بسم الله الرحمن الرحيم

PHYSIOLOGY

وَلَقَدْ خَلَقْنَا ٱلْإِنسَـٰنَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِۦ نَفْسُهُۥ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ ٱلْوَرِيدِ

اللهم إنّا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

MID | Lecture 4

Cardiac Muscle Physiology (Pt.2) & ECG (Pt.1)

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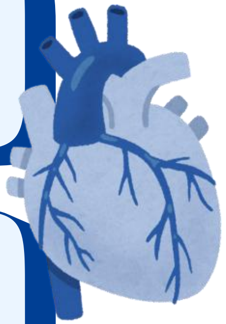
Reviewed by: Almothana Khalil



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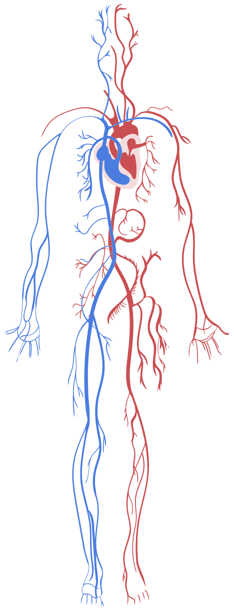


Pre-Studying Notice

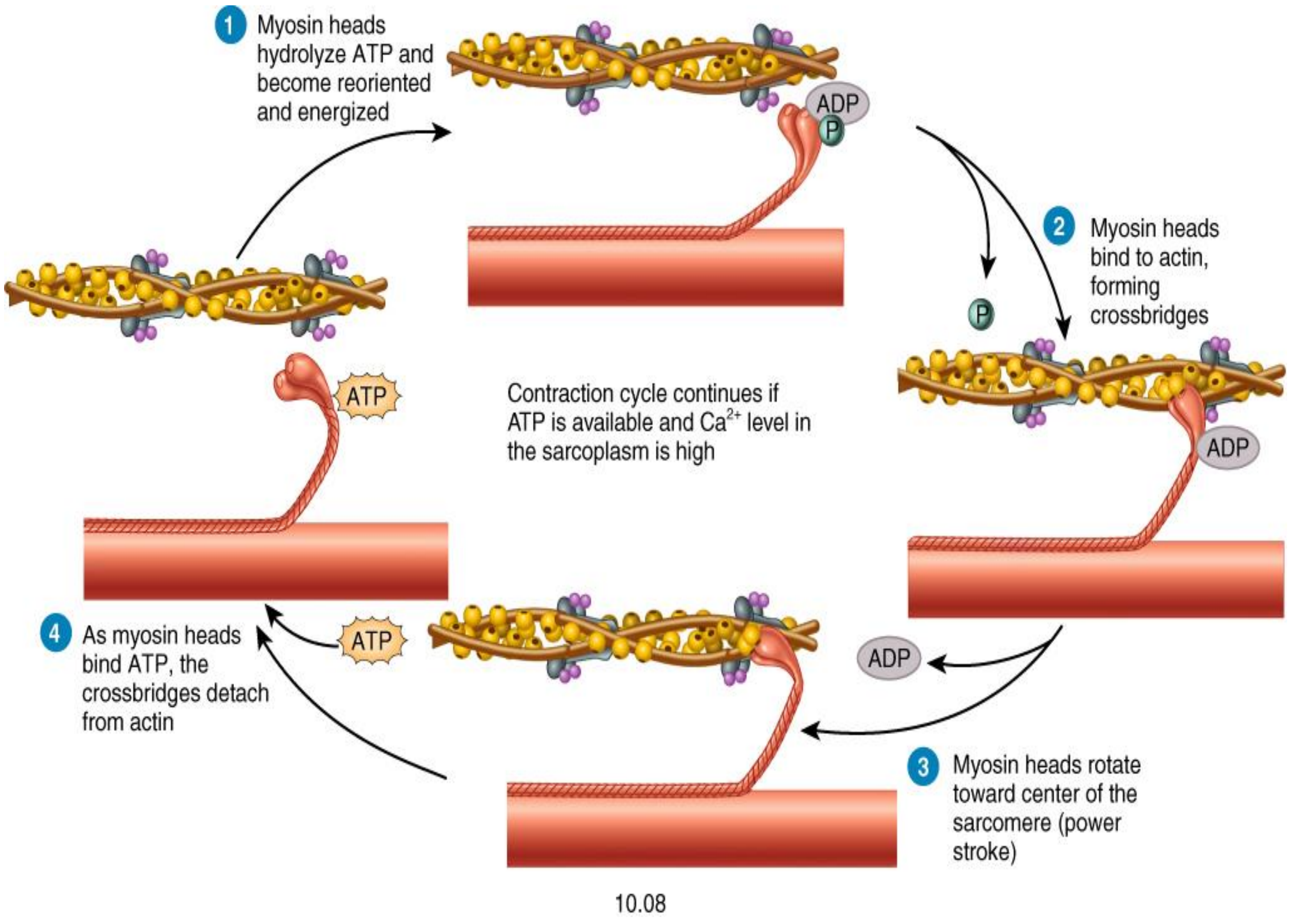
- This lecture is heavy with new information, so feel free to take multiple pauses along the journey.
- Do not hesitate to reach out for additional resources or utilize AI to consolidate your understanding.

استعن بالله ولا تعجز

Cardiac Muscle Vs. Skeletal Muscle Contraction

- Sliding filament hypothesis
- No tetany (Long refractory period because of plateau)
- Fatty acids main source of energy unlike skeletal muscle (Anaerobic and Aerobic)
- Attachment and detachment cycle and ATP dependence is the same

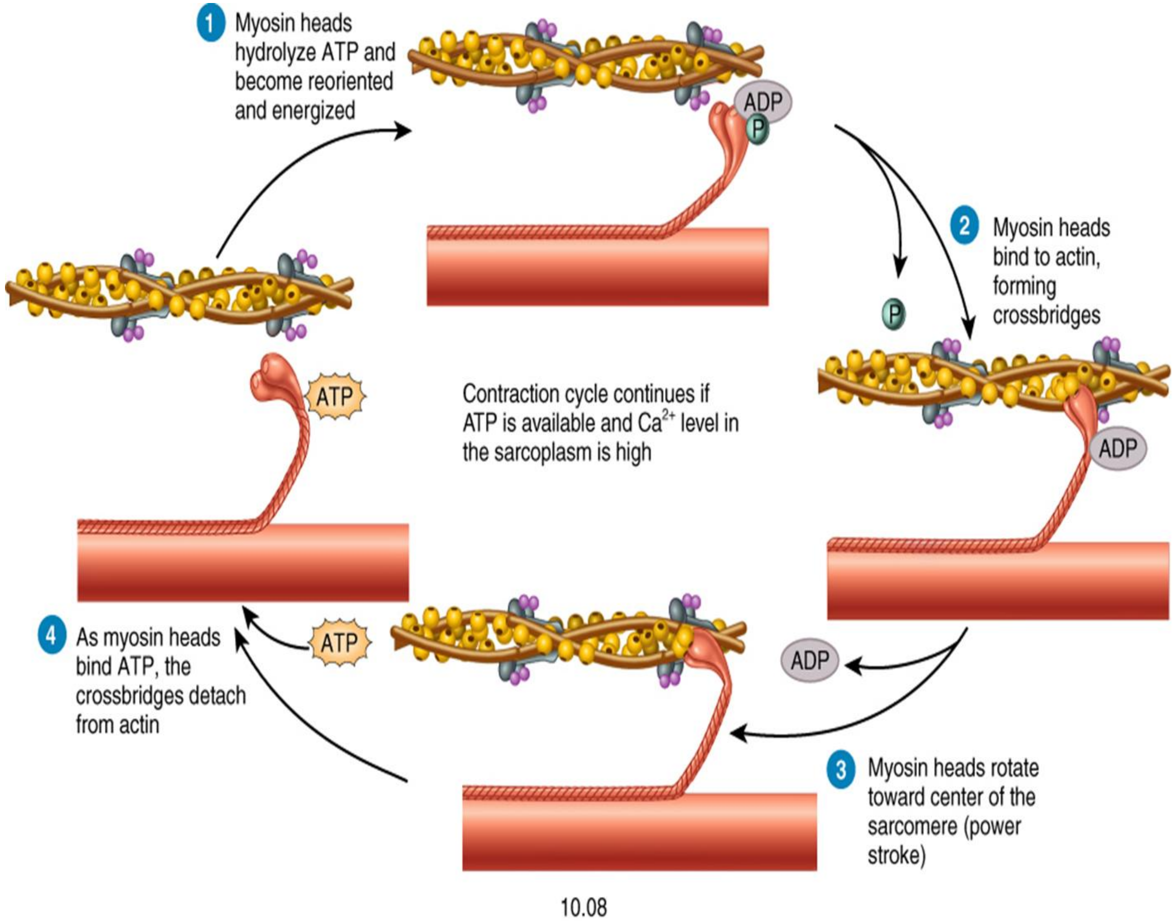
The Contraction-Relaxation Cycle – 1



The Contraction-Relaxation Cycle – 2

- The contraction-relaxation cycle involves the myosin heads, which must be "charged" with energy to interact with actin. This charged state occurs when ATP is hydrolyzed into ADP and inorganic phosphate (Pi), which remain bound to the myosin head. Only in this charged state can the myosin heads attach to actin and initiate the cross-bridge cycle.

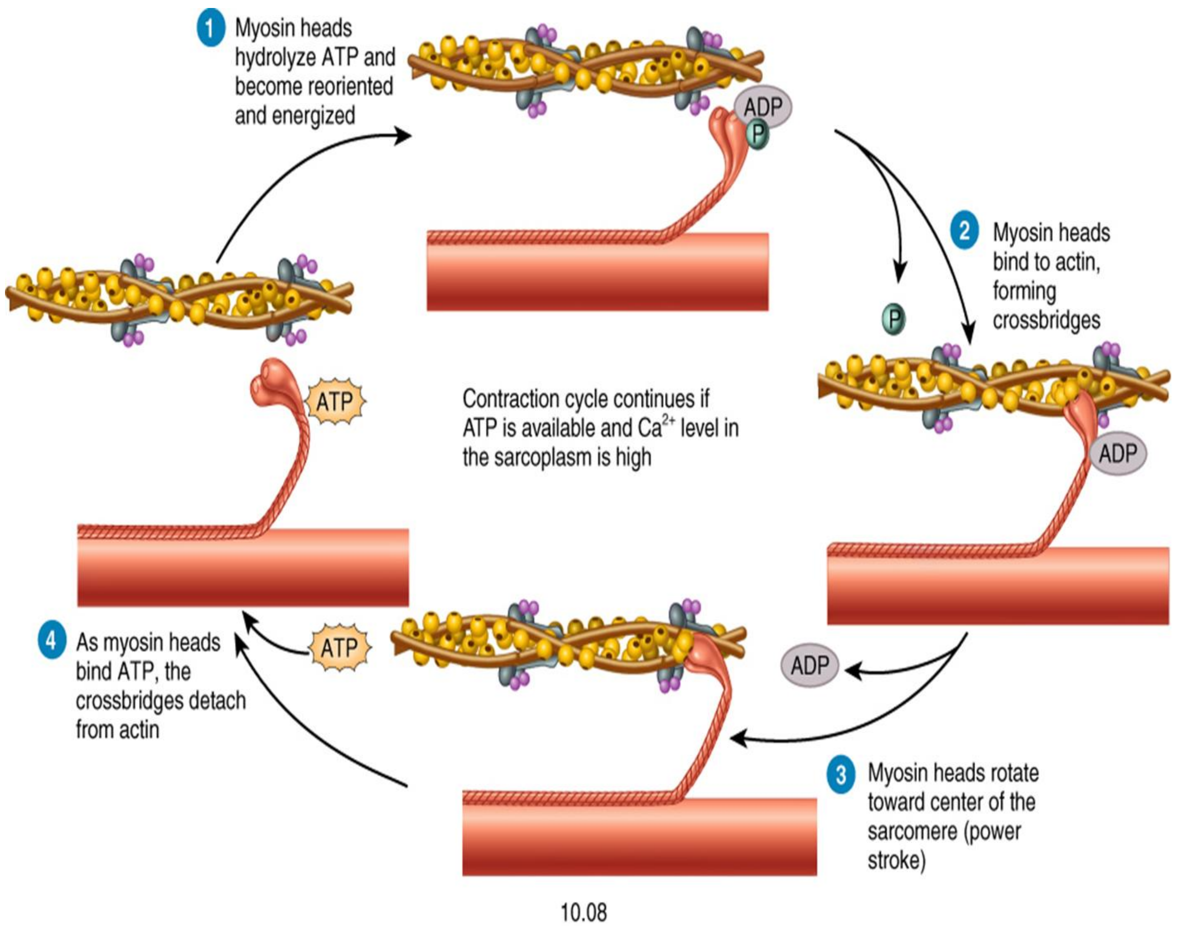
- In the resting state, the actin binding sites are covered by tropomyosin, preventing interaction with myosin. When calcium ions (Ca^{2+}) bind to troponin, a conformational change occurs that shifts tropomyosin away from these binding sites, allowing myosin heads to bind to actin. Once myosin binds to actin, they form a complex called **actomyosin**. The release of ADP and Pi from the myosin head triggers the **power stroke**, during which the myosin head pivots and pulls the actin filament inward, causing shortening of the sarcomere and generation of force.



The Contraction-Relaxation Cycle – 3

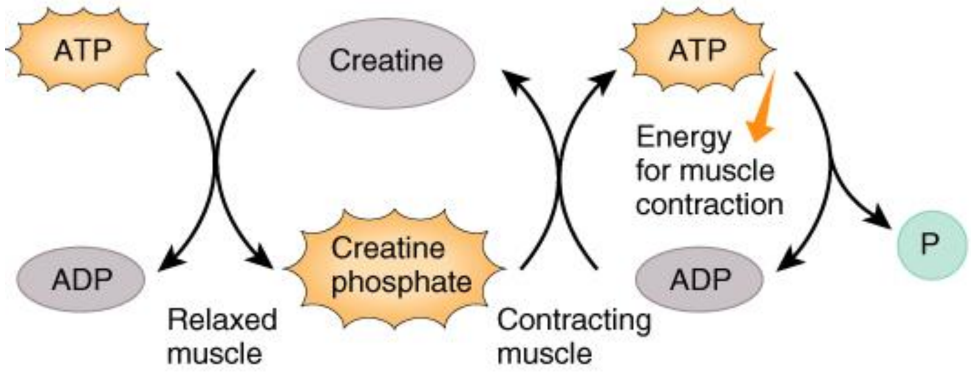
- After the power stroke, the myosin head must detach from actin to begin a new cycle. ATP binding to myosin is essential for detachment of the cross-bridge. Once ATP binds, it is hydrolyzed to ADP and Pi, which re-energizes the myosin head, preparing it for another interaction with actin. Therefore, ATP is required both for detachment and for reattachment of the myosin head during repeated contraction cycles.

- After death, ATP production ceases. Without ATP, myosin heads remain attached to actin filaments and cannot detach, resulting in a condition called **rigor mortis**. This postmortem muscle stiffness occurs due to the lack of ATP required for breaking the actin-myosin cross-bridges.



ATP Sources in Cardiac Muscles – 1

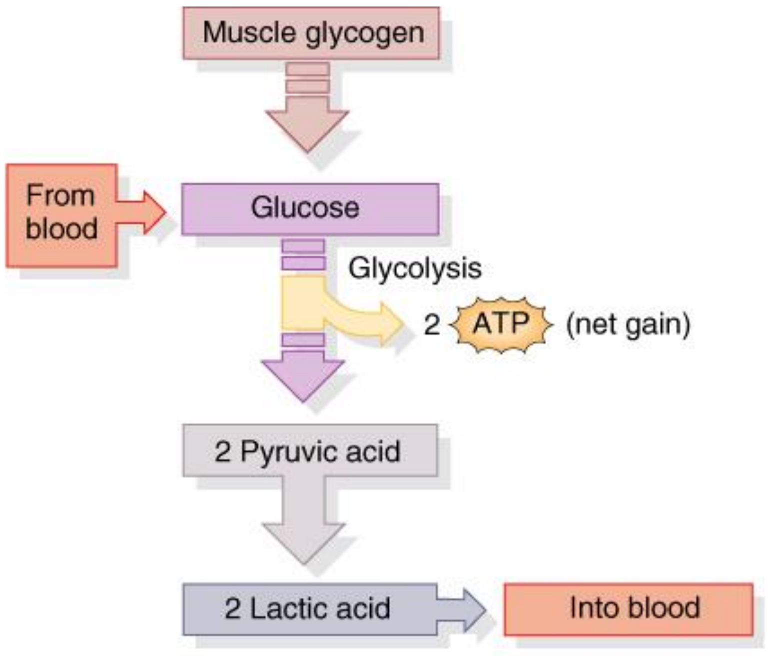
- When discussing ATP levels in muscle contraction, the first source of energy comes from the small amount of ATP already present within the muscle fibers. However, this stored ATP is very limited and can sustain maximal muscle contraction for only a few seconds. To maintain energy supply, the muscle relies on another high-energy compound known as **creatine phosphate** (**phosphocreatine**).
- Creatine phosphate acts as an immediate reserve for the regeneration of ATP. When muscle activity begins, creatine phosphate donates its high-energy phosphate group to ADP to rapidly form ATP through the reaction:
- Creatine phosphate + ADP → Creatine + ATP
- In cases of myocardial infarction, the enzyme creatine phosphokinase (CPK) becomes elevated in the blood.



(a) ATP from creatine phosphate

ATP Sources in Cardiac Muscles – 2

- When activity continues beyond this period, muscles shift to **anaerobic metabolism**, where glycogen is broken down into glucose and then converted into pyruvic acid and subsequently lactic acid in the absence of oxygen. This pathway produces ATP more slowly than the creatine phosphate system but still provides energy rapidly enough for short-term, high-intensity efforts.
- However, anaerobic glycolysis is less efficient and produces limited ATP compared to aerobic metabolism. Additionally, the accumulation of lactic acid can lead to muscle fatigue and, in the heart muscle, may contribute to pain or discomfort due to increased acidity and reduced efficiency of contraction.



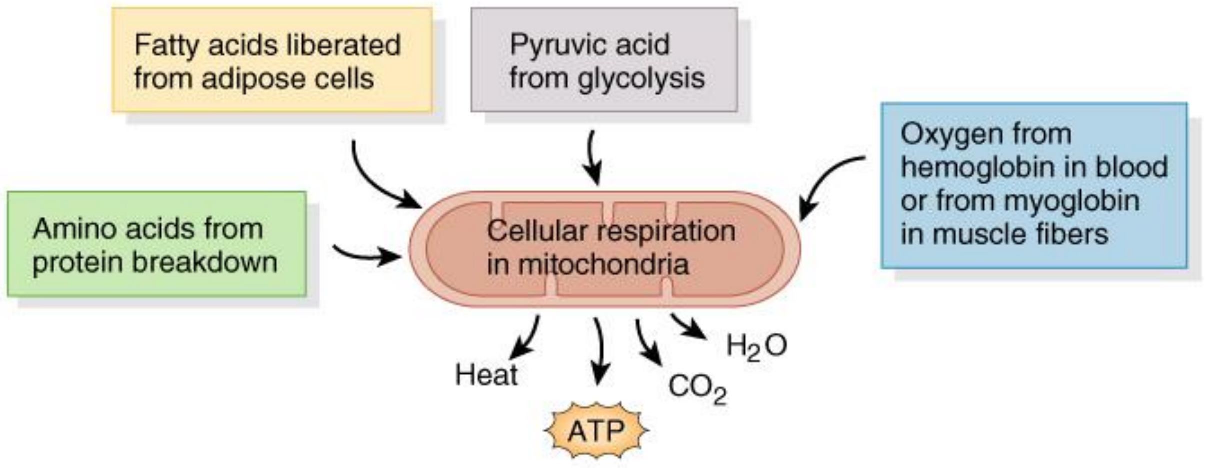
(b) ATP from anaerobic respiration

ATP Sources in Cardiac Muscles – 3

- If sufficient oxygen is available, **aerobic metabolism** becomes the dominant source of ATP, allowing for sustained energy generation during prolonged activities such as endurance exercises.

- Oxidative phosphorylation is the main process responsible for ATP production during aerobic respiration. It primarily utilizes fatty acids (derived from phospholipids and other lipid sources) as substrates for energy generation. This process takes place in the mitochondria and requires oxygen.

- It produces a tremendous amount of ATP compared to anaerobic metabolism, making it **the most efficient energy-generating mechanism** in cardiac and skeletal muscles during prolonged, low-intensity activity.

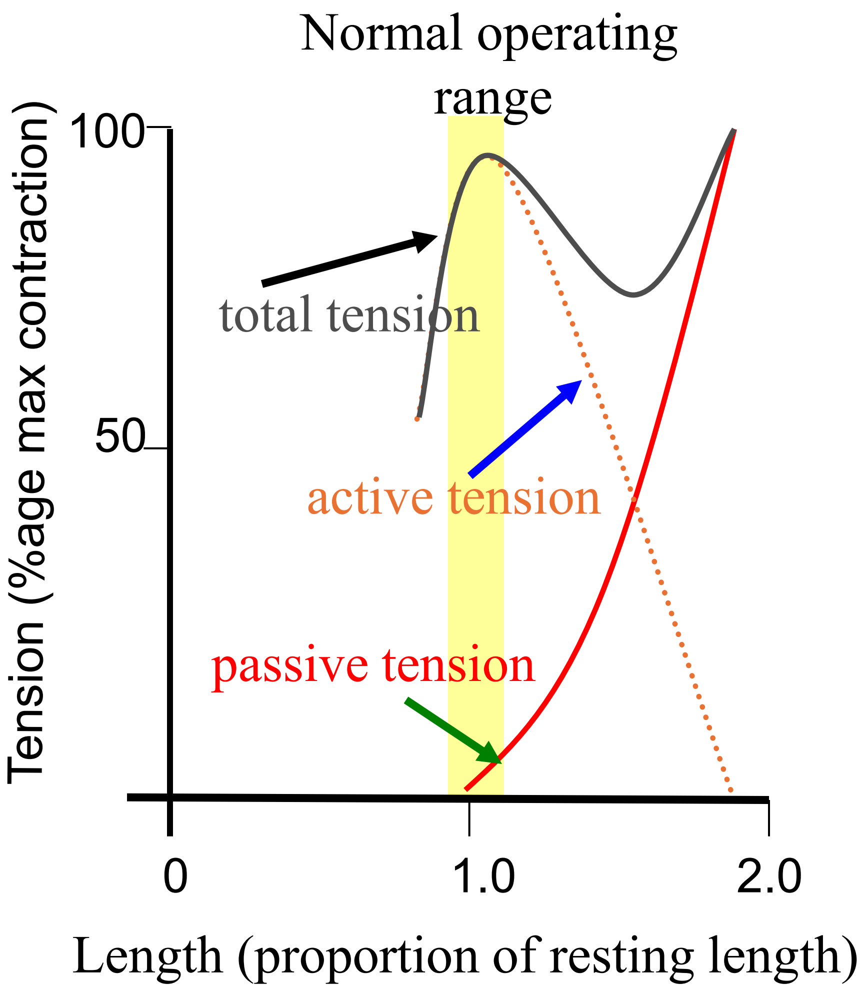


(c) ATP from aerobic cellular respiration

10.13

Length-Tension Relation for Skeletal Muscle

- Active tension cannot be measured directly
- What can be measured?
  - (1) passive tension - *tension required to extend a resting muscle*
  - (2) total tension - *active tension and passive combined*
- Active is calculated from 1 & 2
- (AT = TT – PT)
- Note that active tension falls away linearly with increasing length

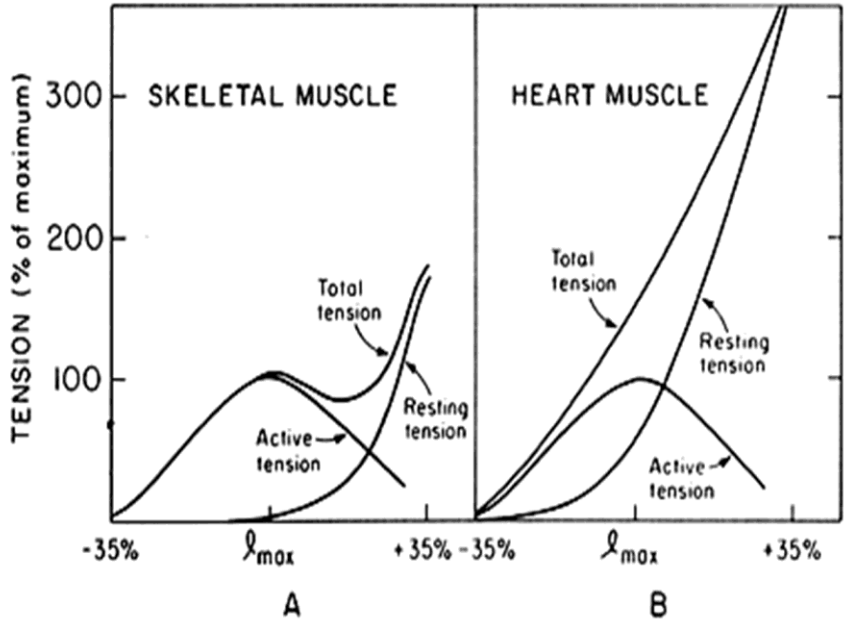


Length-Tension Relation for Skeletal Muscle

- The length–tension relationship in cardiac muscle is explained by Frank–Starling's law of the heart, which states that, within physiological limits, increasing the initial length of cardiac muscle fibers enhances the force of contraction. In simple terms, the more the muscle is stretched before it contracts, the stronger the resulting contraction. This can be compared to stretching a rubber band—the greater the stretch, the more forceful the snap when released. However, if the band is stretched beyond its normal range, it loses elasticity or may even tear, and its recoil force decreases. Likewise, excessive stretching of cardiac fibers beyond their optimal length reduces their ability to contract effectively.
- When the muscle (or rubber band) is stretched, it develops a resistance to that stretch called resting or passive tension. As the degree of stretch increases, both total tension (the overall force during contraction) and active tension (the contractile component) rise—but only up to the physiological limit. Beyond this point, further stretching no longer increases active tension and can actually diminish contractile strength.
- The optimal sarcomere length for normal cardiac function is about 2.2 µm. For contraction to occur, the muscle must produce enough force to overcome the opposing passive tension. The relationship is evaluated using three types of tension:
- Passive tension: the tension present before contraction, caused by muscle stretch.
- Total tension: the overall tension during contraction.
- Active tension: the difference between total and passive tension (Active tension = Total tension − Passive tension).
- In extreme overstretching, the muscle may reach a point where active tension becomes zero, meaning total and passive tensions are equal—indicating that the muscle can no longer generate any additional contractile force.

In the heart, it is not possible to directly measure the muscle fiber length, but it can be indirectly assessed by measuring the volume. An increase in volume corresponds to an increase in muscle fiber length, following the same length-tension relationship.

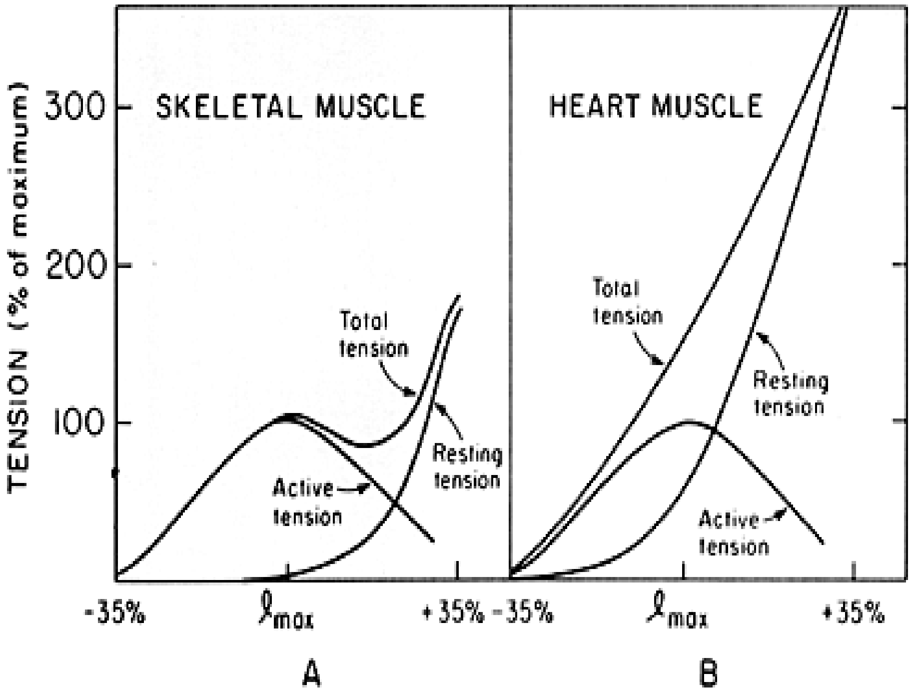
Within the normal physiological range, this length-tension relationship differs between skeletal and cardiac muscle. In skeletal muscle, the relationship shows two peaks (or humps) because skeletal muscle fibers are spindle-shaped and contract mainly in one or two dimensions, and due to the presence of series elastic elements that influence tension development.



In contrast, cardiac muscle fibers are branched and arranged in a three-dimensional network, allowing them to contract in all directions. Therefore, in cardiac muscle, the total tension increases progressively with increasing fiber length, without showing the two distinct peaks observed in skeletal muscle

Why is the tension-length curve different between skeletal and cardiac muscles?

Skeletal muscle shows a **bimodal (bell-shaped)** length–tension curve because its sarcomeres can vary widely in length: force increases as overlap between actin and myosin becomes optimal, then decreases again when overstretched, producing both ascending and descending limbs. In contrast, **cardiac muscle displays only the ascending (unimodal)** portion of this curve, since the myocardium operates within a much narrower sarcomere range (≈1.6–2.2 µm). Strong **passive stiffness from titin, collagen, and the pericardium** prevents overstretching beyond the optimal overlap, so the heart never reaches the descending limb. Functionally, this ensures that as the ventricle fills (greater stretch), contractile force rises predictably—forming the mechanical basis of the **Frank–Starling law**—without risking reduced tension or mechanical failure from excessive length.



The resting length of the sarcomere is not a fixed value but varies within about ±5% of its optimal range. The normal resting length of a sarcomere is approximately 2.2 micrometers, which represents the optimal length for generating maximum tension.

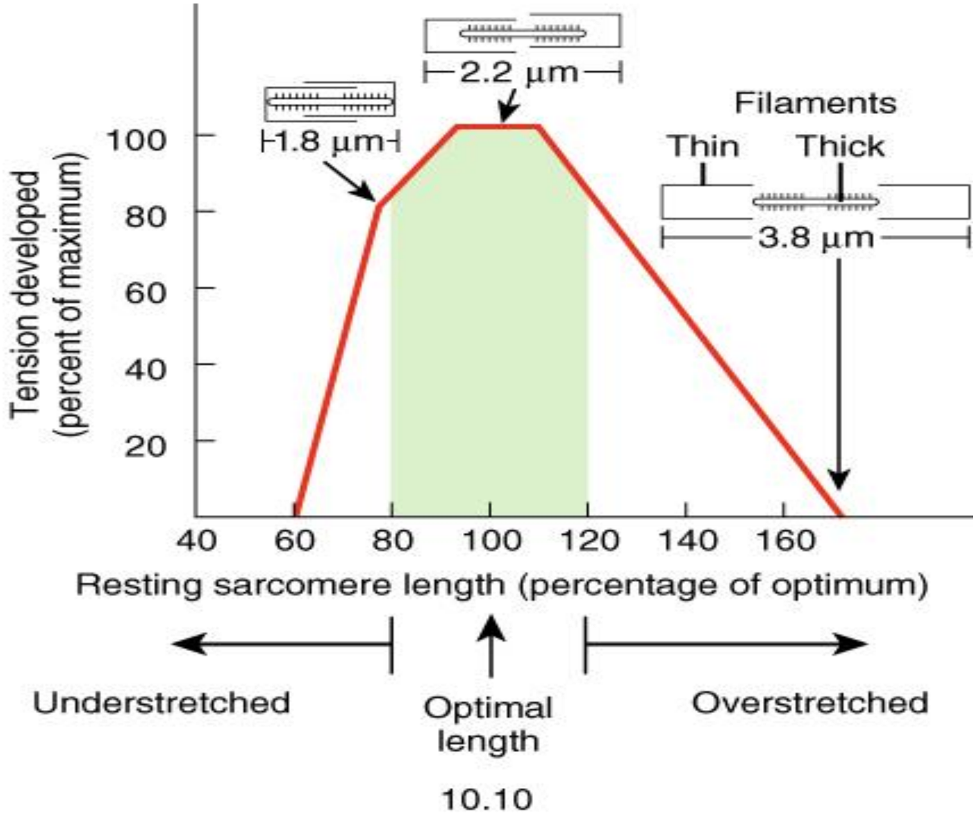
When the sarcomere length is shorter than 2.2 μm, the tension decreases because the actin and myosin filaments overlap too much, reducing the number of effective cross-bridges that can form. Conversely, when the sarcomere is stretched beyond 2.2 μm, the filaments move too far apart, decreasing the number of possible actin-myosin interactions (actomyosin cross-bridges), which also lowers the force of contraction.

At the optimal sarcomere length (2.2 μm), the maximum number of actin-myosin cross-bridges can form, resulting in maximum contractile force.

In the length-tension graph, the X-axis represents sarcomere length, and the Y-axis represents tension. A sarcomere length of 100% corresponds to the normal resting length (2.2 μm).

- At 80% (shorter length), the tension is lower due to excessive overlap.
- At 120% (stretched length), the tension is also lower because the filaments are too far apart.

Thus, maximum tension occurs at the normal resting length (100%, 2.2 μm), where optimal actin-myosin overlap exists.

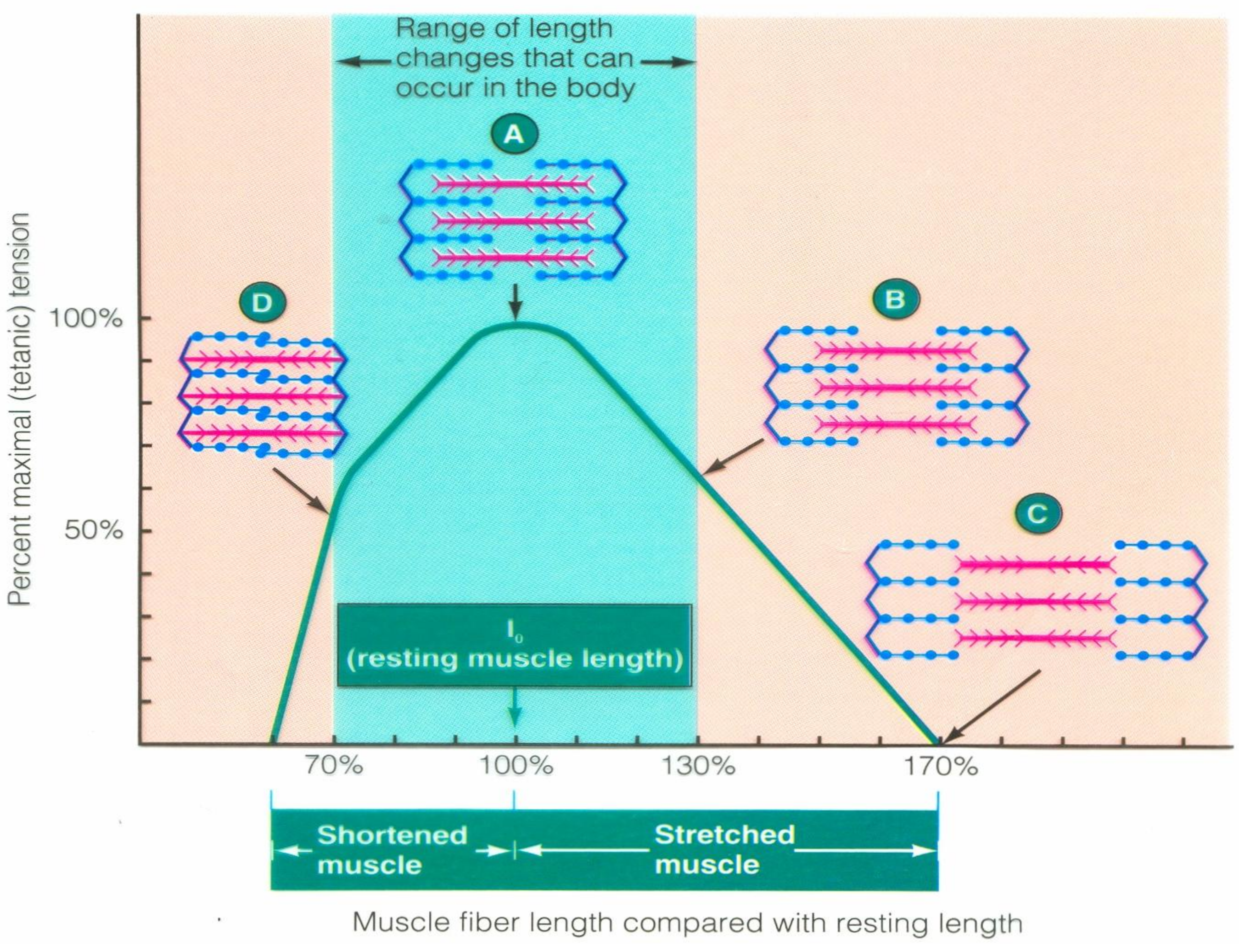


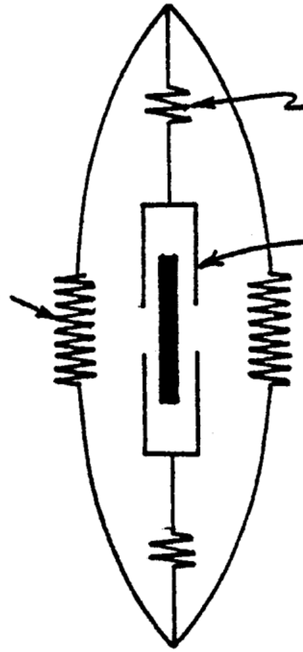
Isometric Contraction

Here, it can be observed that the maximum number of actin and myosin cross-bridges that can form occurs at point A, which represents the optimal sarcomere length. At this point, the force of contraction is at its maximum.

As the sarcomere length deviates from this optimal point—either by shortening or stretching—the meaningful overlap between actin and myosin decreases, making it more difficult for the filaments to interact. Consequently, the force of contraction becomes weaker because fewer cross-bridges can form.

In skeletal muscle, this behavior is also influenced by the presence of series elastic elements, which can stretch and absorb part of the tension during contraction. These elastic components contribute to the overall shape of the length-tension curve in skeletal muscle.





The series elastic elements and parallel elastic elements are linearly present in skeletal muscles. These elastic components influence the length-tension relationship and are responsible for producing the two peaks seen in the skeletal muscle curve.

In contrast, the heart muscle does not have these elastic elements arranged in the same way (linearly in one dimension). When the cardiac muscle contracts, it generates pressure within a hollow chamber rather than tension along a straight fiber. Because the heart is a hollow organ, contraction causes the pressure to increase in the center of the chamber, similar to what happens when you squeeze a balloon—the pressure builds up inside rather than along the sides.

In skeletal muscle, however, contraction occurs by pulling from the sides of the muscle fibers, producing tension along the muscle's length rather than internal pressure.

Cardiac Muscle length-tension relationship

- ☞Cardiac muscle works at much less than its maximum length in contrast to skeletal
- ☞Total, Active and Passive length-tension relationship differ
- ☞Frank-Starling law of the heart

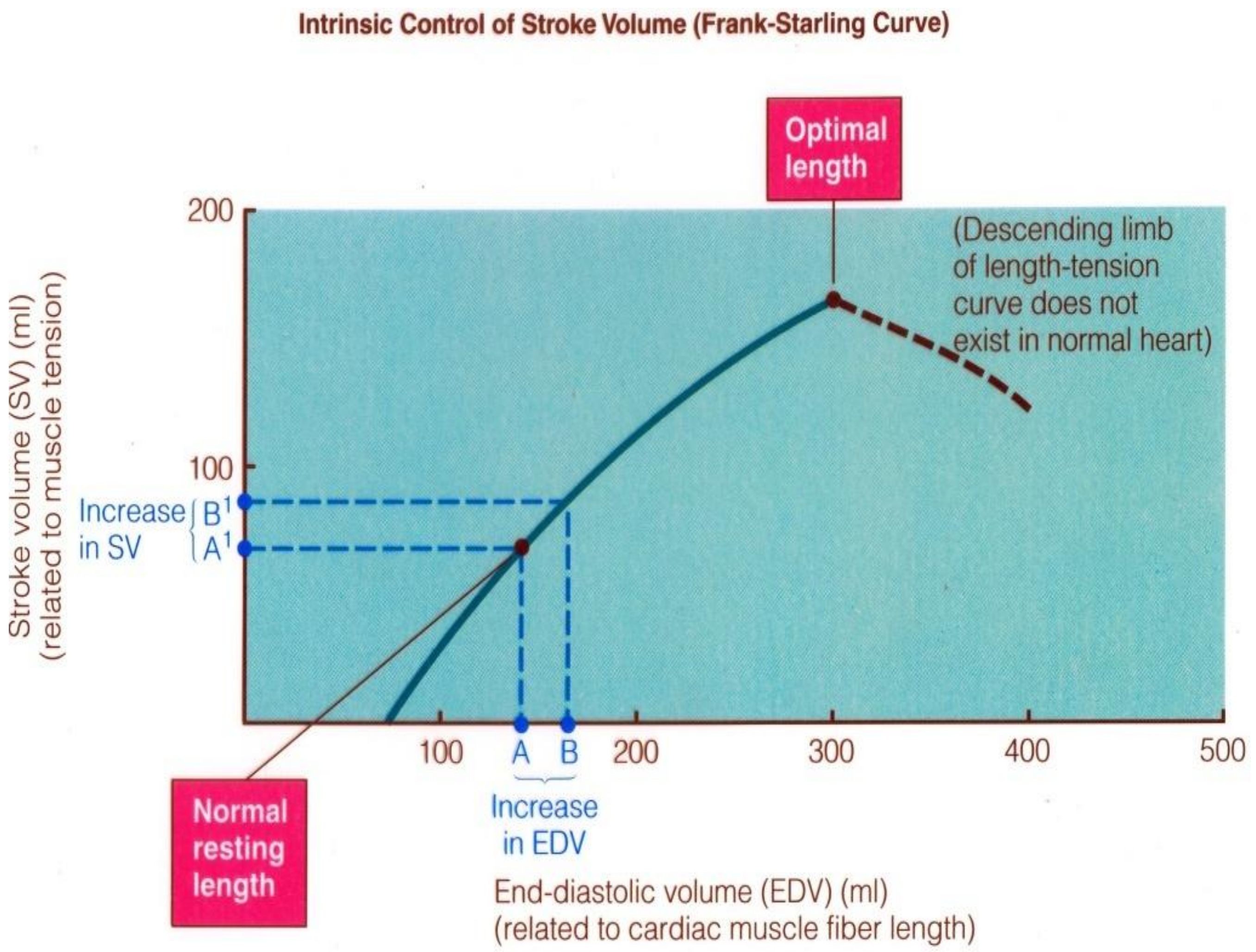
Because the length of the cardiac muscle fibers cannot be measured directly, the Frank-Starling law of the heart relates the volume of blood inside the ventricle to the force of contraction. According to this law, the greater the volume of blood entering the heart during diastole, the greater the volume of blood ejected – within physiological limits.

Excessive Filling & Heart Failure

- If 70 mL of blood enters the ventricle, approximately 70 mL will be pumped out. If 90 mL enters, around 90 mL will be ejected. However, this relationship holds only up to an optimal filling volume (around 180 mL in Dr. Faisal's example). When the heart is stretched beyond this optimal volume — for instance, if 200 mL enters — the heart will not be able to eject the entire amount; it may only pump out 180 mL, leaving 20 mL behind in the ventricle.
- When this happens repeatedly, the heart becomes dilated (cardiac enlargement) because of the retained blood. This condition indicates heart failure, meaning the heart can no longer pump out all the blood that returns to it. Even a small amount of retained blood per beat can accumulate significantly over time.
- For example, if the heart beats 70 times per minute, and just 1 mL remains in the ventricle after each beat, that would result in about 70 mL per minute of retained blood. Over an hour, this adds up to 4.2 liters, which is nearly the total blood volume of the body (around 5-6 liters). Such accumulation is very dangerous.
- That calculation is not physiologically accurate because the heart does not continuously accumulate residual blood after each beat. Instead, it pumps cyclically — each contraction ejects most (not all) of the ventricular blood, and the remaining end-systolic volume simply becomes part of the next filling phase. Blood is constantly recirculated rather than added up beat by beat. True accumulation occurs only if the ventricle fails to eject enough blood over time, as in heart failure, leading to a gradual rise in end-systolic and end-diastolic volumes and eventual congestion — not by simple arithmetic buildup per minute or hour.

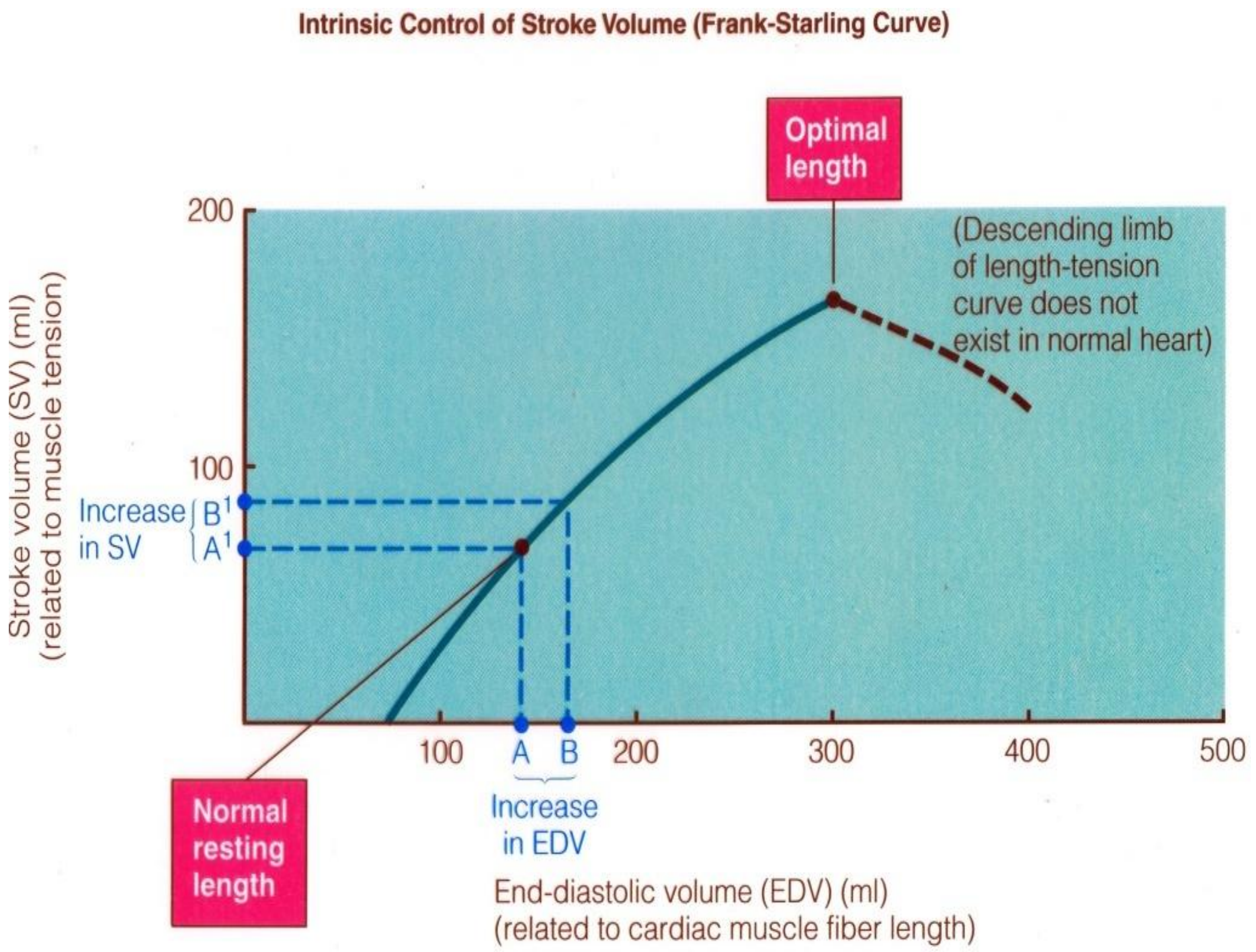
- In this graph, the X-axis represents the end-diastolic volume (EDV), while the Y-axis represents the stroke volume. The end-diastolic volume corresponds to the length of the cardiac muscle fibers, and the stroke volume reflects the tension of contraction.

- As the end-diastolic volume increases, the cardiac muscle fibers stretch, leading to a greater tension of contraction and, consequently, an increase in stroke volume. This relationship continues up to the optimal muscle length, where the heart achieves its maximum efficiency in pumping blood.



- Beyond this optimal point, further increases in end-diastolic volume no longer produce stronger contractions. Instead, the heart begins to fail, meaning that the volume of blood entering (venous return) becomes greater than the volume ejected (stroke volume). This imbalance causes abnormally excess blood to remain in the ventricle, leading to ventricular dilation and heart failure.

- At the optimal length, the cardiac muscle is tense and contracts effectively; however, beyond that limit, the overstretching reduces the overlap between actin and myosin filaments, weakening the contraction and impairing the heart's ability to pump efficiently.



Electrocardiography – Normal

(1)

Faisal I. Mohammed, MD, PhD

Objectives

1. Describe the different “waves” in a normal electrocardiogram.
2. Recall the normal P-R and Q-T interval time of the QRS wave.
3. Distinguish the difference in depolarization and repolarization waves.
4. Recognize the voltage and time calibration of an electrocardiogram chart.
5. Point out the arrangement of electrodes in the bipolar limb leads, chest leads, and unipolar leads.
6. Describe Einthoven’s law.

ECG – Introduction

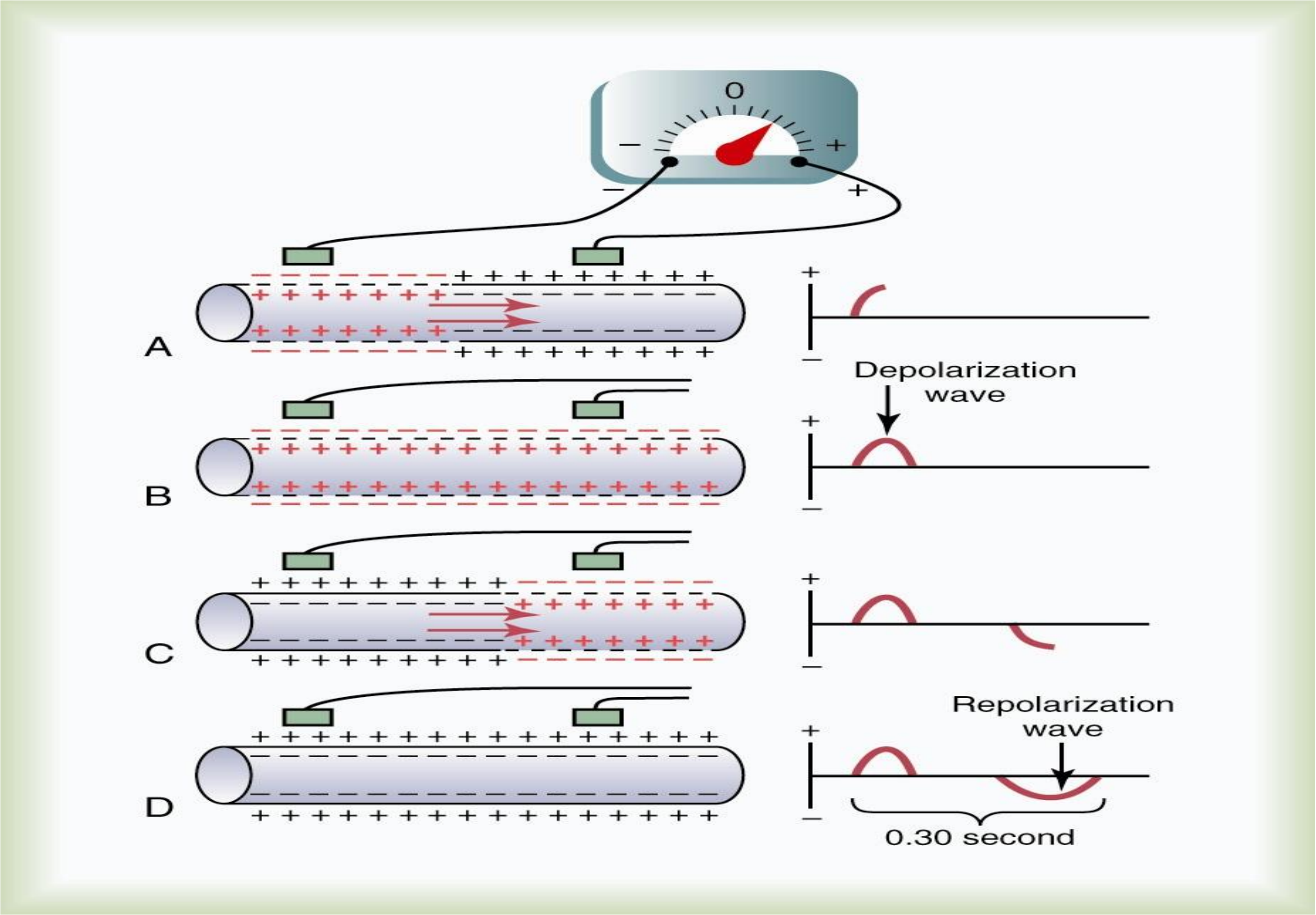
- Electrocardiography (ECG) is the recording of the **electrical changes** that occur in the heart during its activity. It does not record the mechanical events of contraction and relaxation, but rather the electrical events that trigger them.

- The heart functions as two separate syncytia: the atrial syncytium and the ventricular syncytium, each of which undergoes its own sequence of electrical activity. In the atria, there is a process of depolarization followed by repolarization. Similarly, in the ventricles, depolarization occurs first, followed by repolarization.

- In the ECG recording, these electrical changes – atrial depolarization and repolarization**, as well as ventricular depolarization and repolarization – are all represented as specific waves and segments that reflect the sequence of electrical events in the heart.

**: Will be explained

Depolarization and Repolarization Waves

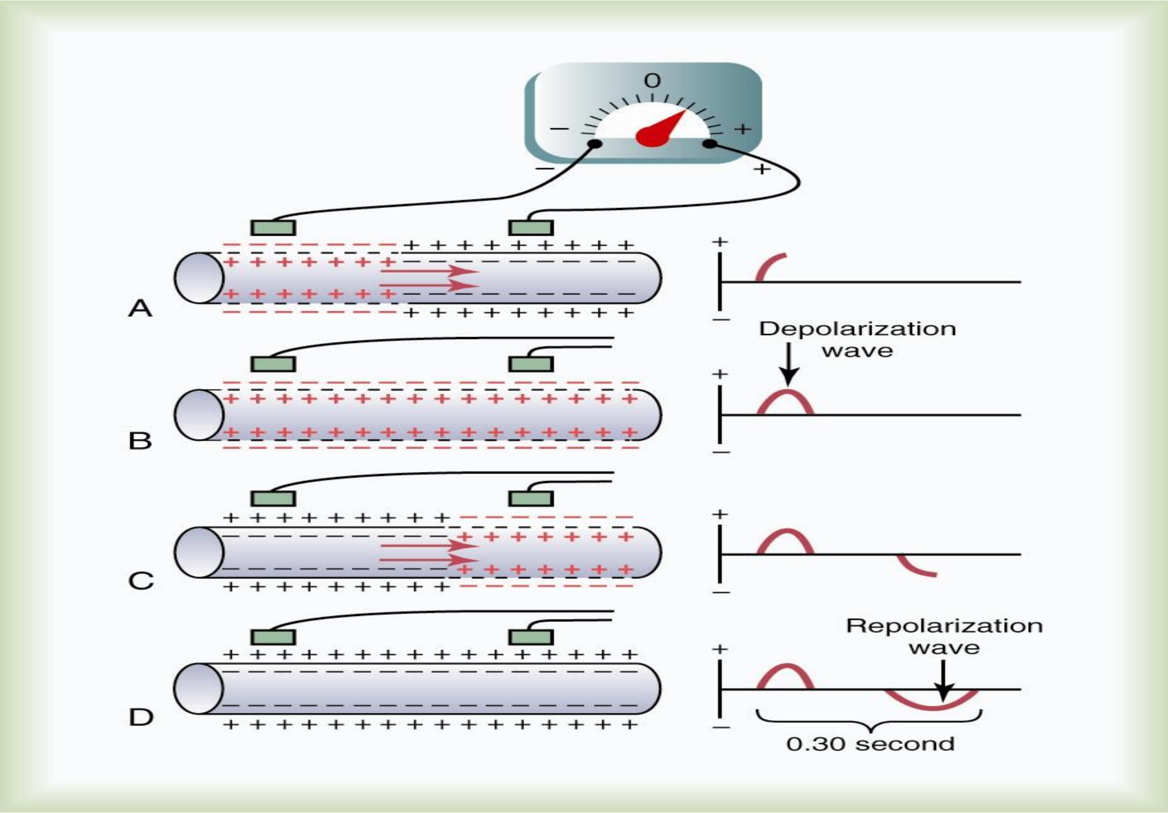


- Note that no potential is recorded when the ventricular muscle is either completely depolarized or repolarized.

Depolarization and Repolarization Waves

- The process of depolarization begins at one end of the muscle and then spreads across. During depolarization, the inside of the cell becomes positive while the outside becomes negative.

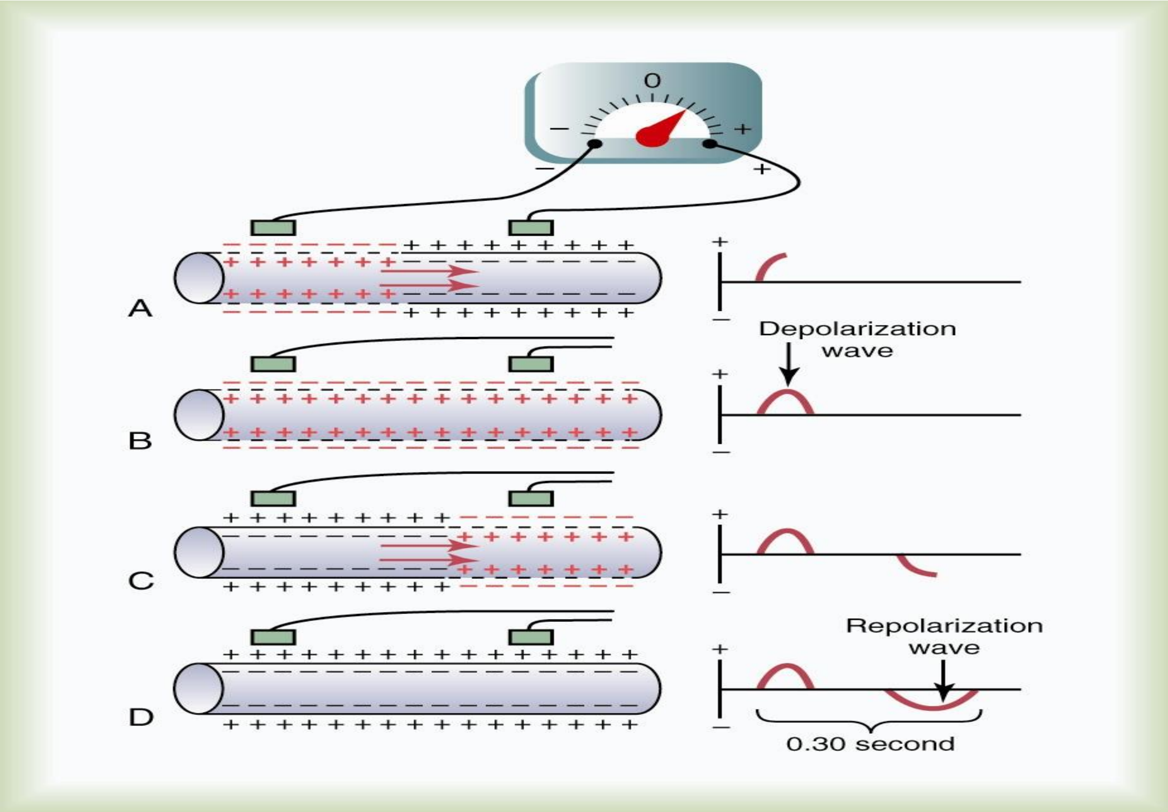
- To record these electrical changes, electrodes connected to a galvanometer are placed on the surface of the muscle. The galvanometer detects the potential difference between the two electrodes. When the entire muscle is in the polarized state (that is, positive outside and negative inside), there is no potential difference, so the galvanometer records a flat line.



Depolarization and Repolarization Waves

- However, when depolarization starts on one side (As seen in A), that region becomes negative outside, while the other side remains positive outside. This creates a potential difference, which the galvanometer detects and records as a deflection on the paper (usually a heat-sensitive paper that records the electrical signal is used).

- When the entire muscle fiber becomes depolarized (As seen in B), both electrodes sense the same potential, so there is no potential difference, and the recording returns to the isoelectric line (a flat baseline on the ECG).

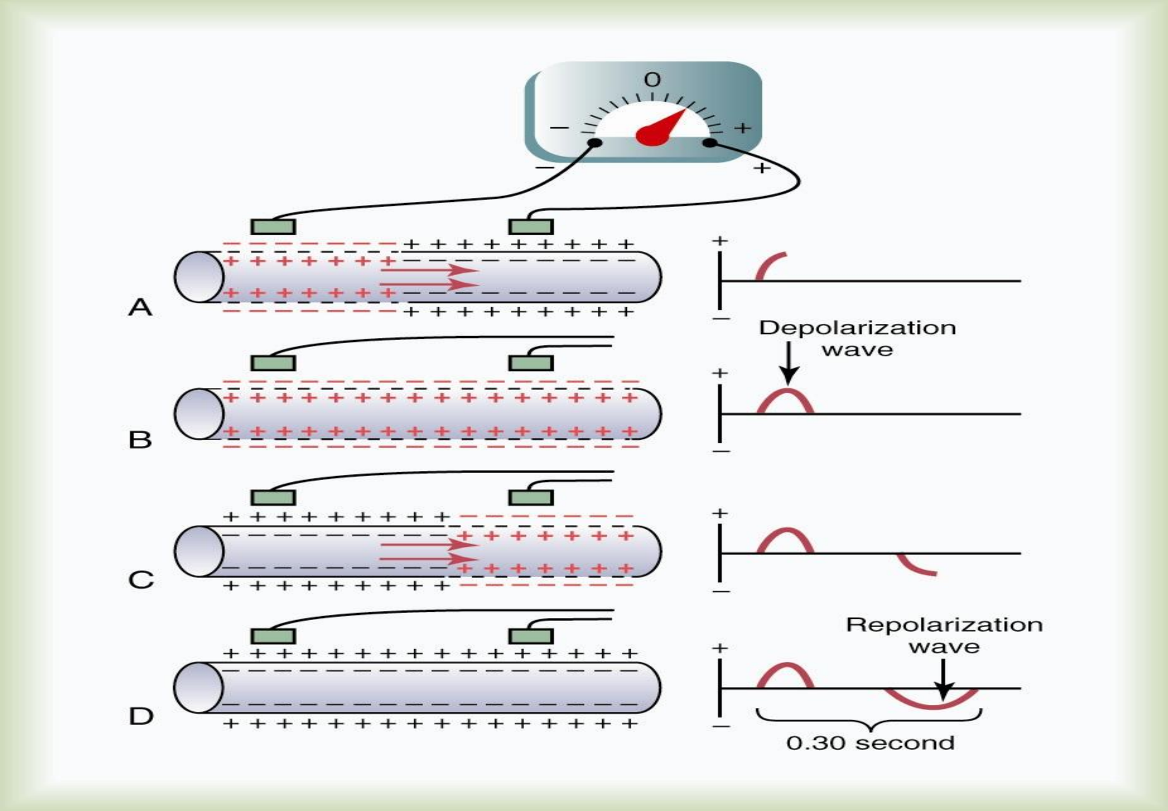


By international convention, depolarization is represented as an upward deflection on the ECG tracing. The maximum deflection occurs when half of the muscle is depolarized and half is still polarized, meaning the potential difference between the two regions is at its highest.

Depolarization and Repolarization Waves

- **When half of the muscle is repolarized and the other half is still depolarized** (As seen in C), **the potential difference between the two regions reaches its maximum, producing the largest deflection on the recording but in the opposite direction.**

- **As the repolarization process continues and the entire muscle becomes completely repolarized** (As seen in D), **both electrodes again detect the same potential, meaning there is no potential difference. At this point, the ECG recording returns to the isoelectric line (the flat baseline).**

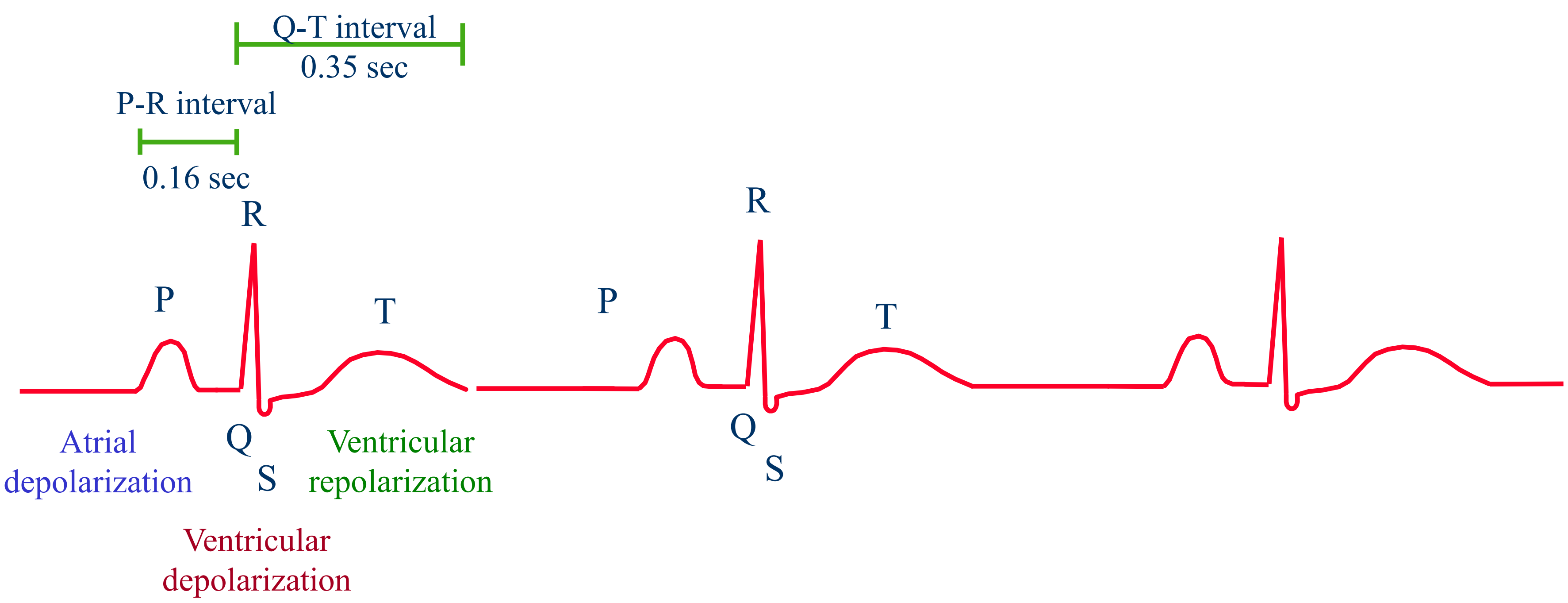


The basis of 'up' and 'down' here is the voltage difference between both electrodes. If you invert the electrodes, depolarization will be a 'down' and repolarization an 'up', but this clearly defies the convention.

Galvanometer Measurements

- The action potential of the cardiac muscle has an amplitude ranging from approximately −90 millivolts (mV) to +30 mV (Guyton says −85 to +20), giving a total potential difference of about 120 mV inside the heart muscle.
- However, when this electrical activity is recorded from the surface of the skin, the detected voltage is much smaller because of the resistance of the body tissues between the heart and the electrodes. As a result, the recorded potential difference on the skin surface is greatly reduced — for example, it may be around 1 to 2 millivolts, not 120 mV.
- To detect and display these small voltage changes accurately, the ECG machine includes amplifiers that increase the signal strength measured by the galvanometer.
- Therefore, an electrocardiograph (ECG machine) consists mainly of a galvanometer (which measures the electrical potential difference) and amplifiers (which magnify these weak signals so they can be recorded clearly).

Normal EKG



This is the electrocardiogram (ECG), which records the electrical activity of the heart during each cardiac cycle.

- The P wave represents atrial depolarization, which is the electrical activity that triggers atrial contraction.
- The QRS complex (composed of the Q, R, and S) represents ventricular depolarization, the electrical event that initiates and precedes ventricular contraction.
- The T wave represents ventricular repolarization, which is the recovery phase of the ventricles before the next cycle begins.

The sequence of these waves – P, Q, R, S, T, P, Q, R, S, T, and so on – repeats with each heartbeat, showing the rhythmic electrical activity of the heart.

A Flat ECG?

- This electrical activity continues in a repeating pattern as long as the heart is functioning normally. If the ECG tracing stops and becomes a flat line, it indicates that there is no electrical activity in the heart, meaning the heart has stopped – this is a sign of cardiac death.

- Similarly, in the brain, when the EEG (electroencephalogram) becomes flat, it indicates brain death because there is no detectable electrical activity in the brain.

- So, a flat ECG represents heart death, while a flat EEG represents brain death.

Repolarization Waves – Strange?

1) **<u>Atrial repolarization</u>** occurs at the same time as ventricular depolarization, which corresponds to the QRS complex on the ECG. Because the electrical activity of the ventricles is much stronger than that of the atria, the atrial repolarization wave is masked by the ventricular depolarization and therefore is not explicitly visible on the ECG tracing.

<u>2) Ventricular repolarization</u> (the T wave):

By convention, repolarization is usually represented as a downward deflection because it is the return of the electrical potential toward the resting (negative) state. However, in the case of the ventricles, the T wave appears upward on the ECG – and here's why:

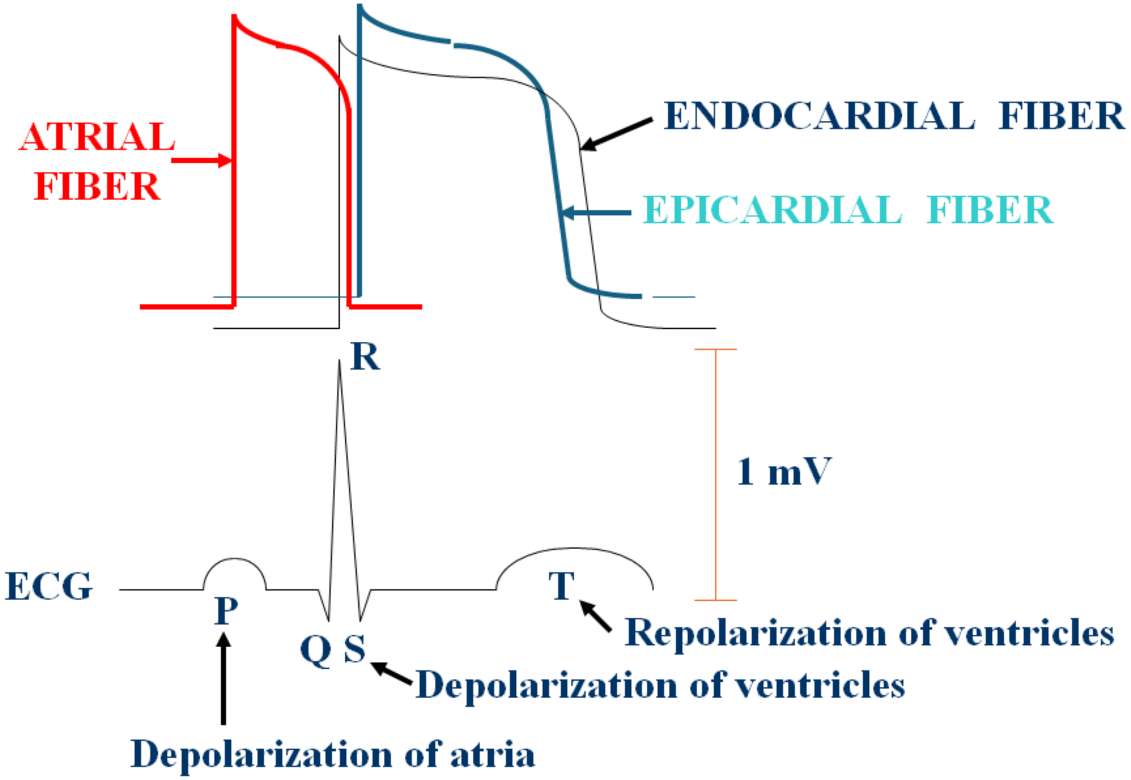
- In the heart, ventricular depolarization begins at the endocardium (the inner layer of the ventricular wall) and spreads outward toward the epicardium (pericardium). It also progresses from the base of the heart toward the apex.
- In contrast, ventricular repolarization begins in the epicardium and moves inward toward the endocardium, and it also proceeds in the opposite direction – from the apex toward the base.
- Because repolarization starts from the opposite side of where depolarization began, the direction of the repolarization current is opposite to that of depolarization. However, since repolarization itself is an opposite electrical process, these two reversals (opposite direction and opposite charge) cancel each other out, producing a positive (upward) deflection on the ECG.

SINGLE VENTRICULAR ACTION POTENTIAL

- **P wave:** Represents **atrial depolarization** (phase 0 of the atrial action potential).
- **T wave:** Represents **ventricular repolarization** (phase 3).

Endocardial (black) vs. Epicardial (blue):

- **Epicardial fibers** repolarize **earlier** than endocardial fibers due to differences in ion channels and lower mechanical stress.
- **Endocardial fibers** experience higher pressure during contraction, delaying their repolarization.
- This difference in timing causes the **T wave to appear as an upward deflection**, since repolarization occurs in the opposite direction to depolarization.
- **Atrial repolarization** can sometimes (abnormally) appear as a **downward deflection**, but it is normally hidden by the **QRS complex** because ventricular activity dominates.



ECG Vs Monophasic action potential

- When recording directly from a single cardiac muscle fiber, the electrical activity obtained is called a monophasic action potential. This recording shows the full change in membrane potential at one point in the muscle — from resting potential, through depolarization, and back to repolarization — as a single, continuous waveform.

- In contrast, the electrocardiogram (ECG) is a biphasic recording because it measures the potential difference between two electrodes placed on the body surface. As the wave of depolarization or repolarization moves toward or away from each electrode, the ECG tracing shows upward and downward deflections depending on the direction of current flow relative to the recording electrodes.

Standardized EKG's

In a standardized ECG recording, the tracing is printed on heat-sensitive paper that contains a grid of 1 mm squares. The axes of this grid represent:

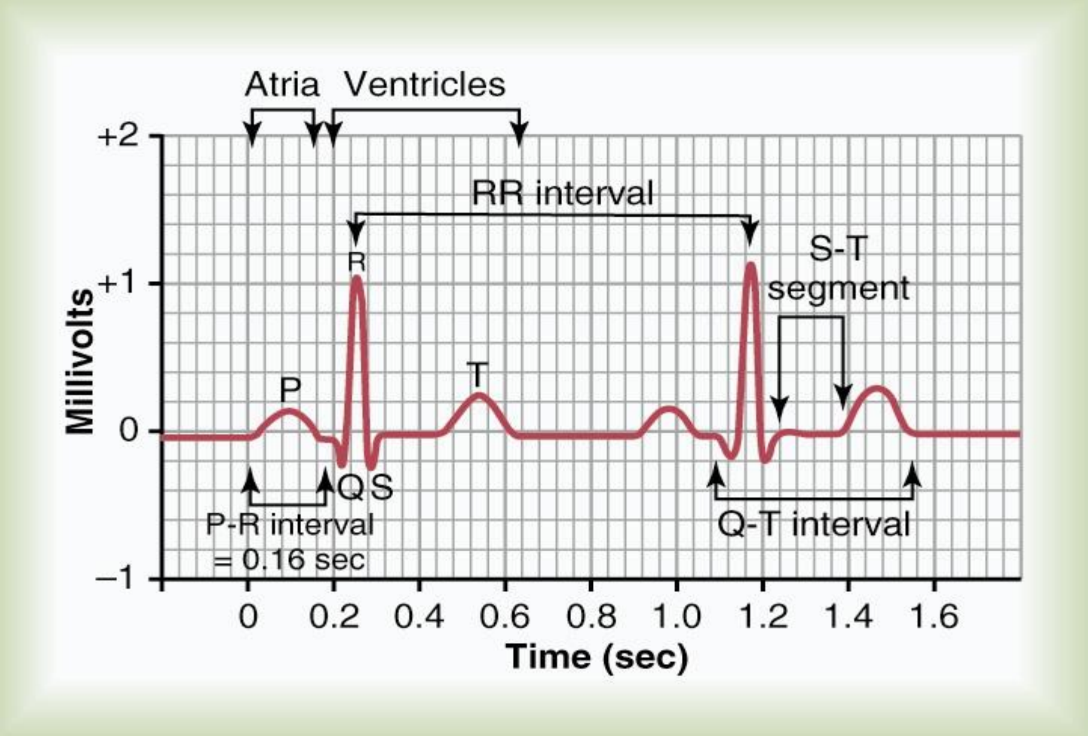
- The X-axis → Time
- The Y-axis → Voltage (millivolts)

The standard paper speed is 25 mm per second, meaning that 25 small squares = 1 second.

- Each small square (1 mm) represents 0.04 seconds (40 milliseconds).
- Each larger square (5 small squares) represents 0.20 seconds.

The distance between one R wave and the next R wave (R-R interval) represents one complete cardiac cycle. By measuring this interval, we can calculate the heart rate.

If each cardiac cycle measures 20 small squares on the ECG paper, this corresponds to 0.8 seconds (since each small square equals 0.04 seconds). Heart rate is 75 bpm.



- Time and voltage calibrations are standardized

For the Y axis:
10 mm for each mV, after amplification.

Electrocardiogram

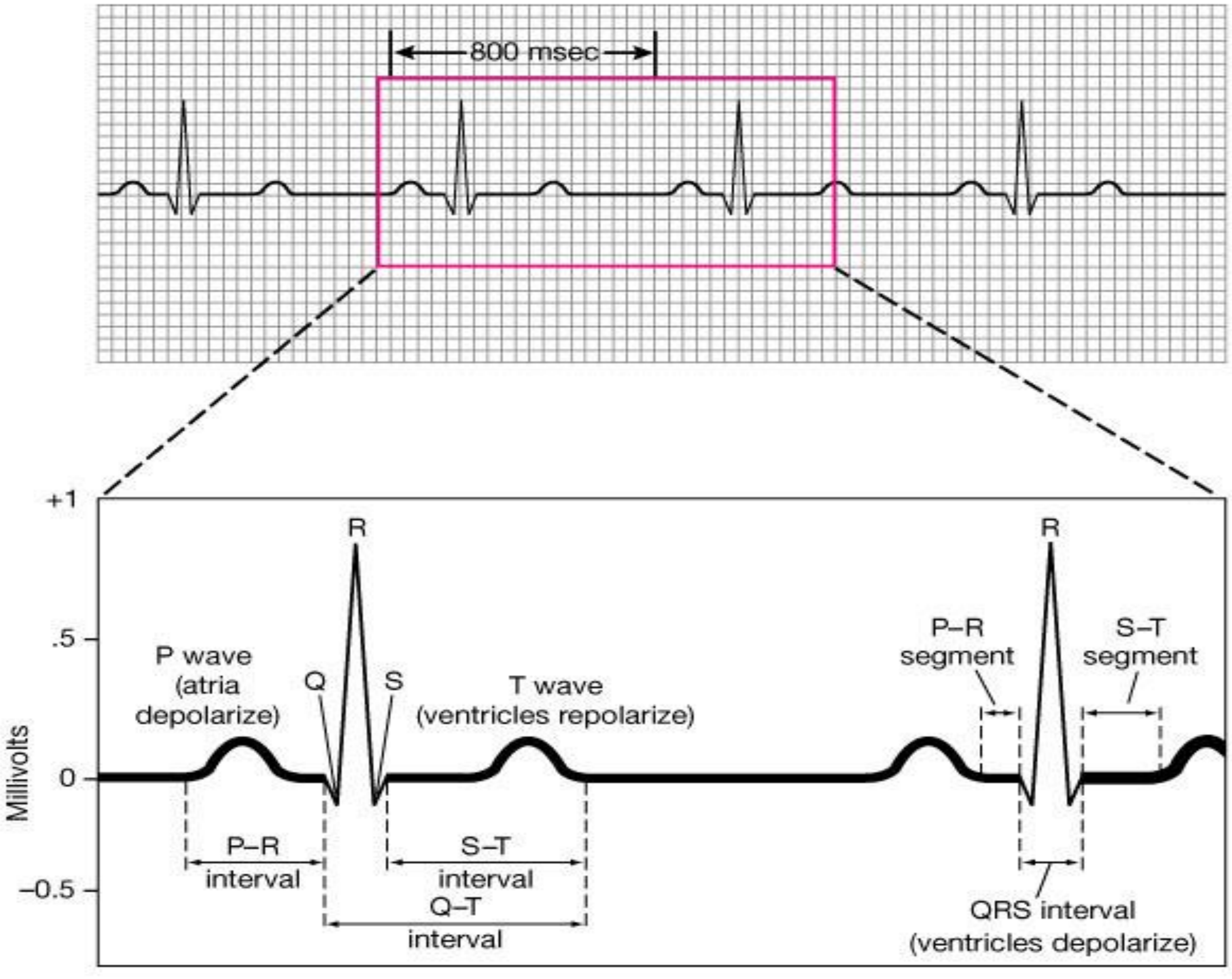
- Record of electrical events in the myocardium that can be correlated with mechanical events
- **P wave**: depolarization of atrial myocardium.
  - Signals onset of atrial contraction
- **QRS complex**: ventricular depolarization
  - Signals onset of ventricular contraction..
- **T wave**: repolarization of ventricles
- **PR interval** or PQ interval: 0.16 sec
  - Extends from start of atrial depolarization to start of ventricular depolarization (QRS complex) contract and begin to relax
  - Can indicate damage to conducting pathway or AV node if greater than 0.20 sec (200 msec)
- **Q-T interval**: time required for ventricles to undergo a single cycle of depolarization and repolarization
  - Can be lengthened by electrolyte disturbances, conduction problems, coronary ischemia, myocardial damage

Electrocardiogram

- The PR interval is measured from the beginning of the P wave to the beginning of the R wave. When we use the term interval, it means that the measurement includes at least one wave and the adjacent segment.

- The R-R interval is easier to measure and represents one complete cardiac cycle. It can also be measured from P to P or T to T, but the R-R interval is the most distinct and commonly used. All these intervals are measured in time units (seconds).

- Other important intervals include the QRS interval (and Q-T interval), which represent the electrical activity of the ventricles.

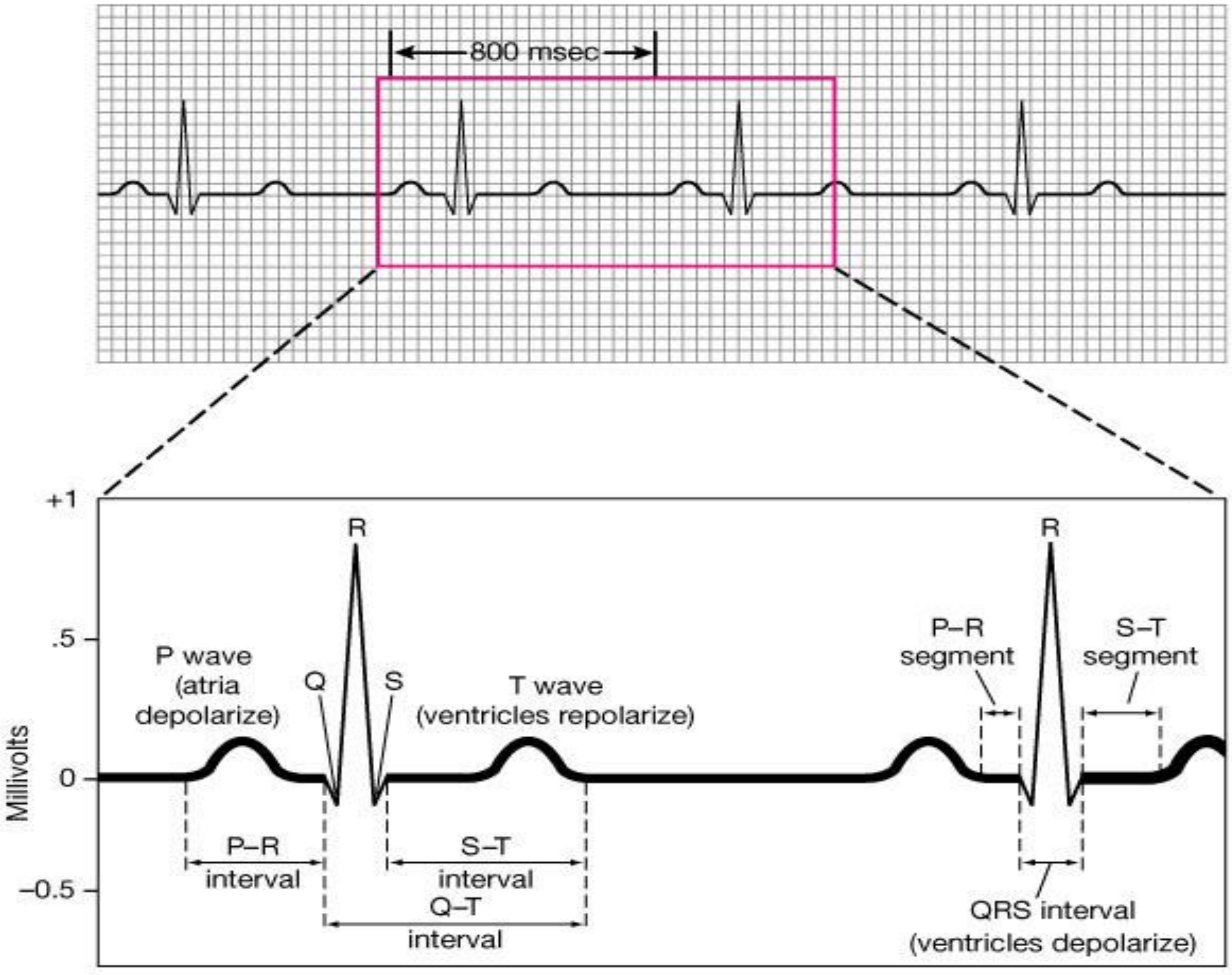


(b)

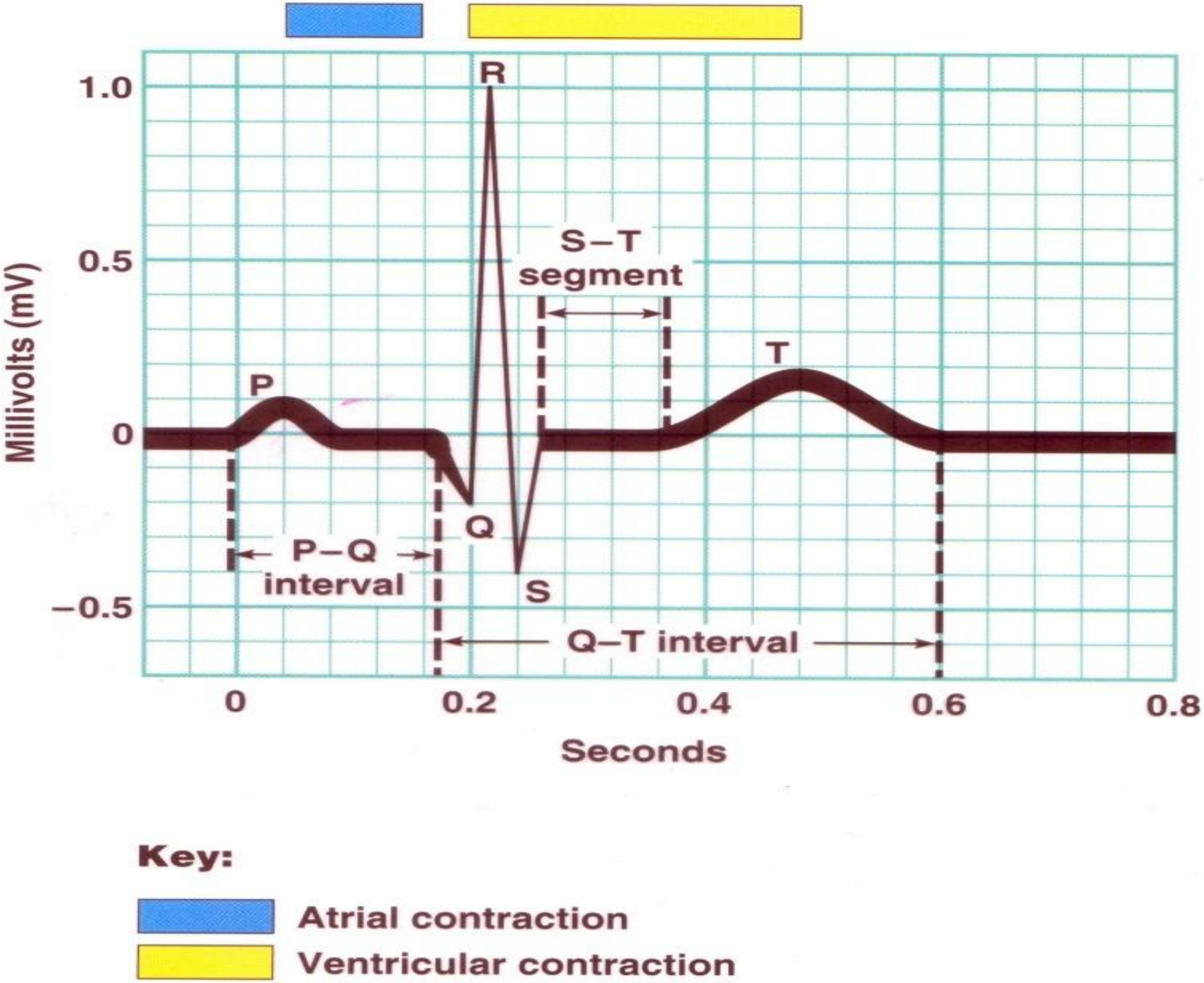
Electrocardiogram

- The ST segment and the PR segment correspond to periods when the ECG tracing lies on the isoelectric line – meaning there is no net electrical difference because the myocardium is either completely depolarized or completely repolarized.

- These segments are clinically very important. Any upward (elevation) or downward (depression) deflection of the ST segment may indicate myocardial infarction or ischemia. Therefore, the ST segment and PR segment are closely examined first when evaluating an ECG to rule out myocardial infarction.



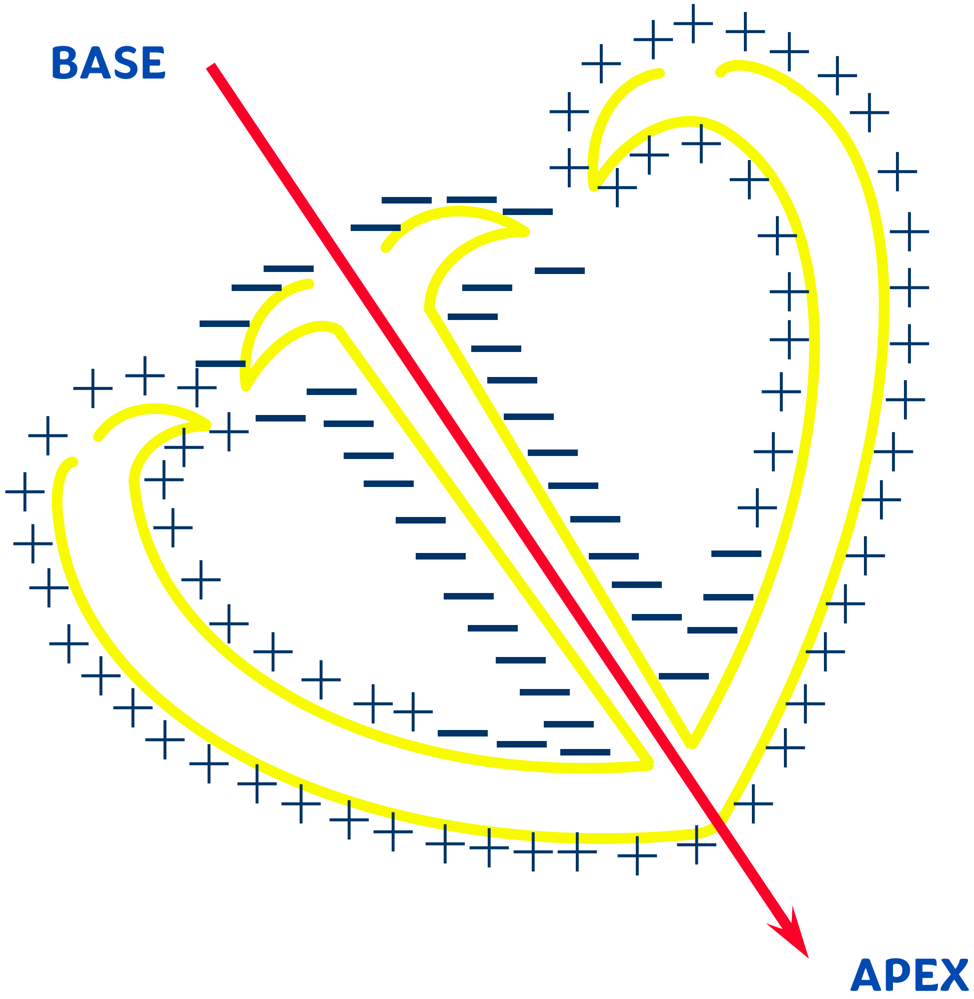
(b)



R
1.0
0.5
0
−0.5
Millivolts (mV)
S–T
segment
T
P
Q
S
P–Q
interval
Q–T interval
0
0.2
0.4
0.6
0.8
Seconds
Key:
Atrial contraction
Ventricular contraction

Flow of Electrical Currents in the Chest Around the Heart

Mean Vector Through the Partially Depolarized Heart



Flow of Electrical Currents in the Chest Around the Heart (cont’d)

- Ventricular depolarization starts at the ventricular septum and the endocardial surfaces of the heart.
- The average current flows positively from the base of the heart to the apex.
- At the very end of depolarization the current reverses from 1/100 second and flows toward the outer walls of the ventricles near the base (S wave).

EKG Concepts

- The P wave immediately precedes atrial contraction.
- The QRS complex immediately precedes ventricular contraction.
- The ventricles remain contracted until a few milliseconds after the end of the T repolarization wave.
- The atria remain contracted until the atria are repolarized, but an atrial repolarization wave cannot be seen on the electrocardiogram because it is masked by the QRS wave.

EKG Concepts (cont'd)

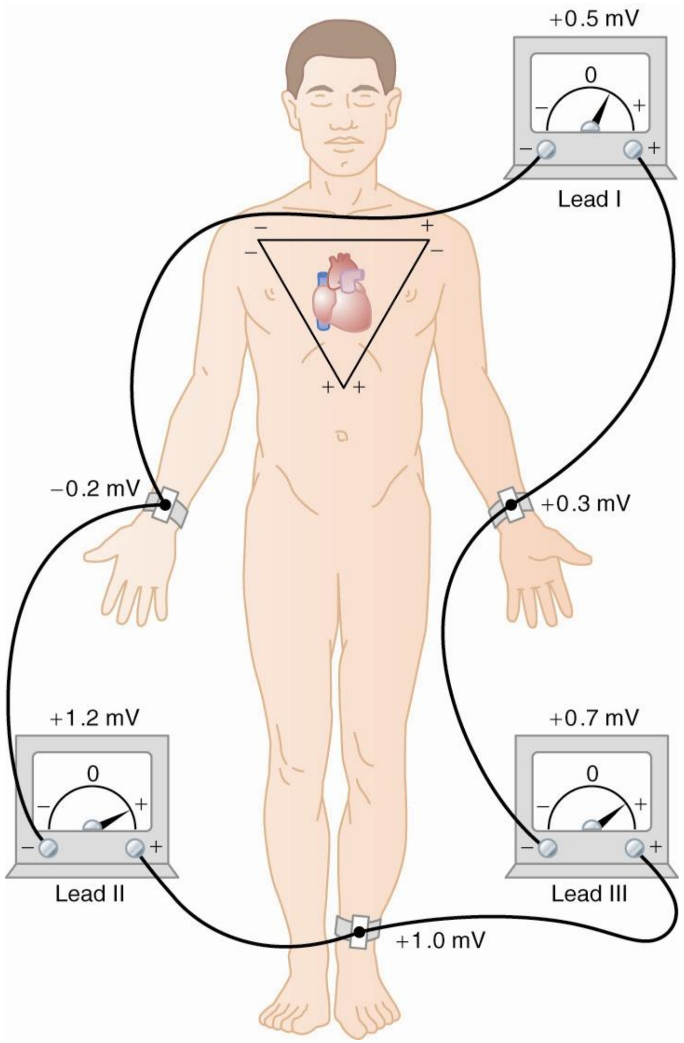
- The P-Q or P-R interval on the electrocardiogram has a normal value of 0.16 seconds and is the duration of time between the beginning of the P wave and the beginning of the QRS wave; this represents the time between the beginning of atrial contraction and the beginning of ventricular contraction.

EKG Concepts (cont'd)

- The Q-T interval has a normal value of 0.35 seconds and is the duration of time from the beginning of the Q wave to the end of the T wave; this approximates the time of ventricular contraction.
- The heart rate can be determined with the reciprocal of the time interval between each heartbeat.

Bipolar Limb Leads

- Bipolar means that the EKG is recorded from two electrodes on the body.



Biphasic Limb Leads

To record the electrical activity of the heart accurately, we must view it from multiple directions, not just from one pair of electrodes. That's why the standard ECG uses 12 leads, which give different views of the heart's electrical activity.

The first three leads are called the bipolar limb leads, because each one records the potential difference between two electrodes:

- Lead I: between the right arm (negative electrode) and the left arm (positive electrode).
- Lead II: between the right arm (negative) and the left leg (positive).
- Lead III: between the left arm (negative) and the left leg (positive).

<u>**In all three leads, the right arm is always negative, and the left leg is always positive.**</u>

This defines the key pathway of cardiac depolarization across the frontal plane.

The right foot is not used for recording but is connected to the ECG as a ground (neutral) to eliminate electrical interference from the environment.

Bipolar Limb Leads (cont'd)

- Lead I - The negative terminal of the electrocardiogram is connected to the right arm, and the positive terminal is connected to the left arm.
- Lead II - The negative terminal of the electrocardiogram is connected to the right arm, and the positive terminal is connected to the left leg.

Bipolar Limb Leads (cont'd)

- Lead III - The negative terminal of the electrocardiogram is connected to the left arm, and the positive terminal is connected to the left leg.
- Einthoven's Law states that the electrical potential of any limb equals the sum of the other two (+ and - signs of leads must be observed). L II= L I + L III
- If lead I = 1.0 mV, Lead III = 0.5 mV, then Lead II = 1.0 + 0.5 = 1.5 mV
- Kirchoff's second law of electrical circuits LI+LII+LIII=0

PHYSIOLOGY QUIZ

LECTURE 4

External Resources

رسالة من الفريق العلمي

Additional sources:

1. Guyton and Hall textbook of medical physiology,
   13th Ed.
   Unit III
   Chapter 9

قال الله تعالى :

نَبِّئۡ عِبَادِيٓ أَنِّيٓ أَنَا ٱلۡغَفُورُ ٱلرَّحِيمُ ٤٩
وَأَنَّ عَذَابِي هُوَ ٱلۡعَذَابُ ٱلۡأَلِيمُ ٥٠

سورة الحجر

أي أخبر-أيها الرسول- عبادي أني أنا الغفور للمؤمنين التائبين, الرحيم بهم ، وأن عذابي هو العذاب المؤلم الموجع لغير التائبين.

(التفسير الميسر)

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			