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 (Glia) 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 neuron**

- Motor info descends spinal cord

**Motor neuron**

- Sensory (afferent) neuron

**Somatic Motor neuron**

- Interneuron

**Autonomic Motor neuron**

---

### 5 Types of Glial Cells

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

2. **Astrocytes**
   - Glial cells that function as part of the blood-brain barrier.

3. **Microglia**
   - Cells which function as the brain's clean up crew. They are phagocytic cells that engulf pathogens or dead cells to destroy them.

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

5. **Satellite cells**
   - Glial cells that cover neurons in PNS (including sensory neurons & neurons of sympathetic & parasympathetic systems).
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 in 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.

**Receptor with ion channel** = on post-synaptic cell. 

*Binding of receptor opens ion channel & ions flood into cell, causing AP.*

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

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

---

**Review of Fri Lecture**

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

**Anatomy of a neuron**
- Cell body, dendrites, axon, myelin sheath, nodes, synaptic knobs, synapse, secretory vesicles, neurotransmitters, pre-synaptic neurons, post-synaptic neurons.

*Neurons communicate with post-synaptic cells by secreting neurotransmitters, which bind to receptor on post-synaptic cell and open up ion channels.*
2. How Neurons Communicate with Cells (Mon Lecture)

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

Pg 57 in Wiki physiology textbook

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. If K\(^+\) channels or Cl\(^-\) open, causes "repolarization" or rest, which inhibits cells.

[Diagram showing Na\(^+\) channel]

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](#) on the PDF copy of this powerpoint for an excellent YouTube video of how neurotransmitter can either stimulates a post-synaptic cell or inhibit it.
Ion channels and cell stimulation or inhibition

Pg 57 in Wiki Physiology textbook

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.

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!

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.

1) Receptor types:

**A. Nicotinic (Ion-gated) receptor**
- 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)

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

**B. Muscarinic (G-protein coupled) receptor for cardiac muscle, smooth muscle, or gland cells:**
- Neurotransmitter binding to cell receptor activates a G-protein
- then opens ion channels.
  - IF Na+ and Ca+2 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

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? **EPSP (causes stimulation)**

Ex. Muscarinic adrenergic (epinephrine, norepinephrine) receptors

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? **EPSP (causes stimulation)**
Ex. Muscarinic GABA receptor

Is this neurotransmitter and receptor going to have an EPSP or IPSP response? __IPSP (causes stimulation)_____
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.

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 = monoamine oxidase (Enzyme that breaks down monoamine neurotransmitters in synapse. Includes dopamine, serotonin, epinephrine)

Click HERE for YouTube video of ACh removal by ACh-E

iii) Glial removal = removal by astrocytes in CNS.

v) 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](#) on the PDF copy of the powerpoint for a YouTube video of ACh release into synapse, binding to receptor on a cell & opening Na+ channel, then breakdown of ACh by ACh-E
Announcement:
UC gym north closed Fri Sep 18th

That’s when Exam 1 was scheduled.

It is rescheduled for Mon Sep 21st.

Review of Mon Lecture

Neurotransmitters @ synapse
- Neurotransmitter released at synapse & binds to receptor on post-synaptic cell.
- If that receptor opens Na+ or Ca+2 channels, it causes an EPSP (cell is stimulated)
- If that receptor opens K+ or Cl- channels, it causes an IPSP (cell inhibited or rests)
- EPSPs can have: Graded potential or summation

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

**Wed Lecture**

<table>
<thead>
<tr>
<th>+ stimulatory</th>
<th>CNS neurotransmitters</th>
<th>PNS neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td>─ inhibitory</td>
<td>ACh is + in CNS</td>
<td>ACh - autonomic Parasympathetic regulation if PNS s + or ─</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>I. Choline-derived:</th>
<th>II. Mono-amine derived (catecholamines):</th>
<th>III. “Other” amino acid derived:</th>
<th>IV. Soluble gas:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>norepinephrine (+)</td>
<td>Glutamate (+) stimulates brain</td>
<td>nitric oxide (NO)</td>
</tr>
<tr>
<td></td>
<td>dopamine</td>
<td>Glycine (-)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serotonin (10% receptors in brain)</td>
<td>GABA (-) (gamma amino butyric acid)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>nitric oxide (NO)</td>
</tr>
</tbody>
</table>

### I. Acetylcholine (ACh)

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

- **Involves 2 types cholinergic receptors:**
  
  1) **Nicotinic cholinergic receptor**
  - ACh released @ neuromuscular junction (between somatic motor neuron & skeletal muscle cells)
  - excitatory only (EPSPs), opens Na+ channels on skeletal muscles.
  - causes skeletal muscles to contract.
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 ACh or epinephrine)
     - 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 Ca²⁺ 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

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

**Ex. 3: Sarin gas (biological weapon - nerve gas) are ACh-EI's**

Sarin attack in subways:
Tokyo, Japan 1995
12 people died, 5,000 injured.

2012 – Syrian government threatening use of sarin chemical warfare against rebels.

2013 – Attack happened by rockets to surface delivery.
3,600 hospitalized patients displayed neurotoxic effects attributed to Sarin gas.

Estimated 281 – 1,729 deaths by neurotoxicity.

“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
Fri Announcement:

• Don’t forget to print out Lab handout & bring to next week’s lab on Osmosis & Diffusion.

• Also, print out and bring Study Outlines and Blank flow diagrams to lab, because after lab we are having a review period for Exam 1.

• Come with any questions you have on Ch 2, parts 1 & 2, and Ch 4, part 1!

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. Stops symptoms of DUMBBELSS

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). Stops skeletal muscle seizures

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.

**Question**: What drug could you give a patient with low ACh (like Alzheimer’s), or have a loss of ACh receptors (like Myasthenia gravis), to improve their functioning and help their symptoms?

**An Acetylcholinesterase inhibitor**!

Ex. For Alzheimer’s: Galantamine, Rivastigmine, Donepezil

Ex. For myasthenia gravis: Neostigmine & Rivastigmine
Review of Wed Lecture (updated 9/10/20)

– Types of Neurotransmitters
  • ACh signaling
    – Voluntary skeletal muscle contraction
    – Involuntary actions on heart and smooth muscle
    – Removal of ACh by ACh-E
    – Cholinergic syndrome (DUMBBELLS) and its treatment
    – Chemicals & Toxins that are ACh agonists (organophosphate pesticides, Permethrin insecticides, sarin gas, and tetanus toxin)
    – Toxins that are ACh antagonists (botulism toxin, saxitoxin, tetrodotoxin)
    – Disorders of ACh signaling in Alzheimers and Myasthenia gravis

II. Monoamine Neurotransmitters

Fri Lecture
Catecholamines
> dopamine, norepinephrine & epinephrine (all made from tyrosine)

Dopamine – plays role in fine motor control And reward for pleasurable activities (also Plays role in psychological addictions)

Serotonin - made from tryptophan. 10% receptors in CNS, 90% receptors in GI system. Plays role in memory, moods, emotions.

Regulated by:
1) Reuptake – primarily with serotonin

    QUES: What are SSRI's? = selective serotonin reuptake inhibitor

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

---

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? S.S.R.I. or M.A.O.I-A

**QUES:**
WHY would there be multiple drugs to treat the same problem???

*not every patient responds to same drug*
II. Monoamine Neurotransmitters

**Dopamine**

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

2 functions:

1) fine motor control (nigrostancial dopamine system)
   > Insufficient dopamine - Parkinson’s – Clinical App 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 (a.k.a noradrenaline & adrenaline)

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

> Norepinephrine 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

Table 6.3

<table>
<thead>
<tr>
<th>Adrenergic Effects of Sympathoadrenal System</th>
<th>Adrenergic Receptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye</td>
<td>α</td>
</tr>
<tr>
<td>Heart</td>
<td>β primarily</td>
</tr>
<tr>
<td>Skin visceral vessels</td>
<td>α1</td>
</tr>
<tr>
<td>Skeletal muscle vessels</td>
<td>α2</td>
</tr>
<tr>
<td>Lungs</td>
<td>β2</td>
</tr>
<tr>
<td>Stomach &amp; intestine</td>
<td>α</td>
</tr>
<tr>
<td>Liver</td>
<td>α1, β2</td>
</tr>
</tbody>
</table>

See Clinical App ONLINE: Beta agonists & 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 influence by dopamine)

2. **Glycine** – inhibitory in spinal cord.

3. **GABA** – inhibitory in brain

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

**Glycine**

“serene like glycine in the spinal cord.”

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

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

**GABA**

- Inhibitory (IPSPs) by opening Cl- channels

- Found primarily in brain synapses (90%) - Coordinates muscle movement in cerebellum (fine motor control and “muscle memory” patterns) — this is why Xanax works to calm panic attacks. Xanax is a GABA agonist in the brain.

- 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)**

Click [HERE](#) to see my writing 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 by Phosphodiesterase (enzyme that breaks down cGMP and stop vasodilation).

Erectile dysfunction drugs like Viagra, Cialis, Levitra work as phosphodiesterase inhibitors. So, these drugs are cGMP agonists
Review of Fri Lecture

– Other Neurotransmitters
  • Monoamines (Dopamine, serotonin, norepinephrine)
    – Their functions & disorders
    – Removal of serotonin by reuptake
    – Low serotonin treated with SSARs
    – Removal of dopamine serotonin, norepinephrine by MAO.
    – Low dopamine or serotonin treated with MAO-I’s
  • Amino acid-based (glutamate, glycine, GABA)
  • Nitric Oxide, cGMP, phosphodiesterase, and ED drugs