# Control of cardiac function

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## Abstract

An understanding of the core cardiovascular physiological principles is fundamental to delivering clinical care to patients. In order to successfully deliver oxygen and other nutrients to end organs whist eliminating waste products, cardiac output is manipulated through the control of heart rate, blood pressure, stroke volume, preload, afterload and contractility. Though controlled through intrinsic physiological mechanisms in health, the delivery of fluids, drugs, external/electrical stimulation and/or mechanical support can allow for optimization of cardiac function in disease. In this article, we outline these core principles and describe how cardiac function is controlled.

**Keywords** Cardiac output; heart rate; inotropes; parasympathetic; Starling's law; stroke volume; sympathetic

**Royal College of Anaesthetists CPD Matrix:** 1A01

## Learning objectives

After reading this article, you should be able to appreciate:

- the physiology underpinning cardiac function
- the factors influencing cardiac output
- the key clinical applications of the intrinsic and extrinsic control of cardiac function

## Introduction

The chief functions of the cardiovascular system are to provide end organs with oxygenated nutrient-rich blood and to facilitate the clearance of waste metabolites. The heart beats as slow as it is able and as fast as it must to achieve these aims, though many other integrated factors determine the resultant cardiac output. Contraction of cardiac muscle increases the pressure within the chambers of the heart thus forcing ejection of blood into the systemic circulation. A pressure gradient is established across the cardiovascular system from areas of high pressure in the arteries and arterioles to areas of low pressure in the venous circulation. Blood flow is directly proportional to the arterial pressure and inversely proportional to the resistance to flow through that system. This article provides an introduction to some of the mechanisms controlling cardiac performance that will be further expanded in the articles that follow in this issue.

## The cardiac action potential

In health, the heartbeat is initiated by pacemaker cells at the sinoatrial (SA) node located at the junction of the superior vena cava and the right atrium. Pacemaker cells do not possess a stable resting membrane potential and instead function with a decaying membrane potential (Figure 1). During the rest phase (phase 4), there is increased permeability to sodium and calcium ions. Both diffuse down their concentration gradient into the cell and this gives rise to the so-called 'funny current'. The inside of the cell gradually becomes more positive with respect to the outside until the membrane potential reaches −40 mV and depolarization is triggered by calcium rapidly entering the cell (phase 0). Following a peak membrane potential of +20 mV, repolarization occurs (phase 3) through potassium extrusion until the membrane potential returns to −60 mV and the rest phase (phase 4) begins again. Of note, there is no phase 1 or 2 associated with a pacemaker potential. The slope of phase 4 is increased by sympathetic stimulation, thus resulting in an increase in heart rate. Likewise, the slope of phase 4 is decreased by parasympathetic innervation, thus resulting in a decrease in heart rate. The balance between these inputs determines heart rate and this in turn has a major influence on cardiac function.

The cardiac muscle action potential differs significantly from the pacemaker action potential (Figure 2). It has five phases (0–4) where phase 4 is the resting membrane potential of −90 mV as maintained by the extrusion of $3Na^+$ in exchange for $2K^+$ by the $3Na^+/2K^+$ ion pump. As the wave of depolarization reaches the myocyte from adjacent myocytes, rapid influx of $Na^+$ triggers rapid depolarization (phase 0). As repolarization begins (phase 1), the $Na^+$ channels close and $K^+$ channels open, but a plateau is achieved (phase 2) through the opening of L-type $Ca^{2+}$ channels. This is of clinical importance, as it prevents the triggering of further depolarizations and therefore cardiac tetany. The membrane potential subsequently tends towards its resting value (Phase 3) through the closure of L-type $Ca^{2+}$ channels and the movement of $K^+$.

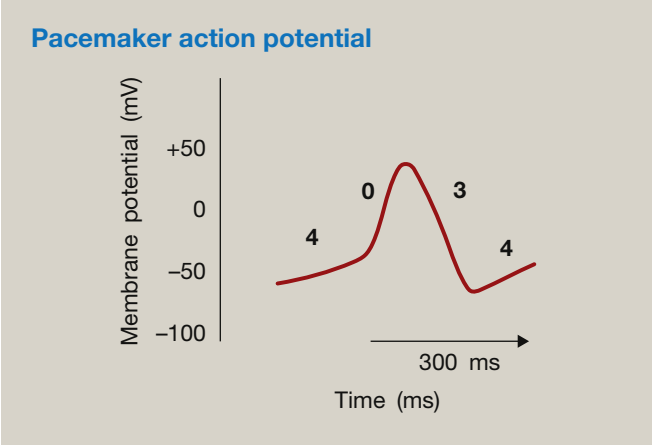


**Figure 1**

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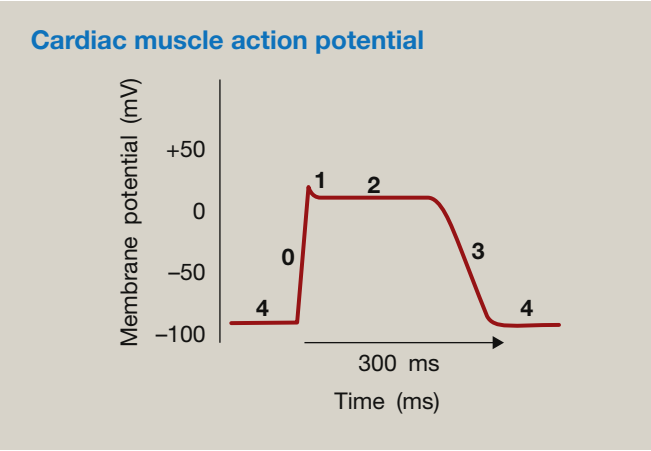


**Figure 2**

## The cardiac cycle

The cardiac cycle is divided into systole and diastole. Diastole is further subdivided into isovolumetric relaxation, rapid ventricular filling, slow ventricular filling and atrial contraction. Systole is subdivided into isovolumetric contraction and ejection. The pressure and volume changes occurring during the cardiac cycle together with corresponding phases, events, heart sounds and measured electrical changes as seen on the electrocardiogram are provided in Figure 3.

## Pressure waveforms

The systemic arterial pressure (Figure 4) can be measured in terms of systolic, diastolic and/or mean arterial pressure (MAP). The best indicator of end organ perfusion is the MAP as derived from the equation:

$$\text{MAP} = [(2 \times \text{Diastolic Pressure}) + \text{Systolic Pressure}]/3$$

Furthermore, MAP is related to cardiac output (CO) and systemic vascular resistance (SVR) through the equation:

$$\text{MAP} = \text{CO} \times \text{SVR}$$

As cardiac output can be defined by the stroke volume (SV) multiplied by the heart rate (HR), cardiac function and therefore MAP is controlled through the manipulation of SV, HR and SVR.

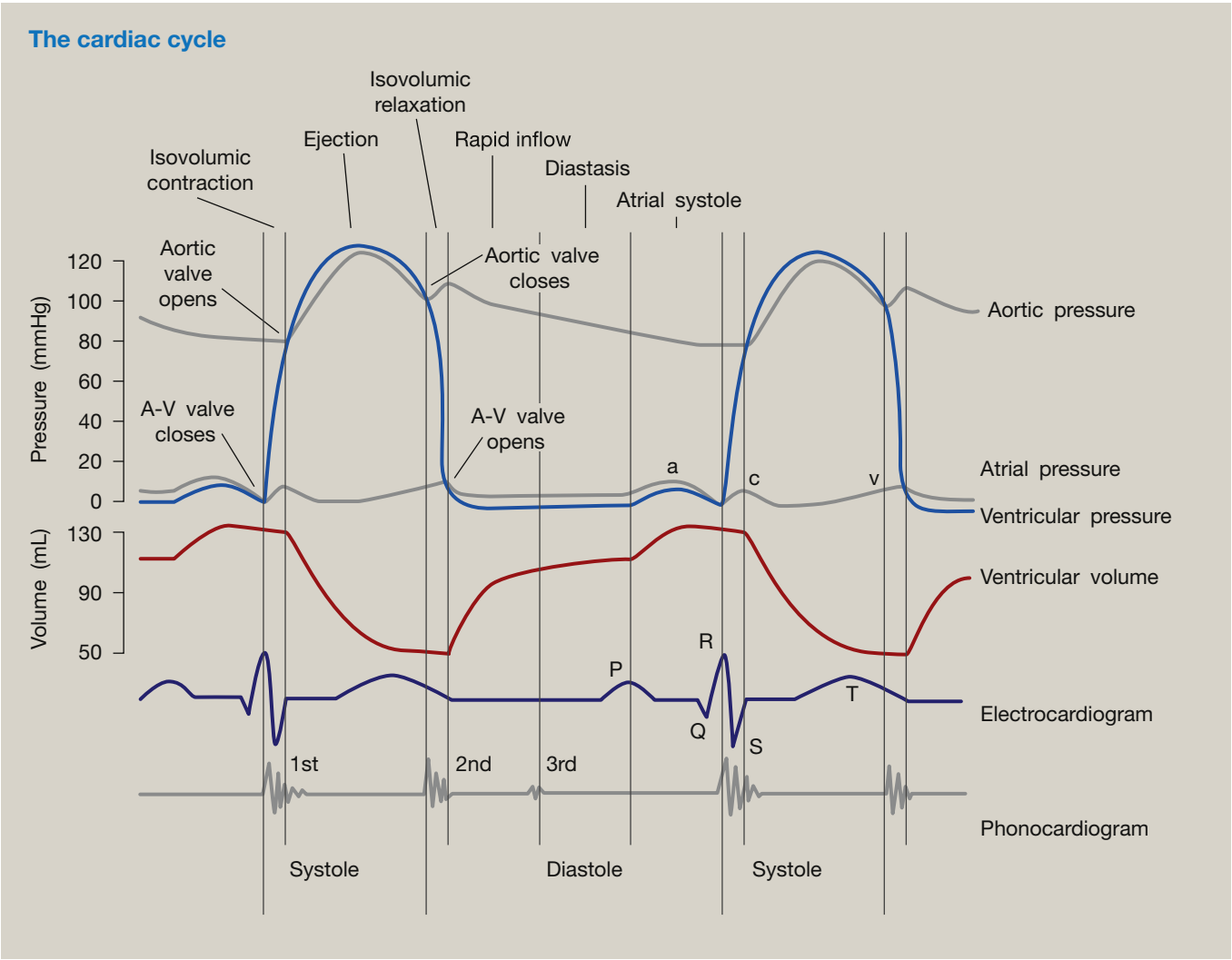


**Figure 3**




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