a higher efficiency of contraction. This principle depends on two variables: preload and afterload. All of this is true only in physiological state, in cardiac hypertrophy and other pathological states the stretching of the cardiac muscle may lead to decrease in contraction force.

Preload

Preload is the force by which the cardiac muscle gets stretched before systolic contraction. Depends largely on venous return - more blood enters the cardiac muscle, the cardiac muscle gets stretched more, the muscle fibers get into a more optimal position for efficient contraction and the next contraction occurs with higher efficiency, thus generating higher force expelling more blood from the heart.

Increased preload increases end-diastolic volume, systolic volume, cardiac output and also end-systolic volume.

Afterload

Afterload is the resistance of the systemic circuit (mainly aorta) against which the heart has to pump. It depends largely on the systemic blood pressure - higher blood pressure means that the resistance in the aorta is higher, hence the heart pumps less blood into the aorta, leaving increased amount of blood in the left ventricle, this additional blood keeps the ventricle dilated allowing the muscle fibers to stay in the optimal position for the next contraction and the next contractions occurs with higher force expelling more blood from the heart. Increased afterload puts unnecessary strain on the cardiac muscle, increasing its oxygen demand and worsening blood supply which can lead to cardiac failure.

Increased afterload increases the end-systolic volume which results in increased next end-diastolic volume and the subsequent systolic contraction has a higher ejection volume.