The Synapses

Summary:
Axon hillock: the very first part of axon
Inside the neuron: figure 1.3 (part 1)
Ions go from high concentration to low concentration
Action Potential: neurons talk with each other
  Depolarization—resting potential—hypolarization

Neurons are ‘simple’ computing devices
Brain functions (including cognitive functions) rely on the activity of interacting neurons
Interactions=synapses

Synaptic Morphology
-pre/post synaptic site
-types of synapse
-synaptic vesicles
-neurotransmitters

Axonal transport
‘stuff’ moves along the axon microtubules (axoplasmic transport)

The parts: figure 2.23
synaptic vesicles are filled with neurotransmitters molecules

3 kinds of synapse locations: figure 2.22
Axo-Dendritic Axo-Somatic Axo-Axon

Synapse Physiology
The synapse is the place where 2 neurons ‘talk’ to each other
Synapse only happened when an action potential
AP—Vesicle fusion—neurotransmitter release in cleft

Neurotransmitter Release: figure 2.24
The action potential of a triggers vesicle fusion at the synapse
Neurotransmitter molecules are released into the synaptic cleft
They are ‘received’ by the postsynaptic membrane in B by 2 kinds of neurotransmitter dependent ion channels

Ionotrophic Receptors: figure 2.25
Transmitter binds—activates receptors—open ion channels
(reflexes, perception, information processing…)

Metabotropic Receptors: figure 2.26
Mediate the influence of hormones and drugs, state-dependent information processing
Second messengers molecules that link receptors to ion channels
Transmitter binds-activate receptor—
    Activate ‘second messengers’—open ion channels
    -intracellular effects

IPSPs and EPSPs: figure 2.27
Excitatory/Inhibitory PostSynaptic potential
Add more +Na would be depolarization
Potassium leave the cell would be more negative, that would be hypolarization
One given neuron release the same neurotransmitter at all of its synapse

Regulation of Release; Re-uptake: figure 2.28
Recycling of molecules
Help with fast, efficient neurotransmission (low signal-to-noise)

Regulation of Release: Autoreceptors, Enzymatic Deactivation
Autoreceptors
-on the presynaptic membrane(aka presynaptic receptors)
-regulate synthesis and release of neurotransmitter (no ion flow)
-mostly metabotropic

Enzymatic Deactivation
Acetylcholine(Ach) vs. Acetylcholine esterase (AChE)

Regulation of Release: axo-axonic synapse: figure 2.30
The AB synapse helps (or interferes with) the BC synapse
The AB synapse exerts a presynaptic facilitation (or inhibition) of the BC synapse

Nonsynaptic Communication
Fun Fact 1
-some neurotransmitters are released diffusely (‘leak-out’): Neuromodulators
-they have slow and diffuse actions (peptides). Influence many postsynaptic targets
eg. Involved in attention, emotions, pain sensitivity

Fun Fact 2
-Most hormones are produced by endocrine glands (adrenal glands, stomach….) in the body
-some neurons produce hormones rather than neurotransmitters
-some neurons have hormone receptors (target cells)
Communication between nervous system and body
eg. Sex hormones and aggression. Stress.

Synaptic Physiology
A.P.—vesicle fusion—Neurotransmitter release
---receptor opening---ion flow---postsynaptic potentials

Neural Integration.
-in space
-in time

Spatial Summation
Temporal Summation
-Postsynaptic potentials from the same synapse (but different action potentials) sum up

Spatio-Temporal Summation: figure 2.29

Preview: when synaptic transmission goes wrong
- not enough neurotransmitter binding: acetylcholine and myasthenia gravis treated by inhibition of enzymatic deactivation
- not enough neurotransmitter binding: depression and selective serotonin reuptake inhibitors
- weakness of postsynaptic receptors: dopamine and drug addiction
- too much binding: dopamine and schizophrenia
- death of presynaptic neurons that produce a specific neurotransmitter: dopamine and Parkinson’s disease
- change in number/sensitivity of postsynaptic receptors: glutamate and learning and memory

Seven steps of neurotransmission
1. neurotransmitter molecules are synthesized from preceptors under the influence of enzymes
2. neurotransmitter molecules are stored in vesicles
3. neurotransmitter molecules that leak from their vesicles are destroyed by enzymes
4. Action Potential causes vesicles to fuse with the presynaptic membrane and release their neurotransmitter molecules into the synapse
5. released neurotransmitter molecules bind with autoreceptors and inhibit subsequent neurotransmitter release
6. released neurotransmitter molecules bind to postsynaptic receptors
7. released neurotransmitter molecules are deactivated either by reuptake or enzymatic degradation