Bioen 3203 Lecture #3 (3/8/06)
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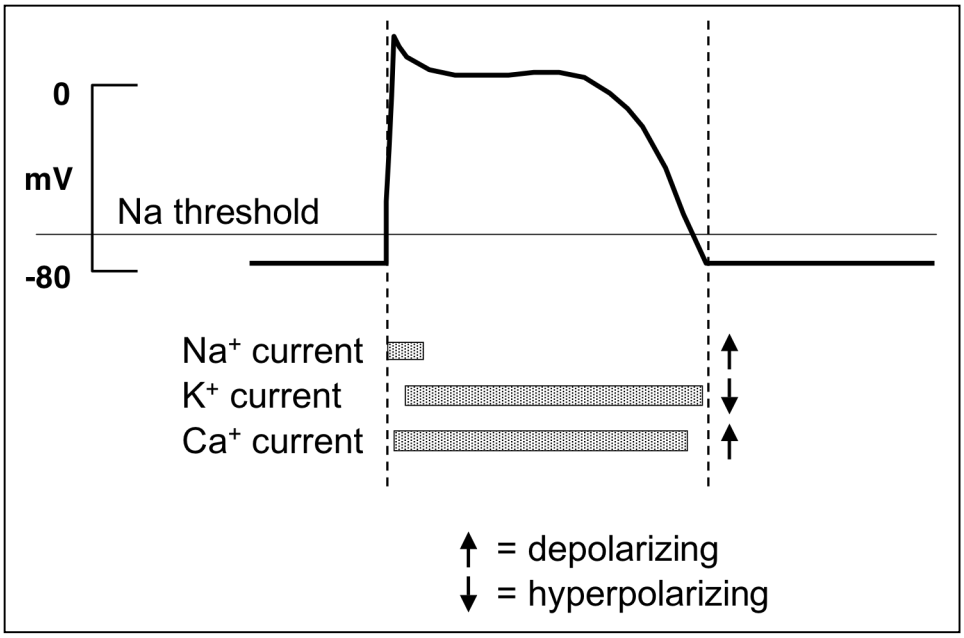
1) Ventricular Cardiac Myocyte Action Potential (A.P.)

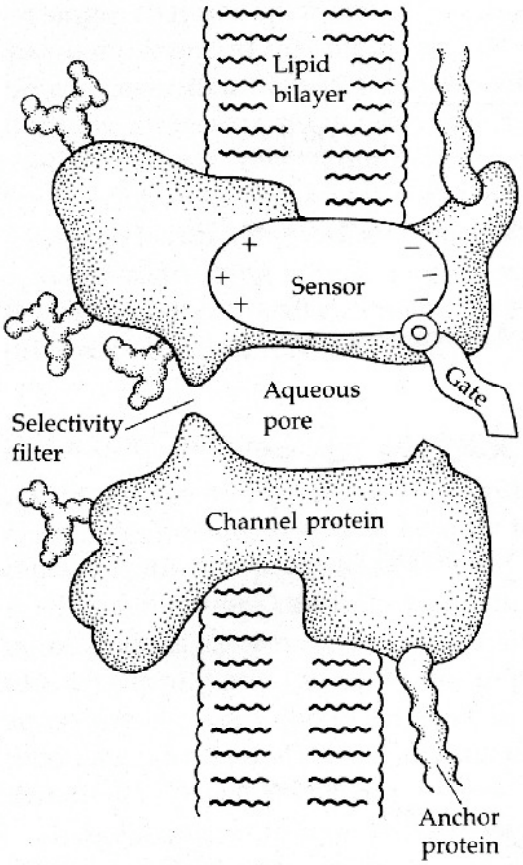
- Comparison to Neuronal A.P.
    a. Stable resting potential (usually).
    b. Much longer action potential.
    c. No hyper polarization.



- A.P. Features
    a. Stimulus - electrical
    b. Ionic Currents – Ca^{++} channels (time dependent with 2 gates).

2) Ion Exchange:

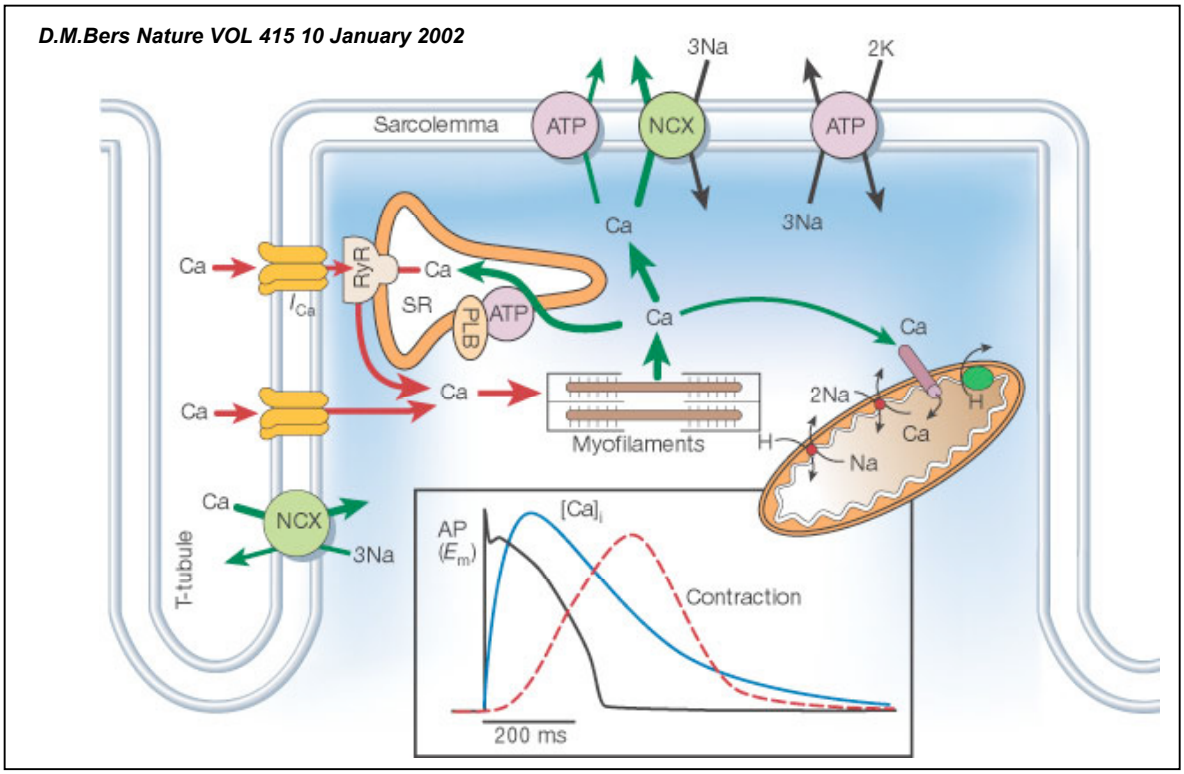
- Na^{+}/ Ca^{++} exchanger.
- ATP drive Ca^{++} pump.
- L-type Ca^{++} channel.
- Na^{+}/ K^{+} pump.
- ~20 different K^{+} channels are activated at different times during the ventricular action potential.
- Channel parts:



3 WORKING HYPOTHESIS FOR A CHANNEL

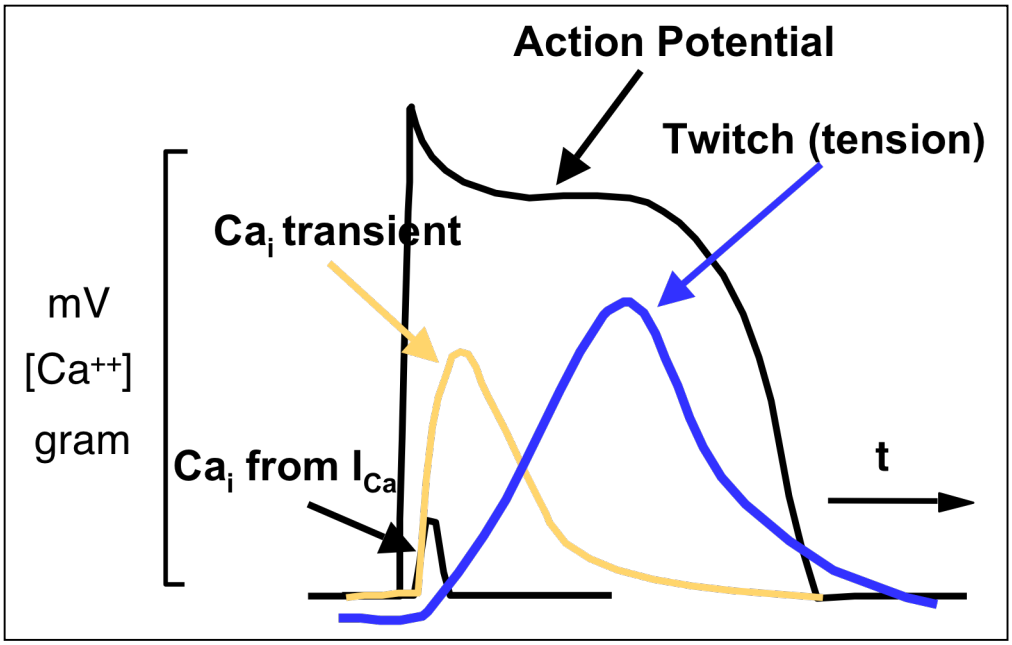
The channel is drawn as a transmembrane macromolecule with a hole through the center. The external surface of the molecule is glycosylated. The functional regions, selectivity filter, gate, and sensor are deduced from voltage-clamp experiments but have not yet been charted by structural studies. We have yet to learn how they actually look.

1) Gates (activating and inactivating)
2) Selectivity filter (allows passage of only certain ions)
3) Charge buildup (depending on the charge on each side of the gate, certain ions will be attracted or repelled).



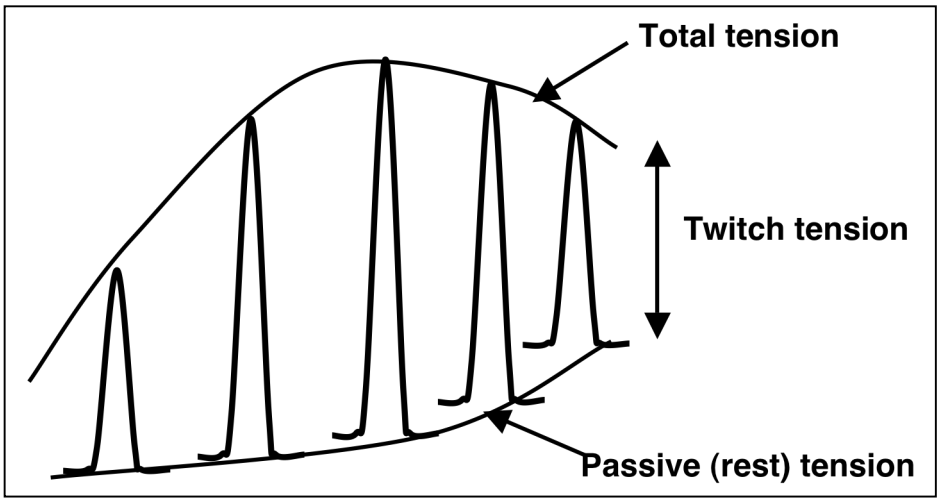
3) Excitation/Contraction coupling:

- Calcium from L-type channels induces large release from SR after interaction with Ryanodine receptor, prolonging action potential. Leads to contraction.
- Ca^{+2} released from the SR interacts with contractile proteins to cause contraction
- Ca^{+2} removed from the intracellular free space soon after it enters by Ca/Na exchanger and Ca pumps in cell and SR membranes.
- Contraction is delayed with respect to AP: about 100 ms
- Regulation of contraction occurs by changes in L-type channel influx, which alters size of release from SR.



4) Frank-Starling Mechanism

- Amount of blood in ventricles will stretch muscle and vary stoke volume depending on how stretched muscles are. (Force-length relationship curve).



- Cellular explanation: individual myocytes contract more strongly at optimal pre-stretch, less strongly for prestreach above and below optimum.

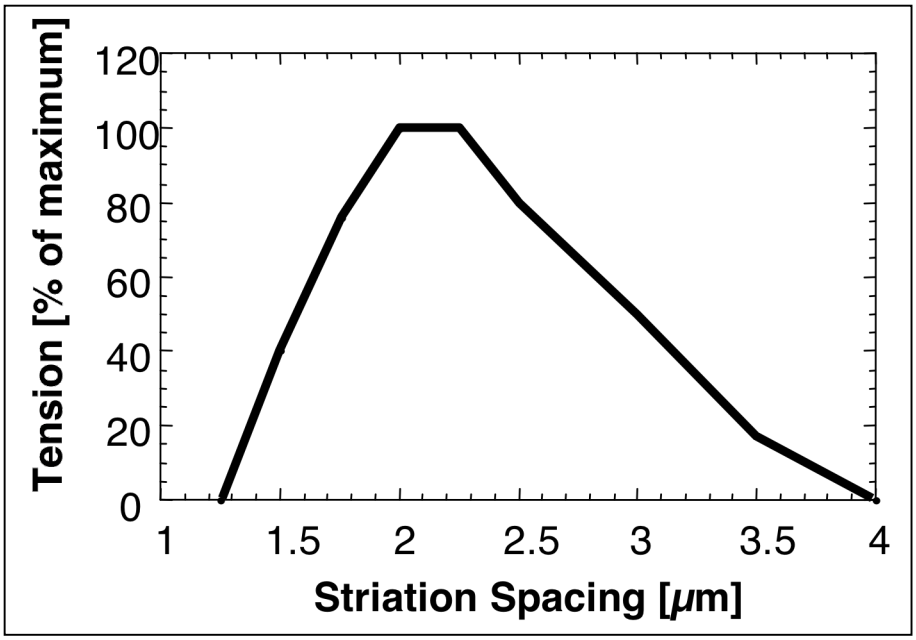


Figure above shows the result for a single myocyte.

For the entire heart, pre-stretch comes from diastolic filling of the chambers and tension development translates to stroke volume. The figure below shows the resulting Frank-Starling curve.