Ch 4, part 1: Neurons, Neurotransmitters, and Cell Communication.

Objectives:
1. Review different types of neurons and neuron anatomy.
2. Understand how neurons communicate.
   - neurotransmitter signaling & action potentials
3. Learn types & functions of neurotransmitters.
4. Become familiar with influence of disease & drugs on neurotransmitter signaling.

See Webpage Neurophysiol. Supplements!

1. Different Types of Neurons and Neuron Anatomy

Anatomy REVIEW!

1. Neurons
   a) Sensory (afferent) neurons = \textit{Send/Sensory info to brain}. (ex. thermoreceptors, baroreceptors)
   b) Motor (efferent) neurons = \textit{Carry motor commands effect from brain out to PNS.}
      to muscle or gland cells.

2. Interneurons \textit{(in CNS)} = strictly in CNS. Relays info. between spinal cord (CNS) & PNS.

3. Neuroglial (Glia) Cells =
1. Different Types of Neurons and Neuron Anatomy

**Fig 4.3**

**Central Nervous System (CNS)**

- **Sensory info ascends spinal cord**
- **Motor info descends spinal cord**

**Peripheral Nervous System (PNS)**

- **Sensory neurons have a dorsal “ganglion”**
- **Sensory (afferent) neuron**
- **Motor neuron**
- **Interneuron**
- **Autonomic Motor neuron**

**5 Types of Glial Cells (4 in CNS & 1 in PNS)**

- **Ependymal cells** = found in choroid plexus of brain ventricles which make CSF.
- **Astrocytes** = make up blood brain barrier in pia mater.
- **Schwan cells (PNS) & Oligodendrocytes (CNS)** = produce myelin sheaths on neuron axons to speed up transmission.
- **Microglia** = brain’s clean up crew. Phagocytizes pathogens.
1. Different Types of Neurons and Neuron Anatomy

**Dendrites** = picks up sensory info from other neurons.
**Cell body** = where cell nucleus of neuron found.
**Axon** = elongated tube that transmits impulse from cell body to synaptic knobs (end of neuron)
**Myelin sheath** = insulated wrappings around axon that keeps signal from dissipating from axon. \[\text{unmylenated axon} = 0.5 \text{m/sec} \quad \text{VS} \quad \text{mylenated axon} = 100 \text{m/sec}\]
**Nodes of Ranvier** = gaps between myelin sheaths where signal jumps to next node (faster conduction)
**Synaptic knobs** = neuron end where electrical impulse turned into a neurotransmitter.

---

**Multiple Sclerosis** – online.

= autoimmune destruction of myelin sheaths of white matter CNS (oligodendrocytes), which creates scar tissue or “scleroses” on the axons.

It slows transmission of electrical impulses, especially in motor neurons involved in movement.

Patients have motor (movement) and many other problems.
1. Different Types of Neurons and Neuron Anatomy

**Secretory vesicles** = vesicles in synaptic knobs that store neurotransmitters.

**Neurotransmitter** = message that crosses the synapse to start an action potential in next cell. (Ex. ACh)

**Synapse** = gap between 2 neurons, or between a neuron and a muscle or gland cell.

**Pre-synaptic neuron** = neuron before synapse.

**Post-synaptic neuron** = neuron or cell after synapse. Has a receptor for neurotransmitter.

---

**Review**

- Types of neural tissue
  - Neurons (sensory/afferent, motor/efferent, and interneurons)
  - Neuroglial (Glial) cells
    - CNS – astrocytes, microglia, ependymal cells, oligodendrocytes
    - PNS – schwan cells, satellite cells
- Anatomy of a neuron
  - Cell body, dendrites, axon, myelin sheath, Nodes, synaptic knobs, synapse, secretory vesicles, neurotransmitters, pre-synaptic neurons, post-synaptic neurons.
2. How Neurons Communicate with Cells

Neurotransmitter signaling = a chemical signal binds to receptor on cell membrane (ex. When acetylcholine binds to a receptor), which:

1. Opens Na+ channel in membrane, Na+ floods into cell causing action potential (AP) or “depolarization”, which stimulates a cell.

2. K+ channels open, K+ exits cell, causes “repolarization”, which inhibits cells.

Neurotransmitter signaling:

1. Pre-synaptic neuron releases neurotransmitter (like ACh) into synapse.

2. Neurotrans. binds to receptor on post-synaptic cell, opens ion (usually Na+) channels on cell membrane.

3. Na+ floods into cell, causes action potential (AP) to form.

4. AP travels through cell.
Neurotransmitter binding to receptor opens ion channel:

**CAN EITHER:**

A) **Cause an Action Potential** *(Excitatory post-synaptic potential or EPSP)*

- IF neurotransmitter binds to receptor that opens Na+ or Ca+2 channels, & causes an AP to form.

  - EPSP can produce “graded potential” = 
    - A lot of neurotrans = big cell response
    - A little of neurotrans = little cell response

  - EPSP can produce “summation” = *Repeated* (high frequency)
    - more frequent stim = big cell response
    - less frequent stim = little cell response

**Example:** the increase in heart rate with epinephrine binding to its *adrenergic* receptor on heart muscle cells is due to opening of Na+ and Ca+2 channels!

B) **Inhibits an Action Potential** *(Inhibitory post-synaptic potential or IPSP)*

- IF neurotransmitter binds to a receptor & opens K+ or Cl- channels, prevents an AP from forming.

**Example:** the decrease in heart rate with ACh binding to its *cholinergic* receptors on heart muscle is due to opening of K+ channels!
Regulation of Neurotransmitter Action:

The 2 R’s:

1) Receptor types

- neurotransmitter effect depends on what kind of receptor it binds to.

   Either:
   
   A. Nicotinic (ion-gated) receptor – for voluntary control of skeletal muscle
   B. Muscarinic (G-protein coupled) receptor – for autonomic control of glands, smooth muscle, & cardiac muscle.

2) Removal systems for neurotransmitters (4 removal systems)

---

1) Receptor types:

A. Nicotinic (Ion-gated) receptor

- For neurotransmitter
- Binding of receptor by ACh causes channels to open.
- Na+ channel opens causes stimulation in a cell.

Thus, skeletal muscle cells have nicotinic cholinergic receptors for ACh for voluntary movement.

Some sensory neurons also have nicotinic cholinergic receptors
1) Receptor types:

A. Nicotinic (Ion-gated) receptor

- For neurotransmitter
- Binding of receptor by ACh causes channels to open.
- Na+ channel opens causes in a cell.

Thus, skeletal muscle cells have nicotinic cholinergic receptors for ACh for voluntary movement.

Some sensory neurons also have nicotinic cholinergic receptors

B. Muscarinic (G-protein coupled) receptor:

- Receptor binding activates enzyme then a G-protein
- G-protein then opens ion channels.
  - IF Na+ and Ca+2 channel opens = stim. or EPSP
  - IF K+ or Cl- channel opens = inhibit or IPSP

- For ACh, norepinephrine & epinephrine, & other neurotransmitters
- Thus, gland cells, and cardiac and smooth muscle cells have muscarinic receptors for involuntary movement.

Ex. Nicotinic cholinergic (ACh) receptors

Fig 4.25

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? EPSP
Ex. Muscarinic adrenergic (epinephrine, norepinephrine) receptors

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? EPSP

Ex. Muscarinic GABA receptor

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? IPSP
For ACh and its receptors:

**ACh & Nicotinic Cholinergic Receptors**

- Postsynaptic membrane of
  - All autonomic ganglia from spinal cord
  - For voluntary control of skeletal muscles

- Neurons Releasing ACh: Somatic motor, preganglionic neurons of ANS
- Location: Skeletal muscles, autonomic ganglia
- Type of ACh Receptor: Nicotinic
- Response: Depolarization, producing action potentials
- Physiological Effect: Muscle contraction

**ACh & Muscarinic Cholinergic Receptors**

- Neurons Releasing ACh: Preganglionic parasympathetic neurons
- Location: Smooth muscles, glands
- Type of ACh Receptor: Muscarinic
- Response: Depolarization, producing action potentials
- Physiological Effect: Contraction of smooth muscles; secretion of glands

<table>
<thead>
<tr>
<th>Neurons Releasing ACh</th>
<th>Location</th>
<th>Type of ACh Receptor</th>
<th>Response</th>
<th>Physiological Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic motor</td>
<td>Skeletal muscles</td>
<td>Nicotinic</td>
<td>Depolarization, producing action potentials</td>
<td>Muscle contraction</td>
</tr>
<tr>
<td>Preganglionic neuron of ANS</td>
<td>Autonomic ganglia</td>
<td>Nicotinic</td>
<td>Depolarization, producing action potentials</td>
<td>Muscarinic postganglionic neurons of the PNS</td>
</tr>
<tr>
<td>Preganglionic parasympathetic neurons</td>
<td>Smooth muscles; glands</td>
<td>Muscarinic</td>
<td>Depolarization, producing action potentials</td>
<td>Contraction of smooth muscles; secretion of glands</td>
</tr>
<tr>
<td>Postsynaptic membrane of autonomic ganglia</td>
<td>Smooth muscles, glands</td>
<td>Muscarinic</td>
<td>Depolarization, producing action potentials</td>
<td>Contraction of smooth muscles; secretion of glands</td>
</tr>
</tbody>
</table>

Produces autonomic parasympathetic (rest and digest) functions in cardiac muscle, smooth muscle, & glands.

- Open Na+ channels
  - Na+ enters cell
  - Cell depolarized (AP forms)
  - Excitation
  - EPSP
  - Muscle contracts
  - ↓ Heart rate

- Open K+ channels
  - K+ leaves cell
  - Cell repolarized (rests)
  - Inhibition
  - IPSP
  - ↑ GI tract activity

- Open Na+ channels
  - Na+ enters cell
  - Cell depolarized (AP forms)
  - Excitation
  - EPSP
1) Receptor Types:

2) Neurotransmitter Removal Systems:

4 Systems:

i) Diffusion = neurotransmitter dissipates out of synapse

ii) Enzyme Breakdown = an enzyme breaks down neurotransmitter into it’s smaller, inactive parts.
   - Ex. ACh-E = acetylcholinesterase = enzyme that breaks down ACh in synapse

   MAO = monoamineoxidase = enzyme that breaks down dopamine, serotonin, epinephrine

iii) Glial removal = removal by astrocytes in CNS.

iv) Reuptake = presynaptic neuron takes back neurotransmitter from synapse (back to vesicles).
   Ex. Prozac, Lexapro, Citalopram are SSRI’s.

   SSRI = Selective serotonin reuptake inhibitor
   \[ \uparrow \text{serotonin levels} \]

Ex. Enzyme breakdown of neurotransmitter:

Acetylcholinesterase (ACh-E) = enzyme that breaks down ACh in synapses. (Ex. Between somatic motor neurons & skeletal muscles)
Ex. Of ACh release into synapse, binding to receptor on a cell & opening Na+ channel, then breakdown of ACh by ACh-E

Review

– Neurotransmitters @ synapse
  • EPSPs & IPSPs are different from APs
  • Graded potential (can undergo summation)
  • No thresh-holds or refractory period

– 2 Ways neurotransmitters regulated:
  • Receptor types (nicotinic & muscarinic)
  • Enzyme removal systems
## 4. Types and Functions of Neurotransmitters

<table>
<thead>
<tr>
<th>Types and Functions</th>
<th>CNS Neurotransmitters</th>
<th>PNS Neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Choline-derived:</td>
<td>- ACh is + stim.</td>
<td>- ACh is + autonomic Parasympathetic regul.</td>
</tr>
<tr>
<td>II. Mono-amine derived (catecholamines):</td>
<td>- norepinephrine (+)</td>
<td>- epinephrine (autonomic Sympathetic regul.)</td>
</tr>
<tr>
<td></td>
<td>- dopamine</td>
<td>- is + or -</td>
</tr>
<tr>
<td></td>
<td>- Serotonin (10% receptors in brain)</td>
<td>Serotonin (90% receptors in intestines)</td>
</tr>
<tr>
<td>III. “Other” amino acid derived:</td>
<td>- Glutamate (+)</td>
<td>- nitric oxide (NO)</td>
</tr>
<tr>
<td></td>
<td>- Glycine (-)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- GABA (-) (gamma amino butyric acid)</td>
<td></td>
</tr>
<tr>
<td>IV. Soluble gas:</td>
<td>- nitric oxide (NO)</td>
<td>- nitric oxide (NO)</td>
</tr>
</tbody>
</table>

### I. Acetylcholine (ACh)

- In both CNS & PNS
- Both excitatory (EPSPs) and inhibitory (IPSPs) – depending on ion channel
- Enzyme breakdown by acetylcholinesterase (ACh-E)

- **Involves 2 types cholinergic receptors:**
  1. **Nicotinic cholinergic receptor**
     - ACh released @ neuromuscular junction (between somatic motor neuron & skeletal muscle cells)
     - excitatory only (EPSPs), opens \( \text{Na}^+ \) channels
     - causes skeletal muscles to ____________.

  2. **Muscarinic cholinergic receptor**
# Acetylcholine (ACh)

- In both CNS & PNS
- Both excitatory (EPSPs) and inhibitory (IPSPs) – depending on ion channel
- Enzyme breakdown by acetylcholinesterase (ACh-E)

## Involves 2 types cholinergic receptors:

1) Nicotinic cholinergic receptor
   - ACh released @ neuromuscular junction (between somatic motor neuron & skeletal muscle cells)
   - excitatory only (EPSPs), opens ___________ channels
   - causes skeletal muscles to ____________________.

2) Muscarinic cholinergic receptor
   - for autonomic parasympathetic regulation of smooth muscles, cardiac muscle, and glands.
   - inhibitory (IPSPs) on cardiac muscle if _____ or _______ channels open.
     - (Ex. ↓ heart rate & contractile strength)
   - stimulatory (EPSPs) in GI smooth muscle & glands if _____ or _______ channels open.
     - (↑ GI activity)

## Drugs/agents that influence activity of a neurotransmitter:

**Agonist** = substance that can increase the levels or activity of a neurotransmitter, or even its receptor.

**Antagonist** = substance that can decrease the levels or activity of a neurotransmitter, or its receptor.
I. Inhibition of enzyme ACh breakdown.

A) Acetylcholinesterase inhibitor (ACh-EI)
- inhibits enzymatic ACh breakdown,
- ACh builds up in synapse with muscles
- causes “cholinergic syndrome”

Question: Is an ACh-EI an ACh **AGONIST?** OR **ANTAGONIST?**

Ex. 1: Organophosphate pesticides are ACh-EI’s
- Malathion – mosquito control
- Carbamate – general insecticide
- Chlordiprifos (dursban) – used in flea & tick meds
  (banned in USA, 2001) **DO NOT USE!!!**

Ex. 2: Non-organophosphate pesticide = Pyrethrins
  (from Chrysanthemum plant)
- Likely an ACh-EI in in **cats** (DO NOT USE!!!), but not dogs

0.20% Pyrethrins

HELL NO!!!
I. Acetylcholine (ACh) - inhibition of enzyme breakdown.

Ex. 3: Sarin gas (biological weapon - nerve gas) are ACh-EI's
– Clinical App Pg 113 AND online

Sarin attack in subways:
Tokyo, Japan 1995

2012 – Syria threatening use of sarin chemical warfare against rebels.

“Clinical Presentation” of someone cholinergic syndrome =

Mnemonic for cholinergic syndrome:

DUMBBELSS - stands for
Diarrhea
Urination
Miosis (constricted pupils)
Brady cardia \[\downarrow HR\]
Bronchoconstriction
Excitation (muscle twitches)
Lacrimation tears
Salivation
Sweating
Treatment for cholinergic syndrome from ACh-EI exposure:

To reverse cholinergic syndrome:

**Pralidoxime (2-PAM)** is the cure for cholinergic syndrome – it stops phosphorylation of ACh-E.

will restore ACh-E

To Treat symptoms:

**Atropine** *(Physiology in Health & Disease Pg 119 and online)*
- ACh antagonist
- blocks ACh muscarinic cholinergic receptors on heart & smooth muscles.

**Question: is Atropine an ACh agonist or antagonist?**

**Valium (benzodiazepine)** Clinical App Pg 114 and online
Works by stimulating GABA inhibition of muscle activity (keeps muscles relaxed).

**Ques: is Valium a GABA agonist or antagonist?**

Toxins that are ACh Agonists:
Clinical App Pg 110
AND online

**A. Tetanus** = toxin produced by **Clostridium tetani**
(found on rusty metal – puncture wound)
- is an ACh agonist
- promotes muscle tetany (”spastic paralysis” OR “hypertonia”)
- **trismus**, or lockjaw
- also a Glycine and GABA antagonist (prevents muscle relaxation).

- prevent w/booster of **tetanus vaccine** every 10 yrs
- suspect exposure, give shot of **tetanus antitoxin**
Toxins that are ACh Antagonists: Clinical Applications online

Clinical “presentation” of someone w/ ACh insufficiency =

A. Botulism = toxin produced by Clostridium
> Prevents ACh from leaving presynaptic vesicles
  (no ACh no skeletal muscle contractions!)
> Causes flaccid paralysis or hypotonia (is an ACh antagonist)

B. Paralytic shellfish poisoning (online)
> Shellfish harvested during red tide have “saxitoxin”
> Blocks ACh nicotinic cholinergic channels (prevent Na+ entry)
> Prevents skeletal muscle contraction
> flaccid paralysis or hypotonia (is an ACh antagonist)

C. Pufferfish poisoning (online)
> Fugu fish have “tetrodotoxin”.
> Blocks ACh nicotinic cholinergic channels (prevent Na+ entry)
> Prevents skeletal muscle contraction
> flaccid paralysis or hypotonia (is an ACh antagonist)

“Other Disorders” of ACh insufficiency a.k.a. ACh antagonists):

Myasthenia gravis AND online
> Autoimmune destruction of ACh receptors.
> Reduced muscle function, weakness, pharyngeal swallowing problems.

Alzheimer’s disease
> loss of ACh producing neurons in brain.
> Excess glutamate production in brain (glutamate toxicity - online)
> memory problems.
> Treatment involves ACh agonists and glutamate antagonists.
Review (added slide)

- Types of Neurotransmitters
  - ACh
  - Nicotinic cholinergic receptors
  - Muscarinic cholinergic receptors
  - ACh-E
  - ACh-EI’s and cholinergic syndrome
  - Toxins as ACh agonists
  - Toxins as ACh antagonists
  - ACh signaling problems with Alzheimer's and myasthenia gravis

End of Exam Material

II. Monoamine Neurotransmitters

Catecholamines
  - dopamine, norepinephrine & epinephrine (all made from tyrosine)

Serotonin - made from tryptophan

Regulated by:
1) Reuptake – primarily with serotonin

QUES: What are SSRI’s?

2) Enzyme breakdown by monoamine oxidase (MAO) – breaks down dopamine, norepinephrine & epinephrine
If have low levels of dopamine, serotonin, or norepinephrine can treat with MAO-I’s to buildup monoamines in synapse.

MAO-I = monoamine oxidase inhibitor (or a monoamine agonist)

\[ \text{MAO-I A} \rightarrow \text{agonist to norepinephrine & serotonin} \]

\[ \text{MAO-I B} \rightarrow \text{agonist to dopamine} \]

**II. Monoamine Neurotransmitters**

**Serotonin**

- 10% of serotonin receptors in brain
  - regulates memory, moods, emotions, behavior, & hallucinations

- 90% of serotonin receptors in intestines (regulates appetite)

- Insufficient serotonin – associated with depression & obesity

**QUES:**
What can you give to build up serotonin in synapses?

\[ \text{SSRI} \rightarrow \text{MAO-I A} \]
II. Monoamine Neurotransmitters

Dopamine

> Produced by substantia nigra neurons in midbrain (of brainstem)

2 functions:
1) fine motor control (nigrostantial dopamine system)
   > Insufficient dopamine – Parkinson’s – Clinical App Pg 134
   > Muscle tremors – Neuromuscular disorder

   > Excess dopamine – “Schizophrenia”

2) emotional reward system (mesolimbic dopamine system)
   “addiction”

Cocaine, Dopamine, & Addiction (Clinical App Pg 118 & 159)

AND online

Cocaine is an agonist to dopamine, serotonin, and norepinephrine (excess amount of these)

Presentation reflects this:
- Hallucinations (too much serotonin)
- Muscle tremors and addiction (too much dopamine)
- High energy, fight or flight. (too much epinephrine)
II. Monoamine Neurotransmitters

**Norepinephrine/epinephrine**

> In PNS for autonomic sympathetic regulation (fight/flight)
  - ↑ heart and respiratory rates, ↓ activity GI tract smooth muscles

> In CNS for general arousal (stimulatory)

Works by 2 types G-protein coupled receptors (Table 6.3)

1) alpha adrenergic receptors (α-adrenergic)
- inhibit smooth muscles & glands of GI tract (slow GI activity).
- vasoconstrict skin and GI tract vessels.

2) beta adrenergic receptors (β-adrenergic)
  i. β1-adrenergic receptor = to increase heart rate.
  ii. β2-adrenergic receptor =
    - brochodilate airways
    - vasodilate arteries to skeletal muscles.

II. Monoamine Neurotransmitters

Table 6.3

<table>
<thead>
<tr>
<th>Organ</th>
<th>Adrenergic Effects of Sympathoadrenal System</th>
<th>Adrenergic Receptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye</td>
<td>Contraction of radial fibers of the iris dilates the pupils</td>
<td>α1-</td>
</tr>
<tr>
<td>Heart</td>
<td>Increase in heart rate and contraction strength</td>
<td>β1, primarily</td>
</tr>
<tr>
<td>Skin and GI arteries</td>
<td>Arterioles contract due to smooth muscle contraction</td>
<td>α1-</td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>Arterioles contract due to sympathetic nerve activity</td>
<td>α1-</td>
</tr>
<tr>
<td>Lungs</td>
<td>Bronchioles (airways) dilate due to smooth muscle relaxation</td>
<td>β2-</td>
</tr>
<tr>
<td>Stomach and intestines</td>
<td>Contraction of smooth muscles stimulates secretion of food</td>
<td>α2-</td>
</tr>
<tr>
<td>Glycogenolysis and secretion of glucose</td>
<td></td>
<td>α1, β2</td>
</tr>
</tbody>
</table>

III. “Other” Amino Acid (NOT monoamine) Neurotransmitters:

1. **Glutamate** (a.k.a glutamic acid)
   - Excitatory (stimulant)
   - found in MSG (monosodium glutamate)
   - Stimulatory (EPSPs in 80 – 90% CNS synapses
   - regulated by glial cell removal (astrocytes)
   - excess glutamate (*glutamate “toxicity”*) associated w/Alzheimer’s (also influenced by ACh)
   & Parkinson’s (also influence by dopamine)

2. **Glycine** – inhibitory in spinal cord.
3. **GABA** – inhibitory in brain

III. “Other” Amino Acid (NOT monoamine) Neurotransmitters:

**Glycine**

“*Serene like glycine in the spinal cord.*”

- Inhibitory (IPSPs) by opening Cl- channels
- Primarily in spinal cord
- Coordinates muscle movement by regulating antagonistic muscle contraction & relaxation (Ex. biceps brachii & triceps brachii)

**Strychnine poisoning** - inhibits glycine relaxation of diaphragm. Diaphragm stays tense, can’t exhale. Die from asphyxiation.
III. “Other” Amino Acid (NOT monoamine) Neurotransmitters:

GABA
- Inhibitory (IPSPs) by opening Cl- channels
- Found primarily in brain synapses (90%)
- Coordinates muscle movement in cerebellum (fine motor control and “muscle memory” patterns)
- Keeps skeletal muscles relaxed in deep (REM) sleep
- Insufficient GABA associated w/ Huntington's disease (autosomal dominant genetic disorder).

QUESTION:
Why is benzodiazepam (Valium) a treatment for Huntington's disease or cholinergic syndrome??

IV. Gaseous Neurotransmitters:

Nitric Oxide (NO)

see my writing assignment example online:

Sexual arousal stimulates parasympathetic response
> causes NO production
> NO activates G protein, guanylate cyclase, & cGMP 2nd messenger
> cGMP causes vasodilation in penile arterioles
> Corpus cavernosa fills w/blood = erection.

Stimulation wanes: enzyme breakdown.
> Phosphodiesterase = enzyme that breaks down cGMP and stop vasodilation

Erectile dysfunction drugs (Viagra, Cialis, Levitra) work by:
- Increasing NO production
- Phosphodiesterase inhibitor (= cGMP agonist)
Review (added slide)

– Types of Neurotransmitters
  • Monoamines
    > Dopamine
    > Serotonin
    > Epinephrine (adrenalin)
      - Autonomic sympathic effects on the body during fight/flight
      - alpha (α) adrenergic receptors
      - Beta 1 (β1) adrenergic receptors
      - Beta 2 (β2) adrenergic receptors
    > Drugs that affect adrenergic receptors