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 different neurotransmitters.
4. Become familiar with influence of disease & drugs on neurotransmitter signaling.

1. Different Types of Neurons and Neuron Anatomy

Anatomy REVIEW! See Pg 56, 73-74 of Wiki physiology book

1. Neurons
   a) Sensory (afferent) neurons = neurons (receptors) that pick up sensory info. and transmit it to an integrating center.

   b) Motor (efferent) neurons = neurons that transmit a motor command from brain to muscle cells or glands.

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

3. Neuroglial (Glial) Cells = neural cells with special functions
1. Different Types of Neurons and Neuron Anatomy

**Sensory (afferent) neuron**

Sensory info ascends spinal cord

Sensory neurons have a dorsal “ganglion”

Motor info descends spinal cord

Somatic Motor neuron

Autonomic Motor neuron

Interneuron

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

- **Ependymal cells** = cells within the choroid plexus of brain ventricles, which produce cerebral spinal fluid (CSF) to circulate within the ventricles.

- **Astrocytes** = glial cells that function as part of the blood brain barrier.

- **Schwan cells (PNS) & Oligodendrocytes (CNS)** = cells that produce a myelin sheath to cover the axon of neurons in either the PNS or CAN. Schwann cells in PNS, oligodendrocytes in CNS

- **Microglia** = cells which function as the brain’s clean up crew. They are phagocytic cells that engulf pathogens or dead cells to destroy them.
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. \[ \text{unmylenated axon} = 0.5 \text{ m/sec} \text{ VS mylenated axon} = 100 \text{ m/sec}! \]
- **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** (Clinical App [online](#) & Pg 74 Wiki book)

= 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 or Ca+2 channel** in membrane, Na+ or Ca+2 floods into cell causing action potential (AP) or **depolarization**, which stimulates a cell.

2. **K+ channels or Cl- open**, causes **repolarization**, which inhibits cells.

Pg 57 in Wiki physiology textbook

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**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.

Click [HERE](#) for YouTube video of how neurotransmitter release stimulates a post-synaptic cell.
Ion channels and cell stimulation or inhibition

IN GENERAL:

If a neurotransmitter binds to a receptor on a cell and it opens sodium (Na+) or calcium (Ca+2) ion channels it will stimulate a cell (cause action potential to form, or depolarize a cell).

- Sodium is always Stimulatory.
- Calcium makes cells Crazy!

If a neurotransmitter binds to a receptor on a cell and it opens potassium (K+) or chloride (Cl-) ion channels, it will inhibit a cell (inhibit action potential or repolarize a cell).

- Potassium (K+) helps cells Kick back and relax.
- Chloride helps cells stay Calm.

Pg 57 in Wiki Physiology textbook

Neurotransmitter binding to receptor opens stimulatory 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” =
  More neurotransmitter released = greater cell response

Less neurotransmitter released = lesser cell response

- EPSP can produce “summation” = Repeated (high frequency)
  More frequent cell stimulation = greater cell response

Less frequent cell stimulation = lesser cell response

Example: the increase in heart rate with epinephrine binding to its Beta-1 andrenergic receptor on heart muscle cells is due to opening of Na+ and Ca+2 channels!
Neurotransmitter binding to receptor opens **inhibitory** 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”
- EPSP can produce “summation”

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 **acetylcholine (ACh)** binding to its **muscarinic cholinergic** receptors on heart muscle is due to opening of K+ channels!

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**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.
        *(Think of this. If you smoke, and I hope you don’t, the cigarette contains nicotine. [Think nicotinic and nicotine]. You must use voluntary muscle movement to pick up the cigarette to bring it to your lips.)*

     B. **Muscarinic (G-protein coupled) receptor** – for autonomic control of glands, smooth muscle, & cardiac muscle. *(Muscarinic means YOU MUST!)*

2) **Removal systems for neurotransmitters** (4 removal systems)
1) Receptor types:

A. Nicotinic (Ion-gated) receptor on skeletal muscles
   - For ACh neurotransmitter
   - Binding of receptor by ACh causes Na+ ion channels to open
   - Na+ channels opening causes stimulation of a cell (muscle cells contract)

Skeletal muscle cells have nicotinic cholinergic receptors for ACh, which open Na+ channels for voluntary muscle contraction.

B. Muscarinic (G-protein coupled) receptor for cardiac muscle, smooth muscle, or gland cells:
   - Neurotransmitter binding to cell receptor activates a G-protein
   - G-protein then opens ion channels.
     - IF Na+ and Ca2+ channel opens = cell is stimulated (muscle cell contracts, gland cells secrete)
     - IF K+ or Cl- channel opens = cell is inhibited (muscle cells stop contracting, gland cells stop secreting)
   - For ACh, norepinephrine & epinephrine, & other neurotransmitters
   - **Muscarinic receptors are for involuntary actions (heart muscle, smooth muscle, and gland cells **MUST** respond.**
Ex. Nicotinic cholinergic (ACh) receptors

Fig 4.25

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

Ex. Muscarinic adrenergic (epinephrine, norepinephrine) receptors

Fig. 4.30

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? _____________________________
**Ex. Muscarinic GABA receptor**

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

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**For ACh and its receptors:**

**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 (voluntary) motor neurons</td>
<td>Skeletal muscles</td>
<td>Nicotinic cholinergic</td>
<td>Depolarization, producing action potentials</td>
<td>Muscle contraction</td>
</tr>
<tr>
<td>Parasympathetic (involuntary) motor neurons</td>
<td>Smooth muscles, glands</td>
<td>Muscarinic cholinergic</td>
<td>Depolarization, producing action potentials</td>
<td>Contraction of smooth muscles; secretion of glands</td>
</tr>
<tr>
<td>Parasympathetic (involuntary) motor neurons</td>
<td>Heart</td>
<td>Muscarinic cholinergic</td>
<td>Hyperpolarization, slowing the rate of automatic production of action potentials</td>
<td>Slowing of heart rate</td>
</tr>
</tbody>
</table>
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 monoamine neurotransmitters in synapse. Includes 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. Pg 65 & 75 Wiki physiology text
Ex. Enzyme breakdown of neurotransmitter:

**Acetylcholinesterase (ACh-E)** = enzyme that breaks down ACh in synapses. (Ex. Between somatic motor neurons & skeletal muscles)

Click **HERE** for YouTube video 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>+ stimulatory</th>
<th>CNS neurotransmitters</th>
<th>PNS neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td>- inhibitory</td>
<td>- ACh is +</td>
<td>- ACh</td>
</tr>
<tr>
<td>I. Choline-derived:</td>
<td>- norepinephrine (+)</td>
<td>- autonomic Parasympathetic 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>- epinephrine (autonomic Sympathetic regul.)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- is + or –</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Serotonin (90% receptors in intestines)</td>
</tr>
<tr>
<td>II. Mono-amine derived (catecholamines):</td>
<td>- Glutamate (+)</td>
<td></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>III.“Other” amino acid derived:</td>
<td>- nitric oxide (NO)</td>
<td></td>
</tr>
<tr>
<td>IV. Soluble gas:</td>
<td></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 Na+ channels on skeletal muscles.
     - causes skeletal muscles to contract.
  2) Muscarinic cholinergic receptor

   - for autonomic parasympathetic regulation of smooth muscles, cardiac muscle, and glands. They MUST respond.
   - inhibitory (IPSPs) on cardiac muscle if K+ or Cl- channels open.
     (Ex. ↓ heart rate & contractile strength)
   - stimulatory (EPSPs) in GI smooth muscle & glands if Na+ or Ca^{2+} 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
- Chlorpyrifos (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

I. Acetylcholine (ACh) - inhibition of enzyme breakdown.

Ex. 3: Sarin gas (biological weapon - nerve gas) are ACh-EI’s
- Clinical App 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)
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 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 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.

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**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
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  
(*think A for Agitation or anxiety, and A for Appetite*)

MAO-I B – agonist to dopamine  
(*think B for Dope Beat*)

See supplemental reading [online for MAO-I’s](#)

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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?
## 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 and [online](#) Neuromuscular disorder
   
   * > Excess dopamine - “Schizophrenia”

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

### Cocaine, Dopamine, & Addiction (Clinical App [online](#) & Pg 76 – 77 Wiki Physiology text)

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.

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**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 visceral vessels</td>
<td>Arterioles constrict due to smooth muscle contraction</td>
<td>α₂</td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>Arterioles dilate due to hormone epinephrine</td>
<td>β₂</td>
</tr>
<tr>
<td>Lungs</td>
<td>Bronchioles (airways) dilate due to smooth muscle relaxation</td>
<td>β₂</td>
</tr>
<tr>
<td>Stomach and intestine</td>
<td>Contraction of sphincters slows passage of food</td>
<td>α₁</td>
</tr>
<tr>
<td>Liver</td>
<td>Glycogenolysis and secretion of glucose</td>
<td>α₂, β₂</td>
</tr>
</tbody>
</table>

See [Clinical App ONLINE](#): Beta blockers.

**B1 agonist** = Dobutamine

↑ HR and cardiac output

good for heart failure patients)

**B1 & B2 agonist** = isoproteronol

↑ HR and cardiac output & Bronchodilate

**B2 agonist** = Albuterol & Terbutaline

Bronchodilates

good for people w/respiratory prob.

**B1 & B2 blocker** = Propranolol

↓ HR and BP & bronchoconstrict

good for hypertension BUT not people w/respiratory prob. (it will cause bronchoconstriction!)

**B1-specific blocker** = Atenolol

↓ HR and BP

no effect on bronchioles

good for hypertension WITH respiratory problems (won’t cause bronchoconstriction)
### 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 influenced 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)
- 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?*

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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

- 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