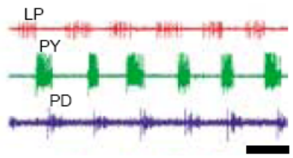
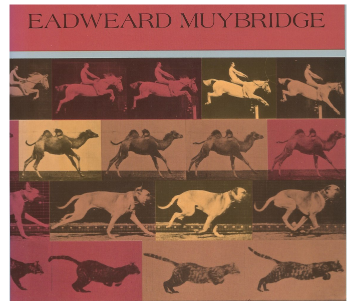
April 4, 2016
Central Pattern Generator Lab



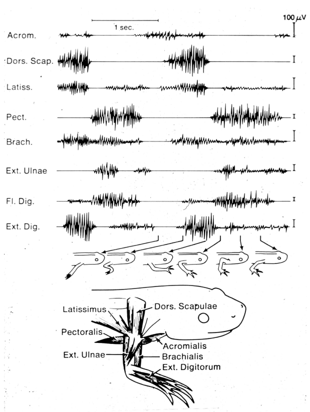
Due Today: Sim 8
Thursday: Lab Results- Synapse 2 Plasticity; Plant
Due Friday: Synapse 1

Neural Networks for Motor Pattern Production

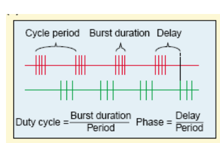


Rhythmic motor patterns important for locomotion, breathing
Easier to study than many motor programs- can be automatically and consistently repetitive

Activity Parameters of Rhythmic Motor Patterns



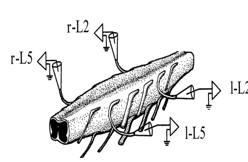
Rhythmic Motor Patterns characterized by Rhythm Frequency and appropriate Activity Phasing



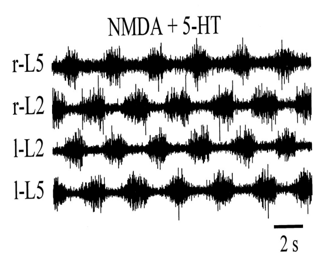
Neural Networks for Rhythmic Motor patterns

Central Pattern Generators (CPGs) produce appropriate rhythm and firing phasing

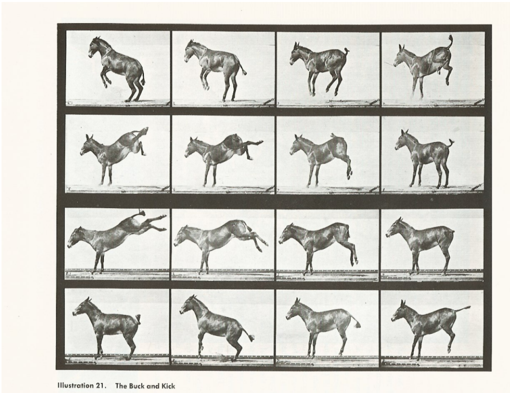
No sensory feedback !
No higher brain activity!



Rodent lumbar spinal segments



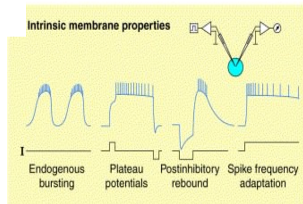
Motor Patterns are Plastic



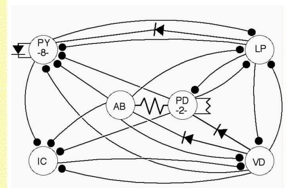
Monty Python movie

What network and cellular properties important for rhythm pattern production? which can be modulated for motor pattern plasticity?

Cellular/synaptic properties important for CPGs
All are modifiable



Marder and Bucher 2001



Lobster pyloric network

Snails as a model systems in neurobiology

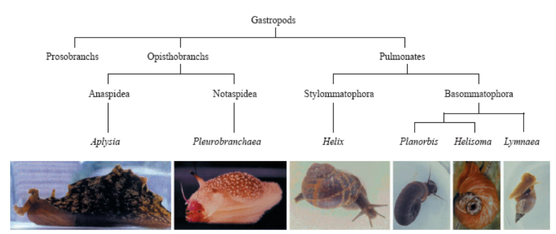
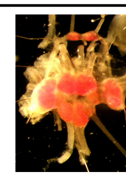


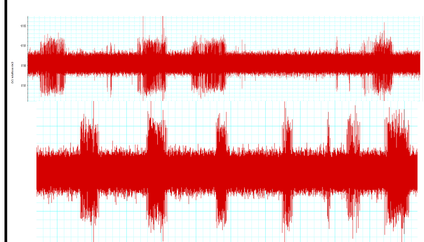
Fig. 1. The herbivorous gastropods studied include the marine opisthobranchs *Aplysia* (10 cm long) and *Pleurobranchaea* (5 cm long, eating a nudibranch, *Flabellina* which is coloured purple and orange) and the pulmonates *Helix* (terrestrial, 2 cm long) and three freshwater genera (*Planorbis*, *Helisoma* and *Lymnaea*, all 1 cm long). The pictures are not to scale. We are grateful to Rhanor Gillette and Andy Bulloch for the pictures of *Pleurobranchaea* and *Helisoma*.

Our lab snail

This week's lab
Snail feeding
CPG



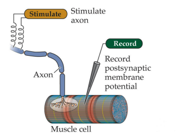
First observe feeding behavior, then record its neural correlates



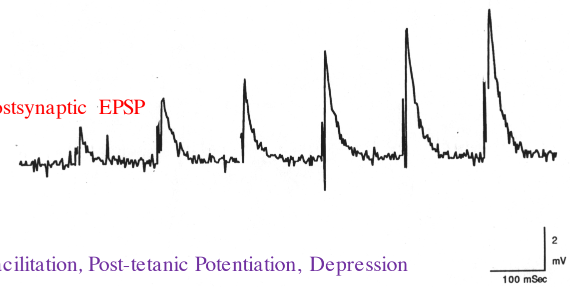
10^{-5} M DA

10^{-4} M DA

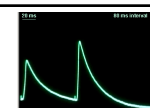
Intrinsic Synaptic Plasticity-
transforms time interval into voltage amplitude



Postsynaptic EPSP



Facilitation, Post-tetanic Potentiation, Depression



Intrinsic Synaptic Plasticity (short term)
Change in synaptic strength due to activity of synapses
Important for:
coincidence detection
gain control
oscillatory networks (phase onset and offset, cycle period)
Habituation, sound localization
Learning and Memory

Extrinsic Synaptic Plasticity
Change in synaptic strength due to neuromodulatory substances
Important for:
Network reconfiguration (active network members,
their excitability and synaptic connections)

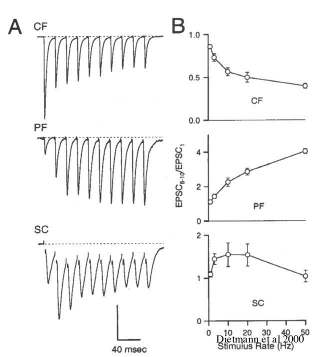
Synaptic changes due to disease

Intrinsic synaptic modulation
highly variable in different brain
networks:

Depression

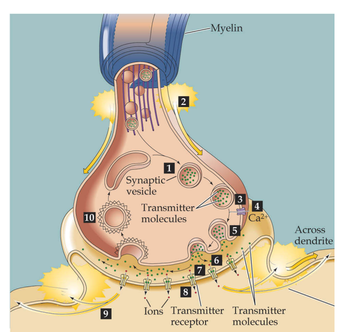
Facilitation

Mixed



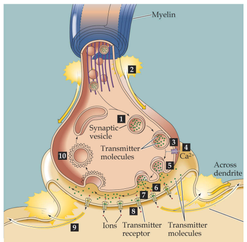
Dietmann et al 2000

Synaptic Modulation

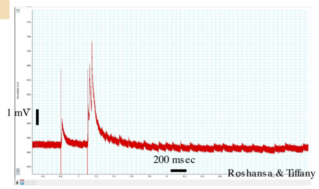


Sites of Possible Synaptic Change?

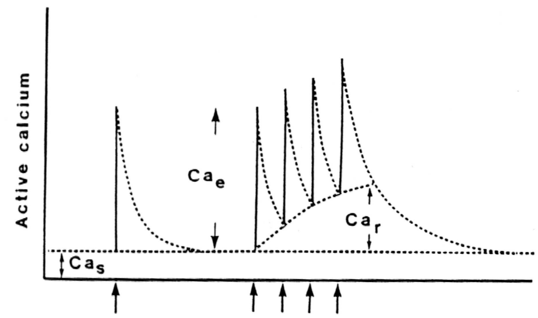
Sites of synaptic change in short term plasticity?



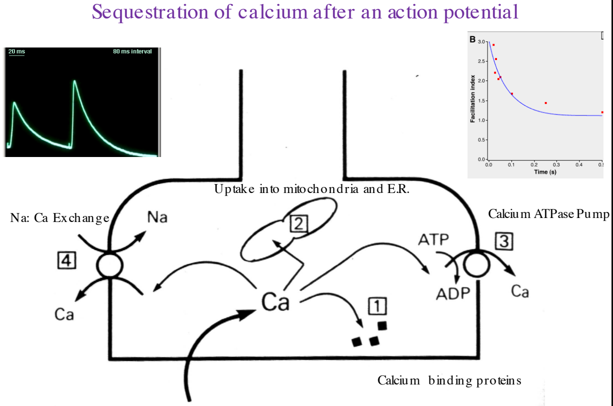
Facilitation:
a) MEPP sizes do not change.
b) MEPP frequency increases.
Quanta content (m = PSP/mini) is increased
pre or post? .



Residual Ca hypothesis for facilitation: Presynaptic Ca builds up with each AP

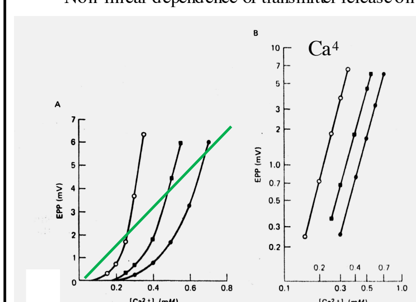


Sequestration of calcium after an action potential



Takes 100-500 msec to bring calcium levels to normal after an AP

Non-linear dependence of transmitter release on $[Ca]_i$

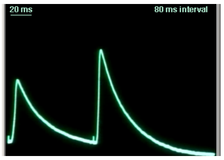


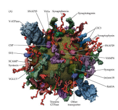
Dependence of EPP amplitude in frog muscle on Ca^{2+} concentration in the bathing solution. A: Linear plot of EPP amplitude versus Ca^{2+} concentration. B: Log (EPP amplitude) versus log (Ca^{2+} concentration). Open circles = $[Mg^{2+}]$ = 0.5 m*M*; squares = 2.5 m*M*; solid circles = 4.0 m*M*. (From Dodge and Rahamimoff, 1967.)

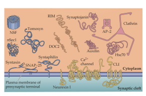
AP brings in
5 units of Ca
$5^4 = 525$
80% uptake before
Next AP

1 unit left= $1^4 = 1$
Next AP = 1 + 5 units
$6^4 = 1296$
(twice as much
NT release!)

Facilitation







BUT:

1) Simulations of expected peak and residual Ca^{2+} levels not able to account for facilitation.

2) Using Ca^{2+} sensitive dyes, pre-synaptic $[Ca^{2+}]$ was not raised enough to account for enhanced synaptic transmission.

3) The time course of $I_{K(Ca)}$ is too fast. The decay of this current should reflect the decay of residual Ca^{2+}.

Ca may be acting at multiple sites of the synaptic machinery