Ch 4: 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.

1. Different Types of Neurons and Neuron Anatomy

Anatomy REVIEW!

1. Neurons
   a) Sensory (afferent) neurons = bring sensory info up spinal cord to sensory cortex
   b) Motor (efferent) neurons = bring motor commands down (from motor cortex) spinal cord.

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

3. Neuroglial (Glial) Cells = helper cells in CNS & PNS
1. Different Types of Neurons and Neuron Anatomy

**Fig 4.3**

Central Nervous System (CNS)

- Sensory (afferent) neuron
- Somatic Motor neuron
- Interneuron
- Autonomic Motor neuron

**Sensory info ascends spinal cord**

- Sensory neurons have a dorsal “ganglion”

**Motor info descends spinal cord**

Peripheral Nervous System (PNS)

- Receptors
- Skeletal muscles
- Smooth muscle
- Cardiac muscle
- Glands

- Autonomic ganglion

1. Different Types of Neurons and Neuron Anatomy

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

<table>
<thead>
<tr>
<th>CSF</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ependymal cells</strong> = make CSF in brain ventricles</td>
</tr>
</tbody>
</table>

| **Astrocytes** = function in blood brain barrier (associated w/ pia mater) |

| **Schwan cells (PNS) & Oligodendrocytes (CNS)** = make myelin sheaths around axon of a neuron |

| **Microglia** = brain phagocytes that seek out & destroy pathogens (a.k.a. clean-up crew) |
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. *unmylenated axon = 0.5 m/sec VS mylenated axon = 100 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 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.
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” = amount of neurotrans.
  - Small amount = weak cell response
  - Large amount = strong cell response

- EPSP can produce “summation” = Repeated (high frequency)
  - Low Frequency of stim = weak cell response
  - High Frequency of stim = strong 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 muscarinic cholinergic receptors on heart muscle is due to opening of K+ channels!

↓ HR.
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 _______ ACh ____ neurotransmitter
   
   - Binding of receptor by ACh causes _______ Na+ _______ 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
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 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

Fig 4.25

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?  

Ex. Muscarinic GABA receptor

Is this neurotransmitter and receptor going to have an EPSP or IPSP response?
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 motor</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 postganglionic 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</td>
</tr>
</tbody>
</table>

ACh & Nicotinic Cholinergic Receptors

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

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

ACh & Muscarinic Cholinergic Receptors

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

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

↓ Heart rate

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

↑ 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 it’s smaller, inactive parts.
   - Ex. ACh-E = \textit{acetylcholinesterase} breaks down Ach
   - MAO = \textit{monoamine oxidase} breaks down \textit{dopamine} and \textit{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 = \textit{Selective serotonin reuptake inhibitors}.

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**Ex. Enzyme breakdown of neurotransmitter:**

\textbf{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

End for Exam 2
### 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><strong>I. Choline-derived:</strong></td>
<td>- ACh is +</td>
<td>- ACh - autonomic Parasympathetic regul. - is + or –</td>
</tr>
<tr>
<td><strong>II. Mono-amine derived (catecholamines):</strong></td>
<td>- norepinephrine (+)</td>
<td>- epinephrine (autonomic Sympathetic regul.) - is + or –</td>
</tr>
<tr>
<td></td>
<td>- dopamine</td>
<td>Serotonin (90% receptors in intestines)</td>
</tr>
<tr>
<td></td>
<td>- Serotonin (10% receptors in brain)</td>
<td></td>
</tr>
<tr>
<td><strong>III. “Other” amino acid derived:</strong></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><strong>IV. Soluble gas:</strong></td>
<td>- nitric oxide (NO)</td>
<td>( \checkmark )</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 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 sympathetic or 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
- Chlorpyrifos (dursban) – used in flea & tick meds (banned in USA, 2001)  **DO NOT USE!!!**

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

**Ex. 2: Sarin gas (biological weapon - nerve gas) are ACh-EI’s**
- Clinical App Pg 113 AND [online](#)

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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** = low H.R.
- **Bronchoconstriction**
- **Excitation** (muscle twitches) = seizures
- **Lacrimation** = tearing
- **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. **and some nicotinic cholinergic receptors**

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 Agonists: Clinical Applications online

Clinical “presentation” of someone w/ACh insufficiency = blocker

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.

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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

**QUEST:** What are SSRI's?

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

MAO-I’s are agonists to these neurotrans. 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 (tx for Parkinson’s)

II. Monoamine Neurotransmitters – inhibiting enzyme breakdown.

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

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? - SSRI’s or - MAO-I A

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

\[ T_X = MAO - S \]

> Excess dopamine - “Schizophrenia”

\[ T_X = \text{dopamine antagonist (blocker)} \]

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)**
   - **β1-adrenergic receptor** = to increase heart rate.
   - **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>( \alpha_1 )</td>
</tr>
<tr>
<td>Heart</td>
<td>Increase in heart rate and contraction strength</td>
<td>( \beta_1 ) primarily</td>
</tr>
<tr>
<td>Skin and visceral vessels</td>
<td>Arterioles constrict due to smooth muscle contraction</td>
<td>( \alpha_1 )</td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>Arterioles constrict due to sympathetic nerve activity</td>
<td>( \alpha_1 )</td>
</tr>
<tr>
<td>Lungs</td>
<td>Bronchioles (airways) dilate due to smooth muscle relaxation</td>
<td>( \beta_2 )</td>
</tr>
<tr>
<td>Stomach and intestine</td>
<td>Contraction of sphincters slows passage of food</td>
<td>( \alpha_2 )</td>
</tr>
<tr>
<td>Liver</td>
<td>Glycogenolysis and secretion of glucose</td>
<td>( \alpha_1, \beta_1 )</td>
</tr>
</tbody>
</table>

**Remember??**
- B1 & B2 blocker = **propanolol**
- B1-specific blocker = **atenolol**
- B1 & B2 agonist = **isoproterenol**
- B1 agonist = **dobutamine**
- B2 agonist = **albuterol**

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**Adrenergic antagonists & agonists**

See Clinical Apps online
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

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

<table>
<thead>
<tr>
<th>Glycine</th>
<th>“Serene like glycine in the spinal cord.”</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Inhibitory (IPSPs) by opening Cl- channels</td>
<td></td>
</tr>
<tr>
<td>- Primarily in spinal cord</td>
<td></td>
</tr>
<tr>
<td>- Coordinates muscle movement by regulating antagonistic muscle contraction &amp; relaxation (Ex. biceps brachii &amp; triceps brachii)</td>
<td></td>
</tr>
</tbody>
</table>

**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??

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

Side effect = priapism = erection lasting > 3 hrs.

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