ECGs for Nurses

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Foreword

I am delighted to write this foreword for a new and exciting ECG book for nurses.

Coronary heart disease is among the biggest killers in the country. More than 1.4 million people suffer from angina. 300 000 people have a heart attack every year. More than 110 000 people die of heart problems in England every year (Department of Health 2000).

The risk of death is the highest within the first hour of myocardial infarction and is usually due to arrhythmias (NICE 2002). Arrhythmias are commonly experienced by patients with coronary heart disease (Jowett & Thompson 2003). It is imperative that nurses not only correctly identify arrhythmias, but also appreciate their significance and necessary management. This book outlines all arrhythmias in a logical fashion using a five-stage approach to analysis with examples followed by possible effects on the patient, and finally, treatment. Chapters exploring arrhythmias include the conduction system, principles of monitoring, arrhythmias originating in the sinoatrial node, atria, atrioventricular junction, ventricles and heart blocks. These chapters will facilitate the development of the practitioner's knowledge of arrhythmias by giving concrete examples of application and practice.

The National Service Framework for coronary heart disease (2000) has identified a target for thrombolysis

to be administered to patients with a myocardial infarction, without contraindications, within 20 minutes of arrival in hospital. Nurses have an increasingly important role to identify patients in this category as they require urgent attention. These situations require skills in 12 Lead electrocardiogram (ECG) recording and interpretation. *ECGs for Nurses* has two chapters dedicated to 12 Lead ECGs, one exploring recording, and the other, interpretation. It is vital for nurses to review their skills of ECG recording to ensure accuracy and thoroughness and include right-sided leads and posterior leads as, and when, necessary. A systematic approach is used for 12 Lead electrocardiogram analysis and a comprehensive exploration of, for example, myocardial infarction, bundle branch blocks with a variety of ECG examples are included for analysis.

The Nursing and Midwifery Council (2002a) supports lifelong learning for nurses and midwives. It is crucial that practitioners keep abreast of new developments in practice, treatments and technology. Nurses must take responsibility for their own learning and cultivate an enquiring approach to identify any deficits in personal knowledge or skills that require development (Nursing and Midwifery Council 2002). The code of conduct stipulates that as a registered nurse or midwife, one must maintain professional knowledge and competence (Nursing & Midwifery Council 2002b).

I believe that a knowledgeable nurse, highly skilled in arrhythmia and 12 Lead ECG interpretation and management, who is not afraid to challenge or indeed admit limitations, works with the multidisciplinary team to ensure that patients receive the care they deserve.

I congratulate Philip Jevon for his systematic and very readable approach to ECG interpretation; it is so often seen as a highly complex and incomprehensible topic.

I hope that you enjoy this book as much as I have, and that you also enjoy your lifelong learning journey in pursuit of excellence in care.

Cynthia Curtis
Senior Lecturer
Northumbria University

Department of Health (2000) *National Service Framework for Coronary Heart Disease*. The Stationery Office, London.

Jowett, N.I., Thompson, D.R. (2003) *Comprehensive Coronary Care*, 3rd edn. Baillière Tindall, Edinburgh.

National Institute for Clinical Excellence (2002) *Guidance on the use of drugs for early thrombolysis in the treatment of acute myocardial infarction*. National Institute of Clinical Excellence, London.

Nursing & Midwifery Council (2002a) *Supporting nurses and midwives through lifelong learning*. Nursing & Midwifery Council Publications, London.

Nursing & Midwifery Council (2002b) *Code of professional conduct*. Nursing & Midwifery Council Publications, London.

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The Conduction System in the Heart

1

INTRODUCTION

The conduction system in the heart comprises specialised cardiac cells, which initiate and conduct impulses, providing a stimulus for myocardial contraction. 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 OBJECTIVES

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;
- ❏ describe the normal ECG and how it relates to cardiac contraction;
- ❏ recognise normal sinus rhythm.

BASIC PRINCIPLES OF CARDIAC ELECTROPHYSIOLOGY

Depolarisation

Depolarisation is the stimulation of the cardiac cell. 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 and QRS complexes.

Repolarisation

Repolarisation is the process by which the cardiac cell returns to its original resting state. Ventricular repolarsiation is represented on the ECG as T waves (atrial repolarisation is not visible on the ECG as it is masked by the QRS complex).

Automaticity

Automaticity is the ability of specialised cardiac cells (automatic or pacemaker cells) to initiate electrical impulses without any external stimulation. The sinus node normally has the fastest firing rate and therefore assumes the role of pacemaker for the heart. If another focus in the heart has a faster firing rate, it will then take over as pacemaker.

Cardiac action potential

Cardiac action potential (Fig. 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, i.e. from the beginning of the P wave to the end of the T wave.

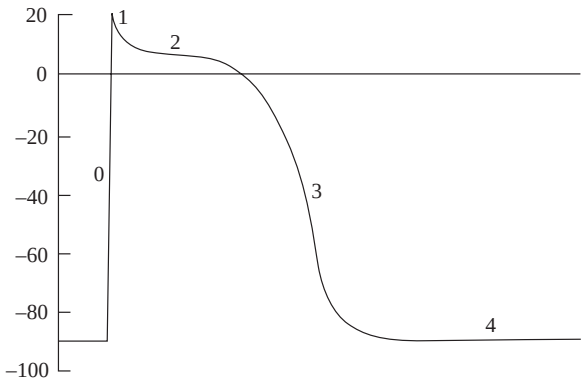

Fig. 1.1 Action potential of cardiac cells (reproduced by kind permission of Scutari Press)

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 level 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 (Fig. 1.2). Automatic cells can initiate an impulse spontaneously without an external impulse.

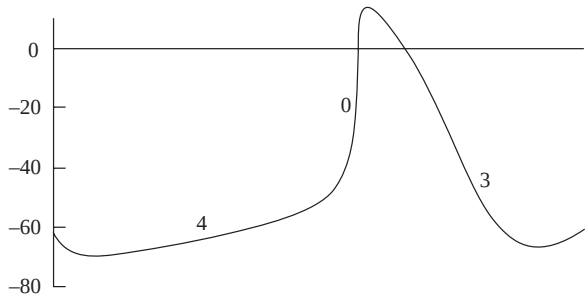

Fig. 1.2 Action potential of automatic or pacemaker cells (reproduced by kind permission of Scutari Press)

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. In normal circumstances, the automatic cells in the SA node have the shortest spontaneous depolarisation time (phase 4) and therefore the quickest firing rate (Julian & Cowan 1993). This is usually about 80 times per minute.

In the atrioventricular junction (AV node and bundle of His), the firing rate is approximately 60 times per minute and in the ventricles 30–40 times per minute. If the SA node firing rate decreases, e.g. a possible complication following an acute inferior myocardial infarction, a subsidiary pacemaker will (hopefully) 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 cells form the conduction system (Fig. 1.3) which comprises the following:

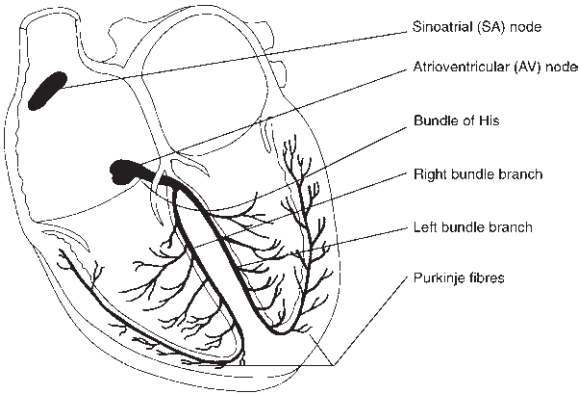

Fig. 1.3 The conduction system in the heart (reproduced by kind permission of Medicotest, manufacturer of 'blue sensor electrodes')

- *Sinoatrial (SA) node or pacemaker*: situated at the junction of the right atrium and superior vena cava. 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).
- *AV junction (AV node and bundle of His)*: this acts as a 'bridge' connecting the atria to the ventricles. 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).
- *Right and left bundle branches*: the left bundle branch divides into the posterior and anterior fascicles. Blood supply is via the left anterior descending artery (Jowett & Thompson 1995).
- *Purkinje fibres*.

Control of heart rate

The heart rate is controlled by the cardiac centre in the medulla through the autonomic nervous system (Green 1991):

- *Parasympathetic or vagus nerve*: 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*: 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.

THE ECG AND ITS RELATION TO CARDIAC CONTRACTION

ECGs relating to the phases of cardiac contraction are shown in Fig. 1.4.

(1) The SA node fires and the electrical impulse spreads across the atria. This results in atrial contraction (P wave).

(2) On arriving at the AV node the impulse is delayed, allowing the atria time to fully contract and eject blood into the ventricles. This brief period of absent electrical activity is represented on the ECG by a straight (isoelectric) line between the end of the P wave and the beginning of the QRS complex. The PR interval represents atrial depolarisation and the impulse delay in the AV node prior to ventricular depolarisation.

(3) The impulse is then conducted down to the ventricles through the bundle of His, right and left bundle branches and Purkinje fibres causing ventricular depolarisation and contraction (QRS complex).

(4) The ventricles then repolarise (T wave).

NORMAL SINUS RHYTHM

Sinus rhythm is the normal rhythm of the heart. The impulse originates in the SA node (i.e. 'sinus') at a regular rate of 60–100 per minute. Each impulse is conducted down the normal pathways to the ventricles

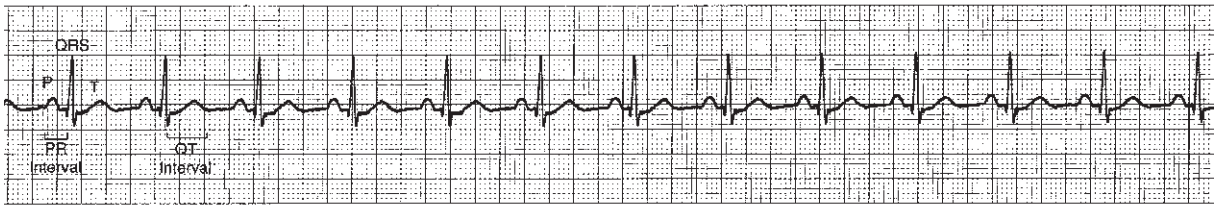

Fig. 1.4 The ECG and its relation to cardiac contraction

without any abnormal conduction delays. It may sometimes present at a cardiac arrest, but without a resultant cardiac output (pulseless electrical activity) (see pages 127–8).

Identifying features on the ECG

- *QRS rate*: 60–100 per min.
- *QRS rhythm*: regular.
- *QRS complexes*: normal width and morphology.
- *P waves*: present and of constant morphology.
- *Relationship between P waves and QRS complexes*: each P wave is followed by a QRS complex and each QRS complex is proceeded by a P wave. PR interval normal and constant.

CHAPTER SUMMARY

The conduction system in the heart comprises specialised cardiac cells, which initiate and conduct

impulses, providing a stimulus for myocardial contraction.

The 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.

REFERENCES

Green, J. (1991) *An Introduction to Human Physiology*. Oxford Medical Publications, Oxford.

Jowett, N.I. & Thompson, D.R. (1995) *Comprehensive Coronary Care*, 2nd edn. Scutari Press, London.

Julian, D. & Cowan, J. (1993) *Cardiology*, 6th edn. Baillière, London.

Thompson, P. (1997) *Coronary Care Manual*. Churchill Livingstone, London.

Principles of Cardiac Monitoring

2

INTRODUCTION

Cardiac monitoring is one of the most valuable diagnostic tools in modern medicine. It is essential if disorders of the cardiac rhythm are to be recognised. It can help with diagnosis and can alert healthcare staff to changes in the patient's condition.

Cardiac monitoring must be meticulously undertaken. Potential consequences of poor technique include misinterpretation of cardiac arrhythmias, mistaken diagnosis, wasted investigations and mismanagement of the patient (Jevon 2000).

The aim of this chapter is to understand the principles of cardiac monitoring.

LEARNING OBJECTIVES

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

- ❏ list the indications for cardiac monitoring;
- ❏ state the common features of a cardiac monitor;
- ❏ describe the positioning of ECG electrodes;
- ❏ discuss the rationale for selecting particular ECG monitoring leads;
- ❏ describe the procedure for cardiac monitoring;
- ❏ discuss the problems associated with cardiac monitoring;
- ❏ outline what the ECG trace records;
- ❏ outline the principles of exercise testing.

INDICATIONS FOR CARDIAC MONITORING

Cardiac monitoring (Fig. 2.1) is required in a variety of clinical situations including:

- chest pain;
- myocardial infarction;
- shock;
- heart failure;
- palpitations;
- history of syncope;
- during CPR.

Jevon 2002

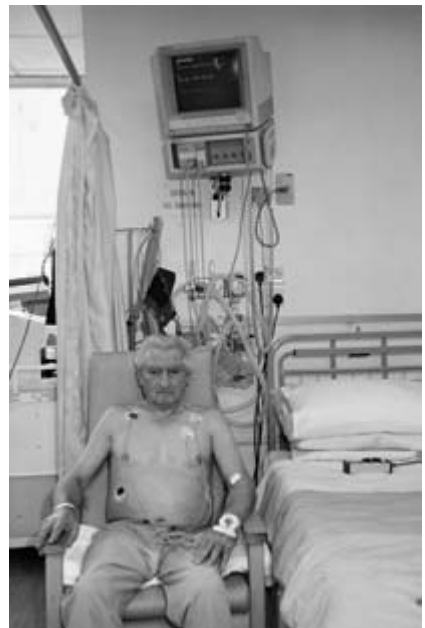

Fig. 2.1 Cardiac monitoring

COMMON FEATURES OF A CARDIAC MONITOR

The bedside cardiac monitor (Fig. 2.2) or oscilloscope provides a continuous display of the patient's ECG and has the following common features:

- *Screen for displaying the ECG trace*, a dull/bright switch can be adjusted if the screen is too light or too dark.

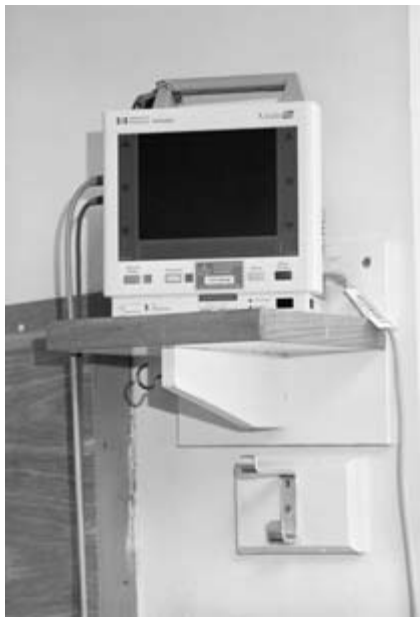

Fig. 2.2 Bedside cardiac monitor

- *ECG printout facility*, to record cardiac arrhythmias (invaluable for both diagnosis and record keeping purposes).
- *Heart rate counter*, to calculate the heart rate (counts the QRS complexes).
- *Monitor alarms*, to alert the healthcare professional to changes in heart rate to outside pre-set limits. Some cardiac monitors can identify important cardiac arrhythmias and changes in the ST segment, and alarm accordingly.
- *Lead select switch*, to select the desired monitoring lead, e.g. lead II.
- *ECG gain*, to alter the size of the ECG complex. If it is set too low or too high the ECG trace can become unrecognisable, either too small or distorted (Fig. 2.3), leading to the possibility of misinterpretation.

POSITIONING OF ECG ELECTRODES

The correct positioning of ECG electrodes is crucial for obtaining accurate information from any monitoring lead (Jacobson 2000). Whether a three or a five ECG

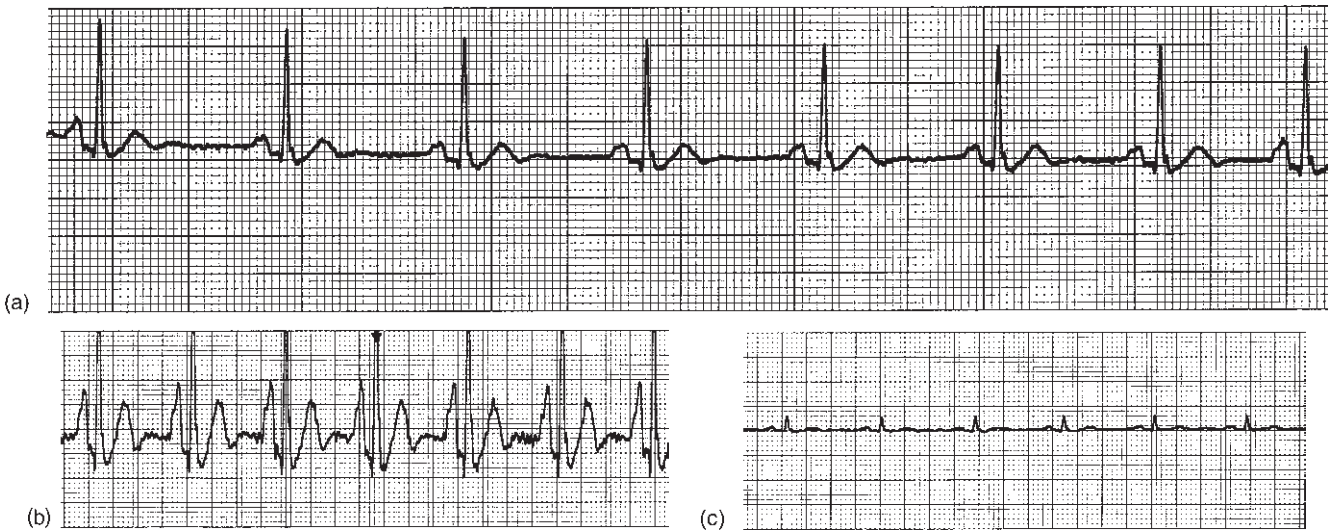

Fig. 2.3 Effects of incorrect ECG gain setting. (a) 10 mm/1 mV – standard setting; ECG complexes adequate size. (b) 40 mm/1 mV – ECG complexes too large; cardiac monitor mistook the P and T waves for QRS complexes resulting in continuous false-high heart rate alarms. (c) 2 mm/1 mV – ECG complexes too small; cardiac monitor did not recognise QRS complexes resulting in continuous false asystole alarms. Also difficulties may be encountered interpreting cardiac arrhythmias when the gain is set too low

cable monitoring system is used will depend upon the patient, the desired monitoring lead(s), local protocols and manufacturer's recommendations.

Three ECG cable system

The standard positioning of ECG electrodes when using a three ECG cable monitoring system is:

- red ECG cable: below the right clavicle;
- yellow ECG cable: below the left clavicle;
- green ECG cable: left lower thorax/hip region.

This electrode position enables the monitoring of lead II. When establishing cardiac monitoring during cardiopulmonary resuscitation (CPR), it is recommended to slightly modify this position: red ECG cable on the right shoulder, yellow ECG cable on the left shoulder and green ECG cable on the left abdominal wall; the precordium should be left unobstructed so as not to hinder defibrillation if it is required (Resuscitation Council UK 2000).

Some clinical areas prefer to monitor on MCL1 (modified chest lead 1, i.e. V1) as recommended by Marriott (1988). The positioning of ECG electrodes is:

- black ECG cable: right shoulder;
- red ECG cable: left shoulder;
- yellow ECG cable: 4th intercostal space, just to the right of the sternum, i.e. corresponding to V1.

Five ECG cable system

Cardiac monitoring using a five ECG cable system is becoming more popular. The main advantage of this system is that different ECG leads can be monitored simultaneously. This is particularly useful when analysing cardiac arrhythmias as it provides an alternative view of the waveform. The standard positioning for ECG electrodes is illustrated in Fig. 2.4 and is as follows:

- RA (red ECG cable): below the right clavicle;
- LA (yellow ECG cable): below the left clavicle;
- RL (black ECG cable): right lower thorax/hip region;

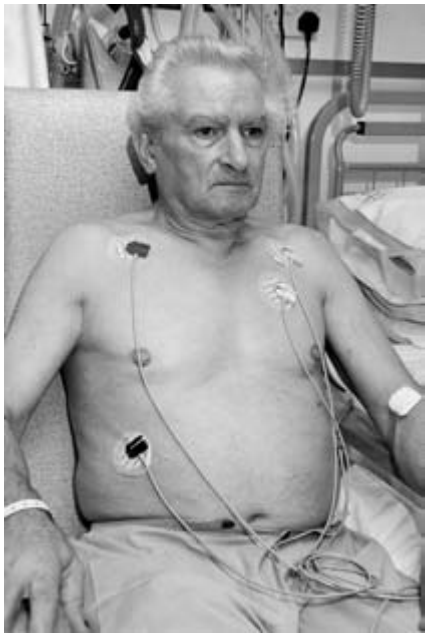

Fig. 2.4 Suggested ECG electrode placement when using a five cable monitoring system

- LL (green ECG cable): left lower thorax/hip region;
- (white ECG cable): on the chest in the desired V position, usually V1 (MCL1, 4th intercostal space just right of the sternum).

Jacobson 2000

EASI 12 lead ECG monitoring

The conventional 12 lead ECG using ten electrodes attached to the limbs and chest is recognised as the current medical standard for the identification, analysis and confirmation of many cardiac abnormalities including cardiac arrhythmias and cardiac ischaemia/infarction.

If 12 lead ECG monitoring is undertaken on a continual basis, the benefits include:

- facilitating the accurate recognition of cardiac arrhythmias;
- enabling the monitoring of the mid-precordial leads which is particularly important for the detection and management of ischaemia;