The Conduction System in the Heart

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INTRODUCTION

The conduction system in the heart is an intrinsic system whereby the cardiac muscle is automatically stimulated to contract, without the need for external stimulation (Waugh & Grant, 2007). It comprises specialised cardiac cells, which initiate and conduct impulses, providing a stimulus for myocardial contraction. It is controlled by the autonomic nervous system; the sympathetic nerves increase heart rate, contractility, automaticity and atrioventricular (AV) conduction, while the parasympathetic nerves have an opposite effect.

Irregularities in the conduction system can cause cardiac arrhythmias and an abnormal electrocardiogram (ECG). An understanding of the conduction system and how it relates to myocardial contraction and the ECG is essential for ECG interpretation.

The aim of this chapter is to understand the conduction system in the heart.

LEARNING OUTCOMES

At the end of the chapter the reader will be able to:

- ❑ Discuss the basic principles of cardiac electrophysiology.
- ❑ Describe the conduction system in the heart.

BASIC PRINCIPLES OF CARDIAC ELECTROPHYSIOLOGY

Depolarisation and repolarisation

The contraction and relaxation of the cardiac muscle results from the depolarisation and repolarisation of myocardial cells (Meek & Morris, 2008):

- *Depolarisation*: can be defined as the sudden surge of charged particles across the membrane of a nerve or muscle cell that

accompanies a physicochemical change in the membrane and cancels out or reverses its resting potential to produce an action potential (McFerran & Martin, 2003); put simply, it is the electrical discharging of the cell (Houghton & Gray, 2003). A change in the cell membrane permeability results in electrolyte concentration changes within the cell. This causes the generation of an electrical current, which spreads to neighbouring cells causing these in turn to depolarise. Depolarisation is represented on the ECG as P waves (atrial myocytes) and QRS complexes (ventricular myocytes).

- *Repolarisation*: can be defined as the process by which the cell returns to its normal (resting) electrically charged state after a nerve impulse has passed (McFerran & Martin, 2003); put simply, it is the electrical recharging of the cell (Houghton & Gray, 2003). Ventricular repolarisation is represented on the ECG as T waves (atrial repolarisation is not visible on the ECG as it coincides with and therefore, is masked by the QRS complex).

Automaticity

Automaticity is the ability of tissue to generate automatically an action potential or current (Marriott & Conover, 1998), i.e. electrical impulses can be generated without any external stimulation. It occurs because there is a small, but constant, leak of positive ions into the cell (Waldo & Wit, 2001).

The sinus node normally has the fastest firing rate and therefore assumes the role of pacemaker for the heart. The speed of automaticity in the SA node can be determined by a number of mechanisms, including the autonomic nervous system and some hormones, e.g. thyroxin (Opie, 1998). If another focus in the heart has a faster firing rate, it will then take over as pacemaker.

Cardiac action potential

Action potential can be defined as the change in voltage that occurs across the membrane of a muscle or nerve cell when a nerve cell has been triggered (McFarran & Martin, 2003). Cardiac action potential (see Figure 1.1) is the term used to describe the entire sequence of changes in the cell membrane potential, from the beginning of depolarisation to the end of repolarisation.

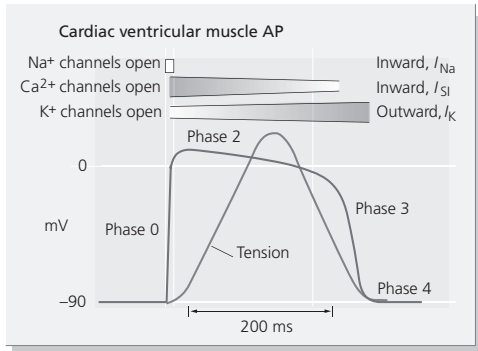

Figure 1.1 Cardiac ventricular muscle AP. Reprinted from Aaronson, P. & Ward J., *The Cardiovascular System at a Glance*, 3rd edn, copyright 2007, with permission of Blackwell Publishing.

Resting cardiac cells have high potassium and low sodium concentrations (140 mmol/l and 10 mmol/l, respectively). This contrasts sharply with extracellular concentrations (4 mmol/l and 140 mmol/l, respectively) (Jowett & Thompson, 1995). The cell is polarised and has a membrane potential of 90 mV.

Cardiac action potential results from a series of changes in cell permeability to sodium, calcium and potassium ions. Following electrical activation of the cell, a sudden increase in sodium permeability causes a rapid influx of sodium ions into the cell. This is followed by a sustained influx of calcium ions. The membrane potential is now 20 mV. This is referred to as phase 0 of the action potential.

The polarity of the membrane is now slightly positive. As this is the reverse pattern to that of adjacent cells, a potential difference exists, resulting in the flow of electrical current from one cell to the next (Jowett & Thompson, 1995).

The cell returns to its original resting state (repolarisation) (phases 1–3); phase 4 ensues. Sodium is pumped out and potassium and the transmembrane potential returns to its resting of 90 mV. Table 1.1 summarises the phases of the cardiac action potential.

Table 1.1 Phases of the cardiac action potential.

Phase	Action
0	Upstroke or spike due to rapid depolarisation
1	Early rapid depolarisation
2	The plateau
3	Rapid repolarisation
4	Resting membrane potential and diastolic depolarisation

Thompson 1997

Action potential in automatic cells

The action potential in automatic cells differs from that in myocardial cells. Automatic cells can initiate an impulse spontaneously without an external impulse.

Automatic cells can be found in the SA node, AV junction (AV node and Bundle of His), bundle branches and Purkinje fibres. The rate of depolarisation varies between the sites:

- *SA node*: has the shortest spontaneous depolarisation time (phase 4) and therefore the quickest firing rate (Julian & Cowan, 1993), usually approximately 60–100 times per minute (Khan, 2004).
- *AV junction (AV node and bundle of His)*: approximately 40–60 times per minute (Sharman, 2007).
- *Bundle branches and Purkinje fibres*: <40 times per minute.

If the SA node firing rate significantly slows or ceases, e.g. a possible complication following an acute inferior myocardial infarction, a subsidiary pacemaker will (it is hoped) provide an escape rhythm. In general, the lower down the conduction system that the pacemaker is sited, the slower the rate, the wider the QRS complex and the less dependable it is (Jowett & Thompson, 1995). When an ectopic pacemaker takes over control of the electrical activity in the heart it is denoted by the prefix 'idio', e.g. an idioventricular rhythm is an escape rhythm originating in the ventricles.

THE CONDUCTION SYSTEM IN THE HEART

The heart possesses specialised cells that initiate and conduct electrical impulses resulting in myocardial contraction. These

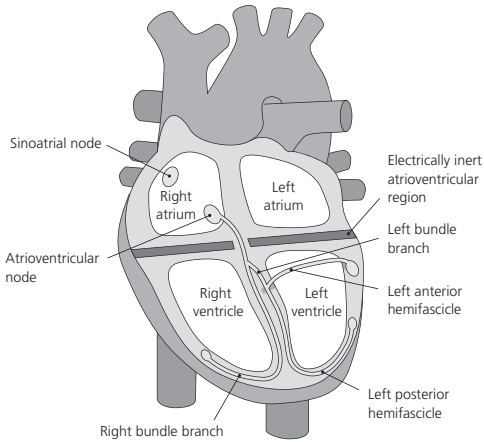

Figure 1.2 The His-Purkinje conduction system. Reprinted from Morris, F. *et al.*, *ABC of Clinical Electrocardiography*, 2nd edn, copyright 2008, with permission of Blackwell Publishing.

cells form the conduction system (see Figure 1.2), which comprises the following:

Sinoatrial (SA) node

The SA node is situated at the junction of the right atrium and superior vena cava (Sharman, 2007). The blood supply is via the nodal artery, which arises from either the right coronary artery (60%) or the left coronary artery (40%) (Jowett & Thompson, 1995). The SA node acts as the natural pacemaker and initiates each cardiac cycle (Meek & Morris, 2008) and is often referred to as the pacemaker (Khan, 2004; Waugh & Grant, 2007).

Internodal pathways

The impulse from the SA node is conducted to the atria via four main atrial pathways; three in the right atrium are referred to as internodal pathways because they carry the impulse from the SA node to the AV node (Khan, 2004) and one to the left atrium (Bachmann's bundle) (Berne & Levy, 1992).

AV node

The AV node is situated near the inferior aspect of the inter-atrial septum (Sharman, 2007). Blood supply is via the nodal artery, which arises from either the right coronary artery (90%) or the left circumflex artery (10%) (Jowett & Thompson, 1995). It acts as a 'bridge' connecting the atria to the ventricles, allowing the impulse to cross the atrioventricular ring (a thick layer of fibrous tissue, which electrically insulates the atria from the ventricles) (Khan, 2004).

The AV node has a slower conduction speed, which delays the conduction of the impulse from the atria to the ventricles (Waldo & Wit, 2001). This allows time for the atria to contract, enabling the ventricles to fill up before contraction (Khan, 2004). Although it does not itself possess the property of automaticity, the AV junction, conduction tissue connecting it to the bundle of His, does (Berne & Levy, 1992).

The AV node has a protective feature, blocking the number of atrial impulses reaching the ventricles (Khan, 2004). This is only seen when the atrial firing rate exceeds 180–200 impulses a minute (Berne & Levy, 1992) which is usually due to an area of abnormal automaticity in the conduction fibres or myocardiam in the atria (Huszar, 2001), e.g. in atrial fibrillation.

Bundle of His

The bundle of His was first discovered in 1893 by Wilhelm His Jr a Swiss cardiologist and anatomist. It is divided into right and left bundle branches. The left bundle branch is divided into two or sometimes three branches:

- *Anterior fascicle*: radiates anteriorly and superiorly across the ventricular wall.
- *Posterior fascicle*: radiates inferiorly and posteriorly across the left ventricular wall.
- *Mid-septal fascicle*: present in approximately a third of the population (Kulbertus & Demoulin, 1976), it usually emerges directly from the left bundle branch but can arise from either the anterior or posterior fascicle, and radiates through the septum (Dhingra *et al.*, 1975).

(Source: Khan, 2004)

Blood supply is via the left anterior descending artery (Jowett & Thompson, 1995).

Purkinje fibres

The Purkinje fibres were first discovered in 1839 by the Czech physiologist Johannes Evangelist Purkinje (Purkyne). They form the final part of the conduction system and result from subdivisions of the bundle branches (Sharman, 2007), enabling ventricular contraction from an inward to outward direction (Khan, 2004).

Control of heart rate

The heart rate is influenced by the cardiovascular centre in the medulla oblongata through the autonomic nervous system (Green, 1991; Waugh & Grant, 2007):

- *Parasympathetic or vagus nerve*: supplies mainly the SA node, AV node and atria (Waugh & Grant, 2007). Continuous vagal activity or vagal tone acts as a brake on the heart. The greater the vagal activity, the slower the heart rate. Increased vagal tone is often associated with an acute inferior myocardial infarction. If vagal activity diminishes, the heart rate will increase. If the vagal tone is completely blocked, the heart rate would be approximately 150 beats per minute (Green, 1991). Atropine blocks the action of the vagus nerve. This causes an increase in heart rate.
- *Sympathetic nerve*: supplies the SA node, AV node, atria and ventricles (Waugh & Grant, 2007). Sympathetic nerve activity ('fight and flight') has a positive chronotropic action on the heart, i.e. it increases the heart rate. It is particularly active in periods of emotional excitement, exercise and stress. Beta blockers shield the heart from sympathetic nerve activity resulting in a decrease in heart rate, blood pressure and myocardial workload.

CHAPTER SUMMARY

The conduction system in the heart comprises specialised cardiac cells, which initiate and conduct impulses, providing a stimulus for myocardial contraction. This chapter has provided an overview to the conduction system. The basic principles of cardiac

electrophysiology have been discussed. The conduction system has been described together with how the ECG relates to cardiac contraction.

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