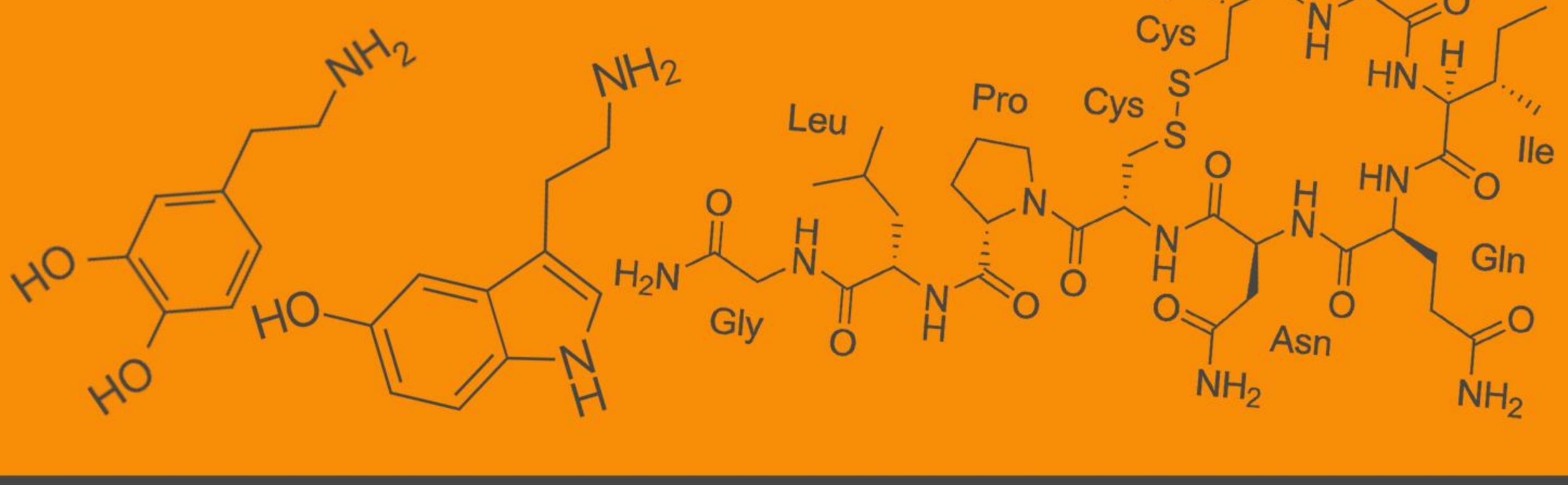

MEDICINE438's CNSPHYSIOLOGY

LECTURE II: Synapse and Receptors

EDITING FILE GSLIDES

IMPORTANT EXTRA LECTURER'S NOTES

1 SYNAPSE AND RECEPTORS

Lecture Two

OBJECTIVES

- Definition and Functions of synapses.
- Structure and Types of synapses: anatomical & functional.
- Synaptic transmission & neurotransmitters.
- What neurotransmitters are, and how they are released and act on their receptors.
- Fate of neurotransmitters.
- Differentiate between neurotransmitter receptors (ionotropic and metabotropic).
- Electrical events at synapses (EPSPs & IPSPs) and the differentiation.
- Properties of synaptic transmission.
- Factors affecting synaptic transmission.

BOX 2-1: GUYTON AND HALL

- A *neurotransmitter* is a chemical substance released from a nerve ending and acts on receptor proteins to elicit a response.
- The neuron releasing the neurotransmitter is called a presynaptic neuron, and its axon terminal from which the neurotransmitters are released is called a “presynaptic terminal”.
- The neuron receiving the neurotransmitter is called postsynaptic neuron, and the part that receives the neurotransmitter on which the receptor resides is called a “postsynaptic terminal”.
- Most neurotransmitters are stored after synthesis in synaptic vesicles in the presynaptic terminal, and are triggered for release by calcium influx which binds release sites to trigger the exocytosis of vesicles.
- The *Intercellular Fluid*, ICF, is slightly more negative than the *Extracellular Fluid*, ECF, this small difference in negativity, which is approximately -90 mV, drives the action potential of excitable tissues.
- Between any two points in space with different electrical charges there exists what is termed an *electric potential* or *voltage* for either repulsion or attraction.
- A negative voltage describes a surplus of electrons with respect to a neutral point in space, and a positive voltage describes a deficiency of electrons also with respect to a neutral point in space, that is for example, when we describe a large nerve fiber with having -90 mV of potential we mean that there is a higher surplus of negative charges in the neuron relative to our neutral point, the ECF, outside the neuron.
- Therefore this negative voltage field tends to attract positive charges. In theory, we could also refer to the ECF of having +90 mV with respect to the ICF (in our example, a nerve fiber), however the consensus is to refer to the ECF as our neutral point in space, and to describe the voltage inside the cell to be negative relative to the outside.
- Potassium is more concentrated within the cell than outside of it, the cell allows potassium to diffuse to the ECF, down its concentration gradient by channels such as sodium/potassium leak channels , and is pumped back into the cell by Na/K pump.
- Potassium diffuses out of the cell until a potential of -94 mV is reached within a cell, this is called the **Nernst potential**. *Which is the potential measured within the cell relative to the outside that stops the net diffusion of an ion.* Since potassium is a positive ion it would make sense that a high negative potential within the cell “a negative voltage field” will pull it back into the cell. Therefore if the resting membrane potential only depended on potassium it will be equal to -94 mV.
- Sodium’s Nernst potential is, calculated at approximately +61 mV. So if the cell only depended on sodium for its membrane potential with zero permeability to all other ions, then the cell will have a resting membrane potential of +61 mV. The cell membrane is less permeable to sodium than potassium, which is why the resting membrane potential is farther from sodium’s Nernst potential, the cell is less permeable to sodium in fact decreasing sodium’s ECF concentration will have little effect on resting membrane potential.

STRUCTURE OF A SYNAPSE

1. Synaptic knobs (presynaptic terminal): It has synaptic vesicles (neurotransmitters).
2. Synaptic cleft: It is the space between the axon terminal and sarcolemma. It has a width of 200–300 angstroms[1].
3. Postsynaptic membrane: It has receptors for neurotransmitters or ion channels.

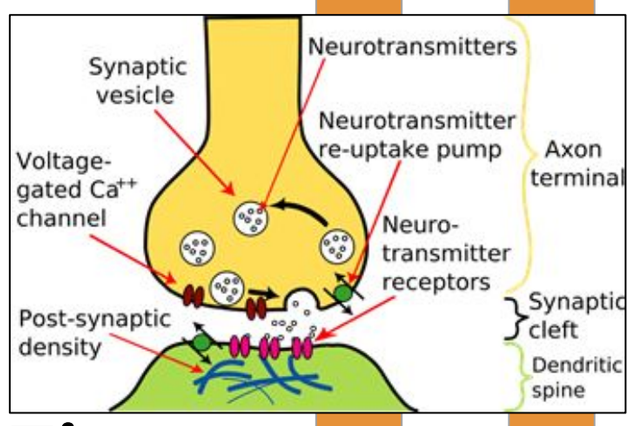

Figure 2-1

GENERAL FACTS ABOUT SYNAPSES

- Definition: It is a junction where the axon or some other portion of one cell **(presynaptic cell)** terminates on the *dendrites*, *soma*, or *axon* of another neuron **(postsynaptic cell).**
- A connection between a neuron and a second cell.
- The **CNS** contains more than 100 billion neurons. The brain has 86 billion neurons.
- Some **CNS** neurons receive 20,000 *synapses*.
- Synaptic input is converted to a nerve impulse (AP) at the *Axon hillock*[2].
- The output signal (AP) travels by way of a single axon leaving the neuron.
- "SYNAPTEIN",from the Greek "syn-" ("together") and "haptein" ("to clasp").

FUNCTIONS OF A SYNAPSE

- In the **CNS**, this other cell is also a neuron.
- In the **PNS**, the other cell may be either a neuron or an effector cell e.g; gland or muscle.
- The synapses determine the directions that the *nervous signals* will spread through the nervous system.
- The synapses perform a selective action, often blocking weak signals while allowing strong signals to pass.

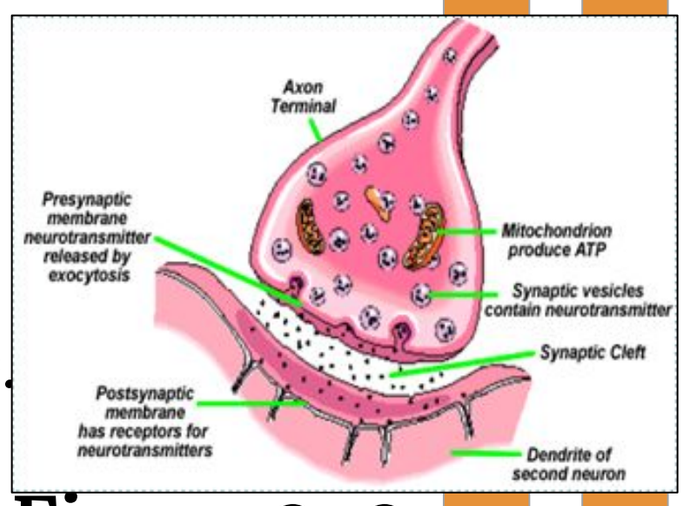

Figure 2-2

FOOTNOTES

1. Equivalent of 10^{-10} m, not an SI unit.
2. The part of a neuron's cell body that connects it to an axon.

ANATOMICAL TYPES OF SYNAPSES

Dendrosomatic
(dendrites to soma)

Dendrodendritic
(dendrite to dendrite)

Axoaxonic
(axon to axon)

Axosomatic
Synapses between the axon of one neuron and the soma of another.

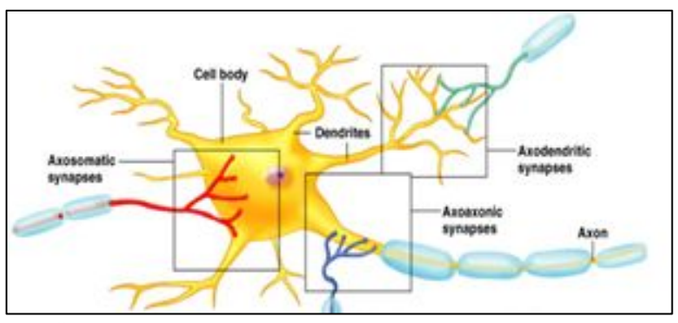

Figure 2-3

Axodendritic
Synapses between the axon of one neuron and the dendrite of another.

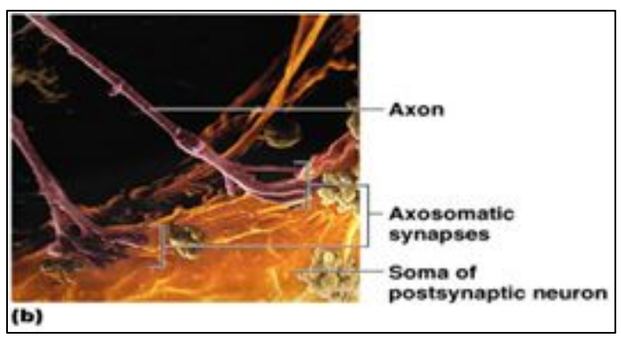

Figure 2-4

Table 2-1 Showing types of synapses according to their anatomical structure.

FUNCTIONAL TYPES OF SYNAPSES

1. CHEMICAL SYNAPSE

Almost all synapses in the CNS. (I.e. first neuron secretes a chemical substance called neurotransmitter at the synapse to act on receptor on the next neuron to excite it, inhibit or modify its sensitivity).

- One direction transmission.
- NTs are released from synaptic vesicles.
- Vesicles fuse with axon membrane and NT released by exocytosis.
- Amount of NTs released depends upon frequency of AP.
- Terminal bouton is separated from postsynaptic cell by synaptic cleft. (<u>20- to 40-nanometer</u>

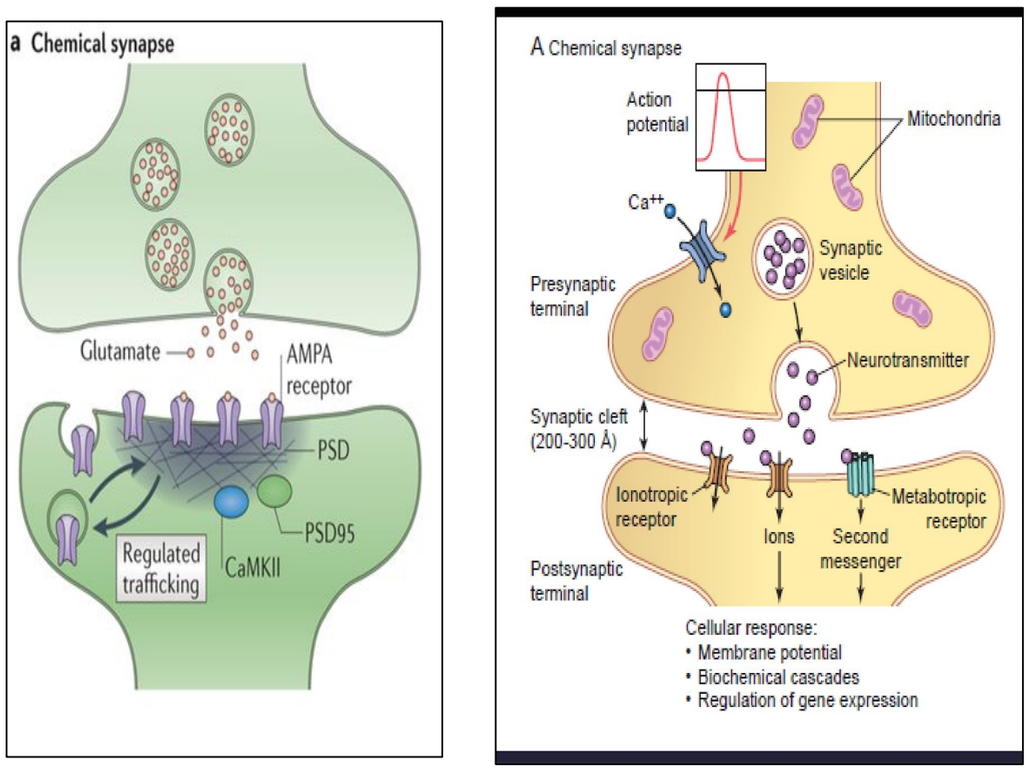

Figure 2-5

2. ELECTRICAL SYNAPSE

Membranes of the pre- and postsynaptic neurons come close together and gap junctions form → low membrane borders which allow passage of ions.

- Are less common than chemical synapses
- Correspond to gap junctions found in other cell types.
- The **bidirectional** transmission of electrical synapses permits them to help coordinate the activities of large groups of interconnected neurons.
- Promotes synchronous firing of a group of interconnected neurons. For example in: <u>mental attention, emotions and memory, arousal from sleep.</u>
- Electrical synapses are present throughout the CNS, smooth and cardiac muscles, brain, and glial cells.

GAP JUNCTIONS

- Adjacent cells electrically coupled through a channel.
- Each gap junction is composed of 12 connexin[1] proteins.

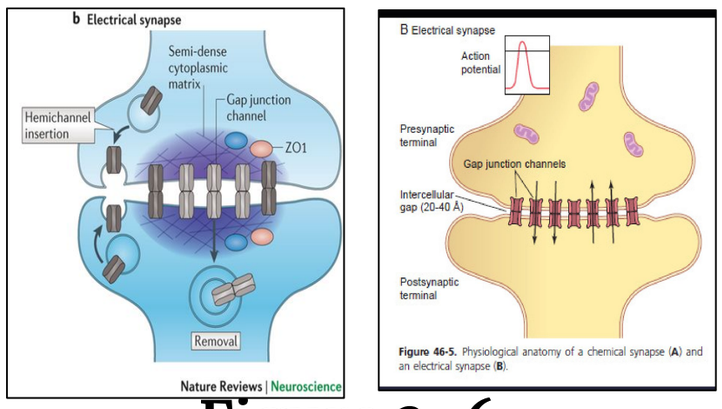

Figure 2-6

3. CONJOINT SYNAPSE

Both electrical and chemical. Examples: neurons in <u>lateral vestibular nucleus.</u>

Table 2-2

EXAMPLES OF SYNAPSE OUTSIDE THE CNS:

1. Neuromuscular junction
2. Contact between: autonomic neurons & smooth, cardiac muscles, & other effector cells.

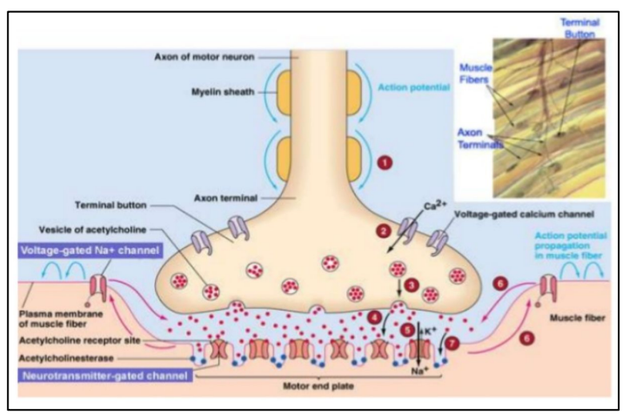

Figure 2-7

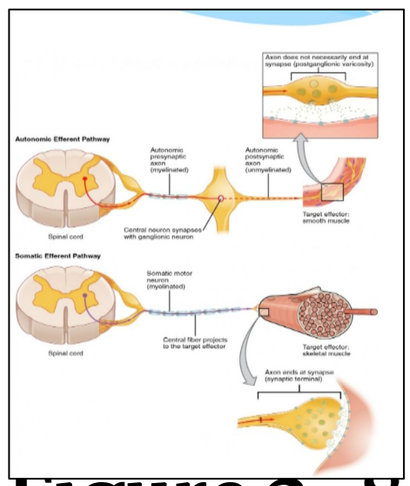

Figure 2-8

FOOTNOTES

1. Gap junctions are formed by membrane pores called connexons, which are made proteins called connexins, connexins permits substances to pass between cells without leaking into the ECF.

MECHANISM OF SYNAPTIC TRANSMISSION

1. Action potential leading to opening of voltage-gated calcium channels.
2. $Ca2+$[1] enters bouton down concentration gradient.
3. Ca2+ activates calmodulin, which activates protein kinase. Protein kinase[1] aid in the fusion of synaptic vesicles[3].
4. Inward diffusion triggers rapid fusion of synaptic vesicles with the presynaptic terminal membrane and release of NTs
5. *Neurotransmitte*r release at docking site and diffuse across synaptic cleft.
6. Binding to postsynaptic receptors (**inhibition** or **excitation**) of the postsynaptic membrane (Depending on the type of the neurotransmitter, i.e. excitatory or inhibitory).
7. Information is transmitted in the central nervous system mainly in the form of nerve action potentials, called *Nerve impulses*, through a succession of neurons, one after another.

- NT release is rapid because many vesicles form fusion-complexes at "docking site."[4]

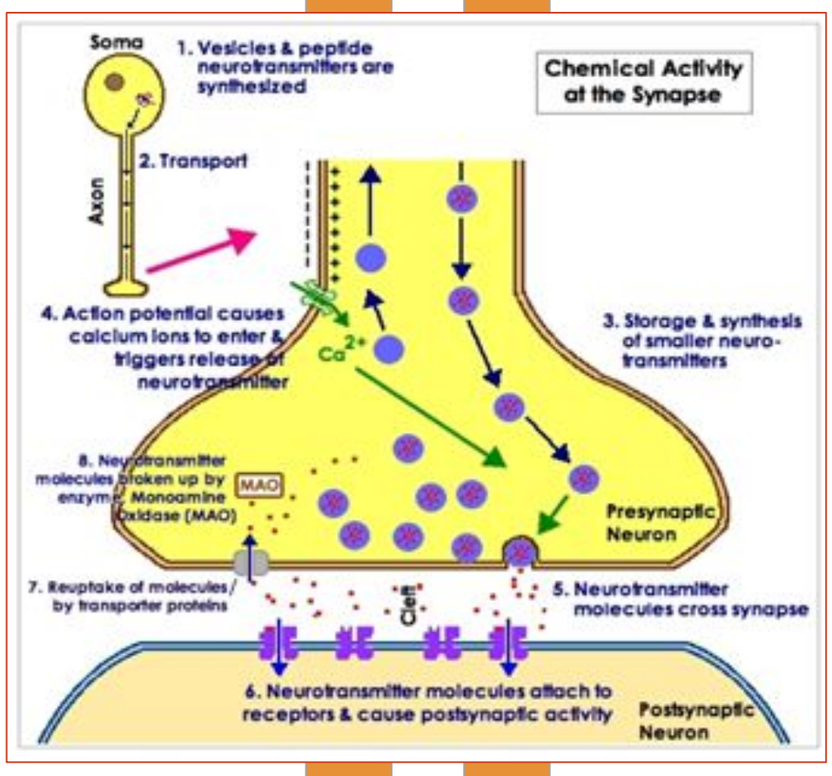

Figure 2-9

FATE OF NEUROTRANSMITTERS

After a transmitter substance is released at a synapse, it must be removed by:

1. Diffusion out of synaptic cleft into surrounding fluid.
2. Enzymatic destruction e.g Ach esterase for Ach.
3. Active transport back into presynaptic terminal itself e.g norepinephrine.

FOOTNOTES

1. Calcium first binds with modulin to form calmodulin, calmodulin acts as a messenger protein to mediate and modify calcium's interactions within the cell.
2. Protein kinase: a protein that modifies other molecules, mostly other proteins, in order to activate or inactivate their metabolic activities.
3. Guyton and Hall: Nitric oxide acts as an exception here, it is not performed and stored synaptic vesicles. Instead it is synthesized and released as needed.
4. Docking sites are sites present on the presynaptic membrane that vesicles "dock" in, following docking the vesicle fuses with the presynaptic membrane, and the neurotransmitter is released. Vesicles are sometimes recycled.

INTERACTIONS AND PHYSIOLOGICAL EFFECTS OF NEUROTRANSMITTERS

Transmitter Substance acts on the Postsynaptic Neuron via "*Receptor Proteins*", these receptors have two components.

COMPONENTS OF RECEPTOR PROTEINS

BINDING SITE

that faces the synaptic cleft to bind the neurotransmitter.

IONOPHORE

It passes all the way through the membrane to the interior.

ION CHANNELS

(IONOTROPIC RECEPTORS)

Anion channels

- Cl- mainly.
- Opening of Cl- channels → diffusion of negative charges into the membrane → decreased membrane potential making it more negative away from threshold level (-) neuron.

Cation channels

- Na+ (most common), K+, Ca++.
- Opening of Na+ channels → an increased membrane potential in positive direction toward threshold level of excitation (+) neuron.

2ND MESSENGER SYSTEM

(METABOTROPIC RECEPTORS)

- Not an ion channel but a molecule that protrudes into the cell cytoplasm and activates one or more substances inside the postsynaptic neuron.
- In the postsynaptic neuron, this mechanism is important where prolonged postsynaptic changes are needed to stay for days, months.. years (memory).
- Effects : intracellular Enzymes activation, gene transcription, etc…

Table 2-3 Showing receptor protein types and how they exert their physiological influence.

NEUROTRANSMITTER ACTIONS

- Neurotransmitter receptors that directly gate ion channels are often called *ionotropic* receptors, whereas those that act through second messenger systems are called *metabotropic* receptors.
- A NT may activate both *Ionotropic* and *Metabotropic* receptors to produce both fast and slow postsynaptic potentials as the same synapse.

Lecturer's notes: Which of the following functions contain conjoint synapse?

A. Vomiting.
B. Sleeping
C. **Balance and equilibrium (the main function of the vestibular system is to maintain equilibrium.)**

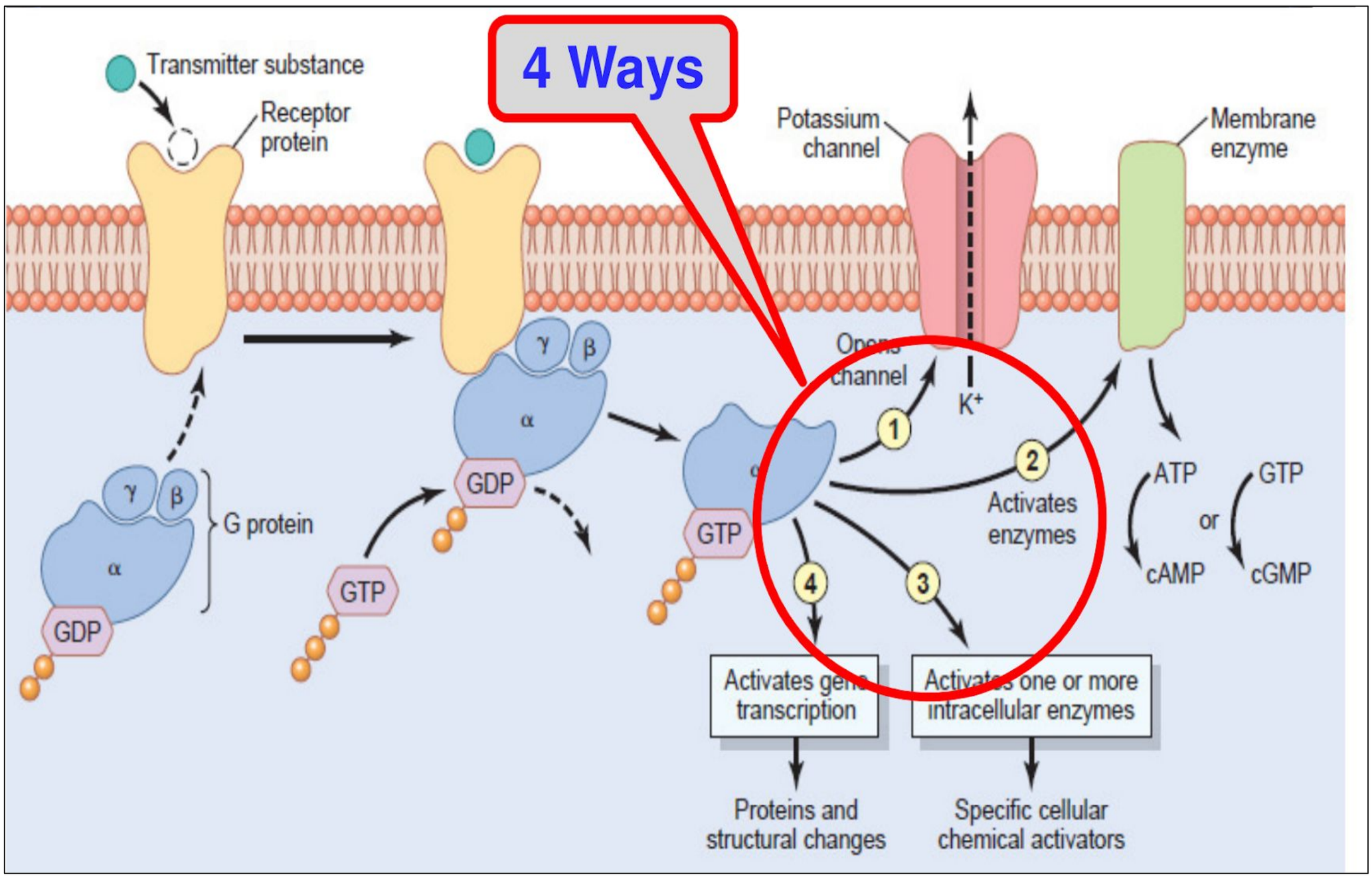

Figure 2-10 2nd messenger system acts in four ways.

BOX 2-2: GUYTON AND HALL

2) *A second messenger system*, the most famous and notable example are **G-protein coupled receptors**. This type of ionophores often helps in achieving physiological effects that last for months or years, in contrast with ionptropic ion channels which remain open for a very brief amount of time.

For example ACh binds for only a brief period of time before it is hydrolyzed by acetylcholinesterases. A 2nd messenger system can activate a cascade that leads to:
(1) opening of ion channel, but in contrast with a ligand-gated ion channel, this one lasts for a prolonged period. (2) Activation of cAMP or cGMP, which can initiate a lot of intracellular functions. (3) Activation of one or or more intracellular enzymes. (4) Activation of gene transcription, which causes the formation of new proteins within the cell thus expanding its physiological significance.

The G-protein contains three subunits alpha the activatory subunit, beta and gamma. The binding of a neurotransmitter produces a conformational change in the G-protein and the alpha subunit detaches and can do one of the following four things mentioned above.

Lecturer's notes: Fastest response occurs by opening of 2nd messenger ion channels, and the slowest and most important is activation of gene transcription since it results in formation of new proteins.

IONOTROPIC	METABOTROPIC
Mediate rapid PSPs	Mediate slower PSPs
Duration of PSPs is 10-30 ms or less.	Duration from 100's ms to minutes or longer.
PSPs (EPSP or IPSP) develop within 1-2 ms after an AP reaching the presynaptic terminal.	There is slowness due to activation of second messengers leading to opening Ion channels.

Table 2-4

BOX 2-3: GUYTON AND HALL

EPSP: The initial positive increase of resting membrane potential to a less negative value, if strong enough it can elicit an action potential.
IPSP: An increase in negativity beyond the cell's resting membrane potential.

BOX 2-4: GUYTON AND HALL

A discharge of a single presynaptic terminal can never increase the membrane potential enough to elicit an action potential. Instead, a simultaneous discharge of 40 to 80 presynaptic neurons is needed at the same time and in rapid succession. The simultaneous discharges will summate, this process is called *summation*, and it allows occurance of APs.

ELECTRICAL EVENTS IN POSTSYNAPTIC NEURONS

1. RESTING MEMBRANE POTENTIAL OF NEURONAL SOMA

- -65 mV i.e. less negative than skeletal muscles (-70 to -90 mV).
- If the voltage is less negative the neuron is excitable.

2. EXCITATORY POSTSYNAPTIC POTENTIALS (EPSPs) (BOX 2-4)

- When excitatory neurotransmitters binds to its receptor on postsynaptic membrane a **partial depolarization** occurs **(increase Na influx)** of postsynaptic cell membrane
- immediately under presynaptic ending, i.e. EPSPs.
- If this potential rises enough to threshold level an AP will develop and excite the neuron.
- This summation *(Refer to* BOX 2-4*)* will cause the membrane potential to increase from -65 mV to -45mV.
- EPSPs = +20mV (an increase strong enough to open enough voltage-gated sodium channels) which makes the membrane reach the firing level and an AP develops at axon hillock.
- **How EPSPs differ from Action Potential ?**

  Proportionate to the strength of the stimulus, can be summated and if large enough to reach firing level an AP is produced.

3. INHIBITORY POSTSYNAPTIC POTENTIALS (IPSPs)

- When an inhibitory NT binds to its receptor on postsynaptic membrane, it causes hyperpolarization of the postsynaptic membrane.
- Increase membrane permeability to Cl- of post-synaptic membrane (produced by inhibitory neurotransmitter) leads to decreased excitability and membrane potential (more negative).

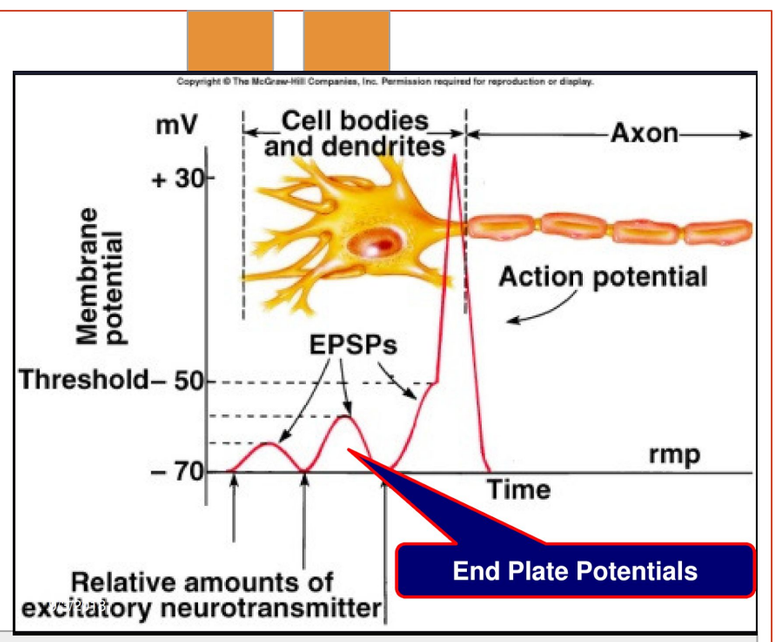

Figure 2-11 Lecturer's notes: End Plate potentials are EPSPs generated at nerve terminals and they are not propagated because they result from subthreshold stimuli.

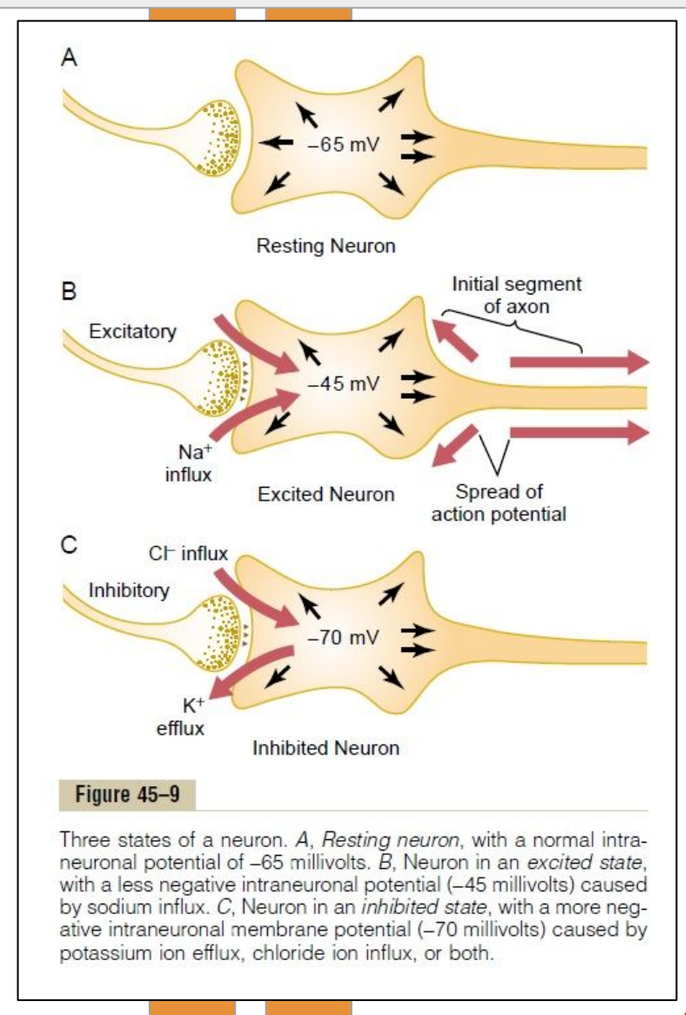

Figure 45–9

Three states of a neuron. *A*, *Resting neuron*, with a normal intraneuronal potential of –65 millivolts. *B*, Neuron in an *excited state*, with a less negative intraneuronal potential (–45 millivolts) caused by sodium influx. *C*, Neuron in an *inhibited state*, with a more negative intraneuronal membrane potential (–70 millivolts) caused by potassium ion efflux, chloride ion influx, or both.

Figure 2-12

EPSPs	IPSPs
1- Opening of Na channels to threshold level (Most Common).	1. Opening of Cl ion channels through the postsynaptic neuronal membrane.
2. Decrease conduction through Cl or K channels, or both.	2. Increase in conductance of K ions out of the Neuron
3. Various changes in the internal metabolism of the postsynaptic neuron to excite or, in some instances, to Increase excitatory membrane receptors or decrease inhibitory membrane receptors	3. Activation of receptor enzymes that inhibit cellular metabolic functions that increase inhibitory membrane receptors or decrease excitatory membrane receptors.

Table 2-5

PRESENT ONLY IN MALE SLIDES

Chemical Synapse	Electrical Synapse
Exhibits synaptic delay eg at NMJ reveal a delay of 0.5 to 4.0 mili sec	Almost no delay in transmission.
20- to 40-nanometer distance that separates cells	Cells approach within about 3.8 nm of each other
Two separate cells that do not touch	Gap junctions are intercellular connection that directly connect the cytoplasm of cells
Slower than Electrical	Faster: many neurons fire synchronously
Mostly unidirectional	Mostly bidirectional
More complex behaviors	Are fast, but can produce only simple behaviors
Act on receptors which are specific	Without the need for receptors to recognize chemical messengers
The response may not be the same as the source.	The response is always the same sign as the source.
The response in the postsynaptic neuron is variable.	Lack Gain the signal in the postsynaptic neuron is the same or smaller than that of the originating neuron

PRESENT ONLY IN MALE SLIDES

CHEMICAL SYNAPTIC PROPERTIES

1. ONE WAY CONDUCTION synapses generally permit conduction of impulses in one-way i.e. from **presynaptic** to **postsynaptic** neuron. **"Bell- Magendie law".**

2. SYNAPTIC DELAY is the minimum time required for transmission across the synapse. It is 0.5 ms for transmission across one synapse.
This time is taken by:
- Discharge of transmitter substance by presynaptic terminal.
- Diffusion of transmitter to post-synaptic membrane.
- Action of transmitter on its receptor.
- Action of transmitter to increase membrane permeability.
- Increased diffusion of Na+ to increase postsynaptic potential.

Clinical Importance is that we can know number of synapses involved in neuronal pathways by time lag

3. SYNAPTIC INHIBITION **Types: Direct inhibition, Indirect inhibition, Reciprocal inhibition & Inhibitory interneuron.**

A. Direct inhibition: Occurs when an inhibitory neuron (releasing inhibitory substances) acts on a postsynaptic neuron leading to hyperpolarization due to opening of Cl^- [IPSPs] and/or K^+ channels. Example: **Glycine** at the level of the spinal cord to block pain impulses.

B. Indirect Inhibition (Presynaptic inhibition): This happens when an inhibitory synaptic knob lie directly on the termination of a presynaptic excitatory fiber. The inhibitory synaptic knob releases a transmitter which inhibits the release of excitatory transmitters from the presynaptic fiber.
e. g. ***GABA*** (Pain modification).

C. Reciprocal inhibition: Inhibition of antagonist activity is initiated in the agonist muscle. Impulses pass directly to the motor neurons supplying the same muscle and via branches to inhibitory interneurons that end on motor neurons of antagonist muscle. **(Figure 2-13)**.

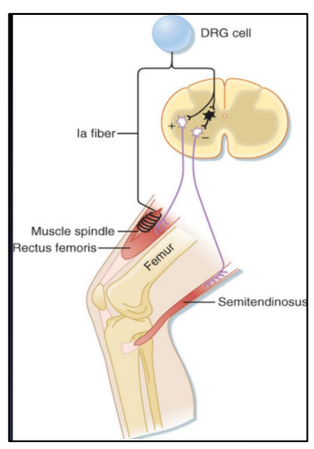

Figure 2-14

D. Inhibitory interneuron[1] (Renshaw cells)
Negative feedback inhibitory interneuron of a spinal motor neuron (Control the strength of contraction) **(Figure 2-14)**.

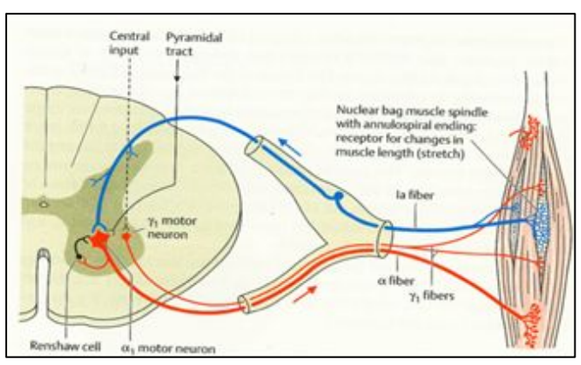

Figure 2-13

BOX 2-5: BASIC AND CLINICAL PHARMACOLOGY

Presynaptic inhibition occurs through receptors present on the presynaptic terminal, these can be autoreceptors or heteroreceptors.
- Autoreceptors, like alpha-2 on adrenergic presynaptic terminals inhibit the release of NT, by binding of the same NT, noradrenaline is inhibited by binding of noradrenaline. Heteroreceptors, like muscarinic receptors found on sympathetic presynaptic terminals can inhibit the release of noradrenaline by binding of ACh.

FOOTNOTES

1. Interneurons generally can refer to any neuron that acts as an intermediary neuron in passing signals between two other neurons. They only function to pass information between neurons without any sensory or motor functions. Interneurons are discussed in-depth in the spinal cord lecture.
2. Glycine acts mainly at synapses in the spinal cord and is believed to act alwats as an inhibitory transmitter.

4. SUMMATION

A. Spatial summation. Eliciting an action potential in a neuron with input from multiple presynaptic cells. (Greater number of fibers)**(Figure 2-15)**

B. Temporal summation: When the frequency of stimulation increased from the same presynaptic fiber. (Increase number of frequency of nerve impulses in each fibers). **(Figure 2-15).**

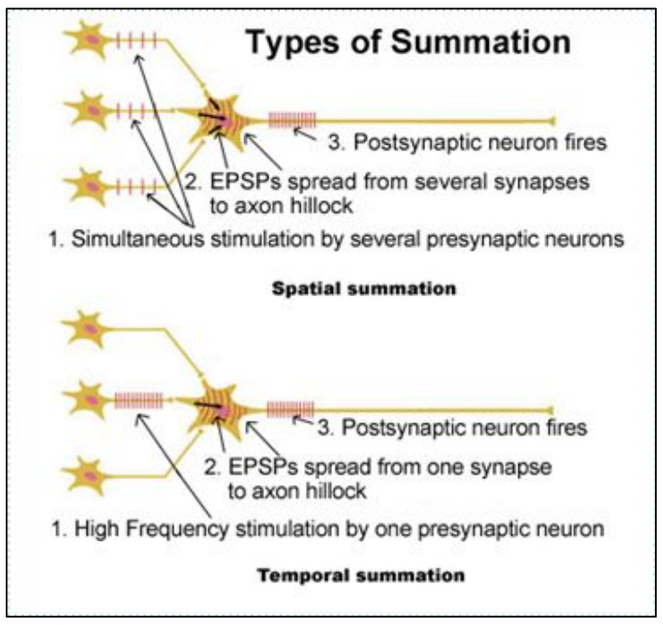

Figure 2-15

5. CONVERGENCE AND DIVERGENCE

A. **Divergence:** axons of presynaptic neurons divide into many branches that diverge to end on many postsynaptic neurons.

B. **Convergence:** When many presynaptic neurons converge on any single postsynaptic neuron. **(Figure 2-16).**

BOX 2-6: ENCYCLOPEDIA OF NEUROSCIENCE

To better visualize divergence, a signal from a preganglionic fiber can excite many different cell bodies in a ganglion to perform a function. The same case can happen with convergence, where each ganglionic neuron receives an input from several preganglionic fibers, this can help to ensure an action potential gets initiated.

6. FATIGUE

- It is due to exhaustion of neurotransmitter. If the presynaptic neurons are continuously stimulated there may be an exhaustion of the neurotransmitter. Resulting in stoppage of synaptic transmission.
- Synaptic fatigue means simply that synaptic transmission becomes progressively weaker the more prolonged and more intense the period of excitation.
- Short-term (synaptic depression), is an activity-dependent form of short term synaptic plasticity that results in the temporary inability of neurons to fire and therefore transmit an input signal.

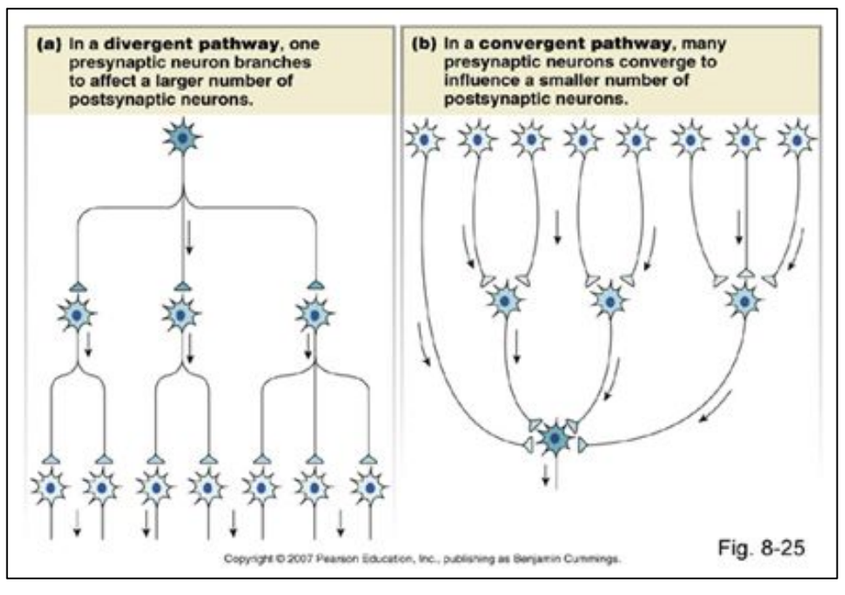

Figure 2-16

Lecturer's notes: For example, in epilepsy there is a post-epileptic fatigue period.

Mechanism of Fatigue

- Exhaustion of the stores of transmitter
- inactivation of many of the postsynaptic membrane receptors
- Abnormal ion concentrations in postsynaptic neuron.

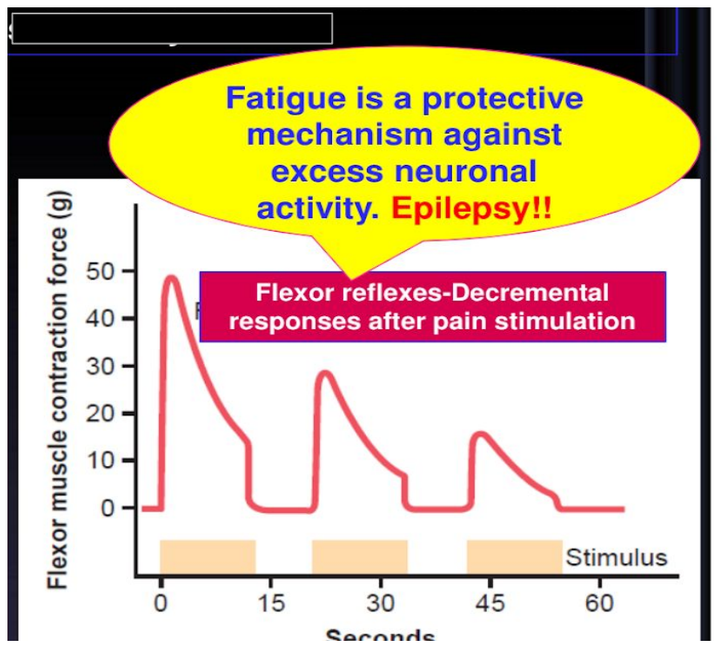

Figure 2-17

FACTORS AFFECTING SYNAPTIC TRANSMISSION

1. ALKALOSIS

- Increases neuronal excitability.
- Causes cerebral epileptic seizures (Increased excitability cerebral neurons).
- e. g. overbreathing in person with epilepsy.
- The over breathing blows off carbon dioxide and therefore elevates the pH of the blood momentarily.

2. ACIDOSIS

- Depresses neuronal activity.
- pH around 7.0 usually causes a coma.
- E.g. severe diabetic or uremic acidosis.

3. DRUGS

- Caffeine depolarizes postsynaptic membrane
- Strychnine: competes with inhibitory transmitters.
- Theophylline and theobromine increases neuronal excitability, by reducing the threshold for excitation of neurons.
- Sedatives, hypnotics & anesthetics: hyperpolarize (increased threshold)
- postsynaptic membrane.

4. HYPOXIA

- Depression of neurons.

5. HYPOGLYCEMIA

- Decreases neuronal activity. (diabetics acidosis is quite common, the reason being that defeciency of glucose uptake causes extensive breakdown of fats which yeald carboxylic acids, therefore acidosis and neuronal depression occurs)

6. HYPOCALCEMIA

- Increases neuronal excitability (tetany) (calcium binds to voltage-gated sodium channels creating a force of repulsion around the channels and delays entry of sodium.)

BOX 2-7: CLINICAL RELEVANCE

- Acetazolamide (a carbonic anhydrase inhibitor) can be used to reduce epileptic seizures by reducing the pH within the vicinity of neurons, this is done by inhibiting the formation of carbonic acid which spontaneously disintegrates into bicarbonate, which is a weak base that raises the pH.
- Acidosis and hypoxia have a relatively similar effect since they both can lead to depression of neurons,, in hypoxia neurons produce excess amounts of lactic acid due to anaerobic glycolysis, precipitating acidosis which eventually leads to coma due to neuronal depression,
- Methylxanthines, such as theophylline and caffeine, inhibit phosphodiesterase, an enzyme that hydrolyzes cAMP, an important 2nd messenger that causes relaxation when activated in bronchial smooth muscles and hence causes bronchodilation. It should be noted that salbutamol, one of the most potent asthmatic drugs is a beta-2 agonist, activation of beta-2 results in increased cAMP levels.

13 Synapse and Receptors

Lecture Two

SUMMARY

- Almost all synapses in the CNS are chemical synapses
- Neurotransmitter is a chemical substance that is secreted by the first neuron at the synapse to act on receptor on the next neuron to excite it, inhibit or modify its sensitivity.
- Chemical synapse is One directional in transmission while electrical synapse transmission can occur in both direction.
- Gap junction is the space between the pre- and postsynaptic neurons which allows the passage of ions.
- Electrical synapses Are important in the CNS in Mental attention,Emotions, memory and Arousal from sleep.
- Neurons in lateral vestibular nucleus have conjoint synapse. .
- Ach esterase is an enzyme that destroys Ach neurotransmitter in a process called Enzymatic destruction.
- Norepinephrine neurotransmitter is actively transported back into the presynaptic terminal.
- Neurotransmitter receptors that directly open gate ion channels are often called ionotropic receptors, whereas those that act through second messenger systems are called metabotropic receptors. .

QUIZ

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1. Which of the following 2nd messenger mechanisms yields the quickest response?

A) Opening of ion channels
B) Activation of gene transcription
C) Activation of one or more intracellular enzymes
D) Formation of cAMP or cGMP

2. Khaled, an asthmatic patient was prescribed a second-line medication for treatment of his symptoms, Khaled showed improvements with notable bronchodilation but with also increased neuronal excitability. Which of the following drugs was prescribed to Khaled?

A) Theophylline
B) Strychnine
C) Salbutamol
D) Theobromine

3. A common mechanism for fatigue:

A) Upregulation of both excitatory and inhibitory receptors
B) Formation of membrane attack complex on the cell membrane
C) Depletion of transmitter stores
D) None of the above

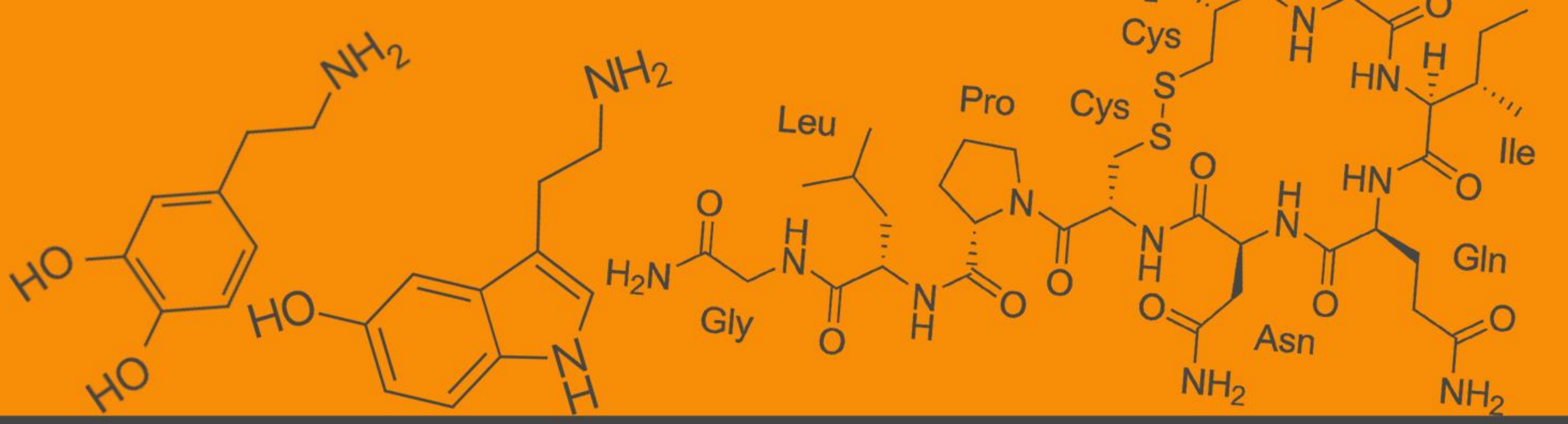
4. If a neurotransmitter substance resulted in formation of new proteins after excitation of a postsynaptic terminal, what type of receptor does the postsynaptic terminal possess?

A) Metabotropic receptor
B) Ionotropic receptor
C) Ligand-gated ion channel
D) No receptor needed

5. An anatomical type of synapse:

A) Axodendritic
B) Electrical synapse
C) Chemical synapse
D) Conjoint synapse

ANSWER KEY: A, A, C, A, A



THIS LECTURE WAS DONE BY

Nayef Alsaber, Hameed M. Humaid

FEMALE PHYSIOLOGY CO-LEADERS

Maha Alnahdi, Ghaliah Salem

MALE PHYSIOLOGY CO-LEADERS

Nayef Alsaber, Hameed M. Humaid

PRESENTED BY

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