Heart physiology

Presented by:
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Place:
Bushehr University of Medical Sciences
The Cardiovascular system consists of:

- A pump
- A series of collecting tubes
- Size of a closed fist
- Shape
  - Apex: Blunt rounded point of cone
  - Base: Flat part at opposite of end of cone
- Located in thoracic cavity in mediastinum
Pericardium

- Fibrous pericardium
- Serous pericardium
  - Parietal pericardium
  - Visceral pericardium (or epicardium)

Pericardial cavity filled with pericardial fluid
Heart Wall

Three layers of heart wall:

- **Epicardium:** The serous membrane of smooth outer surface of heart

- **Myocardium:** Middle layer composed of cardiac muscle cells and responsible for heart contraction

- **Endocardium:** Smooth inner surface of heart chambers
Simple squamous epithelium

Loose connective tissue and fat

Epicardium (visceral pericardium)

Myocardium

Trabeculae carnea

Endocardium
External anatomy

- Four chambers
  - 2 atria
  - 2 ventricles
- Auricles
- Major veins
  - Superior vena cava
  - Pulmonary veins
- Major arteries
  - Aorta
  - Pulmonary trunk
Heart valves

- **Atrioventricular**
  - Tricuspid
  - Bicuspid or mitral

- **Semilunar**
  - Aortic
  - Pulmonary

- **Main function**
  Prevent blood from flowing back
## Comparison of cardiac and skeletal muscle

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<tr>
<th>Muscle type</th>
<th>Cardiac</th>
<th>Skeletal</th>
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<tr>
<td>Appearance</td>
<td>Striated</td>
<td>Striated</td>
</tr>
<tr>
<td>Fiber</td>
<td>Multiple cells</td>
<td>One cell</td>
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<tr>
<td>Excitation</td>
<td>Pacemaker potential</td>
<td>Neuromuscular junc.</td>
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<tr>
<td>Action potential</td>
<td>Long with plateau</td>
<td>Fast without plateau</td>
</tr>
<tr>
<td>Summation</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Tetanus</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Gap junction</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>T tubules</td>
<td>developed</td>
<td>weaker</td>
</tr>
</tbody>
</table>
## Comparison of muscles

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Cardiac Muscle</th>
<th>Skeletal Muscle</th>
<th>Smooth Muscle</th>
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<tr>
<td><strong>Histologic appearance</strong></td>
<td>Striated</td>
<td>Striated</td>
<td>Not striated</td>
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<tr>
<td><strong>Stimulus for excitation</strong></td>
<td>Pacemaker potentials and electrical coupling via gap junctions</td>
<td>Transmission at the neuromuscular junction</td>
<td>Variable: &lt;li&gt;Synaptic&lt;/li&gt; &lt;li&gt;Hormonal&lt;/li&gt; &lt;li&gt;Pacemakers&lt;/li&gt; &lt;li&gt;Coupling via gap junctions&lt;/li&gt;</td>
</tr>
<tr>
<td><strong>Electrical activity</strong></td>
<td>Long action potential plateau</td>
<td>No action potential plateau</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Excitation-contraction coupling</strong></td>
<td>Ca(^{2+})-induced Ca(^{2+}) release from sarcoplasmic reticulum</td>
<td>Voltage sensor triggers Ca(^{2+}) release from sarcoplasmic reticulum</td>
<td>Variable: &lt;li&gt;Ca(^{2+}) entry via L-type channels&lt;/li&gt; &lt;li&gt;IP(_3)-mediated Ca(^{2+}) release from stores&lt;/li&gt;</td>
</tr>
<tr>
<td><strong>Molecular basis of contraction</strong></td>
<td>Ca(^{2+})-troponin C</td>
<td>Ca(^{2+})-troponin C</td>
<td>Ca(^{2+})-calmodulin</td>
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<td><strong>Ending contraction</strong></td>
<td>Repolarization of action potential</td>
<td>Breakdown of acetylcholine in neuromuscular junction</td>
<td>Myosin light chain phosphatase activity</td>
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<td><strong>Control of force developed</strong></td>
<td>Regulation of Ca(^{2+}) entry</td>
<td>Spatial and temporal summation</td>
<td>Latch bridge state</td>
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<tr>
<td><strong>Metabolism</strong></td>
<td>Oxidative</td>
<td>Oxidative and glycolytic fiber types</td>
<td>Oxidative</td>
</tr>
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</table>
Physiology of cardiac muscle

- Structure
- Electrical activities
- Mechanical activities
- Electrocardiography
The heart is composed of three major types of cardiac muscle:

- Atrial muscle
- Ventricular muscle
- Specialized excitatory and conductive muscle fibers
Cardiac muscle as a **syncytium**

- Intercalated discs
- Gap Junctions
- Atrial syncytium
- Ventricular syncytium
Electrical coupling of cardiac myocytes
Gap junctions allow AP propagation from cell to cell
Contraction of cardiac muscle

- Contractile elements
- Role of intercalated discs and cardiac cyncytium
- Transverse-tubules or tubular system
- Role of sarcoplasmic reticulum
T tubules are extensions of plasma membrane, penetrating the muscle cell at two points in each sarcomere: the junctions of the A and I bands.
Membrane depolarization opens the L-type Ca²⁺ channel.

Ca²⁺ entering the cell via L-type Ca²⁺ channels also can activate the Ca²⁺-release channels. However, this pathway is not essential in skeletal muscle.

Mechanical coupling between the L-type Ca²⁺ channel and the Ca²⁺-release channel causes the Ca²⁺-release channel to open.

Ca²⁺ exits the SR via the Ca²⁺-release channel and activates troponin C, leading to muscle contraction.

L-type Ca²⁺ channel (ryanodine receptor) [tiramer]
The process by which the action potential of the cardiac myocyte leads to contraction is termed **excitation-contraction coupling**.
Excitation contraction coupling
به یاد ندارم نابینایی به من تنها زده باش德
اما هر وقت تنم به جمعت نادان خورده گفتند:
"مگه کوری؟"
Electrical activity of the heart
Specialized excitatory and conductive system of the heart

- S-A node
- Internodal pathways
- A-V node
- A-V bundle
- Left and right bundles of Purkinje fibers
Home work for students:

- Accessory AV pathways in wolff-Parkinson-White syndrome
- Sick sinus syndrome
Atrioventricular node, and delay of impulse conduction

- Internodal pathways
- Transitional fibers
- A-V node
- Atrio-ventricular fibrous tissue
- Penetrating portion of A-V bundle
- Distal portion of A-V bundle
- Left bundle branch
- Right bundle branch
- Ventricular septum
Summary of the spread of the impulse through the heart

- The sinus node as the pacemaker of the heart
- Abnormal pacemakers – ectopic pacemaker

SAN: Primary pacemaker (70-80)
AVN: Secondary pacemaker (40-60)
Purkinje system: Tertiary pacemaker (15-40)
Electrical activity of the heart

- Two main types of action potentials occur in the heart:
  
  I. The fast response occurs in:
     - Normal atrial and ventricular myocytes
     - Specialized conducting fibers

  I. The slow response occurs in:
     - Sinoatrial (SA) node
     - Atrioventricular (AV) node
Electrical activity of the heart

Transmembrane Potentials:
- Resting membrane potentials (-85 mv)
- Action potentials

**FIGURE 2-5** Comparison of action potentials from a nerve cell and a nonpacemaker cardiac myocyte. Cardiac action potentials are much longer in duration than nerve cell action potentials.
**Fast voltage-activated Na⁺ channels** can be blocked by tetrodotoxin and lidocaine which is used to treat certain cardiac arrhythmias.

**K**ᵣₒ **(Transient outward) channels** can be blocked by 4-aminopyridine

**L- type Ca⁺⁺ channels are blocked by Ca⁺⁺ channel antagonists** such as verapamil, amlodipine, and diltiazem
Ca\textsuperscript{++} channels

- Two types of Ca channels have been identified in cardiac tissue:
  - L type
  - T type
Effects of diltiazem on action potentials and isometric contractile forces recorded from an isolated papillary muscle
A

Membrane potential (mV)

Time (s)

Relative membrane permeability

Time (s)

$P_{\text{Na}^+}$

$P_{\text{K}^+}$

$P_{\text{Ca}^{2+}}$
Restoration of ionic concentrations (phase 4)

- Na⁺,K⁺-ATPase
- 3Na⁺-1Ca++ antiporter
- Ca++ pump
Ionic basis of the slow response

Phase 0
Phase 2
Phase 3
Phase 4 (Raising phase)
Ionic basis of slow response
Fast response

Ventricular Cell

Ion Conductances

ERP

200 ms

gK⁺
gCa²⁺
gNa⁺

Slow response

SA Node

Ion Conductances

gCa²⁺
gK⁺

Iₚ
PROBLEM 2-2

A drug is found to partially inactivate fast sodium channels. How would this drug alter the action potential in a ventricular myocyte? How would the drug alter conduction velocity within the ventricle?

Because phase 0 of myocyte action potentials is generated by activation of fast sodium channels, partial inactivation of these channels would decrease the upstroke velocity of phase 0 (decrease the slope of phase 0). Partial inactivation also would decrease the maximal degree of depolarization. These changes in phase 0 would reduce the conduction velocity within the ventricle. Blockade of fast sodium channels is the primary mechanism of action of Class I antiarrhythmic drugs such as quinidine and lidocaine.
Differences between the fast and slow-response

1. The slow-response cells lack the early repolarization phase (phase 1)

2. Phase 4 of the fast-response cells is considerably more negative than that of the slow-response cells

3. The slope of the upstroke (phase 0), the amplitude of the action potential, and the overshoot are greater in the fast-response than in the slow-response cells

4. In slow-response cardiac tissue, the action potential is propagated more slowly and conduction is more likely to be blocked than in fast-response cardiac tissue
In the clinic

- Fast responses may change to slow responses under certain pathological conditions

- For example, in coronary artery disease, a region of cardiac muscle may be deprived of its normal blood supply. As a result, $K^+$ in the interstitial fluid that surrounds the affected muscle cells rises because $K^+$ is lost from the inadequately perfused (or ischemic) cells.
The relationships between the action potential and contraction of cardiac muscle
Conduction speed in cardiac fibers

Conduction in atrial, ventricular, internodal, purking fibers and nodal

- Conduction of fast response: (1 to 4 m/s).
- Conduction of the slow response: (0.02 to 0.1 m/s)
Factors affect the conduction velocity

1. Fiber diameter
2. Rate of depolarization (dVm/dt)
3. Amplitude of the action potential
4. The level of resting membrane potential
5. Number of gap junctions
Factors affect the rate (chronotropic effect)

- Change in the slope of phase 4
- Change in resting membrane potentials
- Change in threshold

✓ Tachycardia
✓ Bradycardia
membrane potential, mV

-10
-30
-50
-70
-90

time

sympathetic tone

intrinsic

parasympathetic tone

threshold potential
برای دانشجویان:
پروتکل آزمایشی را طراحی نمایید که در اثر تحريك پاراسمپاتیکی گره دهليزی-بطنی پتانسیل عملی از دهليز ها به دسته های هیس منتقل نمی شود.
<table>
<thead>
<tr>
<th>Current</th>
<th>Channel</th>
<th>Gating mechanism</th>
<th>Functional role</th>
</tr>
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<tbody>
<tr>
<td>$i_{K_{1}}$</td>
<td>$K^+$ channel (inward rectifier)</td>
<td>Voltage</td>
<td>Maintains high $K^+$ permeability during phase 4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Its decay contributes to diastolic depolarization</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Its suppression during phases 0 to 2 contribute to plateau</td>
</tr>
<tr>
<td>$i_{Na}$</td>
<td>$Na^+$ channel (fast)</td>
<td>Voltage</td>
<td>Accounts for phase 0 of action potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Inactivation may contribute to phase 1 of action potential</td>
</tr>
<tr>
<td>$i_{To}$</td>
<td>$K^+$ channel (transient outward)</td>
<td>Voltage</td>
<td>Contributes to phase 1 of action potential</td>
</tr>
<tr>
<td>$i_{Ca}$</td>
<td>$Ca^{2+}$ channel (slow inward, L channels)</td>
<td>Voltage</td>
<td>Primarily responsible for phase 2 of action potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Inactivation may contribute to phase 3 of action potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Is enhanced by sympathetic stimulation and $\beta$-adrenergic agents</td>
</tr>
<tr>
<td>$i_{K}$</td>
<td>$K^+$ channel (delayed rectifier)</td>
<td>Voltage</td>
<td>Causes phase 3 of action potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>May be enhanced by increased intracellular $Ca^{2+}$</td>
</tr>
<tr>
<td>$i_{KATP}$</td>
<td>$K^+$ channel (ATP-sensitive)</td>
<td>Ligand</td>
<td>Increases $K^+$ permeability when [ATP] is low</td>
</tr>
<tr>
<td>$i_{KACH}$</td>
<td>$K^+$ channel (acetylcholine-activated)</td>
<td>Ligand</td>
<td>Responsible for effects of vagal stimulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Decreases diastolic depolarization (and heart rate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hyperpolarizes resting membrane potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Shortens phase 2 of the action potential</td>
</tr>
<tr>
<td>$i_{(\text{&quot;funny&quot;})}$</td>
<td>$Na^+$ channel (pacemaker current)</td>
<td>Both</td>
<td>Contributes to the diastolic depolarization</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Is enhanced by sympathetic stimulation and $\beta$-adrenergic agents</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Is suppressed by vagal stimulation</td>
</tr>
</tbody>
</table>
Cardiac excitability

- Refractory periods

- Absolute or effective refractory period
- Relative refractory period
Fast response

Slow response
- Normal pacemaker
- Automaticity (the ability to initiate its own beat)
- Rhythmicity (the regularity of pacemaking activity)

- Ectopic pacemaker or ectopic foci

- Ectopic foci may become pacemakers when;
  - Their own rhythmicity becomes enhanced
  - The rhythmicity of the higher-order pacemakers becomes depressed
  - All conduction pathways between the ectopic focus and regions with greater rhythmicity become blocked.

- Overdrive suppression
In the clinic

Reentry

- Under certain conditions, a cardiac impulse may re-excite some myocardial region.
- Reentry is responsible for many clinical arrhythmias
- **The conditions that promote reentry:**
  - Prolongation of the conduction time (Block or path elongation)
  - Shortening of the effective refractory period.
هیچ وقت رازت رو به کسی نگو؛ وقتی خودت نمیتونی حفظش کنی، چطور انتظار داری کس دیگه ای برای راز نگه داره؟
Mechanical activity of the heart
Cardiac cycle

- **Ventricular systole**
  1. Isovolumic contraction
  2. Ejection: (Rapid and slow phase)

- **Ventricular diastole**
  1. Isovolumic relaxation
  2. Rapid filling phase
  3. Diastasis
  4. Atrial systole
Cardiac cycle

1. Systole: Period of isovolumic contraction.
   - Semilunar valves closed
   - AV valves closed

2. Systole: Period of ejection.
   - Semilunar valves opened
   - AV valves closed

   - Semilunar valves closed
   - AV valves closed

   - AV valves opened

5. Diastole: Active ventricular filling.
   - AV valves opened
Events during cardiac cycle (Wiggers diagram)
Events during cardiac cycle

- Ventricular pressure wave
  - Systolic and diastolic pressure

- Aortic pressure wave
  - Systolic and diastolic pressure

- Atrial (jugular) pressure wave
  - a, c and v waves

- Left ventricular volume
  - EDV, ESV, SV, EF

- Relationship of atrial and ventricular contraction to the waves of the ECG

- Heart sounds
  - S1, S2, S3, S4
Heart Sounds

- **First heart sound (S1),** which occurs with closure of the A-V valves, is mainly due to oscillation of blood in the ventricular chambers, and vibration of the chamber wall.

- **Second heart sound (S2),** which occurs with closure of the semilunar valves, is composed of higher frequency vibration (higher pitch), is shorter in duration and lower intensity.

- **Third heart sound (S3),** Usually heard in children with thin chest walls or in patients with left ventricular failure, consist of few low-intensity, low frequency vibration heard best in the apex of the heart. It is caused because of blood entering the ventricle and ventricular wall vibration.

- **Forth heart sound (S4),** or atrial sound. Consist of little low-frequency oscillation.
Heart sounds

1. First heart sound (S1) (lub)
2. Second heart sound (S2) (dup)
3. Third heart sound
4. Fourth heart sound

- During inspiration, A2 and P2 are often heard as separate sounds (Splitting)

- **Note:** The physiologic splitting of S2 is caused by delayed closure of the pulmonic valve and earlier closure of the aortic valve.

- **Students:**
  1. Why Pulmonary valve closes later than aortic valve during inspiration?
In the clinic

- Paradoxical splitting of S2
  ✓ When closure of the aortic valve is delayed, causing P2 to occur first, followed by A2.

- The most notable causes are:
  ✓ Aortic stenosis (which prolongs left ventricular systole)
  ✓ Left bundle branch block (which delays the onset of left ventricular contraction).
Pressure-volume relationship
Pressure-volume relationship

ABC: Ventricular Diastole
CD: Isovolumic contraction.
DF: Ventricular Systole
DEF: Ejection (Rapid and slow ejection)
FA: Isovolumic relaxation
ABC: Filling phase
(Rapid filling, diastasis & Atrial systole)

End diastolic volume
End systolic volume
Stroke volume
Ejection fraction = SV/EDV
CO = HR . SV

Example: A healthy man of average size is found to have a resting end-diastolic volume of 140 mL and a resting SV of 70 mL: \( \text{EF} = \frac{70}{140} = 0.5 \text{ (50\%)} \)
Pressure-volume relationship

- Aortic valve opens at point C
- Aortic valve closes at point D
- Mitral valve opens at point A
- Mitral valve closes at point B
- Stroke volume
- Isovolumetric contraction
- Isovolumetric relaxation
- Period of ejection
- End-systolic volume
- End-diastolic volume

Left intraventricular pressure (mm Hg) vs. Left ventricular volume (ml) graph.
Factors affect the ventricular function (SV)

- ↑ Preload → ↑ SV
- ↑ Afterload → ↓ SV
- ↑ Contractility → ↑ SV

Factors affect the heart contractility (inotropic effect)
- Positive inotropic effect
- Negative inotropic effect

Factors affect the heart rate (chronotropic effect)
- Positive chronotropic effect
- Negative chronotropic effect

Factors affect conduction velocity of action potentials through the heart (dromotropic effect)
- Positive dromotropic effect
- Negative dromotropic effect
Pressure-volume curve, preload and afterload
Contractility

- Contractility is the performance of the heart at a given preload and afterload.

- Indices of Contractility
  1. End-diastolic pressure
  2. Rising ventricular pressure (Max. dP/dt)
  3. Ejection fraction (in the clinic)
Question

The figure below shows pressure-volume loops for two situations. When compared with loop A, loop B demonstrates:

(A) Increased preload                    (B) Decreased preload
(C) Increased contractility            (D) Increased afterload
(E) Decreased afterload
از ۳ نفر هر گز متنفر نباش: فرورديني ها، مهری ها، اسفندی ها
چون بهترین هستند

سه نفر را هر گز نرنجون: ارديبهشتی ها، تیری ها، دی ماهی ها
چون صادق هستند

سه نفر رو هیچوقت ندار از زندگیت بیرون برن: شهریوری ها، آذری ها، آبانی ها
چون به درد دلت گوش میدهند

سه نفر رو هر گز از دست نده: مردادی ها، خردادی ها، بهمنی ها
چون دوست واقعی هستند
Regulation of heart function

1. Intrinsic autoregulation

2. Extrinsic regulation
Regulation of heart function

1. Intrinsic autoregulation

- Frank-starling mechanism, is invoked in response to changes in the resting length of myocardial fibers.

Preload (End diastolic volume) vs. Afterload (Arterial pressure)
Rate induced regulation, is evoked by changes in the frequency of the heartbeat. By increasing the rate, the force of contraction will enhance. This is known as *staircase or treppe*, phenomenon.

Cycle length 20 sec and 0.63 sec
Regulation of heart function

2. Extrinsic regulation

✓ Neural control
  - Sympathetic Influences
  - Parasympathetic Influences

✓ Chemical control
  - Adrenomedullary hormones
  - Adrenocortical hormones
  - Thyroid hormones
  - Insulin
  - Glucagon
  - Anterior Pituitary Hormones

✓ Effect of ions

✓ Temperature

✓ Stretch

But main control of the heart beat is by autonomic nervous system.
Neural control

- **Sympathetic (right and left)**
  a. NE (↑ P Na & Ca)
  b. Rate (chronotropic effect)
  c. Conduction velocity (dromotropic effect)
  d. Contractility (ionotropic effect)

- **Parasympathetic (right and left)**
  a. ACH (↑ P K⁺)
  b. Rate
  c. Conduction velocity
  d. Contractility
Sympathetic stimulation

Before stellate ganglion stimulation

During stellate ganglion stimulation

Left ventricular pressure (mm Hg)

dP/dt

Time (sec)
Parasympathetic stimulation

Before vagal stimulation

During vagal stimulation

Left ventricular pressure (mm Hg)

$\text{dP/dt}$
Parasympathetic tone on heat rate predominates in healthy, resting individuals.
Regulation of heart function

- **Effect of ions**
  1. **Effect of sodium**
     - $\downarrow$ Na$^+$ → $\downarrow$ amplitude of the AP and when Na$^+$ is reduced from its normal value of about 140 to about 20 mEq/L, the cell is no longer excitable.
  2. **Effect of potassium**
     - Alterations in extracellular K$^+$ can significantly change Vm, with hypokalemia causing hyperpolarization and hyperkalemia causing depolarization.

- Hyperkalemia
- Pprolonged PR interval
- Widening of the QRS complex
- Blockage of the AV node
- Loss of P waves
- Eventual merging of the QRS complex with the T wave, which produces a “sine wave” pattern that can degenerate into ventricular fibrillation.
3. Effect of Ca$^{++}$
   - ↓ Ca extracellular fluid → ↓ contractile force and eventual arrest in diastole.
   - Conversely, ↑ Ca extracellular fluid → ↑ contractile force, and very high Ca induces cardiac arrest in systole (rigor).

4. Effects of H$^+$
   - ↑ H$^+$ → ↓ contractile force
The fick principle for measuring cardiac output

- **QT = VO2 / (CaO2 − CvO2)**
  - QT = Cardiac output
  - VO2 = O2 consumption
  - CaO2 = Arterial O2 content
  - CvO2 = Mixed venous O2 content

- Example: A person consumes 250 mL of O2 per min. Arterial O2 content is 20 mL of O2 per dL of blood, and the O2 content of mixed venous blood is 15 mL of O2 per dL of blood:

  \[
  QT = \frac{250 \text{ mL/min}}{(20 \text{ mL/dL} - 15 \text{ mL/dL})} = 50 \text{ dL/min}
  \]

  \[
  QT = 50 \text{ dL/min} = 5 \text{ L/min}
  \]
Electrocardiography & Electrocardiogram (ECG)
- Electrocardiography
- Electrocardiograph
- Electrocardiogram
Electrocardiography (EKG)
Relation of the action potential to the QRS and T waves

Note: no potential is recorded in the ECG when the ventricular muscle is either completely polarized or completely depolarized
What are the valuable gains of ECG?

- The anatomic orientation of the heart
- The relative size of its chamber and hypertrophy
- A variety of disturbances of rhythm and conduction
- The extent, location, and progress of ischemic damage to the myocardium
- The effect of altered electrolyte concentration
- The influence of certain drugs (notably digitalis and its derivatives)

*Note: EKG is not the only reference for the above disturbance.*
Principles of ECG recording

- Depolarization waves versus repolarization waves
Principles of ECG recording

- Recording electrical potentials from a partially depolarized mass of syncytial cardiac muscle
Principles of ECG recording

- Flow of electrical currents in the chest around the heart
ECG recording

- P wave
- PQ segment
- Q wave
- R wave
- S wave
- ST segment
- T wave
- TP segment
ECG recording

- P wave
- PQ segment
- Q wave
- R wave
- S wave
- ST segment
- T wave
- TP segment
ECG recording
ECG waveforms

An ECG tracing consists of:

1. P wave
2. QRS complex
3. T wave

- There are four major ECG intervals
  1. PR interval
  2. QRS interval
  3. QT interval
  4. RR interval
Voltage and time calibration of the ECG

- Normal voltages in the ECG
  - QRS: 1 mv, P: 0.1 to 0.3 mv, T: 0.2 to 0.3 mv
The ECG monitoring paper
Determination of HR from ECG

\[ \text{Heart rate} = \frac{300}{R} \]

\[ \frac{1500}{R R} = \text{ضربان قلب} = \text{تعداد مربع های بزرگ بین دو قله} \]

\[ \text{Heart rate} = \frac{60}{R} \text{زمان بین دو قله} \]
Determination of HR from ECG

\[
\frac{1500}{RR} = \text{ضربان قلب} = \text{Tعداد مربع مای کوچک بین دو قله}
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Electrocardiographic leads

- The 12 conventional ECG leads are divided into two groups:
  - Six limb (extremity) leads which record potentials transmitted onto the frontal plane.
    - Bipolar leads (I, II, and III)
    - Unipolar (augmented) leads (aVR, aVL, and aVF).
  - Six chest (precordial) leads which record potentials transmitted onto the horizontal plane
    - V1, V2, V3, V4, V5, V6
Three bipolar limb leads

- I
- II
- III

- Einthoven’s triangle
- Einthoven's law: II = I + III
Three bipolar limp leads
ECG of bipolar leads
Three augmented unipolar limb leads

- aVR (augmented voltage of right hand)
- aVL (augmented voltage of left hand)
- aVF (augmented voltage of left foot)
Three augmented unipolar limb leads

**D. Goldberger limb leads (unipolar)**

**E. Cabrera circle**
Chest leads (Precordial leads)

- V1 to V6
Chest leads (Precordial leads)
Additional chest leads

V3R and V4R
Useful in detecting evidence of acute right ventricular ischemia
Precordial leads ECG
The following rules can be used in interpreting the ECG:

- A wave of depolarization traveling toward a positive electrode results in a positive deflection in the ECG trace or (A wave of depolarization traveling away from a positive electrode results in a negative deflection).

- A wave of repolarization traveling toward a positive electrode results in a negative deflection. (A wave of repolarization traveling away from a positive electrode results in a positive deflection)

- A wave of depolarization or repolarization oriented perpendicular to an electrode axis has no net deflection.

- The instantaneous amplitude of the measured potentials depends upon the orientation of the positive electrode relative to the mean electrical vector.

- Voltage amplitude (positive or negative) is directly related to the mass of tissue undergoing depolarization or repolarization.
Electrocardiographic Interpretation of Cardiac Muscle and Coronary Blood Flow Abnormalities: Vectorial Analysis
Principles of vectorial analysis of ECG

- Use of vectors to represent electrical potentials
  - Arrowhead
  - Length of the arrow

- Resultant vector in the heart at any given instant (*instantaneous mean vector*)
Mean QRS vector

- Mean QRS vector, is about +59 degrees
- This means that during most of the depolarization wave, the apex of the heart remains positive with respect to the base of the heart
Vectorial analysis of potentials recorded in different leads using instantaneous mean vector

**Note:** when the vector in the heart is in a direction almost perpendicular to the axis of the lead, the voltage recorded in the electrocardiogram of this lead is very low. Conversely, when the heart vector has almost exactly the same axis as the lead axis, essentially the entire voltage of the vector will be recorded.
Vectorial analysis of potentials in the limb leads

**Note:** a positive vector in a lead will cause recording in the ECG above the zero line, whereas a negative vector will cause recording below the zero line.
Vectorial analysis of the normal ECG (QRS complex)
ECG during repolarization (T wave)
Depolarization & repolarization of the atria
Determining the electrical axis from standard lead ECGs
Abnormal ventricular conditions that cause **axis deviation**

- Normal axis, 59 degrees
- Normal range, -30 - + 90 (20-100 degrees)
Abnormal ventricular conditions that cause axis deviation

1. Changes in the position of heart in the chest
   A. Shifts to the left
      a. End of deep expiration
      b. Lying down
      c. Fat people
   
   B. Shift to the right
      a. End of deep inspiration
      b. Standing up
      c. Tall lanky people
2. **Hypertrophy of one ventricle**

   a. Right V. (pulmonary hypertension, pulmonary valve stenosis, tetralogy of Fallot, interventricular septal defect)

   b. Left V. (hypertension, aortic regurgitation and stenosis)
Abnormal ventricular conditions that cause axis deviation

3. Bundle branch block
   a. L.B.B.B
   b. R.B.B.B

http://en.ecgpedia.org/wiki/QRS_axis
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