Physiology of the normal heart

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Abstract

The heart is a dynamic organ that beats >2.5 billion times in the average lifetime. Humankind's fascination for its complex yet elegant physiology can be traced as far back as the ancient Egyptians, who first documented the association between abnormalities in the peripheral pulse and the heart. Several thousand years later, in the 17th century, an English physician named William Harvey provided a more comprehensive interpretation of the mechanisms of the systemic circulation. A sound grasp of cardiovascular physiology is pertinent to the understanding of many of the pathophysiological processes in the body and is thus an essential for all aspiring physicians. In this article, we discuss the basic physiological, electrophysiological and haemodynamic concepts of the heart.

Keywords Action potentials; cardiac myocytes; cardiac output; haemodynamics; MRCP; sarcomeres; stroke volume

Key points

- The unstable resting potentials of certain groups of cardiac myocytes result in spontaneous depolarization, a process known as automaticity
- Most ventricular filling from the atria occurs passively, with only 20% occurring via atrial contraction
- The mechanism by which cardiac stroke volume increases in direct relation to increased preload (and therefore myocardial stretch) is known as the Frank–Starling law

Introduction

The cardiovascular system has evolved to deliver blood to perfuse the body's tissues and organs with oxygen and nutrients. The heart is a dual-pump system comprising four chambers. It is organized into the pulmonary and systemic circulation. The right atrium and ventricle deliver blood to the lungs for oxygenation (*pulmonary circulation*), and the left atrium and ventricle deliver oxygenated blood to the rest of the body (*systemic circulation*).

Cardiac muscle

Cardiac myocytes constitute the bulk of the heart mass. They are specialized muscle cells containing the contractile apparatus, called sarcomeres. These contain thick and thin filaments, along with the regulatory proteins troponin and tropomyosin, which together form the contractile apparatus.[1] Contraction occurs by sarcomere shortening, which is produced by the adenosine triphosphate (ATP)-dependent movement of the thick and thin filaments relative to each other. Cardiac myocytes are shorter than skeletal muscle fibres and usually contain only one nucleus. They contain many mitochondria and myoglobin, as ATP is primarily produced through aerobic metabolism. There are differences between the myocytes in the atria, ventricles and conduction system. For example, ventricular myocytes are elongated and packed with sarcomeres while atrial myocytes lack transverse tubules, which are involved in the entry of calcium ions. Myocytes of the conduction system are small cells that possess only rudimentary sarcomeres.

Physiology of cardiac conduction

Electric stimulation is created by a sequence of ion fluxes through channels in the membrane of cardiac myocytes, leading to cardiac contraction. These consist of five phases (Figure 1): the resting phase, depolarization, early repolarization, the plateau phase and repolarization.

Cardiac myocytes generally have a large stable resting membrane potential with a prolonged action potential and plateau phase.[2] Conversely, there are groups of cells that have smaller and unstable resting potentials with a tendency to spontaneously depolarize, which generates electrical impulses.[3] This property is known as automaticity and is unique to cardiac myocytes. These clusters of myocytes are, akin to a metronome, the heart's intrinsic pacemaker. Cardiac myocytes that exhibit this behaviour include the sinoatrial (SA) node, atrioventricular (AV) node, His–Purkinje fibres and ventricular myocytes.

The SA node is located in the upper part of the right atrium at the junction with the superior vena cava, and has an intrinsic depolarization rate of 60–100 beats per minute. Electrical impulses are transmitted by the perinodal cells to the right atrium and to the heart's conduction system, eventually resulting in myocardial contraction. The SA node is tightly regulated by the autonomic nervous system, whereby increased sympathetic and parasympathetic activity increase and decrease the rate of action potential production and heart rate (HR), respectively.

Impulses from the SA node travel through Bachmann's bundle to the left atrium. They travel through the right atrium to the AV node, through the bundles of His, and down the left and right bundle branches. These bundle branches terminate in Purkinje fibres, which project into the myocardium to facilitate synchronized depolarization of the ventricles.

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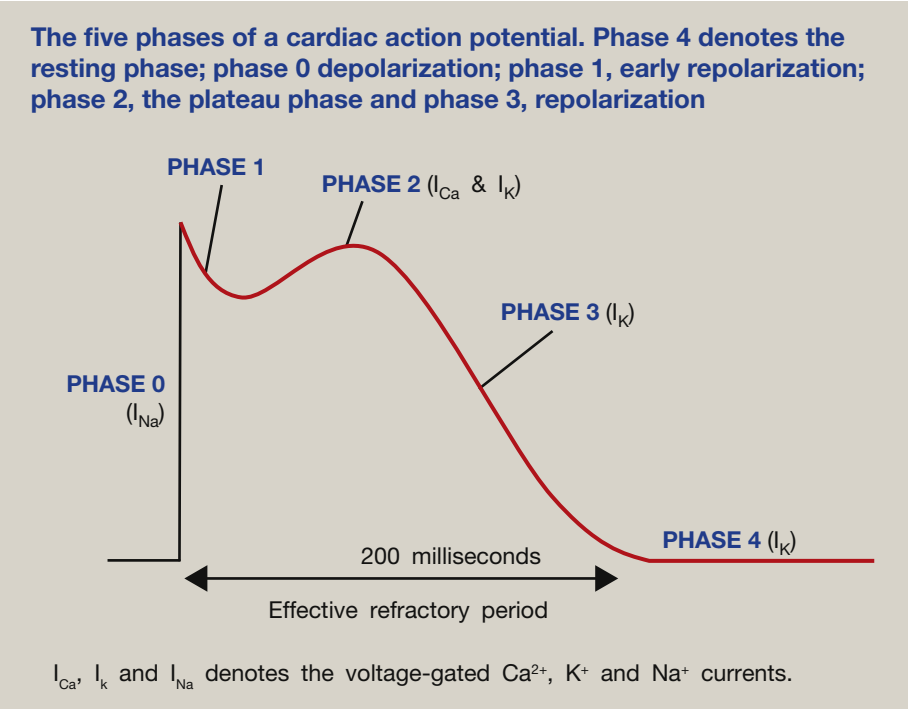

Figure 1

The cardiac cycle

One cardiac cycle is broadly divided into two main phases:[4]

- **systole**, the period of contraction of the ventricles during which blood is pumped into the circulation
- **diastole**, the period of ventricular relaxation and filling.

The electromechanical phases of the cardiac cycle can be seen in Figure 2. The mechanical phases of the cardiac cycle are described below, with similar events occurring in the left and right chambers. Changes in the atrial pressure waveforms are also described.

Atrial contraction

This occurs after a period of passive (usually 80% of) ventricular filling. The final 20% is done by atrial contraction. During this period, while the AV valves are open, the semilunar valves remain closed. The venous 'a wave' is produced within the atrium as a result of atrial contraction.

Isovolumetric contraction

As the ventricles begin to contract, the increasing pressure in the ventricles exceeds that in the atria, resulting in closure of the AV valves (S1, the first heart sound). This period strictly refers to the period when the AV valves have closed but the semilunar valves have not yet opened. The ventricles are thus in the period where they are contracting without a change in the volume of the ventricle, termed isovolumetric contraction. The bulging of the AV valves into the atrium during this period slightly increases the atrial pressures, resulting in a subsequent 'c wave'.

Rapid ventricular ejection

As the ventricular pressure continues to rise (to approximately >80 mmHg in the left ventricle), the semilunar valves open, resulting in the rapid ejection of blood into the aorta and pulmonary artery. The ventricles continue to contract, with a progressive increase in the intraventricular pressure (to approximately 120 mmHg in the left ventricle). As the AV valves are closed and the atria continue to receive blood from the pulmonary veins or vena cava, a 'v wave' is produced.

Reduced ejection

This phase begins with ventricular repolarization, during which ventricular tension and pressure generation are released, leading to a fall in the rate of ejection of blood. Forward flow continues secondary to remnant kinetic energy from previous phase.

Isovolumetric relaxation

With continued relaxation of the ventricles, the pressures drop below the diastolic aortic and pulmonary pressures, leading to closure of the aortic and pulmonary valves (S2, the second heart sound). Although the ventricular pressures decline, they are greater than that of the atria, so the AV valves still remain closed. With all the valves closed, ventricular relaxation occurs without a change in volume, thus being described as isovolumetric relaxation.

Rapid filling

As ventricular relaxation continues, the pressures fall below that of the atria, resulting in opening of the atrioventricular valves and the onset of the passive phase of ventricular filling. Despite filling from the atria, the ventricles are still relaxing, so the ventricular pressure continues to fall off.

Reduced filling

As the ventricles continue to relax, the compliance of the ventricular walls decreases. With continuing filling, the intraventricular pressure subsequently begins to rise, slowing down filling from the atrium.

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