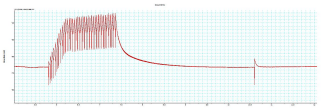
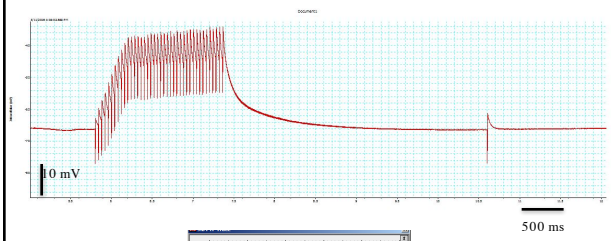
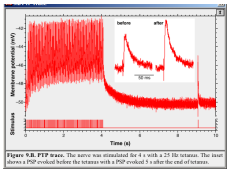
April 6, 2016
PTP/Synaptic Depression
Snail Lab 2- Neuronal Excitability

"Gold Cortex," enamel on composition gold leaf, 18" x 24". Greg Dunn, 2010.

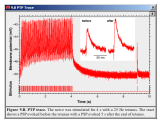


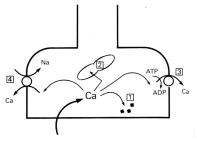
PTP example:



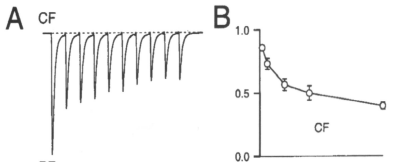


PTP:
1) Correlates with decay of Ca^{2+} image in whole terminal, not just at release sites,
2) reduced by presynaptic Ca^{2+} chelators.
3) In crustacean motor neurons, Na^{+} has a role, perhaps through the Ca/Na pump. Entry of Na^{+} during AP firing may reverse the Na/Ca exchanger to bring in Ca^{2+}.
3) Suggested presynaptic Ca^{2+} unloading from mitochrondria/other internal stores.
4) In Aplysia- Both pre- and post- synaptic Ca^{2+} chelators reduce PTP, as well as postsynaptic hyperpolarization.



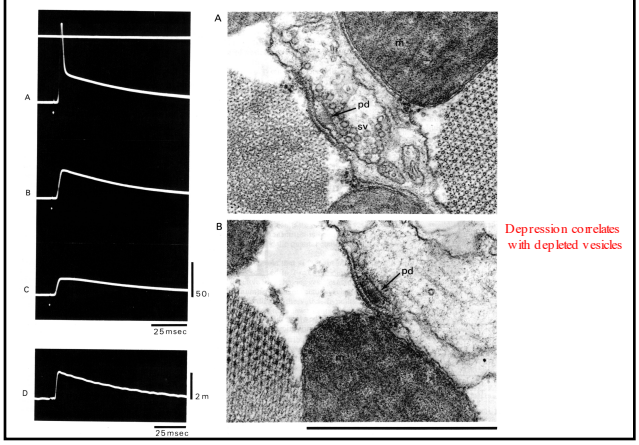


Mechanisms of Synaptic Depression?

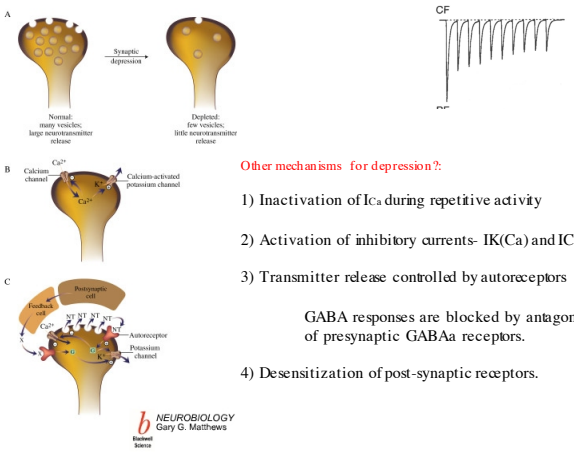


Dietmann et al 2000

Shibere, a temperature sensitive fly mutant, vesicle recycling is blocked



Depression correlates with depleted vesicles



Other mechanisms for depression?:

1) Inactivation of I_{Ca} during repetitive activity

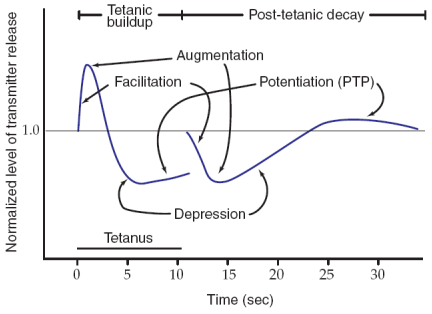
2) Activation of inhibitory currents- IK(Ca) and ICl(Ca)

3) Transmitter release controlled by autoreceptors

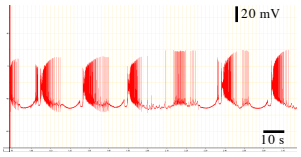
GABA responses are blocked by antagonists of presynaptic GABAa receptors.

4) Desensitization of post-synaptic receptors.

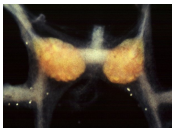
Overlapping Stages of Intrinsic Plasticity

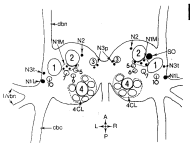


Snail Buccal Ganglia- control of rhythmic feeding movements



Snail bursterneuron





Snail Brain

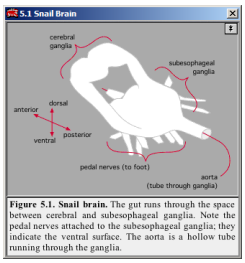


Figure 5.1. Snail brain. The gut runs through the space between cerebral and subesophageal ganglia. Note the pedal nerves attached to the subesophageal ganglia; they indicate the ventral surface. The aorta is a hollow tube running through the ganglia.

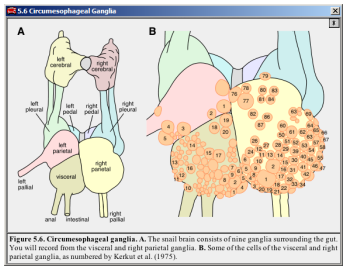


Figure 5.6. Circumesophageal ganglia. A. The snail brain consists of nine ganglia surrounding the gut. You will record from the visceral and right parietal ganglia. **B.** Some of the cells of the visceral and right parietal ganglia, as numbered by Kerkut et al. (1975).

Large neurons ~ 200 micron diameter

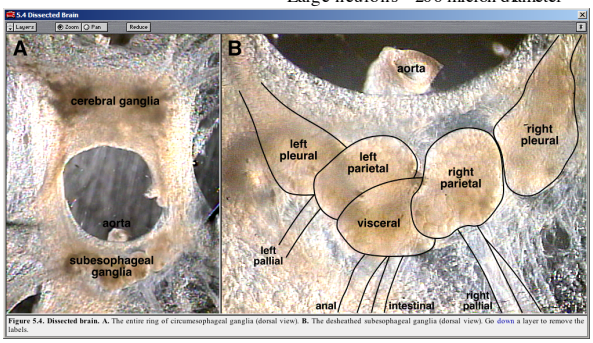
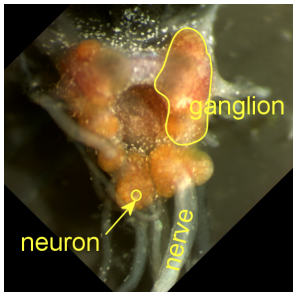
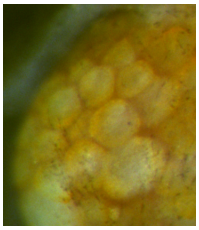


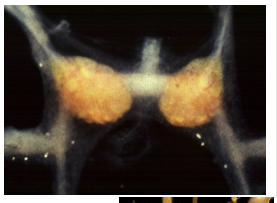
Figure 5.4. Dissected brain. A. The entire ring of circumesophageal ganglia (dorsal view). **B.** The desheathed subesophageal ganglia (dorsal view). Go down a layer to remove the labels.

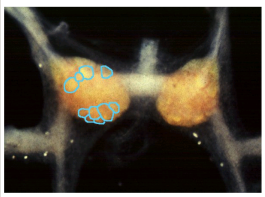
Pond snail brains- Lymnaea, Helisoma



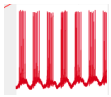


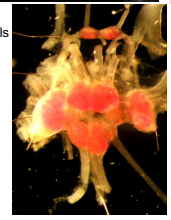
Snail Buccal Ganglia- control of rhythmic feeding movements

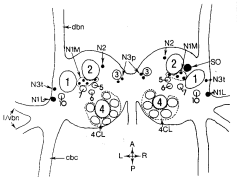




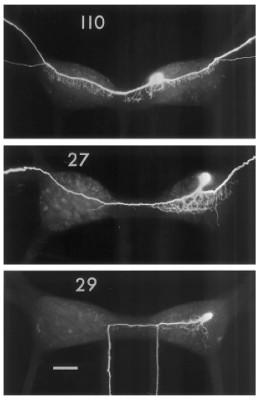
Rhythmically bursting cells







Buccal ganglia neurons



Snail Week 1 Focus: Firing properties of neurons

Action Potentials

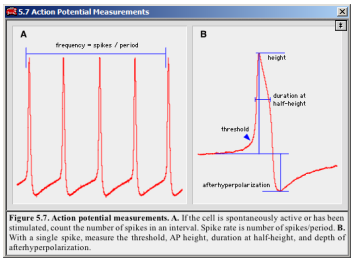


Figure 5.7. Action potential measurements. A. If the cell is spontaneously active or has been stimulated, count the number of spikes in an interval. Spike rate is number of spikes/period. **B.** With a single spike, measure the threshold, AP height, duration at half-height, and depth of afterhyperpolarization.

Responses to current injection

Silent cell

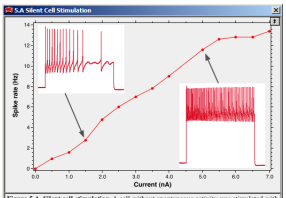


Figure 5.A. Silent cell stimulation. A cell without spontaneous activity was stimulated with depolarizing current for 5 s. The plot shows spike rate during the stimulus vs. the amount of current injected. Insets show activity traces for two of the stimuli.

Tonically firing cell

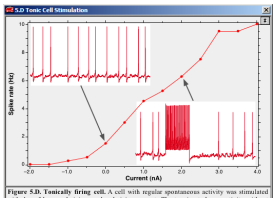


Figure 5.D. Tonically firing cell. A cell with regular spontaneous activity was stimulated with 4 s of hyperpolarizing or depolarizing current. The top inset shows activity without stimulation; the lower inset shows activity with +2 nA of current. Note the period of reduced activity after the stimulus in this inset.

Firing adaptation, also note spike broadening

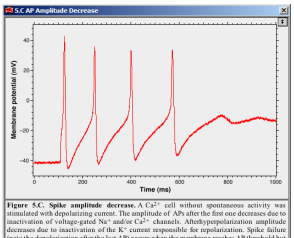


Figure 5.C. Spike amplitude decrease. A Ca^{2+} cell without spontaneous activity was stimulated with depolarizing current. The amplitude of APs after the first one decreases due to inactivation of voltage-gated Na^+ and/or Ca^{2+} channels. Afterhyperpolarization amplitude decreases due to inactivation of the K^+ current responsible for repolarization. Spike failure (note the depolarization after the last AP) occurs when the membrane reaches AP threshold but there are too few active Na^+ channels to trigger a full AP.

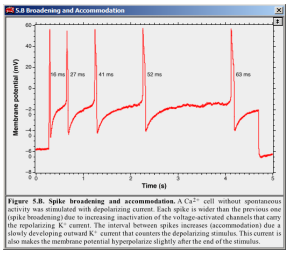


Figure 5.B. Spike broadening and accommodation. A Ca^{2+} cell without spontaneous activity was stimulated with depolarizing current. Each spike is wider than the previous one (spike broadening) due to increasing inactivation of the voltage-activated channels that carry the repolarizing K^+ current. The interval between spikes increases (accommodation) due a slowly developing outward K^+ current that counters the depolarizing stimulus. This current is also makes the membrane potential hyperpolarize slightly after the end of the stimulus.

Post-inhibitory rebound

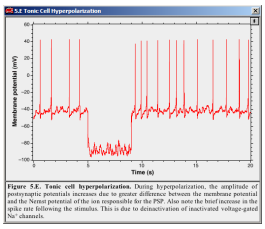


Figure 5.8. Tonic cell hyperpolarization. During hyperpolarization, the amplitude of postsynaptic potentials increases due to greater difference between the membrane potential and the Nernst potential of the ion responsible for the PSP. Also note the brief increase in the spike rate following the stimulus. This is due to deinactivation of inactivated voltage-gated Na^+ channels.

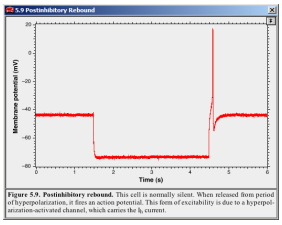


Figure 5.9. Postinhibitory rebound. This cell is normally silent. When released from period of hyperpolarization, it fires an action potential. This form of excitability is due to a hyperpolarization-activated channel, which carries the I_h current.

note enhanced EPSPs

Burster neurons

Burst frequency control

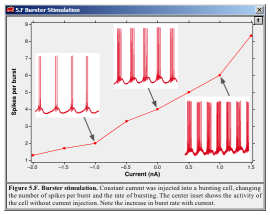


Figure 5.7. Burster stimulation. Constant current was injected into a bursting cell, changing the number of spikes per burst and the rate of bursting. The center inset shows the activity of the cell without current injection. Note the increase in burst rate with current.

Burst characterization

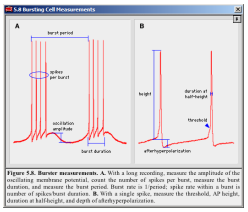
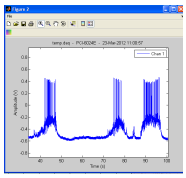
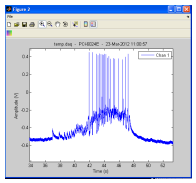


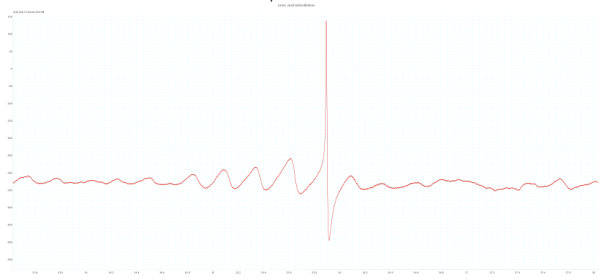
Figure 5.8. Burster measurements. **A.** With a long recording, measure the amplitude of the oscillating membrane potential, count the number of spikes per burst, measure the burst duration, and measure the burst period. Burst rate is 1/period; spike rate within a burst is number of spikes/burst duration. **B.** With a single spike, measure the threshold, AP height, duration at half-height, and depth of afterhyperpolarization.



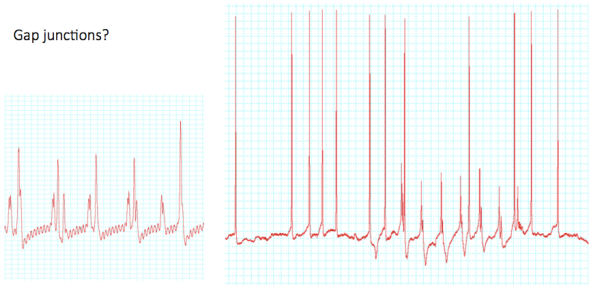
"true" bursters vs. driven bursters



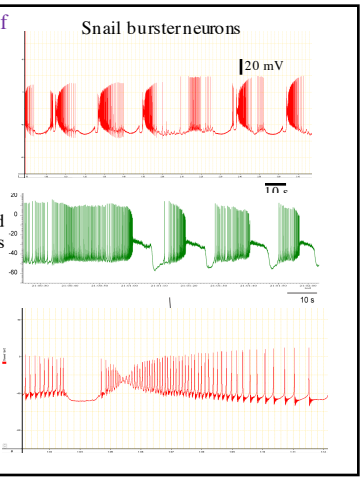
Membrane potential oscillations



Gap junctions?



Interesting firing properties of neurons



Mouse spinal cord burster neurons

Why do APs get smaller and then big again?