We begin with the myocyte resting: The pumps are working to maintain the resting potential; they keep the sodium out and potassium in. The open potassium channel allows some potassium to escape so that there isn’t too much positive charge inside. The fast sodium channels are ready and waiting, as are the voltage-gated calcium channels. The tiny upward deflection of voltage is an action potential arriving from a neighbouring cell, bringing the voltage-gated sodium channels to their opening threshold of -70 millivolts.

**Phase 4**

The Fast Sodium Channels Open. Sodium hoses into the cell. The charge inside the cell rapidly rises. This is what happens in the atrial muscle, the ventricular muscle, and the Purkinje fibers. The SA and AV nodes don’t have such a large voltage across their membranes—they only have about -55mV.

At a voltage of -55mV, the sodium channels are locked shut; no action potential can wake them. The inward-rectifying potassium channels close.

**Phase 0**

At -40 mV, Calcium channels open: Calcium rushes into the cell. This adds to the overall trend of rising positivity. What’s more, as the voltage-gated calcium channels open, so do the sarcoplasmic reticulum channels (triggered by a rising calcium concentration).

After firing for a fraction of a second, the sodium channels shut down and remain inactive until the end of the cycle. That fraction of a second is enough to flood the cell with sodium.

**Half-way through phase 0**

The cell is fully depolarized; Some positive charge is lost via the transient potassium channels which leak a lot of potassium; as the inside of the cell is now largely positive, the potassium is repelled by this charge. These potassium channels close very soon. The sodium channels are also closed now, but the calcium channels have only just started working, and the ongoing positivity of the cell is due to their action.
Phase 2

The longest phase; balance between incoming calcium and outgoing potassium. The potassium continues to leak out via the **outward-rectifying potassium channels**, and the calcium continues to leak in via the L-type calcium channels; the rates of leakage are matched and so in terms of electricity, there is no change in potential difference. Positive ions in, positive ions out. This causes a plateau in the voltage graph. The end of phase 2 is caused by a peak in calcium concentration – this opens the **calcium-gated potassium channels**.

**Plateau due to calcium influx and potassium out-flux**

Phase 3

Repolarisation; the L-type calcium channels close, but the outward-rectifying potassium channels (as well as the calcium-gated potassium channels) are still open, and so the cell is slowly brought back to the equilibrium of Phase 4.

**The ABSOLUTE REFRACTORY PERIOD ends when the membrane reaches around -50 mV.**

ADDITIONALLY:

Contrary to my puerile diagrams, the ion channel picture is far from simple. There are throngs of ion channels besides those mentioned above. **Acetylcholine-activated Potassium Channels** are activated by the actions of the parasympathetic nervous system; it stands to reason – if you want to slow down the heart, you make it more difficult to reach threshold by hyperpolarizing the cell. This hyperpolarity is achieved by opening these Ach-gated potassium channels, and letting even more potassium exit the cell during the resting phase. **Confusingly, these are also activated by Adenosine. – but via a different receptor, the purine A1.**

**Arachidonic Acid-activated Potassium Channels** allow fatty acids to shorten the action potential, for example during an ischaemic event. Acidosis opens these channels, and as a consequence phases 2 and 3 are shorter (repolarisation is faster)

**ATP-sensitive Potassium Channels** are inactive at normal ATP concentration; however as soon as the concentration drops (i.e. in ATP-depleted fatigued heart muscle, or during a coronary artery occlusion). They shorten the action potential and therefore shorten systole. Apparently that plays a protective factor to the ATP-depleted myocytes, by reducing demand. However it also kills people with heart failure and pulmonary oedema.

**Why do we obsess over electrolytes?**

- Changes in external K+ concentration affect the resting membrane potential;
- Changes in external Na+ concentration affect the MAGNITUDE of the action potential
- This stands true for all excitable tissues

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