ELECTRICAL ACTIVITY OF THE HEART

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The heart has the ability to beat outside the human body because the signal for contraction is myogenic, originating from the muscle (heart) itself and not from the nerves.
THE MYOCARDIUM

CARDIAC MUSCLE

Contractile 99%

Auto-rhythmic 1%
Electrical Activity of the Heart

• **Myocardial AUTO RHYTHMIC CELLS (1%)**
These cells are smaller and contain few contractile fibers or organelles. Because they do not have organized sarcomeres, they do not contribute much to the contractile force of the heart.

They include:
- SA node (Sinoatrial Node)
- AV node (Atrioventricular Node)
- Bundle of HIS
- Purkinje fibers

• **Myocardial CONTRACTILE CELLS (99%)**
Contractile cells include most of the heart muscle:
  - Atrial muscle
  - Ventricular muscle

These cells do most of the contraction. They are also known as the Working Myocardium.
DIFFERENT TYPES OF CHANNELS IN THE CARDIAC MUSCLE CELL MEMBRANE
CHANNELS

SODIUM CHANNELS

Voltage-gated Sodium Funny Channels ($I_f$)

Voltage-gated Sodium Channels (Na)

Potassium Channels (Voltage-gated)

Calcium Channels (Voltage-gated)

- Long Lasting Type Channels (L-Type Ca)
- Transient Type Channels (T-Type Ca)
SODIUM CHANNELS

**Typical Voltage-gated SODIUM CHANNELS**

- Typical voltage-gated channels seen in the nerve and muscle cells.
- Have 2 gates:
  - Activation gate &
  - Inactivation gate

**Sodium Funny CHANNELS**

- Discovered quite recently. They are called funny, for 2 reasons:
  - Typically voltage-gated channels open when the membrane becomes less negative (depolarizes), but these unique channels open when the potential becomes more negative (hyperpolarizes) at the end of repolarization from the previous action potential.
  - They allow sodium and calcium ions to pass through, thus, acting as leak channels. When one action potential ends and the If channels open, the resultant depolarizing net inward Na current through these open channels starts immediately moving the pacemaker cell’s membrane potential toward threshold once again.
CALCIUM CHANNELS

Long Lasting Type
CALCIUM CHANNELS

• Also called the Sodium-Calcium channels.
• L-type channels open more slowly, and remain open longer than T-type channels.
• Because of these properties, L-type channels are important in sustaining an action potential (sustained depolarization as seen with the plateau phase of a ventricular muscle cell).
• Thus, the L-type channels are found in both the autorythmic and contractile ventricular cells.

Transient Type CALCIUM CHANNELS

• T-type channels open rapidly and close rapidly as well.
• T-type channels are important in initiating a Pacemaker potential in the auto rhythmic cells.
• T-type channels are commonly found in the auto rhythmic cells.
ACTION POTENTIAL OF A CONTRACTILE CARDIAC CELL

• ATRIAL CARDIAC MUSCLE CELLS
• VENTRICULAR CARDIAC MUSCLE CELLS
The membrane of the contractile cells remains essentially at rest at about -85mv (about −90 mv), until excited by an action potential from the Auto-rhythmic cell.

The Action Potential recorded in a ventricular muscle fiber averages about 105 mv, which means that the intracellular pressure rises from a very negative value, about −85 mv, between beats to a slightly positive value, about +20 mv, during each beat.
ACTION POTENTIAL OF A CONTRACTILE MYOCARDIAL CELL: A TYPICAL VENTRICULAR CELL

- **Depolarization**
  - Opening of fast voltage-gated Na+ channels.
  - Rapid Influx of Sodium ions leading to rapid depolarization.

- **Small Repolarization**
  - Opening of a subclass of Potassium channels which are fast channels.
  - Rapid Potassium Efflux.

- **Plateau phase**
  - 200 msec (about 0.2 sec) duration (while it is only 1msec in neuron)
  - Opening of the L-type voltage-gated slow Calcium channels & Closure of the Fast K+ channels.
  - Large Calcium influx
  - K⁺ Efflux is very small as K⁺ permeability decreases & only few K channels are open.

- **Repolarization**
  - Opening of the typical, slow, voltage-gated Potassium channels.
  - Closure of the L-type, voltage-gated Calcium channels.
  - Calcium Influx STOPS
  - Potassium Efflux takes place.
What causes the long action potential and the plateau in cardiac muscle as compared to the skeletal muscle?

1. The plateau is caused by the L-type (long lasting type) calcium channels which are slow channels and remain open for several tenths of a second. Both sodium and calcium that enter the cardiac muscle maintain the depolarization for prolonged period.

2. The same calcium ions also activate the muscle contractile process.

3. The permeability for potassium decreases about 5 fold, due to excess calcium influx.
Refractory period and it’s significance in Cardiac muscle:

The refractory period of the heart is the interval of time, during which normal cardiac impulse cannot re-excite an already excited area of cardiac muscle.

- The normal refractory period of the ventricle is 0.25 to 0.30 second (the duration of the plateau).
- The refractory period of the atrial muscle is much shorter than that for the ventricles (about 0.15 second for the atria compared with 0.25 to 0.30 second for the ventricles).
- It prevents the heart from undergoing tetanus.
- It lasts as long as the cardiac contraction and thus prevents irregular heart beat (Arrhythmias).
ACTION POTENTIAL IN AN AUTO-RHYTHMIC CARDIAC CELL

The human heart has a special system for rhythmic self-excitation and repetitive contraction approximately 100,000 times each day, or 3 billion times in the average human lifetime.

- SA NODE
- AV NODE
- BUNDLE OF HIS
- PURKINJE FIBERS
The Auto-rhythmic cardiac cells

The auto rhythmic cells **DO NOT** have a stable resting membrane potential like the nerve and the skeletal muscles. Instead they have an **unstable** membrane potential that starts at \(-55\) to \(-60\)\,mV and slowly drifts upwards towards threshold.

Because the membrane potential never rests at a constant value, it is called a **Pacemaker Potential** rather than a resting membrane potential.
At the negative potential of $-55$ to $-60$mv, the fast sodium channels mainly have become “inactivated”, which means that they have become blocked. This is because the inactivation gates have become closed and remain so.
What causes the lesser negativity and the membrane potentials of these cells to be unstable?

Auto rhythmic cells contain channels different from other excitable cells.

The **cause of the lesser negativity** is that the cell membranes of the sinus fiber are naturally leaky to sodium and calcium ions, and positive charges of the entering sodium and potassium ions neutralize some of the intracellular negativity.
Thus, the inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation.
Why does the leakiness to sodium and calcium channels not cause the sinus nodal fibers to remain depolarized all the time?

1. The L-type calcium channels become inactivated within about 100 to 150 seconds after opening.
2. A large number of potassium channels open.
IONIC BASIS OF ACTION
POTENTIAL OF AUTORRYTHMIC CELLS

Phase 1: Pacemaker Potential:
- Opening of voltage-gated Sodium channels called Funny channels (I_f or f channels).
- Closure of voltage-gated Potassium channels.
- Opening of Voltage-gated Transient-type Calcium (T-type Ca^{2+} channels) channels.

Phase 2: The Rising Phase or Depolarization:
- Opening of Long-lasting voltage-gated Calcium channels (L-type Ca^{2+} channels).
- Large influx of Calcium.

Phase 3: The Falling Phase or Repolarization:
- Opening of voltage-gated Potassium channels
- Closing of L-type Ca channels.
- Potassium Efflux.
Comparing the Action Potentials

- Nerve
- Skeletal Muscles
- Cardiac ventricular muscle