The Electrocardiogram: Etiology, Interpretation & Acquisition

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The heart is an electrical organ, and its activity can be measured non-invasively.

Wealth of information related to:

- The electrical patterns proper
- The geometry and mechanical properties of the heart’s tissue
- The metabolic state of the heart

Standard tool used in a wide-range of medical evaluations.
A heart

- Myocardium: a muscle made of millions of cells – contract in a sequence to pump blood
- Blood circulates, passing near every cell in the body, driven by this pump (actually, two pumps) - Atria = turbochargers
  - Ox blood from lungs into left side of heart (left atrium through to ventricle) - 30% pumped to left ventricle
  - After a short delay, the ventricle contracts and pumps blood to aorta
  - At the same time the deoxygenated blood is being pumped through the right side of heart to the pulmonary artery (and to the lungs)
- Heart Valves to prevent backflow against one-way flow through heart
Blood flow through the heart

- **Animation of flow**
  (Follow this link)
Pressure, ECG and sound of a beat

- Isovolumic contraction
- Isovolumic relaxation
- Ejection
- Rapid inflow
- Diastasis
- Atrial systole

Pressures (mmHg):
- Aortic pressure
- Atrial pressure
- Ventricular pressure

Volumes (mL):
- Ventricular volume

Electrocardiogram:
- P
- Q
- R
- S
- T

Phonocardiogram:
- 1st
- 2nd
- 3rd

Sounds:
- ‘lub’ Systole
- ‘dub’ Diastole
- Systole
Moving echocardiogram (loop)
To understand the ECG:

- Electrophysiology of a single cell
- How a wave of electrical current propagates through myocardium
- Specific structures of the heart through which the electrical wave travels
- How that leads to a measurable signal on the surface of the body

Figure by MIT OCW.
Part I: Cell electrophysiology

- Each **myocyte** (muscle cell) contains myofibrils (long chains of sarcomeres), the contractile units of the cell.

- There are various specialized forms of myocytes: cardiac, skeletal, and smooth muscle cells, with various properties.

- Cardiac myocytes (like those at the SA node) are responsible for generating the electrical impulses that control the heart rate, among other things.

- There are around $2 \times 10^9$ muscle cells in normal hearts of children and adult humans, and may rise to $4 \times 10^9$ in excessively hypertrophied hearts.
Cell dynamics

ATPase

\[ 2 \text{K}^+ \rightarrow \text{K}^+ 
\]

\[ 3 \text{Na}^+ \rightarrow \text{ATPase} \]
a myocyte

Intracellular millivoltage

Resting comfortably

-90
Depolarizing trigger
Intracellular millivoltage

Na channels open, briefly

time
Intracellular millivoltage

In: Na+

Mystery current

time
In: Na\(^+\) is in balance with K\(^+\) out
In: Na$^+$

\textbf{Excitation/Contraction Coupling:} Ca$^{++}$ causes the Troponin Complex (C, I & T) to release inhibition of Actin & Myosin.
In: Na$^+$ + Ca$^{++}$ in; K$^+$ out
More K$^+$ out; Ca$^{++}$ flow halts
In: Na$^+$; Out: K$^+$

In: Ca$^{++}$; Out: K$^+$

Sodium channels reset
Higher resting potential
Few sodium channels reset
Slower upstroke

In: Na⁺
Summary: Four phase of the action potential

1. **Initial repolarization**: opening of a K channel.

2. **Plateau** in the action potential and a delay in repolarization: inward-going calcium current and outward-going potassium current approx balanced.

3. **Repolarization**: Conductance $K^{\uparrow}$ Ca$^{\downarrow}$ … … encourages cell to repolarize.

4. **Resting condition**: open K channels & -ve transmembrane potential.
The dipole field due to current flow in a myocardial cell at the advancing front of depolarization.

\( V_m(x,t) \)

Direction of propagation

\( V_m \) is the transmembrane potential.
So how does the heart **start** to beat?

Specialized auto-rhythmic ‘pacemaker’ cells:

- Located in the SA node
- The AV node
- The ventricles

- The AV node and ventricles are ‘back-ups’ to the SA node.
a pacemaker cell

Slow current of Na\(^+\) in; note the resting potential is less negative in a pacemaker cell
a pacemaker cell

Intracellular millivoltage

Threshold voltage

-40

time
Ca++ flows in
... and K+ flows out
...and when it is negative again, a few Na+ channels open
Typically, an impulse originating anywhere in the myocardium will propagate throughout the heart. Cells communicate electrically via “gap junctions.” Behaves as a “syncytium.” Think of the “wave” at a football game!
Important specific structures

- Sino-atrial node = pacemaker (usually)
- Atria
- After electrical excitation: contraction
- Atrioventricular node (a tactical pause)
- Ventricular conducting fibers (freeways)
- Ventricular myocardium (surface roads)
- After electrical excitation: contraction
But how do we represent the activity of so many cells?

- Need to have simple model...
The Idealized Spherical Torso with the Centrally Located Cardiac Source (Simple dipole model)

- Laplace’s equation gives potential distribution on the torso as:
  \[ \Phi(t) = \cos\theta(t)3|\mathbf{M}(t)|/4\pi\sigma R^2 \]

- \( \theta(t) \) is the angle between the direction of the heart vector \( \mathbf{M}(t) \),
- \( \mathbf{OA} \) the lead vector joining the center of the sphere, \( O \), to the point of observation, \( A \)
- \( |\mathbf{M}| \) is magnitude of the heart vector.

- The potential difference between the two points on the surface of the torso would be:
  \[ V_{AB}(t) = \mathbf{M}(t) \cdot \mathbf{L}_{AB}(t) \]

- Where \( \mathbf{L}_{AB} \) is the ‘lead’ connecting points \( A \) and \( B \) on the body surface

- Central terminal averages all ‘limb’ leads:
  \[ \Phi_{CT}(t) = \Phi_{RA}(t) + \Phi_{LA}(t) + \Phi_{LL}(t) \]
  LA=left arm, RL=right leg (should always be zero) RA=right arm.
Excitation of the Heart
Excitation of the Heart
Cardiac Electrical Activity

Putting it all together:
Normal features of the electrocardiogram.

Recording Conventions, Waveform Nomenclature, and Normal Values for the Electrocardiogram.
Temporal changes: rhythms

Normal sinus rhythm

Figure by MIT OCW.
Sinus arrhythmia

Sinus Arrhythmia

Figure by MIT OCW.
Arrhythmias

- Abnormal conduction of the heart leading to abnormal patterns in the ECG:
  - Not firing when you should (e.g. bradycardia, late ectopic)
  - Firing when you shouldn’t (e.g. tachycardia, premature ventricular beats)
  - Both (e.g. re-entrant arrhythmias)

- Consequences / symptoms:
  - Cardiac arrest: Some arrhythmias are life-threatening medical emergencies
  - Palpations (abnormal awareness of heart beat)
    - atrial/ventricular fibrillation, ‘wiring’ faults,
    - mechanical issues in cardiac pacemakers

- Potentially lead to life threatening problems like stroke or embolism
Sinus Tachycardia

Sinus Tachycardia—Rate 122

Figure by MIT OCW.
Sinus bradycardia

Sinus Bradycardia—Rate 48

Figure by MIT OCW.
Abnormal electrical conduction

- What happens if the pacemakers don’t fire at the ‘right’ time?
- We observe ‘ectopic’ beats
  - i.e. ‘out of place’ (in time and space)
Atrial premature contractions

(see arrowheads)
Ventricular premature contractions
Wave-front Trajectory in a Ventricular Premature Contraction.
AV node conduction block

Complete A-V Block with Junctional Escape Rhythm

Figure by MIT OCW.
e.g.: Ventricular escape beat

Figure 1.23  Ventricular escape beat. Note the atrial P wave (black arrowhead) followed by an evident pause, indicating a failure to conduct through the AV node. The ventricular escape beat (white arrowhead) is a fail-safe mechanism so that conduction blocks do not cause ventricular cardiac arrest. Also see Figure 1.21. (From: [2]. © 2004 MIT OCW. Reprinted with permission.)
Atrial Fibrillation

Atrial Fibrillation (2 examples)

Figure by MIT OCW.
Non-sustained ventricular tachycardia (3 episodes)
Ventricular Fibrillation

Three Examples of Ventricular Fibrillation

Figure by MIT OCW.
Understanding the ECG: A Cautionary Note

- Basic cell electrophysiology, wavefront propagation model, dipole model: powerful, but incomplete
- There will always be electrophysiologic phenomena which will not conform with these explanatory models
- Near-field effects
- Examples:
  - Localised ischemia
  - metabolic disturbances
  - anti-arrhythmic medications
  - need for 12-lead ECG to record a 3-D phenomenon (spatially over-sample)
Questions?
Part II

- How to record the ECG:
  - Instrumentation
  - Safety & testing
  - Transmission & storage
  - Lead placements
  - Electrodes & skin preparation
Recording the surface ECG

- (Defib Protect)
- Isolate
- Filter
- Amplify
- Anti-alias
- Digitize
For use in medical situations, the ECG must be able to recover from a 5kV, 100A impulse (defibrillation)
Use large inductors and diodes
Isolation

- Opto-isolators
- DC-DC converters
Electromagnetic compatibility (EMC) — the ability of a device to function (a) properly in its intended electromagnetic environment, and (b) without introducing excessive electromagnetic energy that may interfere with other devices

Electromagnetic disturbance (EMD) — any electromagnetic phenomenon that may degrade the performance of equipment, such as medical devices or any electronic equipment. Examples include power line voltage dips and interruptions, electrical fast transients (EFTs), electromagnetic fields (radiated emissions), electrostatic discharges, and conducted emissions

Electromagnetic interference (EMI) — degradation of the performance of a piece of equipment, transmission channel, or system (such as medical devices) caused by an electromagnetic disturbance

Electrostatic discharge (ESD) — the rapid transfer of electrostatic charge between bodies of different electrostatic potential, either in proximity in air (air discharge) or through direct contact (contact discharge)

Emissions — electromagnetic energy emanating from a device generally falling into two categories: conducted and radiated. Both categories of emission may occur simultaneously, depending on the configuration of the device
RF testing
Transmission and storage

- Speed? 15 leads, @ 1kHz, 16 bits = 30 kb/s
- Data formats – headers, checksums
- Open/proprietary?
- Protocols (HL7)
- Synchronization between devices, clock drift, network timing, daylight savings
- Annotations, meta-data
- Encryption
- Storage, backup, remote access
Clinical Lead Placement

- **Einthoven Limb Leads:**

  The Einthoven **limb leads** (standard leads) are defined in the following way:
  
  \[
  \begin{align*}
  \text{Lead I: } & \quad V_1 = \Phi_L - \Phi_R \\
  \text{Lead II: } & \quad V_{II} = \Phi_F - \Phi_R \\
  \text{Lead III: } & \quad V_{III} = \Phi_F - \Phi_L
  \end{align*}
  \]

  where
  
  - \( V_1 \) = the voltage of Lead I
  - \( V_{II} \) = the voltage of Lead II
  - \( V_{III} \) = the voltage of Lead III
  - \( \Phi_L \) = potential at the left arm
  - \( \Phi_R \) = potential at the right arm
  - \( \Phi_F \) = potential at the left foot

  (The left arm, right arm, and left leg (foot) are also represented with symbols LA, RA, and LL, respectively.)

  According to Kirchhoff's law these lead voltages have the following relationship:

  \[ V_1 + V_{III} = V_{II} \]

  hence only two of these three leads are independent.
The temporal pattern of the heart vector combined with the geometry of the standard frontal plane limb leads.
Precordial leads

The location of these leads is as follows:

\( V_1 \): on the fourth intercostal space at the right sternal margin

\( V_2 \): on the fourth intercostal space at the left sternal margin

\( V_3 \): midway between leads \( V_2 \) and \( V_4 \)

\( V_4 \): on the fifth intercostal space at the midclavicular line

\( V_5 \): on the anterior axillary line at the horizontal level of lead \( V_4 \)

\( V_6 \): on the midaxillary line at the horizontal level of lead \( V_4 \)
- Frontal Plane Limb Leads

\[ \begin{align*}
& \text{aVR} \\
& -150^{\circ} -120^{\circ} -90^{\circ} -80^{\circ} -30^{\circ} 0^{\circ} +30^{\circ}
\end{align*} \]

\[ \begin{align*}
& \text{aVL} \\
& +150^{\circ}
\end{align*} \]
Full clinical 12 Lead ECG

SAGITTAL

FRONTAL

TRANSVERSE
The 12 lead ECG: A heart attack
Choosing a lead set

- Ischemia – at least 5 precordial leads
- Holter 3 lead
- VCG
- EASI
- Mason-Likar
- 12 lead diagnostic

- Derived ECGs
  - the Dower transform ...
And now ... the lab

- Record your own ECG
- Examine time domain properties
  This afternoon (1-5pm):
- Examine spectral properties
- Filter it
  Tomorrow afternoon (1-5pm):
- Build a VF detector (wed & thurs)
- Build a peak detector (next week)
- Extract respiration (next week)
- Compare it to the pulse ox (next week)
And now … the lab

- Start a Google doc - your lab diary – and share it with me
- Break into 4 groups of 4 (~ per placements)
- Choose a corner of the room.
- Connect equipment to each machine in each corner
- Follow instructions on handout:
  - Turn on the equipment, connect it
  - Start Labview and make sure it is working – make a noise recording (square wave)
  - Load the data in Matlab and view it
  - Choose a (first) volunteer from your group
  - Prep the electrode areas
  - Attach and look for characteristic pattern
  - Follow questions in ECG_lab_day1-4.pdf at: http://www.robots.ox.ac.uk/~gari/BSP/labs/data1/
  - and make notes (in your Google doc lab book!)
Connecting yourself up

- Lead I = ch 1: brown +ve (Left Arm), red -ve (Right Arm),
  Lead II = ch 2: black +ve (Left Leg), white (Right Arm) -ve
  Common = Earth: green (Right Leg) - just above right hip

Standard 5 lead connection box
Skin preparation

- Shave off hair (not today)
- Abrade skin (to remove dead skin and improve conductivity)
- Clean with alcohol
- Wait for alcohol to evaporate
- Place electrode patches on clinical locations
References

Physiology:

Hardware:

ECG lead systems:

Regulations
- FDA, *Good Clinical Practice VICH GL9*.
- FDA, *General Principles of Software Validation; Guidance for Industry and FDA Staff*. 