Unit 4 Lecture 3 Notes

I. Nerve Regeneration
   1. Mature neurons cannot regenerate so severe damage close to the cell back can cause neuron to die
      i. May kill neurons with connections to injured neuron due to lack of stimulation
   2. Injured peripheral axons regenerate
      i. Proximal axon end seals off and swells up
      ii. Organelles and materials accumulate
      iii. Wallerian Degeneration - distal axon begins to degenerate
      iv. If reconnected the axon regains its function
   3. In CNS regeneration is almost never successful
      i. Microglia and astrocytes phagocytize debris
      ii. Oligodendrocytes surrounding fibers will die (no channels, glial scare tissue blocks path)

II. Transmission of Information
   1. Path from receptor (modified dendritic end) to effector
      i. Information sent from receptor across afferent neuron
      ii. To synapse with association neuron
      iii. To another association neuron OR efferent neuron
      iv. Moves across efferent neuron to effector (muscle or gland)
   2. Neurons respond to stimulation (electrical impulse along axon)
      i. Impulse is the same regardless of the source or type
   3. Resting membrane potential
      i. More K+ inside cell and more Na+ outside cell → movement through channels in response to electrical and chemical gradient (membrane more permeable to K+)
      ii. Sodium potassium pump maintains potential difference across membrane to generate nerve impulse (info from one neuron to next)
      iii. Depolarization of Membrane
         1. Reduction in membrane potential → inside becomes less negative and + ions move to cause depolarization
      iv. Hyperpolarization of membrane – increase membrane potential (more negative)
      v. Local or Graded Potentials
         1. Short-lived, local changes in membrane potential
         2. Magnitude varies – stronger stimulus=greater change
         3. Local changes cause flow of current to adjacent regions – set up different potential difference in adjacent regions
         4. Local currents die with increasing distance – important in initiating action potential

III. Action Potentials – how neurons communicate
   1. Brief, large depolarization of plasma membrane that requires threshold stimulus

            i. Threshold stimulus causes depolarization of membrane to trigger zone of axon hillock
            ii. -55 to -50 mv becomes all-or-none
                1. Spike Potential - potential will become less and less negative and overshoot to +30mv
        2. After stimulus depolarization is driven by influx of Na+ ions
            i. Na+ ions enter → membrane potential less negative → voltage-gated Na+ channel opens → Na+ comes in
            ii. Called Hodgkin Cycle
            iii. Spike Potential occurs
            iv. Repolarization - Na+ gates close and K+ gates open to move toward RMP
        3. After hyperpolarization (undershoot)
            i. Potassium gates open longer so more leaves (Na+ diffusion stops)
            ii. Interior becomes more negative than RMP and Na+/K+ pump helps restore RMP
        4. Latent Period – time between stimulus and depolarization
        5. Absolute Refractory – time when no stimulus will cause depolarization (Na+ gates open – guarantees rest interval so neuron is not continuously fired)
        6. Relative Refractory – time larger than normal stimulus will cause depolarization (after hyper-polarization stage - Na+ gates closed and K+ gates open)

IV. Propagation (conduction) of impulse
    1. Stimulus creates local currents (graded potential) that's spread along membrane of dendrites and cell body toward the axon hillock
        i. Motor Neurons – Action Potential generated at axon hillock
        ii. Unmyelinated fibers – Action Potential takes place over limited area of axon
    2. Process
        i. Stimulus
        ii. Inward Na+ causes region to become less negative (eventually positive)
        iii. Na+ ions move toward axon area (still Negative)
        iv. This sets up a local current that depolarizes next section of axon
        v. Impulses travel in one direction – axon refractory behind the leading edge of depolarization
    3. New Action Potential is created at each depolarization point – different from conduction of electrical current

V. Saltatory Conduction (propagation)
    1. Occurs in Myelinated axons
        i. Action potential occurs at Nodes of Ranvier
        ii. Channels for Na+ and K+ all crowded at bare part of axon (nodes)
        iii. Ionic current flows through extracellular fluid and axoplasm (current jumps from node to node)
            1. Occurs faster than unmyelinated fibers
        iv. Speed depends of fiber diameter and presence of myelin

1. Type A – myelinated fibers; 1-20 microns in diameter; 5-150 meters per second (F<u>ASTEST</u>)
    a. Motor to skeletal muscles
    b. Sensory fibers for muscle stretch and localized pain
2. Type B – finely myelinated; up to 3 microns in diameter; 3-15 meters per second
    a. Visceral nerve fibers
3. Type C – unmyelinated; 3-5 microns in diameter; 0.6-2 meters per second (<u>SLOWEST</u>)
    a. Poorly localized pain

VI. Synapse - region of functional but not actual contact (nerve impulses cross)
1. Axondendritic – between axon and dendrite
2. Axosomatic – between axon and cell body
3. Axoaxonic – between axon and axon

2. Electrical synapses – (relatively few) gap junctions bridges between neurons
    i. Multiple neurons synchronize response
    ii. Like pacemakers in single unit smooth muscles
    iii. In brain regions responsible for stereotyped movements
3. Chemical Synapses – synaptic neuron in telodendron of axon
    i. Synaptic knob contain synaptic vesicles that hold neurotransmitter and large numbers of mitochondria
    ii. Synaptic cleft – fluid filled space (1millionth inch across) that does not have direct contact with neurons
        1. Neurotransmitter (NT) diffuses across synaptic cleft
    iii. Postsynaptic neuron or effector cell
        1. Dendrites
        2. Cell body
        3. Effector – muscle or gland
            a. Postsynaptic membrane with NT receptors
            b. Protein specialized to react with NT
            c. Different NT may effect postsynaptic neuron
4. Functional types of Synapses
    i. Excitation – NT causes partial depolarization →membrane potential of postsynaptic membrane is decreased
    ii. <u>Excitatory Postsynaptic Potential (EPSP)</u>
    iii. Synaptic resistance to firing is reduced (facilitation)
5. Inhibition
    i. NT causes partial hyperpolarization → Sometimes – Chloride gates open, Cl^- enters →Membrane potentials increases
    ii. Called an <u>Inhibitory Postsynaptic Potential (IPSP)</u>
    iii. Resistance to firing is increased

VII. Neurotransmitter (over 100+ kinds)
1. Chemical compounds that diffuse across aynaptic cleft
2. Cause depolarization (impulse transmitted) or hyperpolarization (transmission blocked)