1. Different Types of Neurons and Neuron Anatomy

**Anatomy REVIEW!**

1. **Neurons**
   
   a) **Sensory (afferent) neurons** = neurons that pick up sensory info and transmit it UP the spinal cord to brain for interpretation.
   
   b) **Motor (efferent) neurons** = neurons that carry a motor command from the brain DOWN the spinal cord to an effector (muscle cell or gland).

2. **Interneurons (in CNS)** = strictly in CNS. Relays info. between spinal cord (CNS) & PNS.

3. **Neuroglial (Glial) Cells** = specialized cells that have distinct functions, and help neurons.
1. Different Types of Neurons and Neuron Anatomy

**Fig 4.3**

Central Nervous System (CNS)

Peripheral Nervous System (PNS)

**Sensory info ascends spinal cord**

**Sensory neurons have a dorsal “ganglion”**

**Motor info descends spinal cord**

**Sensory (afferent) neuron**

**Somatic Motor neuron**

**Autonomic Motor neuron**

**Interneuron**

---

1. Different Types of Neurons and Neuron Anatomy

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

**Ependymal cells** = glial cells that are found within choroid plexus of brain ventricles and produce cerebrospinal fluid (CSF)

**Astrocytes** = glial cells in the pia mater that make up the blood brain barrier, which restricts entry of some things to brain, but allows other small permeable substances to diffuse into brain.

**Schwan cells (PNS) & Oligodendrocytes (CNS)** = cells that make the myelin sheath around axons of neurons in white matter of the brain.

**Microglia** = glial cells that function as the brain’s clean-up crew and migrate through phagocytizing pathogens or dead/damaged tissue.
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.  
  
**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.

\[
\text{unmylenated axon} = 0.5 \text{ m/sec} \quad \text{VS} \quad \text{mylenated axon} = 100 \text{ m/sec}!
\]

\[
\text{Nodes of Ranvier} = \text{gaps between myelin sheaths where signal jumps to next node (faster conduction)}
\]

---

**Multiple Sclerosis – Clinical App Pg 102 and 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.

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**Review**

- Types of neural tissue
  - Neurons (sensory/afferent, motor/efferent, and interneurons)
  - Neuroglial (Glial) cells
    - CNS – astrocytes, microglia, ependymal cells, oligodendrocytes
    - PNS – schwann 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.

Click image for a YouTube video
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” = *deals with amount of neurotrans.*
    - The more neurotransmitter = the greater the cell response
    - The less neurotransmitter = the weaker the cell response
  
  - EPSP can produce “summation” = *Repeated* (high frequency)
    - the higher the frequency of the stimulation = the greater the cell response
    - the lower the frequency of stimulation = the greater the cell response

**Example:** the increase in heart rate with epinephrine binding to its **Beta-1 (B1) 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 **muscarnic 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)

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1) Receptor types:

A. Nicotinic (ion-gated) receptor
- For \( \text{ACh} \) neurotransmitter
  - Binding of receptor by ACh causes \( \text{Na}^+ \) channels to open.
  - \( \text{Na}^+ \) channel opens causes \underline{st}imulation or \underline{A}P \underline{Po}r 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 actives and enzyme then a G-protein
   - G-protein then opens ion channels.
     IF Na+ and Ca+2 channel opens = _______________________________
     IF K+ or Cl- channel opens = _______________________________
   - 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

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

**Nicotinic Cholinergic Receptor**

**Muscarinic Cholinergic Receptor**

#### TABLE 6.4: Effects of Acetylcholine (ACh) in the PNS

<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 neurons</td>
<td>Skeletal muscles</td>
<td>Nicotinic</td>
<td>Depolarization, producing action potentials</td>
<td>Muscle contraction</td>
</tr>
<tr>
<td>Preganglionic neurons of ANS</td>
<td>Autonomic ganglia</td>
<td>Nicotinic</td>
<td>Depolarization, producing action potentials</td>
<td>Stimulates preganglionic neurons of the ANS</td>
</tr>
<tr>
<td>Postganglionic 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>Postganglionic parasympathetic neurons</td>
<td>Heart</td>
<td>Muscarinic</td>
<td>Hyperpolarization, slowing the rate of automatic production of action potentials</td>
<td>Slowing of heart rate; production of GI tract activity</td>
</tr>
</tbody>
</table>

**ACh & Nicotinic Cholinergic Receptors**

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

**ACh & Muscarinic Cholinergic Receptors**

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

Open Na+ channels
Na+ enters cell
Cell depolarized (AP forms)

Open K+ channels
K+ leaves cell
Cell repolarized (rests)

Open Na+ channels
Na+ enters cell
Cell depolarized (AP forms)

↓ Heart rate
GI tract activity

↑ Heart rate
GI tract activity
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 its smaller, inactive parts.
   - Ex. ACh-E = *acetylcholinesterase* breaks down Ach & J Ach in synapse
   - MAO = *monoamine oxidase* (dopamine, serotonin, epinephrine)

iii) **Glial removal** = removal by astrocytes in CNS.

iv) **Reuptake** = presynaptic neuron takes back neurotransmitter from synapse (back to vesicles).
   - Ex. Serotonin removed this way.
   - SSRI = selective serotonin reuptake inhibitory
   - Ex. Prozac, Lexapro, Citalopram are SSRI’s.

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

Click image for a YouTube video

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**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>+ stimulatory</th>
<th>CNS neurotransmitters</th>
<th>PNS neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td>- inhibitory</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I. Choline-derived:</td>
<td>ACh is +</td>
<td>- ACh</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- is + or −</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.&quot;Other&quot; amino acid derived:</td>
<td>- Glutamate (+)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Glycine (-)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- GABA (-)</td>
<td></td>
</tr>
<tr>
<td>(gamma amino butyric acid)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV. Soluble gas:</td>
<td>- nitric oxide (NO)</td>
<td></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 $\sqrt{Na^+}$ channels
     - causes skeletal muscles to contract.

  2) Muscarinic cholinergic receptor
### 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 __________ 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.

**Blocker**

**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
- Chlropyris (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

*Added slide!*

0.20% Pyrethrins

**OK to use**

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:

**DUMBELSS** - stands for
- Diarrhea
- Urination
- Miosis (constricted pupils)
- Bradycardia
- Bronchoconstriction
- Excitation (muscle twitches)
- Lacrimation
- 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.

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 botulinum*
   - 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** (Clinical App Pg 113)
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.
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? Drugs that are serotonin agonist work best for ↑ serotonin

2) Enzyme breakdown by monoamine oxidase (MAO) – breaks down dopamine, norepinephrine & epinephrine

II. Monoamine Neurotransmitters – inhibiting enzyme breakdown.

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)
2 types MAO-I’s:
   - MAO-I A - agonist to norepinephrine & serotonin
   - MAO-I B – agonist to dopamine

Dope Beat

Read Physiology in Health & Disease Pg 119 and online for MAO-I’s
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?

SSRIs, MAO-I

*Read Physiology in Health & Disease Pg 119 for SSRI’s*

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 and online
   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 =
    - bronchodilate airways
    - vasodilate arteries to skeletal muscles.
### 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

---

### 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 creates the pupils</td>
<td>α1, β1</td>
</tr>
<tr>
<td>Heart</td>
<td>Increase in heart rate and contraction strength</td>
<td>β1, primarily</td>
</tr>
<tr>
<td>Skin and visceral vessels</td>
<td>Arterioles constrict due to smooth muscle contraction</td>
<td>α1, β1</td>
</tr>
<tr>
<td>Arterioles constrict due to sympathetic nerve activity</td>
<td>α1, β1</td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>Arterioles dilate due to hormone epinephrine</td>
<td>β2, β1</td>
</tr>
<tr>
<td>Lungs</td>
<td>Bronchioles (airways) dilate due to smooth muscle relaxation</td>
<td>α1, β1</td>
</tr>
<tr>
<td>Stomach and intestine</td>
<td>Contraction of sphincters slows passage of food</td>
<td>α1, β1</td>
</tr>
<tr>
<td>Liver</td>
<td>Gluconeogenesis and secretion of glucose</td>
<td>α1, β1</td>
</tr>
</tbody>
</table>

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

**Glycine**

- 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)
  * in deep sleep, inhibits skeletal muscle & brain awareness
- 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)

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**Review**

- Types of Neurotransmitters
  - ACh
  - Monoamines (Dopamine, serotonin, norepinephrine)
  - Amino acid-based (glutamate, glycine, GABA)
  - Nitric Oxide
- Poisons that affect ACh
- Disorders of ACh system
- Disorders of other neurotransmitter systems