Review 14

Nerves, con't.

- What causes action potential to travel down length of axon?
  - influx of sodium depolarizes adjacent area, causing same set of events to occur "next door
- Four things to remember about action potentials:
  - ① don't occur until CRITICAL LEVEL OF DEPOLARIZATION achieved
  - ② are ALL OR NONE phenomena
  - ③ travel lengths of axons WITHOUT DECREMENT
  - ④ presence of REFRACTORY PERIOD prevents action potentials from going in both directions
- Transmission of action potential to next cell (across SYNAPSE)
  - ➔ Neurotransmitter released from pre-synaptic membrane
  - ➔ Neurotransmitter diffuses across synapse; binds with BINDING PROTEIN
  - ➔ Binding protein-neurotransmitter complex allows sodium to leak into post-synaptic cell
  - ➔ If critical level of depolarization reached, new action potential spreads from synaptic region
  - Acetylcholine (ACh) is a very common neurotransmitter
  - Removal of ACh from synapse--ACETYLCHOLINESTERASE (AChase)
    - also embedded in post-synaptic membrane
    - breaks ACh into acetate and choline
    - choline reabsorbed into nerve end plate

Muscle stuff (mostly reminders of previous material):

- Motor neuron feeds into muscle, triggers muscle contraction
  - AP in muscle causes release of Ca^{++} into muscle cytoplasm
  - Presence of Ca^{++} activates contractile mechanism; uses lots of ATP
  - Reabsorption of Ca^{++} inactivates contractile mechanism, allowing relaxation of muscle, and also uses ATP

Review 14, con't

Problems with ACh at the neuromuscular junction:

Botulism and tetanus toxins:

- block release of ACh from pre-synaptic terminal
- result is paralysis due to lack of synaptic transmission

Myasthenia gravis:

- autoimmune disease: produce antibodies (IgG) to ACh receptor protein in post-synaptic membrane, causing loss of receptors and reduction in response to ACh
- result is progressive muscle weakness and easy tiring

Military nerve gases:

- cholinesterase inhibitors
- allow overstimulation of post-synaptic cells
- result is loss of nerve function and paralysis

Inhibitory neurons: act by *hyperpolarizing* post-synaptic cells

e.g. effect on motor neurons

- normally require 2 – 3 almost simultaneous incoming messages to launch AP (which will then cause muscle contraction)
- with hyperpolarization, requires 4 – 5 almost simultaneous incoming messages to stimulate action potential

Strychnine: blocks inhibitory receptor proteins on post-synaptic membranes, resulting in loss of inhibitory activity; result is uncontrolled tremors and death due to asphyxiation