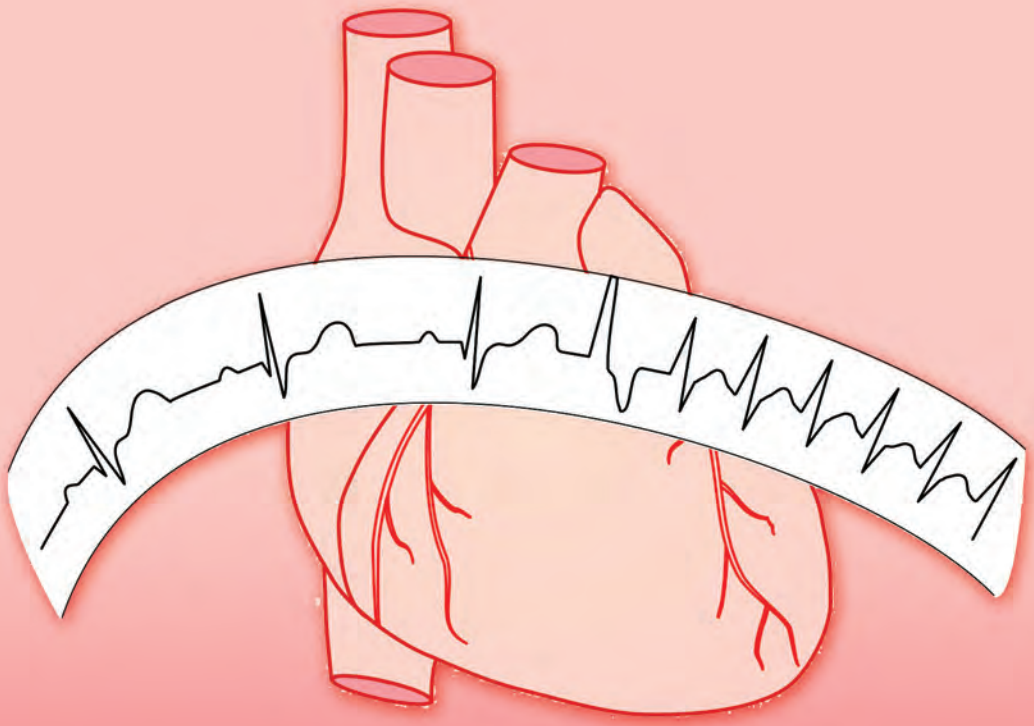
ECG

from Basics to Essentials

Step by Step



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with website

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Preface

Before deciding to write this book, we examined many of the multitude of books on electrocardiography to determine whether there was a need for a new book with a different approach focusing on graphics. In our experience the success of our "step by step" books on cardiac pacemakers and implanted cardioverter-defibrillators was largely due to the extensive use of graphics according to feedback we received from many readers. Consequently in this book we used the same approach with the liberal use of graphics. This format distinguishes the book from all the other publications. In this way, the book can be considered as a companion to our previous "step by step" books. The publisher offers a large number of PowerPoint slides obtainable on the Internet. Based on a number of suggestions an accompanying set of test ECG tracings is also provided on the Internet. We are confident that our different approach to the teaching of electrocardiography will facilitate understanding by the student and help the teacher, the latter by using the richly illustrated work.

The authors would also like to thank Garant Publishers, Antwerp, Belgium /Apeldoorn, The Netherlands for authorizing the use of figures from the Dutch ECG book, *ECG: Uit of in het Hoofd*, 2006 edition, by E. Andries, R. Stroobandt, N. De Cock, F. Sinnaeve and F. Verdonck,

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About the companion website

This book is accompanied by a companion website, containing all the figures from the book for you to download: www.wiley.com/go/stroobandt/ecg

CHAPTER 1

ANATOMY AND BASIC PHYSIOLOGY

* What is an ECG?
* Blood circulation – the heart in action
* The conduction system of the heart
* Myocardial electrophysiology
    - About cardiac cells
    - Depolarization of a myocardial fiber
    - Distribution of current in myocardium
* Recording a voltage by external electrodes
* The resultant heart vector during ventricular depolarization

ECG from Basics to Essentials: Step by Step. First Edition. Roland X. Stroobandt, S. Serge Barold and Alfons F. Sinnaeve.
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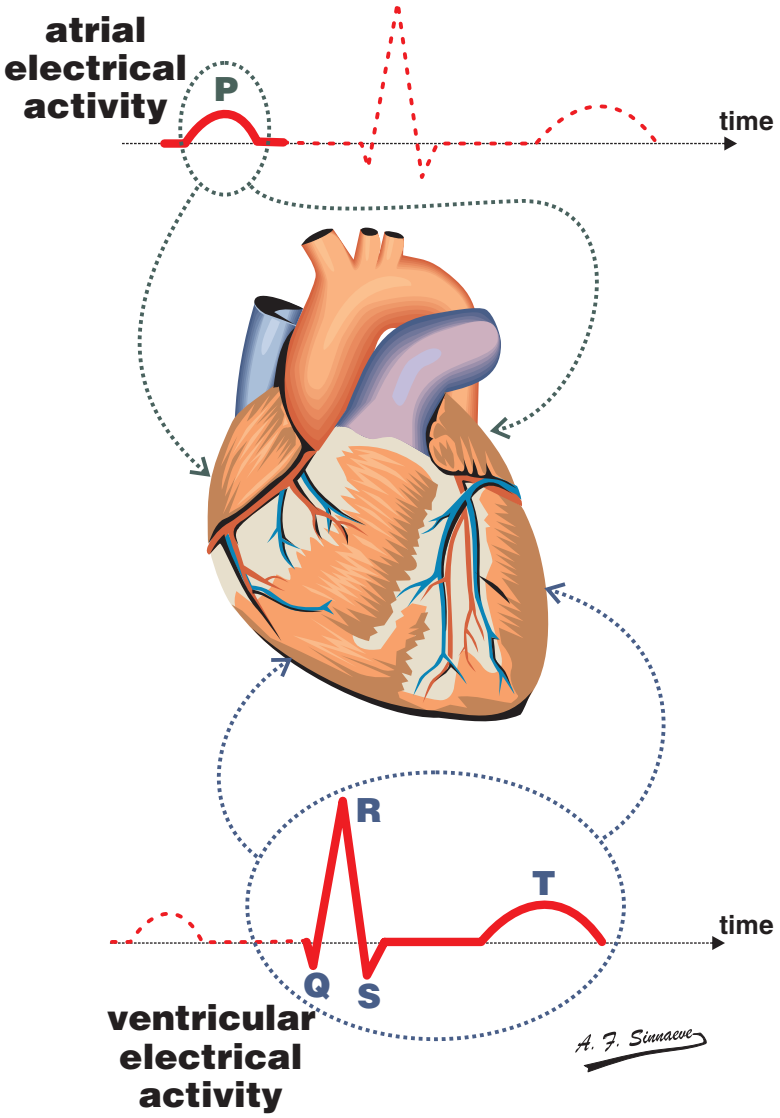
WHAT IS AN ECG?



The *electrocardiogram (ECG)* is the recording of the electrical activity generated during and after activation of the various parts of the heart. It is detected by electrodes attached to the skin.

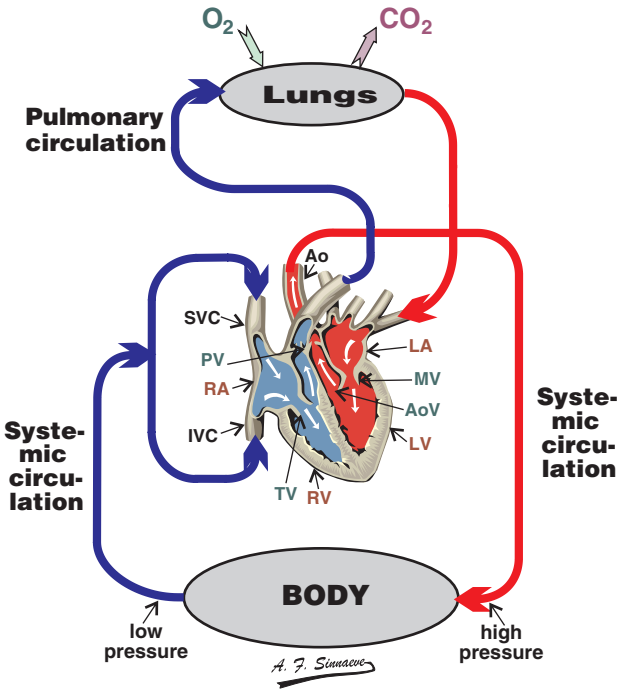
The ECG provides information on:

* **the heart rate or cardiac rhythm**
* **position of the heart inside the body**
* **the thickness of the heart muscle or dilatation of heart cavities**
* **origin and propagation of the electrical activity and its possible aberrations**
* **cardiac rhythm disorders due to congenital anomalies of the heart**
* **injuries due to insufficient blood supply (ischemia, infarction, ...)**
* **malfunction of the heart due to electrolyte disturbances or drugs**

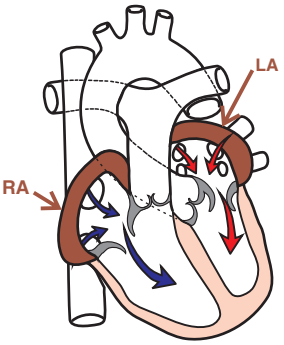
History

The Dutch physiologist Willem Einthoven was one of the pioneers of electrocardiography and developer of the first useful string galvonometer. He labelled the various parts of the electrocardiogram using P, Q, R, S and T in a classic article published in 1903. Professor Einthoven received the Nobel prize for medicine in 1924.

BLOOD CIRCULATION – THE HEART IN ACTION

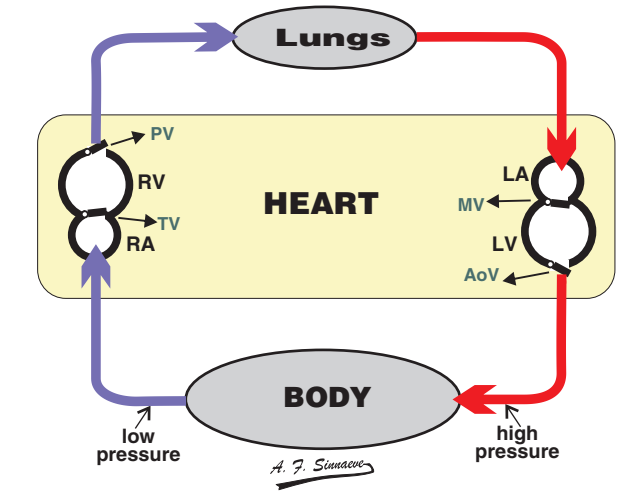


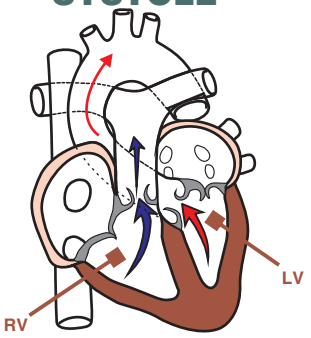
VENTRICULAR DIASTOLE



ATRIAL CONTRACTION
VENTRICULAR RELAXATION

VENTRICULAR SYSTOLE





VENTRICULAR CONTRACTION
ATRIAL RELAXATION

Abbreviations : Ao = aorta ; AoV = aortic valve ; LA = left atrium ; LV = left ventricle ; MV = mitral valve ; PV = pulmonary valve ; RA = right atrium ; RV = right ventricle ; TV = tricuspid valve ; IVC = inferior vena cava ; SVC = superior vena cava ; O_2 = oxygen ; CO_2 = carbon dioxide

The heart is a muscle consisting of four hollow chambers. It is a double pump: the left part works at a higher pressure, while the right part works on a lower pressure.

The right heart pumps blood into the *pulmonary circulation* (i.e. the lungs). The left heart drives blood through the *systemic circulation* (i.e. the rest of the body).

The *right atrium* (RA) receives deoxygenated blood from the body via two large veins, the superior and the inferior vena cava, and from the heart itself by way of the coronary sinus. The blood is transferred to the *right ventricle* (RV) via the *tricuspid valve* (TV). The right ventricle then pumps the deoxygenated blood via the *pulmonary valve* (PV) to the lungs where it releases excess carbon dioxide and picks up new oxygen.

The *left atrium* (LA) accepts the newly oxygenated blood from the lungs via the pulmonary veins and delivers it to the *left ventricle* (LV) through the *mitral valve* (MV). The oxygenated blood is pumped by the left ventricle through the *aortic valve* (AoV) into the aorta (Ao), the largest artery in the body.

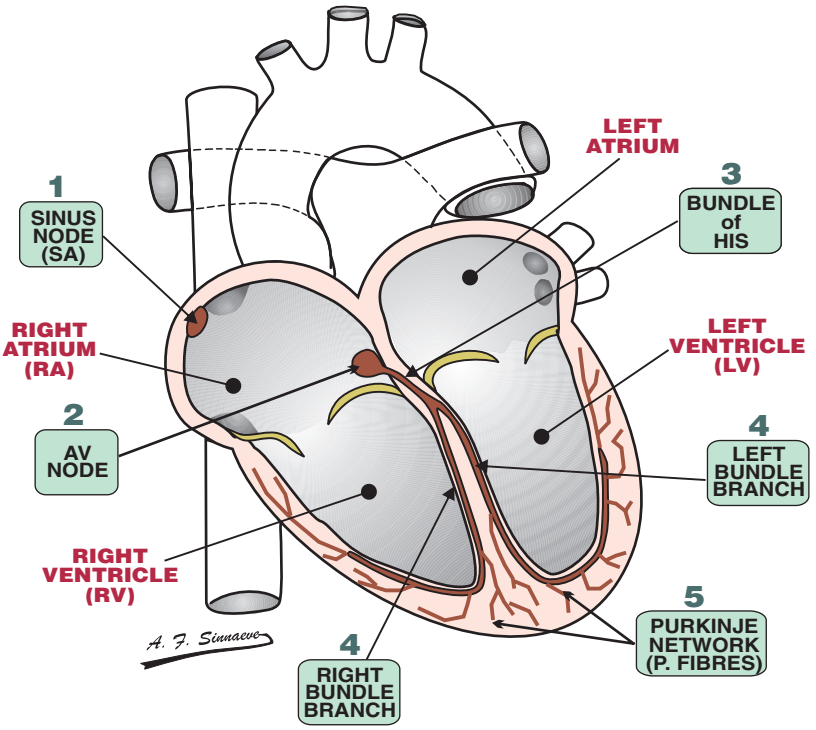
The blood flowing into the aorta is further distributed throughout the body where it releases oxygen to the cells and collects carbon dioxide from them.

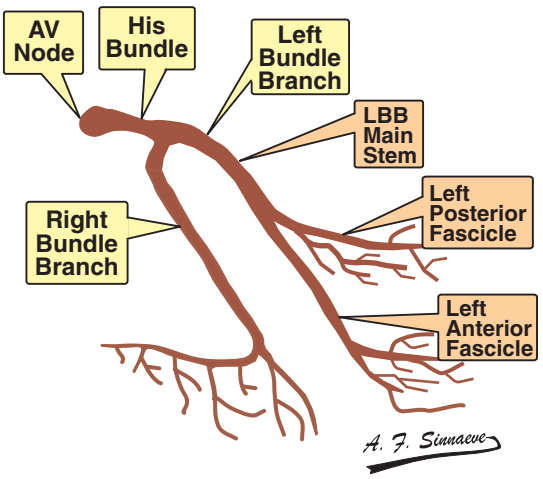
The cardiac cycle consists of two primary phases:

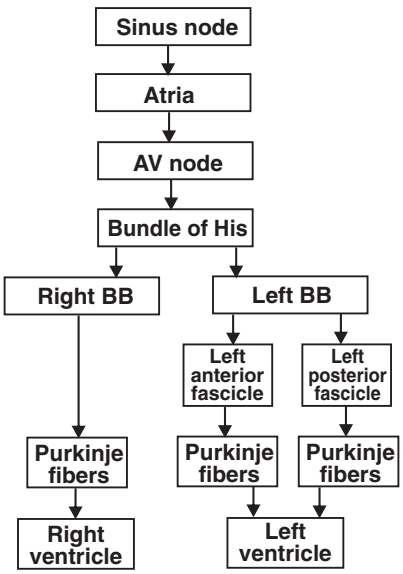
1. VENTRICULAR DIASTOLE is a period of myocardial relaxation when the ventricles are filled with blood.
2. VENTRICULAR SYSTOLE is the period of contraction when the blood is forced out of the ventricles into the arterial tree.

At rest, this cycle is normally repeated at a rate of approximately 70–75 times/minute and slower during sleep.

THE CONDUCTION SYSTEM OF THE HEART





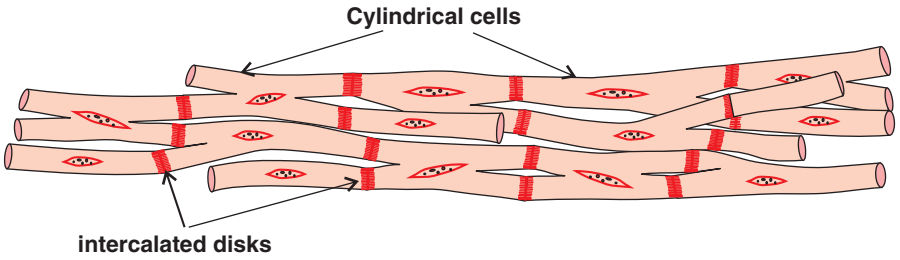


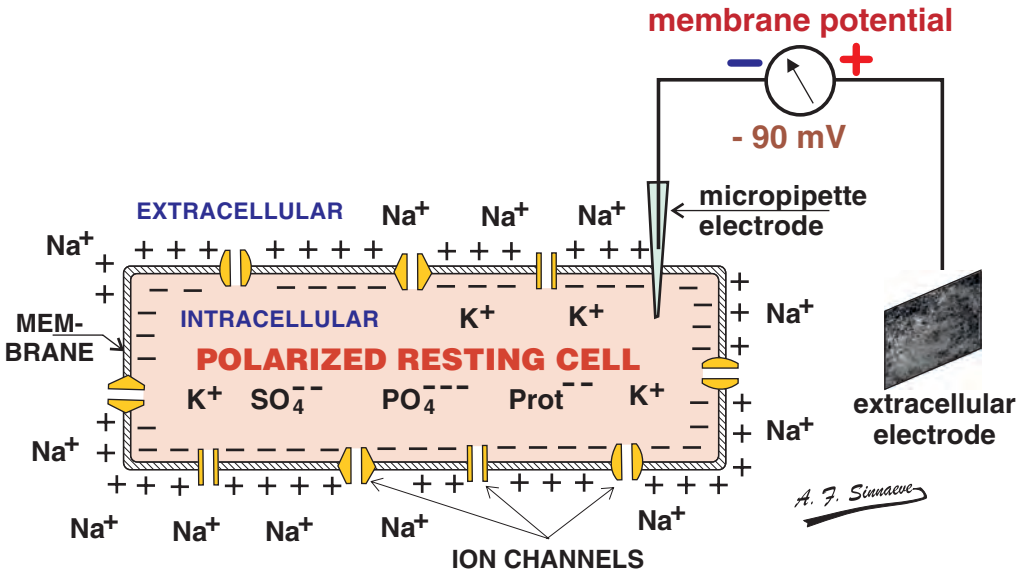
The contractions of the various parts of the heart have to be carefully synchronized. It is the prime function of the electrical conduction system to ensure this synchronization. The atria should contract first to fill the ventricles before the ventricles pump the blood in the circulation.

1. **The excitation starts in the sinus node consisting of special pacemaker cells. The electrical impulses spread over the right and left atria.**
2. **The AV node is normally the only electrical connection between the atria and the ventricles. The impulses slow down as they travel through the AV node to reach the bundle of His.**
3. **The bundle of His, the distal part of the AV junction, conducts the impulses rapidly to the bundle branches.**
4. **The fast conducting right and left bundle branches subdivide into smaller and smaller branches, the smallest ones connecting to the Purkinje fibers.**
5. **The Purkinje fibers spread out all over the ventricles beneath the endocardium and they bring the electrical impulses very fast to the myocardial cells.**

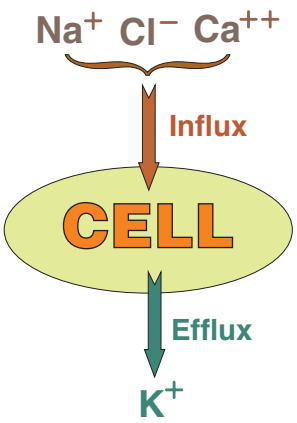
All in all it takes the electrical impulses less than 200 ms to travel from the sinus node to the myocardial cells in the ventricles.

ABOUT CARDIAC CELLS 1





ION	$[ion]_e$ Extracellular concentration in mmol/liter, mM	$[ion]_i$ Intracellular concentration in mmol/liter, mM
K^+	4	150
Na^+	145	10
Ca^{++}	1.8	10^{-4}
Cl^-	120	20



Cardiac muscle cells are more or less cylindrical. At their ends they may partially divide into two or more branches, connecting with the branches of adjacent cells and forming an anastomosing network of cells called a *syncytium*. At the interconnections between cells there are specialized membranes (*intercalated disks*) with a very low electrical resistance. These *"gap-junctions"* allow a very rapid conduction from one cell to another.

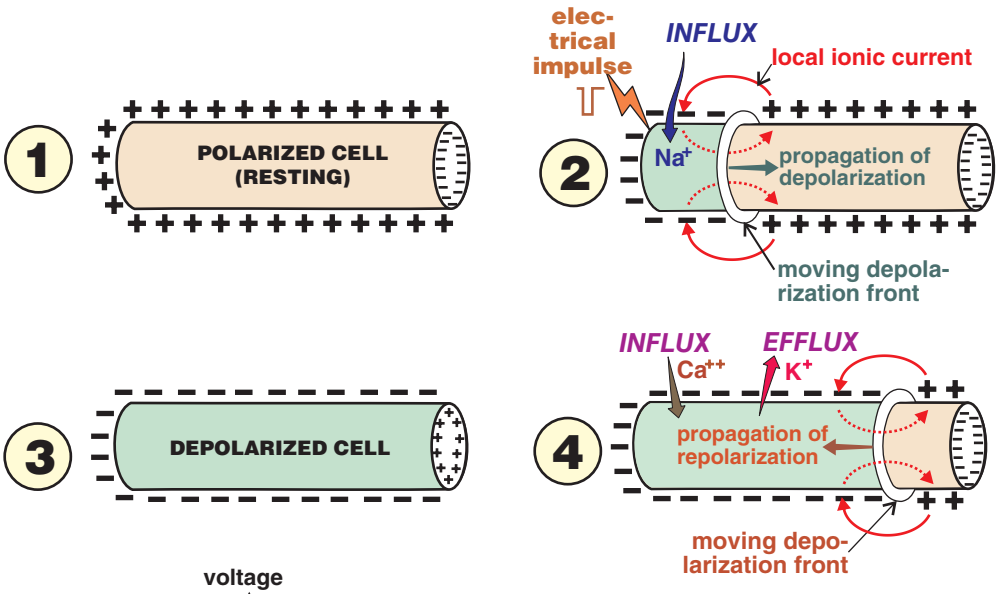
All cardiac cells are enclosed in a *semipermeable membrane* which allows certain charged chemical particles to flow in and out of the cells through very *specific channels*. These charged particles are *ions* (positive if they have lost one or more electrons, such as sodium Na^+, potassium K^+ or calcium Ca^{++} and negative if they have a surplus of an electron, e.g. Cl^-).

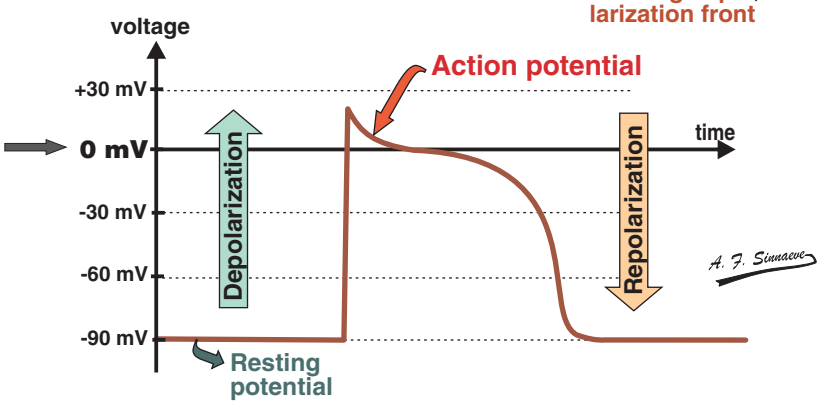
The ion channels are very selective. Larger ions such as phos-phate ions (PO_4^{---}), sulfate ions (SO_4^{--}) and protein ions are unable to pass through the channels and stay in the inside making the inside of the cell negative. A voltmeter between an intracellular and an extracellular electrode will indicate a potential difference. This voltage is called the *resting membrane potential* (normally about –90 millivolts).

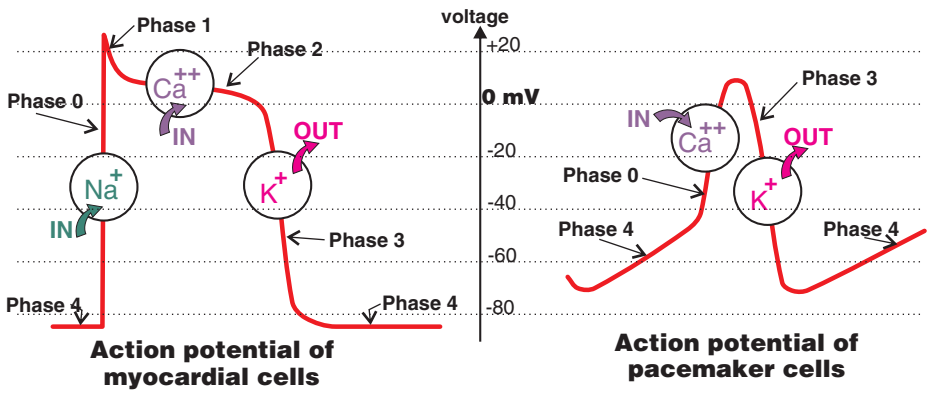
In the resting state, a high concentration of positively charged sodium ions (Na^+) is present outside the cell while a high concentration of positive potassium ions (K^+) and a mixture of the large negatively charged ions (PO_4^{---}, SO_4^{--}, $Prot^{--}$) are found inside the cell.

There is a continuous leakage of the small ions decreasing the resting membrane potential. Consequently other processes have to restore the phenomenon. The Na^+/K^+ pump, located in the cell membrane, maintains the negative resting potential inside the cell by bringing K^+ into the cell while taking Na^+ out of the cell. This process requires energy and therefore it uses adenosine triphosphate (ATP). The pump can be blocked by digitalis. If the Na^+/K^+ pump is inhibited, Na^+ ions are still removed from the inside by the Na^+/Ca^{++}exchange process. This process increases the intracellular Ca^{++} and ameliorates the contractility of the muscle cells.

ABOUT CARDIAC CELLS 2







Action potential of myocardial cells

Action potential of pacemaker cells

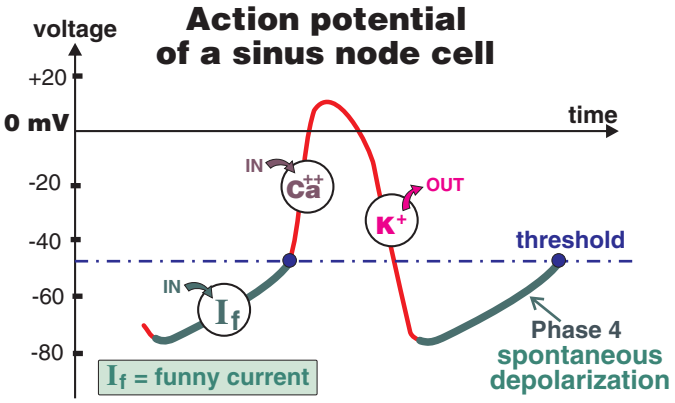
An external negative electric impulse that converts the outside of a myocardial cell from positive to negative, makes the membrane permeable to Na^+. The influx of Na^+ ions makes the inside of the cell less negative. When the membrane voltage reaches a certain value(called the threshold), some fast sodium channels in the membrane open momentarily, resulting in a sudden larger influx of Na^+.Consequently, a part of the cell depolarizes, i.e. its exterior becomes negative with respect to its interior that becomes positive.Due to the difference in concentration of the Na^+ ions, a local ionic current arises between the depolarized part of the cell and its still resting part. These local electric currents give rise to a depolarization front that moves on until the whole cell becomes depolarized.

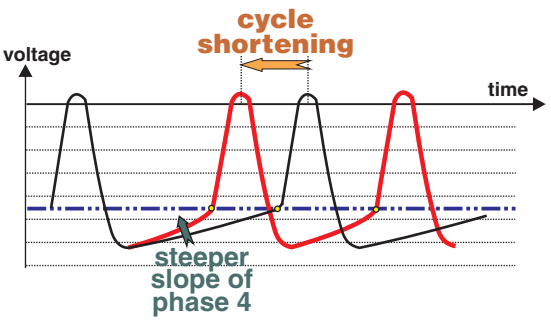
As soon as the depolarization starts, K^+ ions flow out from the cell trying to restore the initial resting potential. In the meantime, some Ca^{++} ions flow inwards through slow calcium channels. At first, these ion movements and the decreasing Na^+ influx nearly balance each other resulting in a slowly varying membrane potential. Next the Ca^{++} channels are inhibited as are the Na^+ channels while the open K^+ channels together with the Na^+/K^+ pump repolarize the cell. Again local currents are generated and a repolarization front propagates until the whole cell is repolarized.

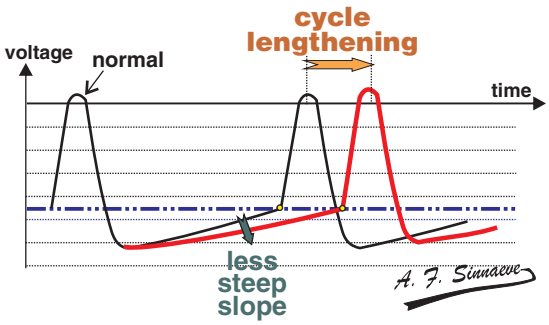
The action potential depicts the changes of the membrane potential during the depolarization and the subsequent repolarization of the cell. The intracellular environment is negative at rest (resting potential) and becomes positive with respect to the outside when the cell is activated and depolarized.

The cells of the sinus node and the AV junction do not have fast sodium channels. Instead they have slow calcium channels and potassium channels that open when the membrane potential is depolarized to about –50 mV.

ABOUT CARDIAC CELLS 3







Dominant Pacemaker
Sinus Node (SAN)
60–80 /min

↓

Latent or Escape Pacemakers

↓

AV Junction including the His Bundle
40–60 /min

↓

Right and Left Bundle Branches
30–40 /min

↓

Purkinje Fibers
20–40 /min

Common myocardial cells only depolarize if they are triggered by an external event or by adjacent cells.
However, cells within the sinoatrial node (SAN) exhibit a completely different behavior. During the diastolic phase (phase 4 of their action potential) a spontaneous depolarization takes place.

The major determinant for the diastolic depolarization is the so-called "funny current" I_f. This particularly unusual current consists of an influx of a mix of sodium and potassium ions that makes the inside of the cells more positive.

When the action potential reaches a threshold potential (about –50/–40mV), a faster depolarization by the Ca^{++} ions starts the systolic phase. As soon as the action potential becomes positive, some potassium channels open and the resulting outflux of K^+ ions repolarizes the cells. The moment the repolarization reaches its most negative potential (–60/–70mV), the funny current starts again and the whole cycle starts all over.

The funny current I_f is most prominently expressed in the sinoatrial node (SAN), making this node the natural pacemaker of the heart that determines the rhythm of the heart beat. Hence I_f is sometimes called the "pacemaker current".

Spontaneous depolarization may be modulated by changing the slope of the spontaneous depolarization (mostly by influencing the I_f channels). The slope is controlled by the autonomic nervous system.

Increase in sympathetic activity and administration of catecholamines (epinephrine, norepinephrine, dopamine) increases the slope of the phase 4 depolarization. This results in a higher firing rate of the pacemaker cells and a shorter cardiac cycle. Administration of certain drugs decreases the slope of the phase 4 depolarization, reducing the firing rate and lengthening the cardiac cycle.

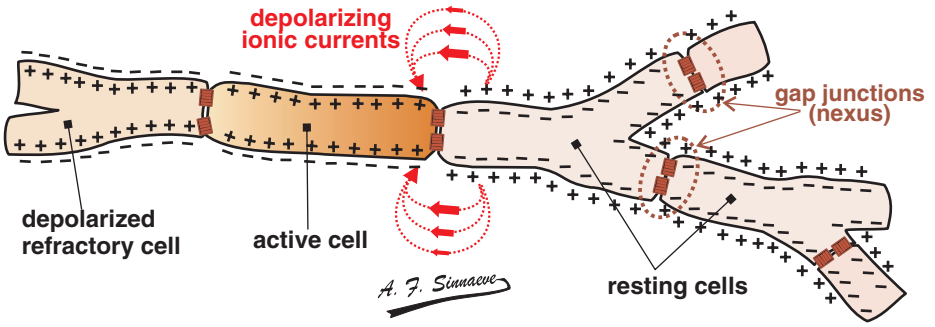
Spontaneous depolarization is not only present in the sinoatrial node (SAN) but, to a lesser extent, also in the other parts of the conduction system. The intrinsic pacemaker activity of the secondary pacemakers situated in the atrioventricular junction and the His-Purkinje system is normally quiescent by a mechanism termed overdrive suppression. If the sinus node (SAN) becomes depressed, or its action potentials fail to reach secondary pace-makers, a slower rhythm takes over.

Secondary pacemakers provide a backup if the activity of the SAN fails

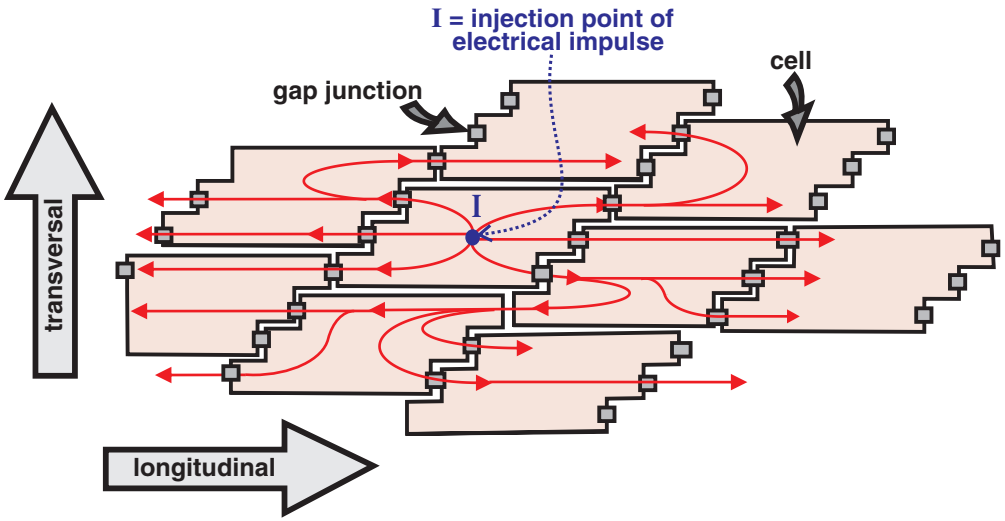
Overdrive suppression occurs when cells with a higher intrinsic rate (e.g. the dominant pace-maker) continually depolarize or overdrive potential automatic foci with a lower intrinsic rate thereby suppressing their emergence.

Should the highest pacemaking center fail, a lower automatic focus previously inactive because of overdrive suppression emerges or "escapes" from the next highest level.
The new site becomes the dominant pacemaker at its inherent rate and in turn suppresses all automatic foci below it.

DEPOLARIZATION OF A MYOCARDIAL FIBER



DISTRIBUTION OF CURRENT IN MYOCARDIUM AND RAPID SPREAD OF ELECTRICAL ACTIVITY

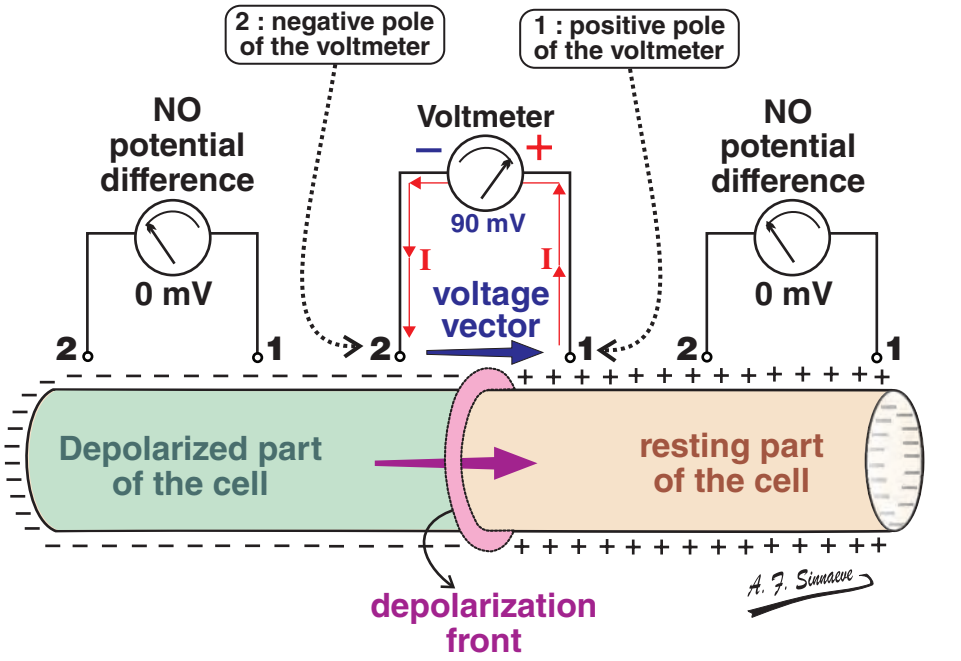


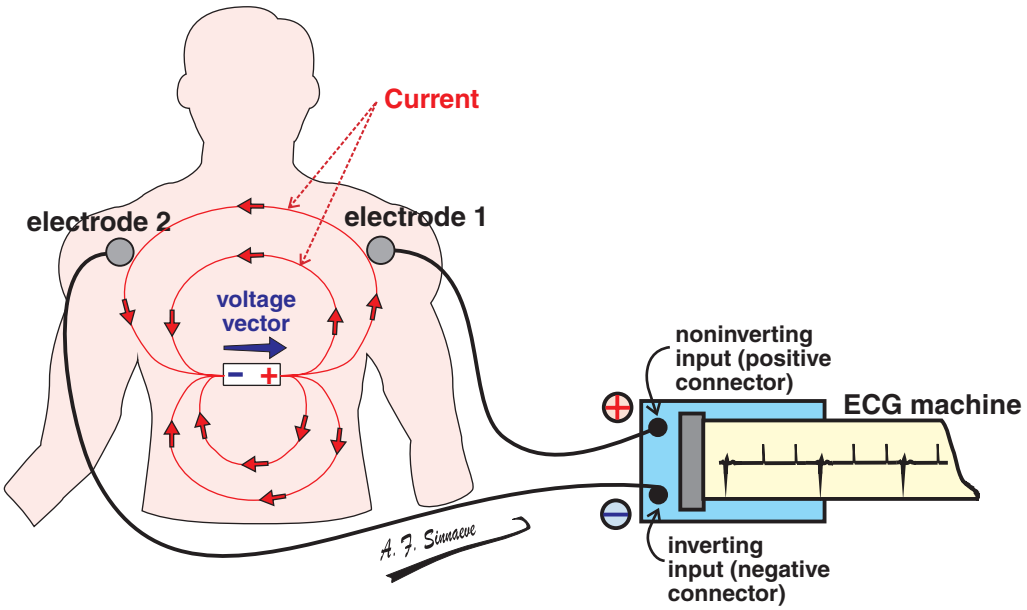
A depolarization front can propagate through the fibers of the heart muscle in the same way as the depolarization front moves through a single cylindrical cell. Local ionic currents between active cells and resting cells depolarize the resting cells and activate them.

- **Very rapid conduction of electrical impulses from one cell to another is due to *"gap junctions"* with a low electrical resistance between the cylindrical cells.**
- **Cardiac cells partially divide at their ends, forming an anastomosing network or "syncytium" causing fast depolarization of the whole myocardium.**

Due to the intercalated disks with their gap junctions, a depolarizing electrical impulse spreads out rapidly in all directions. However, the gap junctions with their very low electrical resistance are only present at the short ends of the myocardial cells. Hence, depolarization propagates very fast in the longitudinal direction of the fibers and less fast in the transversal direction.

RECORDING A VOLTAGE BY EXTERNAL ELECTRODES





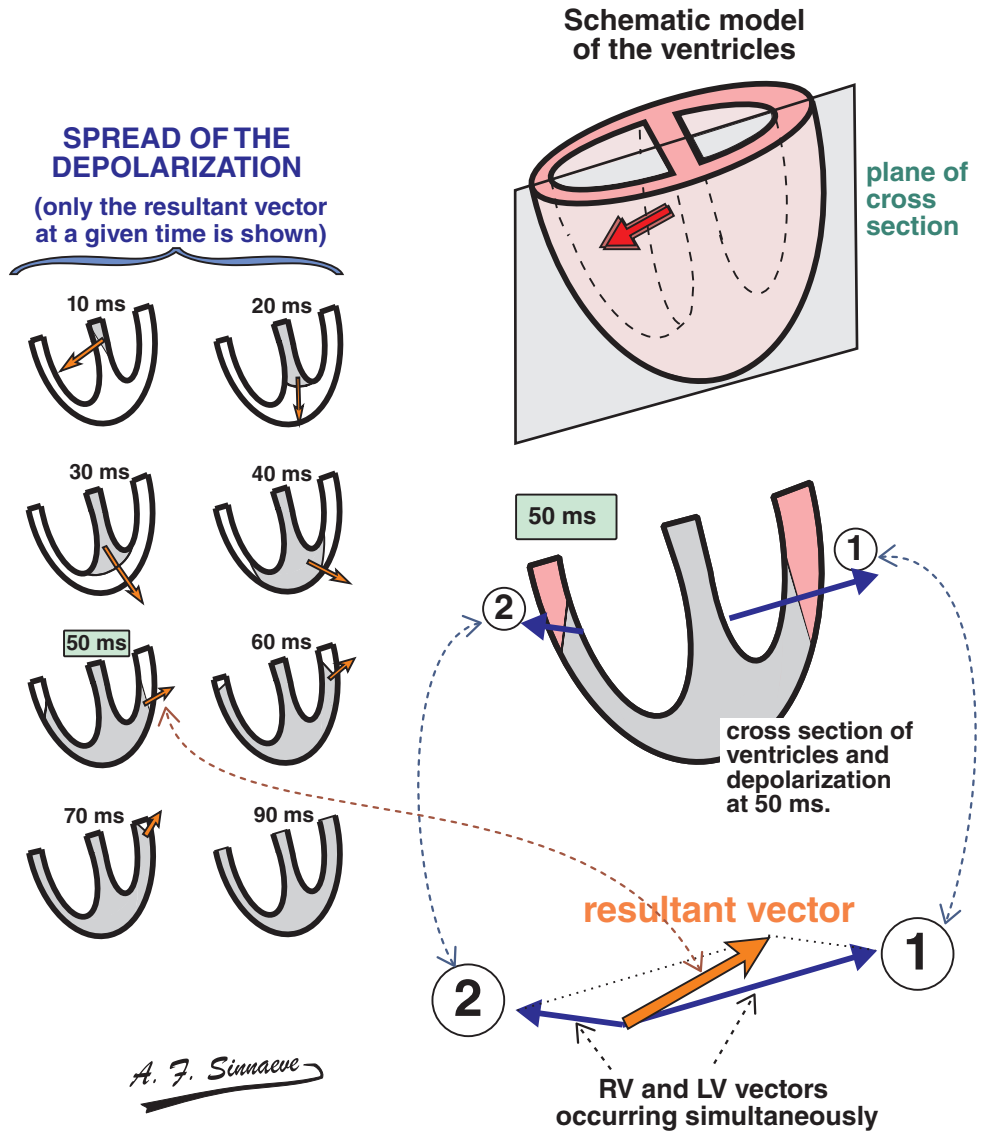
A voltage is always measured between TWO electrodes.

A potential difference or voltage is only caused by a propagating front (either depolarization or repolarization). A resting cell or a depolarized cell does not give rise to a deflection of the voltmeter.

The voltmeter shows a positive deflection if the voltage vector points towards its positive pole !
A very small current flows through the voltmeter from its positive pole to its negative pole. The internal resistance of the voltmeter has to be extremely high since the small current may not influence the condition of the source, i.e this weak current may not affect the distribution of the ions around the cell.

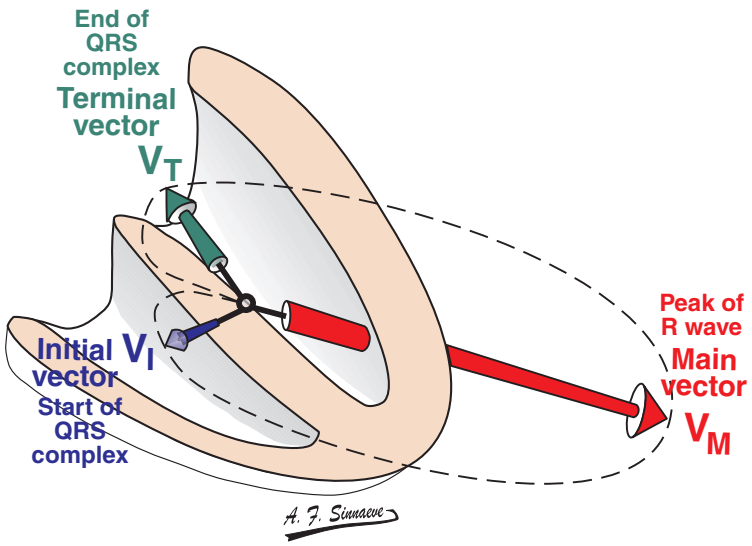
Due to the high degree of electrical interaction between the branched cells, many cells are depolarizing simultaneously in different regions of the ventricles during the ventricular activation process. The voltage vectors of these many cells may be combined into one resultant vector. When a depolarization front or a repolarization front moves rapidly through a region of the heart it generates a voltage vector and a tiny electrical current flows through the body (which is a good conductor). The ECG recorder acts in the same way as a voltmeter and when the voltage vector points to its positive connector, the ECG registers a positive (+) deflection.

THE RESULTANT HEART VECTOR DURING VENTRICULAR DEPOLARIZATION



Ventricular activation consists of a series of sequential activation fronts. At each particular time, the vectors of these activation fronts may be combined to form one resultant vector. The resultant vector changes continually as the ventricles are being progressively depolarized. However, at each point in time the multiple activation fronts can be represented by a single resultant vector.

THE RESULTANT HEART VECTOR IS NOT CONSTANT
* its direction in space changes continuously
* its magnitude changes all the time



V_I, V_M and V_T occur sequentially

The point of the resultant heart vector traces a closed loop in space. The projection of this path is the vectorcardiogram.

20 Further Reading

Barold SS. Willem Einthoven and the birth of clinical electrocardiography a hundred years ago. Card Electrophysiol Rev. 2003;7:99-104.
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