Mechanical properties of the cardiac muscle

The mechanical properties of the cardiac muscle help to allow it to contract efficiently without having to waste energy through excess or weak contractions. The mechanism of contraction in the cardiac muscle is based on the same principles as the contraction in skeletal muscle

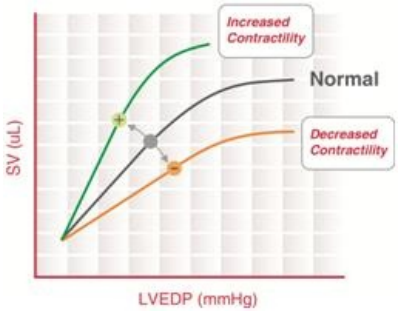
Structure of Cardiac Muscle

- Cardiac muscle is a branched, interconnected array of cells
- Individual cardiac muscle cells are held together by collagen fibres
- Mechanical forces are easily transmitted between cells
- Myocardial cells are interconnected by **intercalated discs**, which permit the transmission of electrical signals and transmit mechanical forces between adjacent cells
- In each cell, almost half of the volume is filled with myofibrils arranged in a regular repeating pattern - **sarcomeres**
- Each sarcomere ends in a Z-line that provides mechanical continuity with the next sarcomere
- Thin actin filaments extend from the Z-lines and to the center of the sarcomere and interdigitate with the thick myosin filaments in the centre of the sarcomere

Cardiac Mechanics

The functional properties of cardiac sarcomeres can be described by a three-component model:-

- **Contractile element** - actin-myosin interacton
- **Parallel elastic component** - causes resting fibres to remain in a shortened state; it contributes to the increase of contractile force along with increased ventricular filling (**Frank-Starling mechanism**)
- **Series elastic component** - this component must be stretched out through internal shortening before tension can develop



Frank-Starling Curve

Elementary forms of contraction:-

- **Isotonic contraction** - active shortening under a constant load (constant pressure)
- **Isometric contraction** - length is kept constant (constant volume)
- **Afterloaded contraction** begins with an isovolumetric phase (the pressure rises and the volume remains constant) and is followed with an isotonic decrease in volume

Length-Force Relationship

- Skeletal and cardiac muscle show similar length-force relationship
- Maximum developed force is observed when the thick and thin filaments overlap optimally
- When sarcomeres are stretched beyond their optimal length, the force of contraction decreases due to less overlap of the thick and thin filaments, which means less cycling of cross-bridges
- When the ventricles fill with blood, this causes the muscle fibres to stretch, causing overlap of filaments, therefore causing cycling of cross-bridges and this stimulates contraction: too much blood in the ventricles will cause the muscle fibres to over-stretch and therefore less cycling occurs, meaning the contraction becomes weaker
- **Frank-starling mechanism** - increased diastolic filling augments the contractile response, leading to increased ability to eject the blood

Force-Velocity Relationship

Force and velocity are inversely related - with no load (afterload), force is negligible and velocity is maximal: in an isometric contraction, where no external shortening occurs, force is maximal and velocity is zero

1. A certain weight preloads the muscle (blood in ventricles), stretching the elastic elements
2. When the muscle is stretched in this way, it must first undergo internal shortening to remove the slack in the series elastic element before tension develops (isometric phase of contraction)
3. When the developed tension equals the load (afterload), the weight is lifted without further stretch of the elastic elements (isotonic phase of contraction)

- The **preload** refers to the stretch of the ventricle just before the onset of contraction due to the blood in it (aka. the **end-diastolic volume**)
- The **afterload** refers to the aortic pressure during the period when the aortic valve is open (the remaining amount of blood in the ventricles at this time is referred to as the **end-systolic volume**)
- Increase in preload will increase contractility of the heart up until the limit of the Frank-Starling Law
- Increase in afterload will increase the systolic pressure in the heart, until the afterload becomes so great that

the ventricles cannot produce a greater force to surpass the afterload in order to open the aortic valve
- The **ejection fraction** is the ratio of the volume of blood ejected from the left ventricle at the end of diastole (**EF=SV÷EDV**) and is an index of contractility

Effects of Cardiac Glycosides

- Cardiac glycosides are examples of drugs that increase contractility (**inotropic** drugs) e.g: Digitalis, Stophanthin and Ouabain
- Inotropic drugs may engage in different mechanisms to achieve increased contractility:-

1. Na^+-K^+ pump blocking: this elevates intracellular Na^+ levels, meaning the gradient for removing Ca^{2+} by the Na^+-Ca^{2+} exchanger is reversed, causing an increase in intracellular Ca^{2+} ions
2. Release of Ca^{2+} ions from the smooth endoplasmic reticulum can be directly increased by some glycosides
3. Glycosides increase the slow inward current carried by Ca^{2+} ions, increasing the height of the plateau of action potentials, causing an increased concentration of Ca^{2+} inside the myocardial cells

Links

Bibliography

HALL, John E. *Guyton and Hall Textbook of Medical Physiology.* 11th Edition edition. 2006. ISBN 978-1-4160-4574-8.

POKORNY, . *Circulation* [lecture for subject Mechanical Properties of the Cardiac Muscle, specialization Physiology, First Faculty of Medicine Charles University]. Prague. 2011.