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42 Aero Camino, Goleta, CA 93117
www.biopac.com

Biopac Student Lab® Lesson 5

ELECTROCARDIOGRAPHY (ECG) I
Introduction

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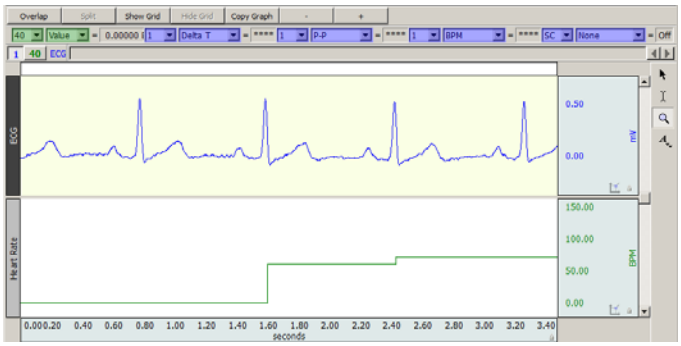
Richard Pflanzer, Ph.D.
Associate Professor Emeritus
Indiana University School of Medicine
Purdue University School of Science

William McMullen
Vice President, BIOPAC Systems, Inc.



I. INTRODUCTION

The main function of the heart is to pump blood through two circuits:

1. **Pulmonary circuit**: through the lungs to oxygenate the blood and remove carbon dioxide; and
2. **Systemic circuit**: to deliver oxygen and nutrients to tissues and remove carbon dioxide.

Because the heart moves blood through two separate circuits, it is sometimes described as a dual pump.

In order to beat, the heart needs three types of cells:

1. Rhythm generators, which produce an electrical signal (SA node or normal pacemaker);
2. Conductors to spread the pacemaker signal; and
3. Contractile cells (myocardium) to mechanically pump blood.

The Electrical and Mechanical Sequence of a Heartbeat

The heart has specialized **pacemaker** cells that start the electrical sequence of **depolarization** and **repolarization**. This property of cardiac tissue is called **inherent rhythmicity** or **automaticity**. The electrical signal is generated by the **sinoatrial node (SA node)** and spreads to the ventricular muscle via particular conducting pathways: **internodal pathways** and **atrial fibers**, the **atrioventricular node (AV node**,) the **bundle of His**, the right and left **bundle branches**, and **Purkinje fibers** (Fig. 5.1).

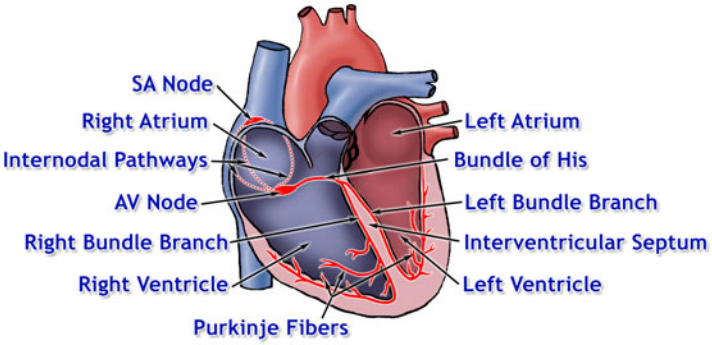


Fig. 5.1 The Heart

When the electrical signal of a depolarization reaches the contractile cells, they contract—a mechanical event called **systole**. When the repolarization signal reaches the myocardial cells, they relax—a mechanical event called **diastole**. Thus, the electrical signals cause the mechanical pumping action of the heart; mechanical events always follow the electrical events (Fig. 5.2).

The **SA node** is the normal pacemaker of the heart, initiating each electrical and mechanical cycle. When the SA node depolarizes, the electrical stimulus spreads through atrial muscle causing the muscle to contract. Thus, the SA node depolarization is followed by atrial contraction.

The SA node impulse also spreads to the **atrioventricular node (AV node**) via the **internodal fibers**. (The wave of depolarization does not spread to the ventricles right away because there is nonconducting tissue separating the atria and ventricles.) The electrical signal is delayed in the AV node for approximately 0.20 seconds when the atria contract, and then the signal is relayed to the **ventricles** via the **bundle of His, right** and **left bundle branches**, and **Purkinje fibers**. The Purkinje fibers relay the electrical impulse directly to ventricular muscle**,** stimulating the ventricles to **contract** (ventricular **systole**). During ventricular systole, ventricles begin to repolarize and then enter a period of diastole (Fig. 5.2).

Although the heart generates its own beat, the heart rate (beats per minute or BPM) and strength of contraction of the heart are modified by the **sympathetic** and **parasympathetic** divisions of the autonomic nervous system.

- The sympathetic division increases automaticity and excitability of the SA node, thereby increasing heart rate. It also increases conductivity of electrical impulses through the atrioventricular conduction system and increases the force of atrioventricular contraction. Sympathetic influence increases during inhalation.
- The parasympathetic division decreases automaticity and excitability of the SA node, thereby decreasing heart rate. It also decreases conductivity of electrical impulses through the atrioventricular conduction system and decreases the force of atrioventricular contraction. Parasympathetic influence increases during exhalation.

The Electrocardiogram (ECG)

Just as the electrical activity of the pacemaker is communicated to the cardiac muscle, "echoes" of the depolarization and repolarization of the heart are sent through the rest of the body. By placing a pair of very sensitive receivers (**electrodes**) on other parts of the body, the echoes of the heart's electrical activity can be detected. The record of the electrical signal is called an **electrocardiogram (ECG).** You can infer the heart's mechanical activity from the ECG. Electrical activity varies through the ECG cycle as shown below (Fig. 5.2):

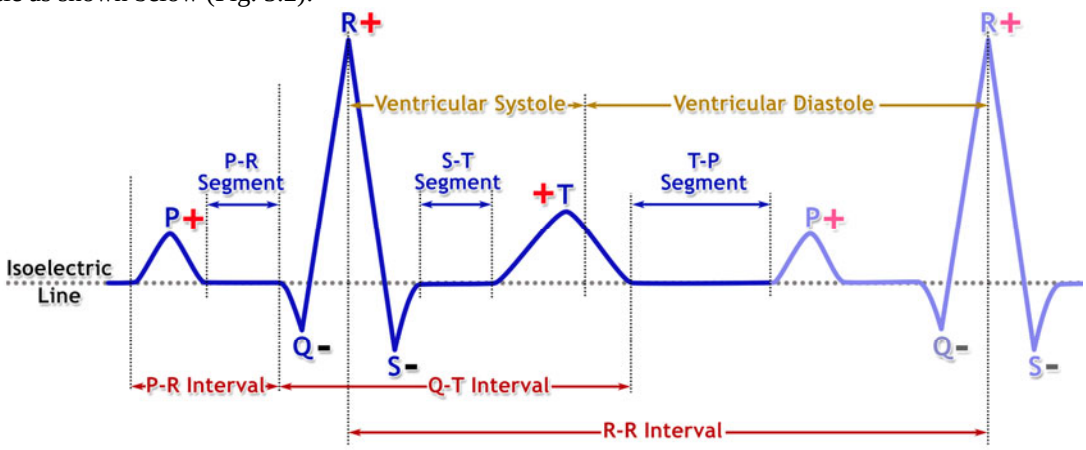


Fig. 5.2 Components of the ECG & Electrical and mechanical events of the cardiac cycle

Because the ECG reflects the electrical activity, it is a useful "picture" of heart activity. If there are interruptions of the electrical signal generation or transmission, the ECG changes. These changes can be useful in diagnosing changes within the heart. During exercise, however, the position of the heart itself changes, so you cannot standardize or quantify the voltage changes.

Components of the ECG

The electrical events of the heart (ECG) are usually recorded as a pattern of a baseline (isoelectric line,) broken by a **P** wave, a **QRS** complex, and a **T** wave. In addition to the wave components of the ECG, there are intervals and segments (Fig. 5.2).

- The **isoelectric line** is a point of departure of the electrical activity of depolarizations and repolarizations of the cardiac cycles and indicates periods when the ECG electrodes did not detect electrical activity.
- An **interval** is a time measurement that includes waves and/or complexes.
- A **segment** is a time measurement that does not include waves and/or complexes.

Table 5.1 Components of the ECG & Typical Lead II Values*

ECG COMPONENT		*Measurement area…*	*Represent…*	**Duration** *(seconds)*	**Amplitude** (millivolts)
Waves	**P**	begin and end on isoelectric line (baseline); normally upright in standard limb leads	depolarization of the right and left atria.	0.07 – 0.18	< 0.25
	QRS complex	begin and end on isoelectric line (baseline) from start of Q wave to end of S wave	depolarization of the right and left ventricles. Atrial repolarization is also part of this segment, but the electrical signal for atrial repolarization is masked by the larger QRS complex (see Fig. 5.2)	0.06 – 0.12	0.10 – 1.50
	T	begin and end on isoelectric line (baseline)	repolarization of the right and left ventricles.	0.10 – 0.25	< 0.5
Intervals	**P-R**	from start of P wave to start of QRS complex	time from the onset of atrial depolarization to the onset of ventricular depolarization.	0.12-0.20	
	Q-T	from start of QRS complex to end of T wave	time from onset of ventricular depolarization to the end of ventricular repolarization. It represents the refractory period of the ventricles.	0.32-0.36	
	R-R	from peak of R wave to peak of succeeding R wave	time between two successive ventricular depolarizations.	0.80	
Segments	**P-R**	from end of P wave to start of QRS complex	time of impulse conduction from the AV node to the ventricular myocardium.	0.02 – 0.10	
	S-T	between end of S wave and start of T wave	period of time representing the early part of ventricular repolarization during which ventricles are more or less uniformly excited.	< 0.20	
	T-P	from end of T wave to start of successive P wave	time from the end of ventricular repolarization to the onset of atrial depolarization.	0.0 – 0.40	

* Notes: Tabled values represent results from a typical Lead II setup (wrist and ankle electrode placement) with Subject heart rate ~75 BPM. Values are influenced by heart rate and placement; values for torso placement would be different.

Leads

The particular arrangement of two electrodes (one **positive**, one **negative**) with respect to a third electrode (the **ground**) is called a **lead**. The electrode positions for the different leads have been standardized. For this lesson, you will record from **Lead II**, which has a positive electrode on the left ankle, a negative electrode on the right wrist, and the ground electrode on the right ankle. Typical Lead II values are shown in Table 5.1.

The dominant ECG component in any normal standard lead record is the QRS complex. Usually, in a Lead II record the Q and S waves are small and negative and the R wave is large and positive as shown in Fig. 5.2. However, it is important to note many factors, normal and abnormal, determine the duration, form, rate, and rhythm of the QRS complex.

- Normal factors include body size (BSA) and distribution of body fat, heart size (ventricular mass,) position of the heart in the chest relative to lead locations, metabolic rate, and others.

For example, in a person who has a high diaphragm, the apex of the heart may be shifted slightly upward and to the person's left. This change in the position of the heart alters the "electrical picture" of ventricular depolarization seen by the Lead II electrodes, resulting in decreased positivity of the R wave and increased negativity of the S wave. In other words, the positive amplitude of the R wave decreases and the negative amplitude of the S wave increases.

Similar changes in the Lead II QRS complex may be observed in a person, an athlete for example, who has no cardiac disease but does have a larger than normal left ventricular mass. In fact the decrease in R wave positivity coupled with the increase in S wave negativity may be so extreme as to give rise to the mistaken impression that the R wave has become inverted, when in reality the inverted spike is an enlarged S wave preceded by a much smaller but still positive R wave. When the amplitudes of Lead II Q, R, and S waves are all negative, the result is an abnormal inverted QRS complex.

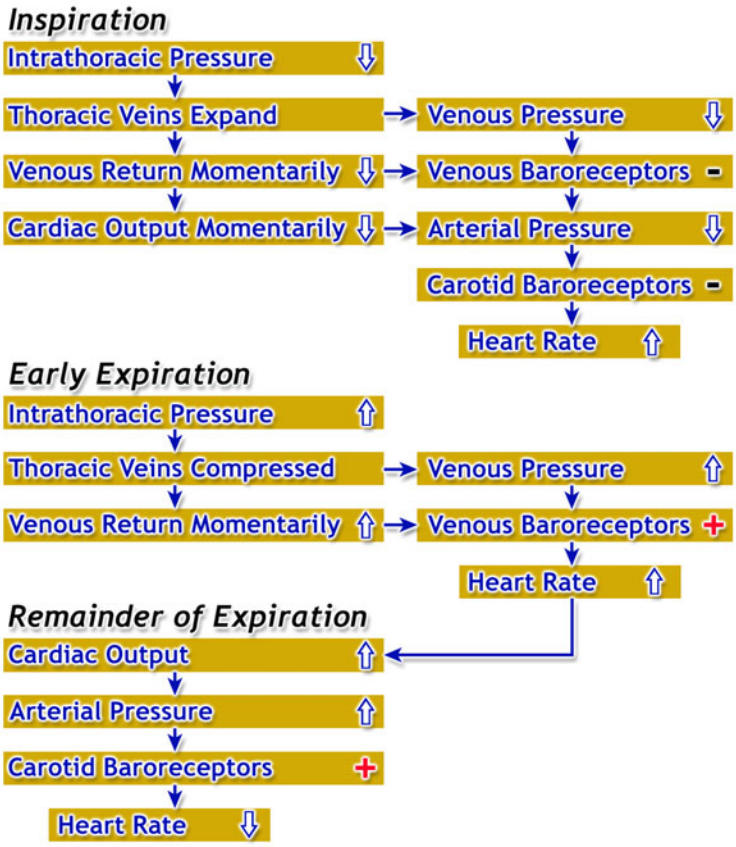
- Abnormal factors include hyper- and hypothyroidism, ventricular hypertrophy (observed for example, in chronic valvular insufficiency,) morbid obesity, essential hypertension and many other pathologic states. A more detailed discussion of QRS changes in response to normal and abnormal factors requires an introduction to cardiac vectors, for which the reader is referred to Lesson 6.

Effects of the Resting Respiratory Cycle on Heart Rate

Temporary minor increases and decreases in heart rate associated with the resting respiratory cycle reflect heart rate adjustments made by systemic arterial and systemic venous pressure receptor (baroreceptor) reflexes in response to the cycling of intrathoracic pressure (Fig. 5.4).

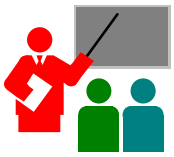
When inspiratory muscles contract, pressure within the thorax (intrathoracic pressure) decreases, allowing thoracic veins to slightly expand. This causes a momentary drop in venous pressure, venous return, cardiac output, and systemic arterial blood pressure. The carotid sinus reflex normally decreases heart rate in response to a rise in carotid arterial blood pressure. However, the momentary drop in systemic arterial blood pressure during inspiration reduces the frequency of carotid baroreceptor firing, causing a momentary increase in heart rate.

When inspiratory muscles relax, resting expiration passively occurs. During early resting expiration, intrathoracic pressure increases causing compression of thoracic veins, momentarily increasing venous pressure and venous return. In response, systemic venous baroreceptors reflexively increase heart rate. However, the slight increase in heart rate is temporary because it increases cardiac output and systemic arterial blood pressure, which increases carotid baroreceptor firing causing heart rate to decrease.



Source: Richard Pflanzer, Ph.D., Associate Professor
Indiana University School of Medicine, Purdue University School of Science

Fig. 5.3 Effects of the Resting Respiratory Cycle on Heart Rate



The average resting heart rate for adults is between 60-80 beats/min. (Average 70 bpm for males and 75 bpm for females.) Slower heart rates are typically found in individuals who regularly exercise. Athletes are able to pump enough blood to meet the demands of the body with resting heart rates as low as 50 beats/min. Athletes tend to develop larger hearts, especially the muscle in the left ventricle—a condition known as "left ventricular hypertrophy." Because athletes (usually) have larger and more efficient hearts, their ECGs may exhibit differences other than average resting heart rate. For instance, low heart rate and hypertrophy exhibited in sedentary individuals can be an indication of failing hearts but these changes are "normal" for well-trained athletes.

Because ECGs are widely used, basic elements have been standardized to simplify reading ECGs. ECGs have standardized grids of lighter, smaller squares and, superimposed on the first grid, a second grid of darker and larger squares (Fig. 5.4). The smaller grid always has time units of 0.04 seconds on the x-axis and the darker vertical lines are spaced 0.2 seconds apart. The horizontal lines represent amplitude in mV. The lighter horizontal lines are 0.1 mV apart and the darker grid lines represent 0.5 mV. In this lesson, you will record the ECG under four conditions.

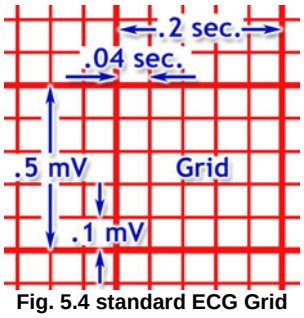


Fig. 5.4 standard ECG Grid