MEDICAL ELECTRONICS

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The goal of the course is to learn how to develop solutions to measurement problems, in particular non−invasive measurements of physiological parameters. The course will introduce some basic physiology (underlining the importance of understanding what is being measured and why) and will cover in detail the electronic implementation of solutions to measurement problems. Discussion of the balance between hardware and signal processing will be included where appropriate.

We will cover four devices currently in use for measuring physiological parameters:

1. The Electrocardiogram (ECG)
   - Basic physiology of the heart and its electrical activity
   - Electrodes and the conversion of ionic currents to electrical currents
   - ECG instrumentation amplifiers
   - Noise reduction using the "driven right leg" circuit
2. Respiration measurement using Impedance Plethysmography
   - Electrical impedance changes of the chest cavity due to breathing and blood flow
   - 4−electrode method of measuring electrical impedance
   - 2−electrode measurement principle, amplifier and signal processing circuit
   - Limitations of 2−electrode method
3. Oxygen Saturation using Pulse Oximetry
   - The optical characteristics of oxygenated and deoxygenated blood
   - Principles of pulse oximetry
   - Electronic implementation of subsystems for pulse oximetry, including separation of the d.c. and a.c. components.
   - Detailed circuits for pulse oximetry: constant−current source, current−voltage converter, amplifiers.
4. Non Invasive Blood Pressure
   - The physiology of blood transmission and pressure
   - Theory and circuitry for three methods:
     1. The traditional method: Korotkoff sounds
     2. The current method: Oscillometry
     3. The future: beat−by−beat measurement using Pulse Transit Time.

The electrocardiogram (ECG)

If a pair of surface electrodes, attached to the left and right arms of a human subject, are connected to a high impedance differential amplifier, an electrical signal which varies in time with the heart beat will be observed at the output of the amplifier (see Figure 1). This signal, which has a peak amplitude, before amplification, of the order of 1mV, is known as the electrocardiogram (ECG).

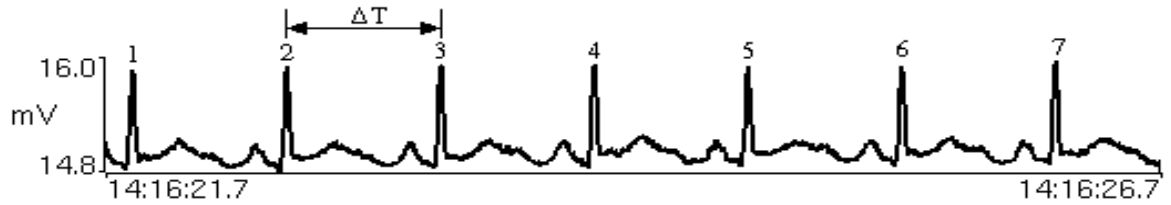

Figure 1: An ECG trace. 1, 2, 3 ... are consecutive heart beats. The heart rate, in beats per minute, is 60/T

What is the origin of the ECG signal? Before we can answer that question, we need to consider briefly some of the physiological background.

Basic physiology of the heart and its electrical activity

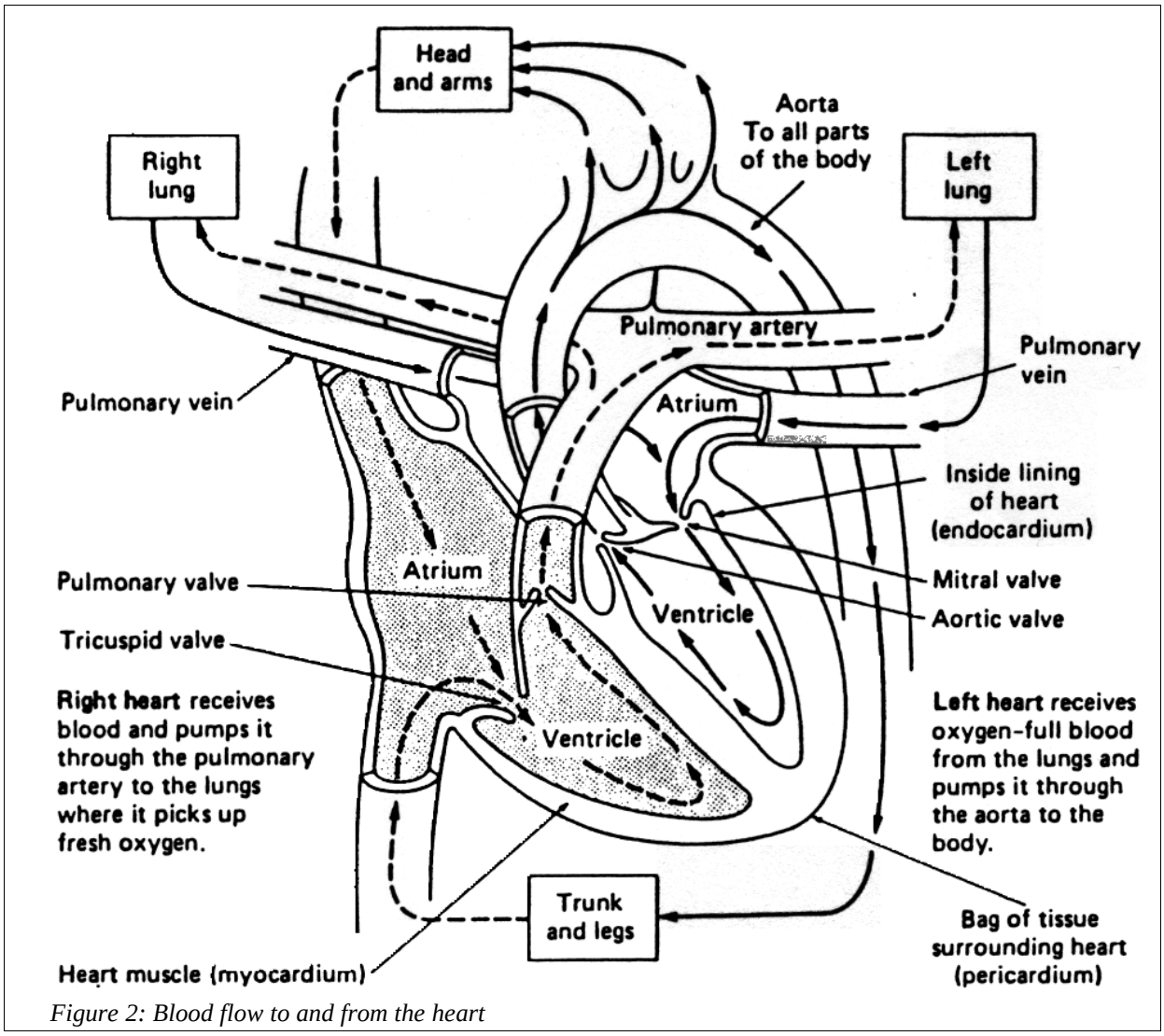

Figure 2: Blood flow to and from the heart

The heart is a four chambered pump which provides the driving force for the circulation of blood around the body. A wall divides the heart cavity into a double−pump configuration,

each side being further divided into an upper chamber, the *atrium*, and a lower chamber, the *ventricle* (see Figure 2). The main pumping function is supplied by the ventricles and the atria are merely antechambers to store blood during the time the ventricles are pumping. The resting or filling phase of the heart cycle is referred to as *diastole*; the contractile or pumping phase is called *systole*.

Blood returns to the right heart after it has delivered nutrients and oxygen to the cells of the body. This blood is pumped to the lungs where the waste gas (carbon dioxide) is expired and new oxygen is picked up for cellular use. The left heart receives the oxygenated blood from the lungs and then pumps it out through the aorta for distribution by smaller arteries to all parts of the body.

Cell Potentials

The heart is made up of different types of muscle. Muscle cells share an important property with nerve cells: they both have *excitable* membranes. The cell membrane is the dividing medium between the extracellular and intracellular fluids which have different ionic concentrations. The membrane has a different permeability to the various ions and therefore acts as a selective ionic filter.

The principal ions involved in the mechanism of producing cell potentials are Na^+, K^+ and Cl^-. *Metabolic* energy is used to drive 'pumps' in the membrane which actively expel sodium ions from the cell and draw potasium ions into the cell. This results in an internal environment rich in K^+ but low in Na^+ compared with an extracellular component of high Na^+ and low K^+. There are therefore concentration and electric field gradients caused by the uneven distribution of charge. The different concentrations of potassium are sustained because the electical field and concentration gradients have equal and opposite effects. There is, however, a small but nonzero flow of sodium ions which would, over a period of hours, result in a loss of these concentration differences were the pumps not operating. A steady state potential therefore exists across the cell membrane, the inside being some 82 mV negative relative to the outside (see Figure 3). This potential remains constant until the cell dies, is disturbed or is stimulated by some other nerve or muscle cell or, in the case of sensing cells, by an electrical, mechanical, thermal or chemical stimulus.

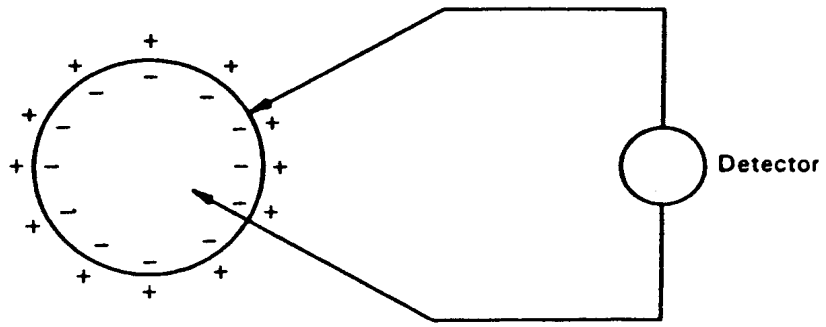

Figure 3: A polarised cell with its resting potential

Cell stimulation

When a cell is stimulated, the permeabilities of the membrane to ionic transfer are modified. The first phase of this modification is a greatly elevated permeability to sodium which results in a large influx of Na^+ into the cell. The ionic current due to the flow of Na^+ into the cell increases and this causes the transmembrane potential to increase from its resting value of −80 mV in an attempt to bring the sodium and potassium currents back into equilibrium. When equilibrium is achieved, the cell membrane potential has risen to approximately +20 mV.

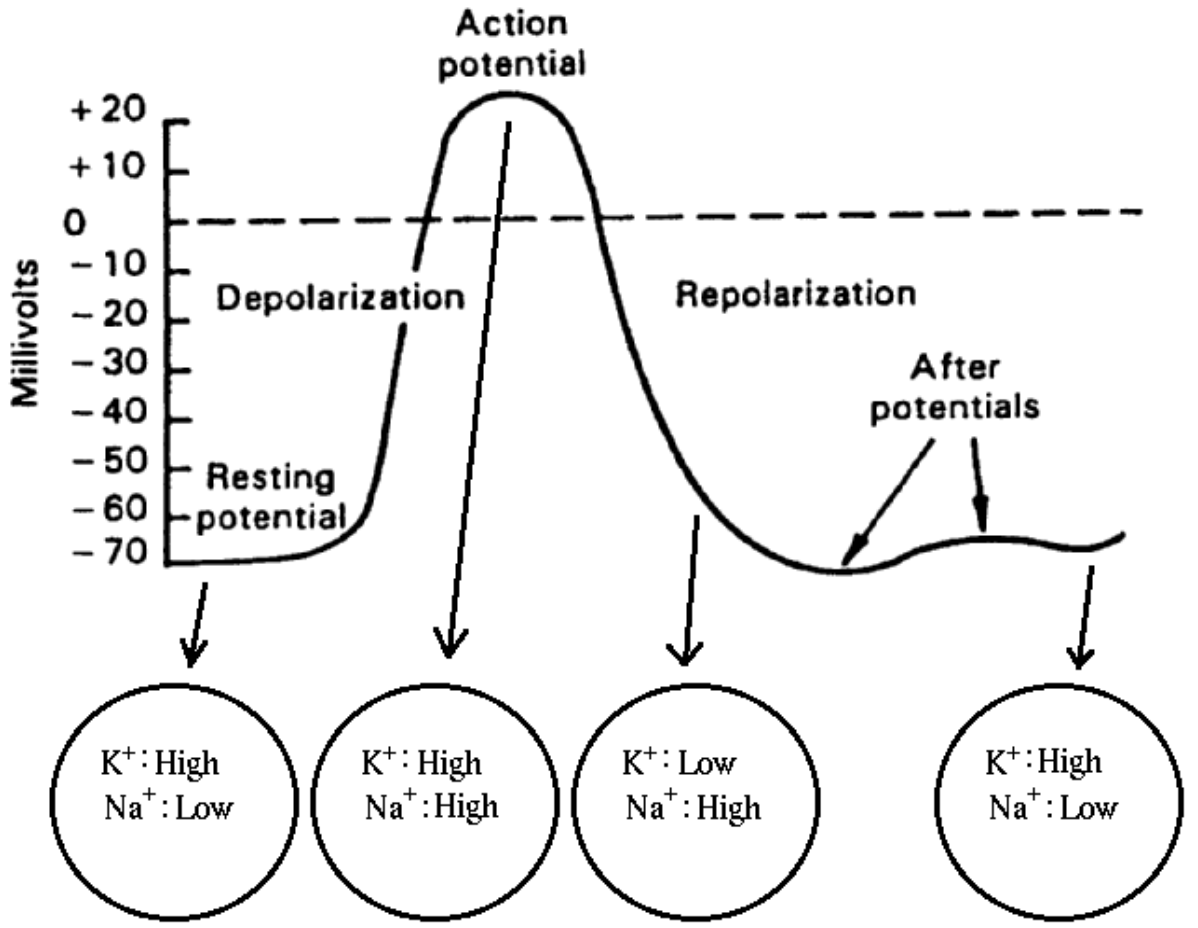

Figure 4: Waveform of an action potential and the relative ionic concentrations in the cell. Note that the timescale varies with the type of cell.

At this point in the cycle the cell is said to be *depolarised* (see Figure 4). The membrane permeability to sodium then returns to normal but the permeability to potassium increases about thirty−fold. K^+ ions diffuse out in sufficient quantity to bring the cell membrane potential back to its resting level (*repolarisation*). Metabolic processes once again take over in order to pump out the Na^+ ions interior to the cell and allow the K^+ ions back in again so that the cell may be brought back to its resting ionic concentrations. Once the membrane has fully recovered, the cell is again at rest and capable of being re−stimulated. The time course of these changes of membrane potential is known as the *action potential*.

All or nothing law

The form and amplitude of the action potential for a given cell is the same regardless of the magnitude of the stimulus, provided that the stimulus is above the threshold value required to produce the action potential.

Cardiac Muscle

In a tissue, the depolarisation disturbance of one cell is propagated to the next until the entire tissue depolarises. In muscle tissue, the action potential causes a mechanical response: the tissue contracts and becomes shorter in length after some delay following a depolarisation.

In mammalian heart tissue, depolarisation lasts about 2ms but repolarisation lasts 200ms or more. The contractile response of the cardiac muscle begins just after the start of depolarisation and lasts about 1.5 times as long as the action potential (see Figure 5).

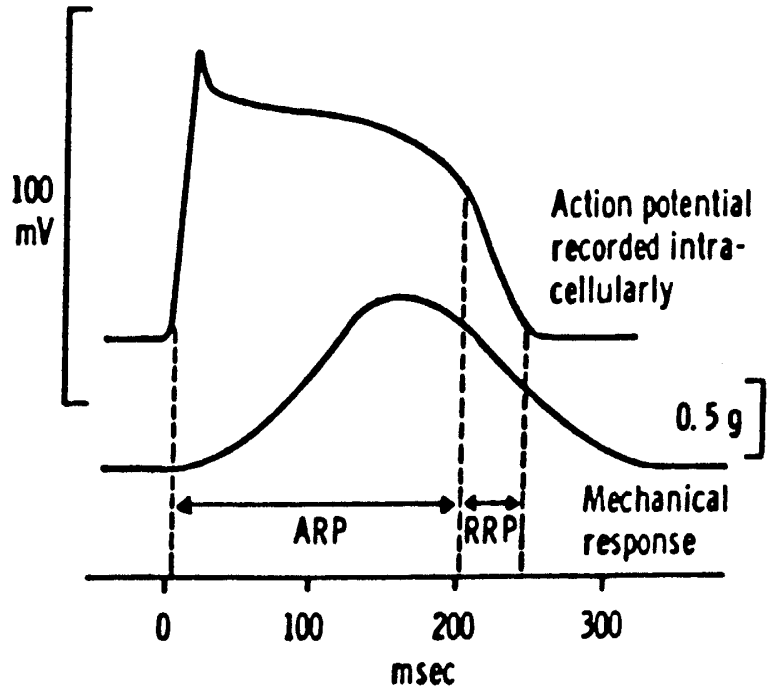

Figure 5: Action potentials and contractile response of mammalian cardiac muscle plotted on the same axis

How are the cardiac muscles cells stimulated in the first place? The answer is to be found by considering a group of cardiac cells known as the *pacemaker* cells.

Pacemaker cells

While the resting potential can be maintained indefinitely in ordinary muscle cells, for pacemaker cells (which are found in a region of the heart known as the sinoatrial (SA) node − see Figure 6), the transmembrane potential *spontaneously* increases because of *ionic leakage* in the smooth muscle membrane. Eventually, the threshold for excitation is reached and so the pacemaker cells are said to be *self−excitatory*. As the SA node cells depolarise they stimulate the adjacent atrial cells, causing them to depolarise. Thus a depolarisation wave spreads over the atria in an outward−travelling wave from the point of origin.[1]

A fibrous barrier of non−excitable cells prevents the propagation of the depolarisation wave from continuing beyond the limits of the atria. The only excitable tissue that crosses this barrier is the *Bundle of His*. At the origin of this bundle is a mass of specialised tissue about 2cm long and 1cm wide called the atrio−ventricular (AV) node. The conduction velocity through the AV node is approximately 0.1m/s, 10% of that of the atrial cells. The transmission delay that this causes in the propagation of the atrial depolarisation wave is the key to the proper time relationship between the atria and the ventricles. This delay is very important as it allows the atria to complete their contraction before there is any ventricular contraction.

Brief Summary (so far)

The contraction of the atria and ventricles is set up by specific patterns of electrical activation in the musculature of these structures. The patterns themselves are initiated by a coordinated series of events in a very localised and specialised conduction system.

1 Such self−excitatory oscillations are not, however, confined to the SA node. Tissue in other regions of the heart, such as the AV node, has the same properties but a lower frequency of oscillation than that of the SA node. The oscillations of the AV node therefore determine the heart rate since it causes stimulation of the other tissue regions before they can reach their self−pacing threshold.

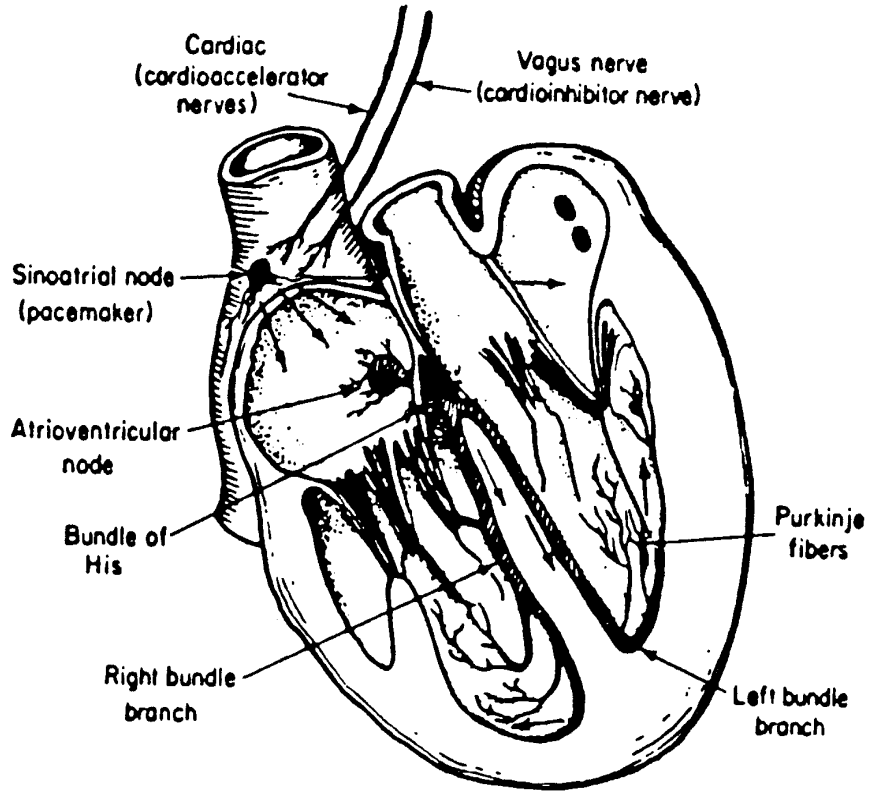

Figure 6: Basic conducting system of the heart

Measurement of the potentials on the body surface

The current densities generated by the membrane activity of the heart muscle cells cause current changes in the surrounding medium. These ionic currents flow in the thorax which can be considered to be a purely passive medium since it contains no other sources or sinks. Thus, to a first approximation, the heart may be modelled as an electrical generator with the thoracic medium as its resistive load. The *electrocardiogram* (ECG), introduced at the beginning of these lecture notes, is a record of the potentials measured at the outer surface of this medium (*ie* on the surface of the body). A typical ECG waveform is shown in Figure 7.

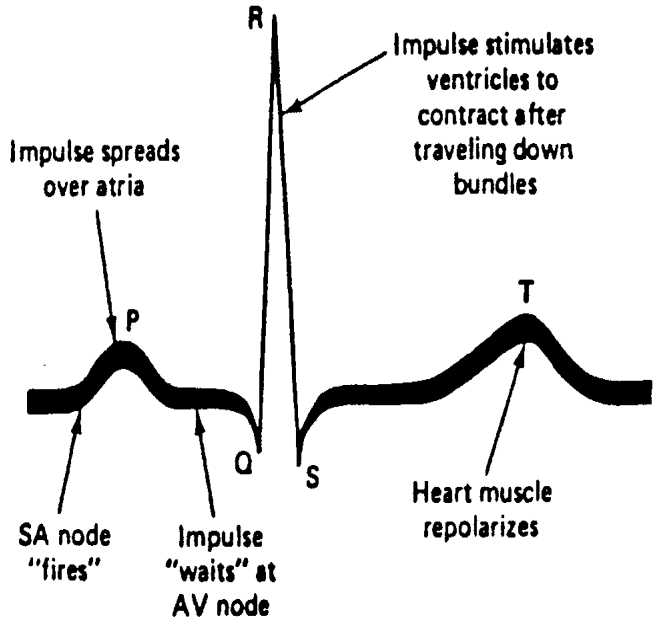

Figure 7: Typical ECG waveform obtained at the body's surface

Description of ECG waveforms

The ECG is characterised by five different segments of the waveform: the P, Q, R, S and T waves. The analysis of these segments gives information on the different events of the cardiac cycle:

- P−wave: a small low−voltage deflection caused by the depolarisation of the atria prior to atrial contraction.
- QRS complex: the largest−amplitude portion of the ECG, caused by currents generated when the ventricles depolarise prior to their contraction. Although atrial repolarisation occurs before ventricular depolarisation, the latter waveform (*ie* the QRS complex) is of much greater amplitude and atrial repolarisation is therefore not seen on the ECG.
- T−wave: ventricular repolaristaion
- P−Q interval: the time interval between the beginning of the P wave and the beginning of the QRS complex. It is the time between the beginning of the atrial contraction and the beginning of ventricular contraction; as such it represents the delay of the electrical impulse from the atria to the ventricles.

Relationship between the ECG and the mechanical activity of the heart

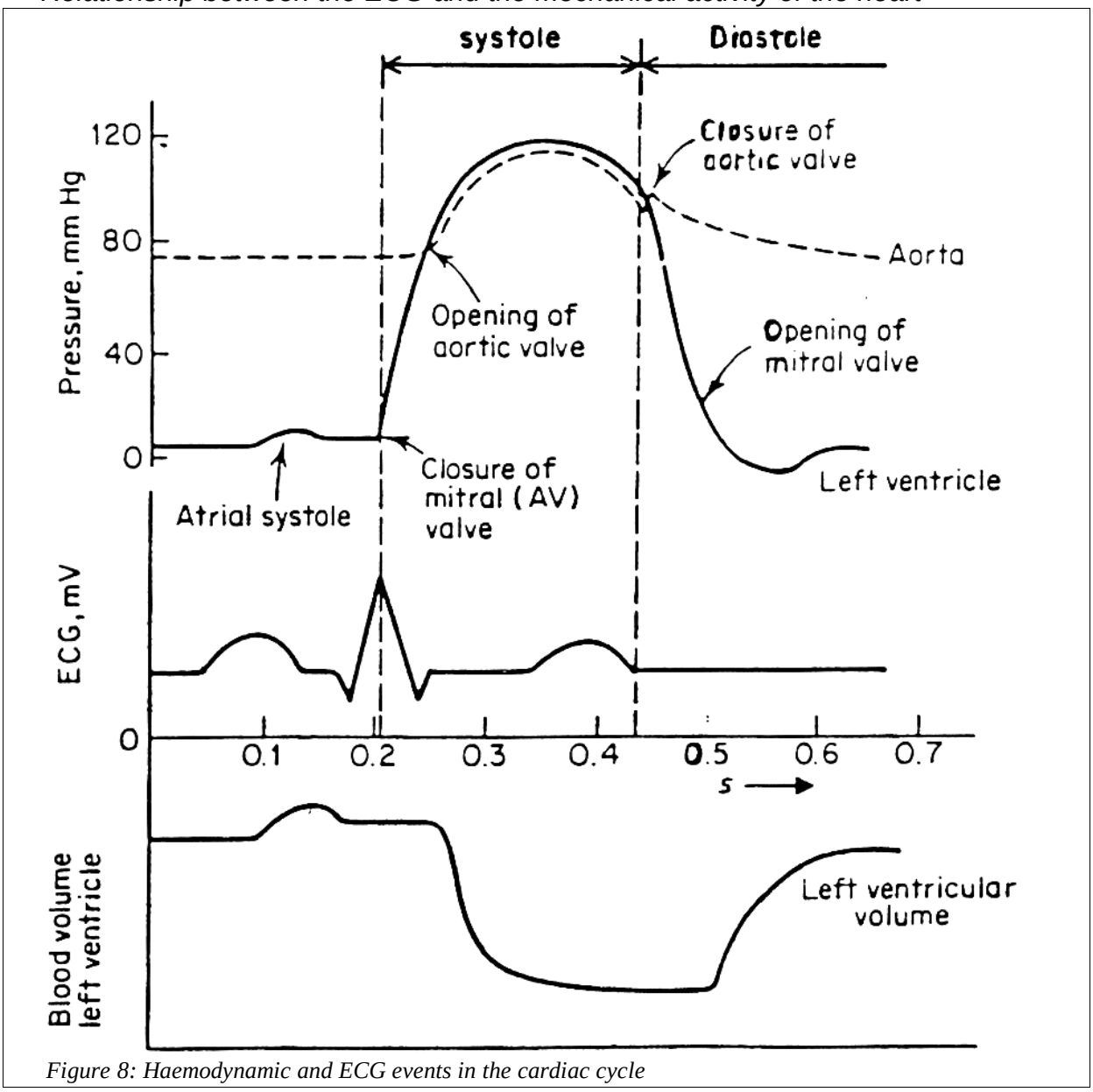

Figure 8: Haemodynamic and ECG events in the cardiac cycle

Obviously enough, the function of the heart is to pump blood. Electrical activity is important only because it initiates muscular contraction. The ECG can nevertheless provide much information to a skilled clinician on the history of a heart attack for example, although it cannot give information about the pumping capacity of the heart except in terms of rhythm. Under normal circumstances, the correlation between electrical and mechanical events is as shown in Figure 8.