Physiology Labs Protocols

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Labs aim: Explore biology in context through brain and hands

ECG

AIMS OF THE LAB

- Practical demonstration of ECG and its lead system, arrangement, and placement of electrodes on the surface of the body in 12 lead system
- Observation and reduction of artefacts interfering with the ECG signal (muscle contraction, improper contacts of electrodes with the skin, role of conductive gel or other medium improving the contact between the electrode and skin, etc.
- Detailed analysis of recorded ECG curve

REQUIRED KNOWLEDGE

- Dynamics of Ionic currents during action potential (AP) in various myocardial cells (SA and AV cells vs. working myocardium cells)
- Understanding of events of the cardiac cycle and their relationship to individual phases of AP and individual phases (waves) of ECG
- Understanding the principles of summation of vectors and the basics of vector calculus
- Physical principles of lead systems, Ohm law and volume conductor (electrical resistance of various tissues)

PRACTICAL TASKS: **record 12 lead ECC:**

a. in resting laying subject

b. during increased ventilation

c. during patient movement

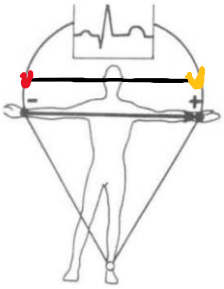
d. in max inspiration and max expiration

Electrocardiography:

Electrocardiography, commonly abbreviated as ECG, is a widely used non-invasive method applied to diagnose cardiac problems that are associated with the characteristics of heart as an excitable tissue. Disorders or conditions (e.g., ischemia, ion disbalance) affecting the excitability and related electrical attributes of the heart tissue interfere with the cardiac electrical activity and affect a normal ECG signal. ECG captures, amplifies, and records the electric activity of the heart that changes in time in a pretty periodic manner. By understanding what a normal ECG looks like, and knowing the mechanisms determining its normal physiological shape, we can recognise and properly interpret variations from the normal ECG. If such deviations from the norm are specific enough, repeatable, and sufficiently strong, then a proper interpretation of those abnormalities significantly contributes to the diagnosis. This, along with the clinical symptoms, help us arrive at a proper diagnosis in time regarding the diseased heart.

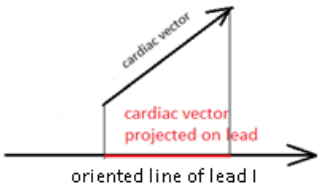
During each cardiac cycle, an electric field of the heart is generated by the electric activity of excitable cardiomyocytes. This field is recorded by a system of electric **leads** arranged by attaching **electrodes on the skin** (at least two electrodes must be used to measure an electrical potential between two points!).

So, what is a **lead**?



Regarding the ECG theory, it is the notion of electric potential between **at least 2 electrodes**. The leads themselves are not necessarily physical objects and could be understood as an abstract concept**.** Mind, that the lead can **be formed by a combination of more than TWO electrodes in case each such electrode contributes to the resulting potential.**

There is another meaning of the **lead** in ECG theory – each lead can be imagined as an **oriented line in 3D space** onto which the 3D electrical vector of the instantaneous electric field is projected. Then, from the physical point of view, the actual (or instantaneous) voltage measured by a given lead is in fact a "dot product" of **TWO** vectors: **1)** the actual 3D cardiac vector of the electric field of the heart, and **2)** the vector representing the position of a given lead in 3D space (see picture to the right for illustration). Geometrically speaking, the amplitude of recorded voltage from a given lead corresponds to the length of projection of the cardiac vector onto the oriented line of that lead (as opposed to a cardiac vector that is changing in time, the vector of the lead has a constant orientation in space; picture to the right represents first limb lead that is oriented horizontally in a frontal plane).



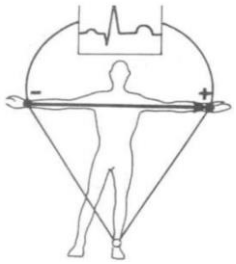
What is an **electrode**?

It is a physical conductive pad/clamp etc., which is attached commonly to the skin (can be also attached directly to the endocardium – intracardial electrodes, or to other body tissues – e.g., mucosa, or be inserted as a needle electrode into certain tissues – e.g., muscles - **E**lectro**M**yo**G**raphy). Using two (or more than two) electrodes, we can form one lead as already explained.

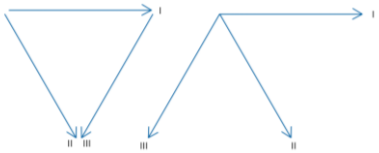
There is a special electrode that is not involved directly in the recording system reading and amplifying voltages from the skin. It is a **ground electrode** whose role is to deflect or divert a noisy signal (e.g., charges generated by static electricity or other, externally induced electrical signal) away from recording leads. In principle, it can be placed anywhere but should be away from active recording electrodes. Thus, most commonly, it is attached to the right leg, far away from the recording electrodes. Mind that ground electrode **IS NOT** providing a neutral or reference point for other leads as its name could tempt us to think. These reference points (or neutral potentials against which exploring electrodes measure voltage) are provided by central (common) terminals made up of a combination of more recording electrodes as discussed later in this protocol.

By using the briefly outlined concept of electrodes and leads, we can understand how to record voltages from various places of our body, provided an optimized system of electrodes and leads is used to collect electrophysiological signals originating from the heart. A total of 9 recording electrodes are used to obtain the most standard **12 leads ECG recordings** (mind, that the last 10th electrode is a ground electrode that is not involved in the recording lead system). The leads in ECG are **commonly classified as**:

3 STANDARD BIPOLAR LIMB LEADS (I, II, AND III) – EINTHOVEN'S LEAD SYSTEM



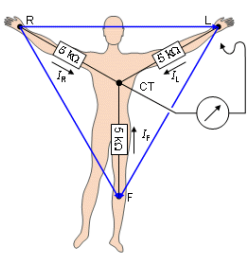
Einthoven recorded differences in electric potentials between two locations on the human body. As there were no adhesive electrodes and serious limitations in amplification systems at that time, the optimal way to contact the body electrically to a pioneered ECG system was to place extremities into metal buckets filled with salt solution connected via wires to form ECG leads. Einthoven could thus measure the electric potentials between the right and left arm (lead I), the right arm and left leg (lead II), and the left arm and left leg (lead III).

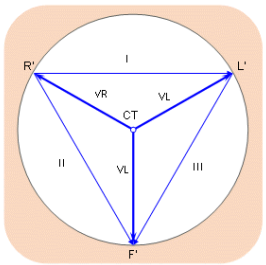


These 3 limb leads form an imaginary triangle that can be projected onto the human chest **– the Einthoven's Triangle – where each leg of that triangle represents a lead of two exploring electrodes having equivalent roles in recording – hence such leads are commonly called bipolar** (both el. poles having equal roles).

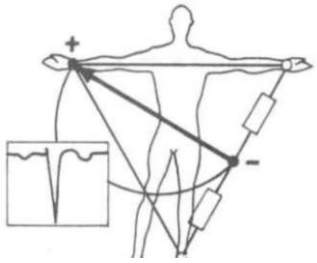
3 PSUEDOUNIPOLAR LIMB LEADS (aVR, aVL AND aVF) - GOLDBERGER'S LEAD SYSTEM

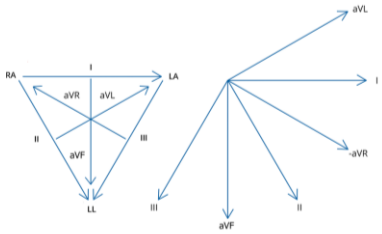
As mentioned, Einthoven's bipolar limb leads (I, II, III) measure the potential between respective extremities and are aligned within the frontal plane, subtending 120° from each other (see top picture to the right). However, one could wish to have other orientations of leads in 3D space (still within the frontal plane), onto which the instantaneous 3D vector of electric field will be projected. These orientations were first conceived by Wilson, who connected all three extremities through three 5 Ohm resistors, forming a single electrical point at which voltages from collected extremities converge and average. He thus created the **C**ommon **T**erminal (**CT**), called nowadays the **Wilson Central Terminal (WCT**, see the left picture above**).** The position of WCT could





figuratively be localised somewhere in the middle of the chest, very close to the assumed position of the heart. Then, if the voltage between the given extremity (R hand, L hand, or F food) and the WCT is measured, it will correspond (as it was already explained), to the dot product of **1)** the instantaneous 3D electric vector generated by the heart and **2)** the vector corresponding with the newly created lead (VR, VL, or VF pointing from the assumed location of CT (middle of the chest - see the right picture above) towards the recorded extremity (R, L or F)). VR, VL, and VF obtained thus new lead orientations, shifted by an angle of 30° from I, II, and III bipolar lead orientations. The problem with these new leads is however that their voltages are way smaller than voltages coming from the bipolar limb leads. Goldberger had an idea to eliminate this drawback and removed a wire connecting the recorded extremity with the WCT. This results into a circuit (shown the right), measuring the potential difference between, e.g., the right hand and the so-called Goldberger's Central Terminal (GCT), which is in fact the Wilson Central Terminal that is lacking the 5 Ohm connection going to the right hand in this case. Removing the 5 Ohm wire that was diverting the charge from the right hand towards the CT increases, or augments the measured voltages between the right hand and GCT, giving prefixes *a-augmented* to those leads: aVR, aVL, aVF. Simply said, these leads in fact measure the potential between a given extremity against the potential that averages potentials from opposite extremities:

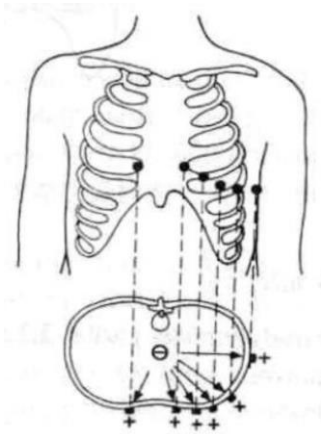




-aVR = (I + II) / 2; aVL = (I - III) / 2; aVF = (II + III) / 2

With three Einthoven's bipolar limb leads, we now have six leads available "to scan" the electrical activity of the heart in the frontal plane (see the picture to the left).

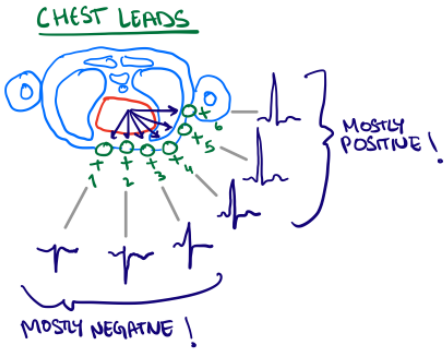
6 UNIPOLAR CHEST LEADS (V1 TO V6)- WILSON'S LEAD SYSTEM



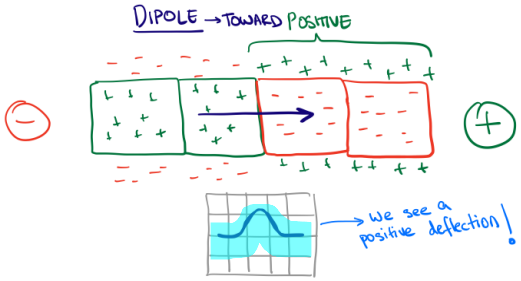
Wilson further developed the ECG system by adding a lead system placed in a horizontal plane that is sectioning through the heart. He accomplished this by attaching electrodes directly to the chest wall and measured their voltages against a virtual reference point, located in the middle of the heart, the point that was already described in the abovementioned paragraph - the Wilson Central Terminal. This is a reference point, as already explained, constructed by the resistor network connected to three extremities against which the voltages are measured from six chest leads C1-C6, forming the leads **V1-V6**.

Together with the Einthoven limb leads and Goldberg augmented limb leads, the 12-lead ECG system is now created! This 12-lead ECG system is known today as the gold standard for ECGs.

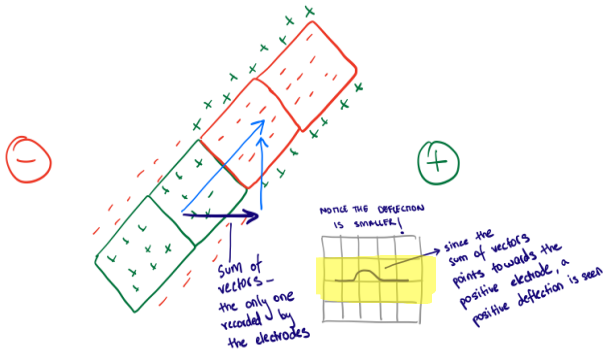
Concept of ECG - genesis of the ECG signal**:**



Each myocardial cell generates an action potential after receiving suprathreshold electrical stimulation. This is accompanied by a genesis of a miniature electric field represented by an electric vector of each cell that is being depolarised (and consecutively repolarised). If we add up together all those individual vectors of a given moment, we speak about the so-called instantaneous 3D cardiac vector. Then, the instantaneous voltage measured by a given lead can be viewed as a dot product of two vectors, one being the cardiac vector, the other one the vector aligned with the orientation of the recording lead (in the Einthoven triangle).



Simply speaking, if a positive deflection is recorded by the particular lead, the cardiac vector is pointing towards the positive electrode. Whereas, if a negative deflection is recorded, the cardiac vector is pointing toward the negative electrode. If the cardiac vector runs parallel to the lead line (lead vector), we register maximum deviation on the ECG. If the cardiac vector is directed perpendicular to the lead line, we record zero deflection despite the actual cardiac vector is not zero! As cardiomyocytes experience a wave of depolarisation, followed by a wave of repolarisation during each cardiac cycle, they contribute to the resulting everchanging cardiac vector. Its amplitude and its direction change many times during each cardiac cycle. This alternation of depolarisation, followed by repolarisation, the course of conduction pathways along which the excitation propagates through the myocardium, as well as the leads' orientations, determine the recorded shape of ECG.

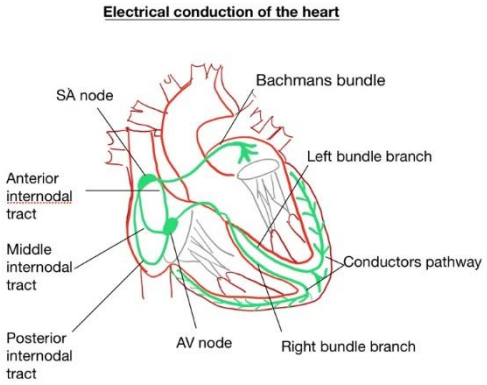


Finally, resulting ECG curves are evaluated by us in the time domain based on analysing the shapes of waves (P waves, QRS, T waves), the intervals between them, and the regularity of their occurrences over a few consecutive cardiac cycles.

Origin of the electrocardiogram:

<u>The conductive system</u> (electrical conduction system of the heart composed of modified muscle fibres) serves to generate and to convey purposefully the excitation through the myocardium, optimising its mechanical contraction, mainly in terms of effective hemodynamics.
SA node (sinoatrial node found in the right atrium)→ Internodal pathway→ AV node (atrioventricular node)→ delay of more than 0.1 second in conducting from atria to ventricles → bundle of His (interventricular septum)→left and right Tawar bundles (carry the impulses to left and right ventricles)→Purkinje fibres (found in sub-endocardium layer) bring the action potentials throughout the ventricular muscle into working cardiomyocytes.

SA node



the pacemaker of the heart that is responsible for the spontaneous physiological activity of the heart (based on spontaneous diastolic depolarization of SA node cells), allowing the heart to function without any external stimulation. The SA cells depolarize the fastest in terms of spontaneous change of resting membrane potential, thus they set the pace of the heart—determining the main pacemaker - frequency is around 60-90 BPM at rest). The consecutive parts of conduction system spread that signal over the whole heart and induce contraction in all contractible cardiomyocytes. On normal ECG, the activity of the SA node is not visible because it is very small, and thus its depolarization is not visible by electrodes placed on the body surface (but intracardial electrodes can "see" it).

AV node

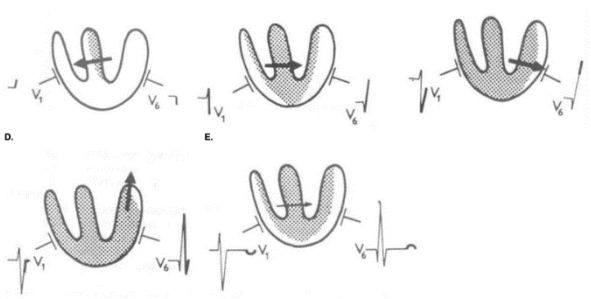
Secondary pacemaker (can take the role of a pacemaker if SA node is damaged) – second fastest spontaneous frequency ranging 40-60 BPM. Normally, the AV node is only responsible for **slowing down the impulses** passing through it (delaying thus the conduction of action potentials from atria to ventricles), allowing the ventricles to fill optimally with blood by atrial contraction before the depolarization hits the ventricles, causing them to contract optimally to eject the blood. However, if the SA node ceases to function for some reason, the AV takes over its function and becomes the main pacemaker giving a pace of 40-60 BPM.

If the SA node and AV nodes are destroyed both, the cells of conducting system of ventricles (frequency 30-40 BPM) act as the next pacemaker which results in severe bradycardia. The more peripherally we go, the lower the spontaneous frequency of cells of conductive system. Understanding that, we can infer which area of conductive system is damaged, taking into consideration the actual heart rate and other information related to patient's clinical picture (example: a 25 YO athlete versus an 85 YO man both having bradycardia. We can understand that the bradycardia of 25 YO man is probably physiological (why?), whereas the bradycardia of 85 YO man could potentially be associated with a problem in a conductive system).

Electrocardiography curve:

Paper speed - 25mm/sec, for better time resolution could be 50mm/sec. This is very important parameter because all time intervals on ECG could be properly calculated only if this parameter is known. E.g., with the speed of 25 mm/sec the 1 mm segment lasts 40ms (smallest square), 5mm segment lasts 200ms (middle square, e.g., 1 minute spans 300 middle squares, thus the expression 300/"No of middle squares between two events" gives us the frequency of those events per minute, e.g., 300/"No of middle squares between two adjacent R waves" gives us the BPM).

P wave - atrial depolarization (followed by an atrial mechanical contraction that is delayed by 50-100ms – mechanical contraction is due to the Ca^{2+} release postponed by roughly tens of millisecond in striated muscle or heart when measured from the onsets of electrical events).



PQ segment - isoelectric line – AV node delay – from the end of the P wave to the beginning of the QRS complex.
The velocity of depolarization inside the heart is relatively fast (0.5 m/s in working myocardium cells, 4 m/s in Purkinje fibres), with the only exception that is the AV node. Here, the spreading is very slow (0.05 m/s), giving ventricles an extra time (roughly 50 – 100ms, depending on the heart rate) to be filled with blood by atrial contraction before they start their own contraction.
The PQ interval, in addition to the PQ segment, comprises the duration of the P wave and should last from 120ms to 200ms.

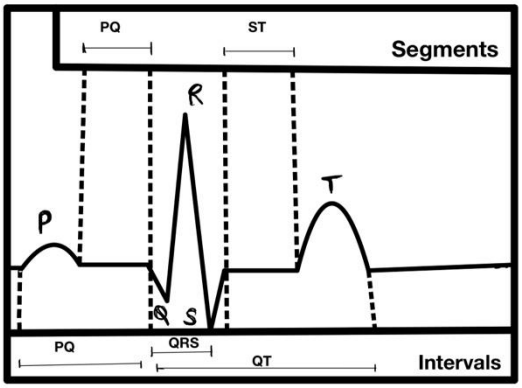
QRS complex - ventricular depolarisation (mechanical contraction of ventricles is delayed by roughly 100ms from their electrical depolarisation, which is a very similar delay as in atria). It is usually multiphasic (having usually two – three waves, depending on the "viewing" lead and on the particular trajectory of cardiac vector during ventricular depolarization). This contrasts with a rather monophasic or biphasic P wave (as seen by most of leads), which is due to the simpler trajectory of cardiac vector of atrial depolarization (or ventricular repolarisation), compared to the more complex trajectory of ventricular depolarization.
In lead II (most referenced lead), the ventricular complex is usually made up of 3 waves: q(Q) - negative deflection, R(r) - positive deflection, and s(S) - negative deflection
Deflections that do not exceed 0.3 mV are denoted by lower-case letters, whereas deflections bigger than 0.3 mV (which are also necessarily wider) are denoted by capital letters.
Mind, that the atrial repolarisation occurs within the QRS complex and is thus completely electrically masked or overridden by much bigger voltages coming from massive walls of ventricles.

ST segment - isoelectric line occurring when ventricles are completely depolarized (plateau phase – membrane potential of ventricular cells is rather constant during this interval, so the resulting cardiac vector is very close to zero). This is a very important phase of ECG that is very susceptible to the ischemia (and also to the other factors) affecting the plateau phase of APs of ventricular cells suffering from ischemia, deviating (elevating or depressing) the otherwise isoelectric line of ST segment.

T wave - ventricular repolarisation – as the wave of repolarization travels from the epicardium to the endocardium (ventricular depolarization goes from the endocardium to epicardium), but yet also having the opposite direction of transmembrane current (in regard to depolarisation), its polarity should be identical to an overall polarity of QRS complex (works for limb leads as well as chest leads – in aVR, and in V1 the QRS complex and T waves are mostly negative, whereas in others leads rather positive).

When assessing the electrocardiographic curve, we evaluate several parameters. In the routine description of the ECG, the following data should be included as an essential part of the clinical evaluation:

• **Rhythm** – the source of heart pace. We refer to the rhythm coming physiologically from the SA node as the sinus rhythm or the SA node rhythm. If the SA node isn't working, the AV node becomes a primary pacemaker and then we speak about the AV node rhythm.



• **Heart rate** (normally 60–80 BPM at rest) – the most logical, possibly intuitive way of calculating it would be to divide the speed of paper[mm/s] / the length of average representative R-R interval[mm]=frequency [Hz], which could be further multiplied by 60 to get BPM. The length of average R-R interval could be found also by measuring the length of optimally chosen train of R-R intervals divided by the number of those R-R intervals (e.g., the length of consecutive 4 R-R intervals/4). There are also other simple ways of estimating the ECG heart rate described by various textbooks or other sources (e.g. BPM = 300 /"No of middle squares spanning an average R-R interval" as already mentioned a few pages above)

• **Duration of the P wave** (from the beginning of the P wave to the end of the P wave, normally 60–100ms)

• **PQ interval** (from the beginning of the P wave to the beginning of the QRS complex, normally 120–200ms, but BEWARE, in trained athletes with bradycardia may be longer). It is a very important parameter the deviation of which reveals AV node conduction problems.

• **QRS width** (from the beginning to the end of the ventricular complex, normally 60–100ms). Also a very important parameter whose prolongation reveals problems in the conduction system that can be caused by localised ischemia, remodelation of the heart tissue, or other pathologies interfering with the propagation of ventricular depolarisation.

• **The QT interval** (from the beginning of the QRS complex to the end of the T wave, its length depends on the heart rate, with the normal value being 0.34-0.42s at rest, which could increase with age).

• **Inclination of the electrical axis of the heart** (direction of the overall QRS vector is normally –15° to +105°). Overall QRS vector is a vector that averages all instantaneous cardiac vectors over the whole ventricular depolarisation. This procedure can be done automatically by an ECG device, or manually by calculating average QRS deflections in each of two leads that are mutually perpendicular (e.g., in lead I and in lead aVF; perpendicular limb lead pairs are I and aVF, II and aVL, III and aVR). To calculate the net QRS amplitude of a given lead, we need to subtract the Q wave and the S wave amplitudes from the R wave amplitude. This net QRS deflection must then be aligned along the respective line vector of a given lead from which it was calculated (oriented according to the Einthoven triangle). If the net QRS deflection is negative the alignment must be done in opposite direction to a given lead vector.