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 = pick up info, ascend spinal cord to brain cortexes to be interpreted
   b) Motor (efferent) neurons = deliver motor command from brain, descends down spinal cord to effectors (muscle cells or glands).
2. Interneurons (in CNS) = strictly in CNS. Relays info. between spinal cord (CNS) & PNS.
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**

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

- **Ependymal cells** = found in choroid plexus of ventricles.  
  
  *Make CSF*

- **Astrocytes** = found in blood brain barrier (Pia mater)  
  
  *(cell body)*

- **Schwan cells (PNS) & Oligodendrocytes (CNS)** = make myelin sheath around axon of white matter neurons.

- **Microglia** = brain macrophages  
  
  *(Find & destroy 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. \( [\text{unmyelened axon} = 0.5 \text{ m/sec VS mylenated axon} = 100 \text{ m/sec!}] \)
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 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.

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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.
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” =
  \[
  \text{large amt neurotrans} \rightarrow \text{large response} \\
  \text{small amt} \rightarrow \text{small response}
  \]

- EPSP can produce “summation” = Repeated (high frequency)
  \[
  \text{high freq stim} \rightarrow \text{large response} \\
  \text{low freq} \rightarrow \text{small response}
  \]

**Example:** the increase in heart rate with epinephrine binding to its \( \beta \) 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 \( \text{cholinergic} \) 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 __________________________ 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
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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

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

Fig 4.25
Ex. Muscarinic adrenergic (epinephrine, norepinephrine) receptors

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

Fig. 4.30

Ex. Muscarinic GABA receptor

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

Fig. 4.27
For ACh and its receptors:

<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>Postganglionic neurons</td>
<td>Autonomic ganglia</td>
<td>Nicotinic</td>
<td>Depolarization, producing action potentials</td>
<td>Stimulates postganglionic potentials</td>
</tr>
<tr>
<td>Prostaganglionic, parasympathetic</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>Prostaganglionic, parasympathetic</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

**ACh & Muscarinic Cholinergic Receptors**

Produces autonomic parasympathetic (rest and digest) functions in cardica 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)
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 = \( \text{Acetylcholinesterase} \) = enzyme breaks down ACh
   - MAO = monoamine oxidase = enzyme breaks down monoamines (dopamine **
   epinephrine **
   Serotonin *)

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

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>CNS neurotransmitters</th>
<th>PNS neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I. Choline-derived:</strong></td>
<td><strong>- ACh is +</strong></td>
</tr>
<tr>
<td><strong>- ACh</strong></td>
<td><strong>- autonomic Parasympathetic regul.</strong></td>
</tr>
<tr>
<td><strong>- Epinephrine (autonomic Sympathetic regul.)</strong></td>
<td><strong>- is + or -</strong></td>
</tr>
<tr>
<td><strong>- noradrenaline</strong></td>
<td><strong>- norepinephrine (+)</strong></td>
</tr>
<tr>
<td><strong>- dopamine</strong></td>
<td><strong>- Serotonin (10% receptors in brain)</strong></td>
</tr>
<tr>
<td><strong>- Glutamate (+)</strong></td>
<td><strong>- Serotonin (90% receptors in intestines)</strong></td>
</tr>
<tr>
<td><strong>- Glycine (-)</strong></td>
<td><strong>- GABA (-)</strong></td>
</tr>
<tr>
<td><strong>- GABA (-)</strong></td>
<td><strong>(gamma amino butyric acid)</strong></td>
</tr>
<tr>
<td><strong>- nitric oxide (NO)</strong></td>
<td><strong>- nitric oxide (NO)</strong></td>
</tr>
</tbody>
</table>

| **II. Mono-amine derived (catecholamines):** | **- Adrenaline** |
| **- norepinephrine (+)** | **- dopamine** |
| **- Serotonin (10% receptors in brain)** | **- Epinephrine (autonomic Sympathetic regul.)** |
| **- is + or -** | **- is + or -** |
| **- Serotonin (90% receptors in intestines)** | **- Serotonin (90% receptors in intestines)** |

| **III. “Other” amino acid derived:** | **- Glutamate (+)** |
| **- Glycine (-)** | **- GABA (-)** |
| **- GABA (-)** | **(gamma amino butyric acid)** |

| **IV. Soluble gas:** | **NO** |
| **- nitric oxide (NO)** | **- nitric oxide (NO)** |

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### 1. Acetylcholine (ACh)

- **Acetylcholine always stimulates**
- 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) **Muscarnic 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)

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**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

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 ______________________
(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 ________________
   > 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? Are serotonin agonist*

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

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### 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

*anxiety, appetite, mild drowsiness*

*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?

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

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II. Monoamine Neurotransmitters

Dopamine

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

**2 functions:**
1) fine motor control (nigrostantial dopamine system)
   - Insufficient dopamine
     \[ \text{Tx} = \text{dopamine agonist} \]
     *Parkinson’s – Clinical App Pg 134 and online*  
     *Neuromuscular disorder*
   - Excess dopamine
     \[ \text{Tx} = \text{dopamine antagonist} \]
     “Schizophrenia”

2) emotional reward system (mesolimbic dopamine system)
   “addiction”
Cocaine, Dopamine, & Addiction (Clinical App Pg 118 & 159)

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

2) **β-adrenergic receptors (β-adrenergic)**
   i. **β1-adrenergic receptor** = to increase heart rate.
   ii. **β2-adrenergic receptor** =
      - brochodilate airways
      - vasodilate arteries to skeletal muscles.
Table 6.3
Selected Adrenergic Effects in Different Organs

<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 mucosal vessels</td>
<td>Arterioles constrict due to smooth muscle relaxation</td>
<td>$\alpha_1$, $\beta_1$</td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>Arterioles dilate due to sympathetic nerve activity</td>
<td>$\alpha_1$</td>
</tr>
<tr>
<td>Lungs</td>
<td>Bronchioles (alveoli) 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>$\alpha_1$, $\beta_2$</td>
</tr>
<tr>
<td>Liver</td>
<td>Glycogenolysis and secretion of glucose</td>
<td>$\alpha_1$, $\beta_2$</td>
</tr>
</tbody>
</table>


Adrenergic antagonists & agonists
See Clinical Apps online

Remember??

- B1 & B2 blocker = propranolol
  - ↓HR ↓BP
- B2 specific blocker = atenolol
  - ↓HR ↓BP
  - won’t affect bronchioles
- B1 & B2 agonist = isoprotenerol
  - ↑HR, ↑BP
  - bronchodilation
- B1 agonist = dobutamine
  - ↑HR, ↑BP
  - (makes the heart beat faster)
- B2 agonist = albuterol
  - bronchodilation
  - (for asthma)

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

![Serene in spinal cord](image)

![Beat time in brain](image)
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.

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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

**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