Chapter 12: *Nervous Tissue and Homeostasis*
Pages 403 - 438

Neurology – branch of medicine that deals w/ the normal functioning and disorders of the nervous system.

I. Structures of the nervous system:
* 4.5 lbs (2 kg), or 3% of total body weight; it is the smallest and yet most complex of all the 11 body systems.
* Intricate network of billions of neurons.

A. Structures included are: **(See figure 12.1; page 405)**
1. ***Brain*** and its *cranial nerves I* through *XII* emerge from the brain.
2. ***Nerve*** – a bundle of 1000's of axons (neurons); nerves follow a specific path or region in the body, e.g. *cranial nerve I* signals smell.
3. ***Spinal cord*** – connects to the brain via the brain stem and enclosed in a bony canal (vertebral column).
Contains ~ 100 million neurons. Thirty-one pairs of spinal nerves emerge from the spinal cord all serving both sides of the body.
4. ***Enteric plexus*** – a network of neurons in the walls of the GI tract that help regulate the digestive system.
5. ***Sensory receptors*** – the dendrites of *sensory neurons*; these neurons monitor changes in the internal and external environment, e.g. photoreceptors, pressure receptors, touch receptors, etc..

II. 2 main systems control homeostasis; both control conditions w/in limits that maintain health
1. endocrine - hormonal; slow response
2. nervous - nerve impulses; fast response

A. Functions of the Nervous system: 3 basic functions.
1. Sensory input - senses external & internal changes.
eg. sound, BP, blood chemistry, and rain drops. (***sensory afferent***)
2. Integrative - analyzes the sensory info. (***perception***); stores some info.
& makes decisions regarding an appropriate behavior or response.
3. Motor output - **response to stimuli** by initiating muscular contraction or glandular secretions.
(***motor efferent***)
III. Organization of the Nervous System: **(See figure 12.2; page 406)**
2 Main divisions - 1) CNS 2) PNS

1. **CNS** - includes brain & spinal cord
* sensory info. is integrated here; memories formed & stored.
* most nerve impulses involving motor response originate here.
CNS is connected to sensory receptors, muscles & glands via PNS.

2. **PNS** - includes cranial nerves (arise from brain), and spinal nerves (arise from spinal cord)
* portions of both cranial nerves and spinal nerves carry nerve impulses into & out of the CNS
A. Organization of PNS: Hint: remember "**SAME**"
SA = **S**ensory > **A**fferent (***adds*** to CNS)
ME = **M**otor > **E**fferent (***exits*** CNS)

1. **S**ensory/**A**fferent Neurons (**SA** pathway) - ***(adds** to CNS)*
* conduct nerve impulses from sensory receptors to CNS
(e.g. touch, smell, taste, pain, GI tract, blood chemistry)

2. **M**otor/**E**fferent Neurons (**ME** pathway) – ***(exits** CNS)*
* conduct nerve impulses from CNS to skeletal muscles.

3. **E**nteric **N**ervous **S**ystem (**ENS**) – regulation of the "gut"
* Once considered part of the ANS (autonomic nervous system).
* Involuntary control of the GI chemistry & stretching of walls.
* Communicates with the CNS via sympathetic & parasympathetic

Motor/Efferent System is subdivided into 3 divisions:
a. **S**omatic *(limbs/non-internal organs)* **N**ervous **S**ystem **(SNS)** – *voluntary control*
* info. from CNS to skeletal muscles .ie, you can control motor responses. ie. somatic motor

b. **A**utonomic **N**ervous **S**ystem **(ANS)** - *involuntary(self) control*
* info. from CNS to smooth muscle (organs), heart & glands
ie. visceral(organs) motor

2 divisions of **ANS**: they have opposing actions
1. Sympathetic - stimulatory/excitatory; gas pedal.
eg. ↑ HR
2. Parasympathetic - inhibitory; brake pedal. eg. ↓ HR
NOTE: there are neurotransmitters that are released from neurons that act as inhibitors or stimulators!

c. **E**nteric **M**otor **N**eurons **(ENS)** – *involuntary control*. Governs the release of digestive hormones and movement GI tract.

IV. Anatomy of a Neuron *aka. A nerve cell*: (**See fig 12.3; page 408)**
* various sizes; from < 1mm to 1 meter in length
* various nerve impulse speeds; from 0.5 to 395 m/sec
(1 to 280 mi/hr.)
A. 3 basic parts of a neuron - (**See fig 12.8; page 412)**
1. *Cell body* - contains nucleus; typical organelles
2. *Dendrites* - emerges from cell body
3. *Axon* - long, thin, cylindrical, that may be *mylinated*
(a protein/lipid coat) that insulates the axon & can ↑ speed of nerve impulse. nerve impulse travels **away** from the cell body along the **axon** to another neuron, a muscle cell, or gland cell.
- nodes of *Ranvier* – gaps in the myelin sheath along the axon.
- axons end by dividing into many processes called
axon terminals (synaptic end-bulb); contain synaptic vesicles that store neurotransmitters.

NOTE: there are neurotransmitters that are released from neurons that act as inhibitors or stimulators!

Clinical App*: *Demyelination – the loss or destruction of the myelin sheaths around the axons. It may result from Multiple Sclerosis, or Tay Sachs (hereditary), radiation therapy & chemotherapy.
Leads to degeneration of the nervous system.

***Clinical App*:** Rabies, Polio, Herpes **viruses** – travels from PNS → CNS via **axonal transport**; the virus travels up the axon towards the cell body where it reproduces.

B. Gray and White Matter: (**See fig 12.9; page 413)**
Freshly dissected brain & spinal cord has regions that appear white & dark
White matter – composed of *myelinated axons*.
Gray matter – *unmyelinated axons*, cell bodies, dendrites.

C. 3 Functional Classifications of Neurons:
Based on according to the direction in which the nerve impulse travels relative to the CNS.
1) *sensory neurons* (adds to CNS)
2) *motor neurons* (exits CNS)
3) *interneurons aka. association neurons*(found between motor & sensory neurons)

Nerve - a bundle of many neurons (sensory & motor) that follow the same path in **PNS**; eg. ulnar nerve, sciatic nerve

Tract - a bundle of nerve fibers in the **CNS**; connects regions of the brain, or extends the length of the spinal cord to brain.

V. Electrical Signals in Neurons: (**See fig 12.10; page 415; pencil scenario)**
Nerve cells are electrically *excitable!*
They communicate with one another using two types of electrical signals:
1) Graded potential – short-distance communication.
2) Action potentials (nerve impulse) – long and short-distance communication.

A. Neurophysiology:
- Communication between neurons depends on the cell membrane - Why is this so??
- Neurons are highly irritable (easily stimulated).
- When a neuron is stimulated, an electrical impulse travels down the length of the axon, i.e. an *Action Potential* is generated!

B. Role of Membrane Ion Channels: (**See fig 12.11; page 416)**
- neuron's membranes are peppered w/ diff. ion channels made of proteins embedded in the cell membrane. They regulate the movement of ions into & out of the cell.

Ion Channels - 2 types:
- each type of channel is selective about what ions it allows to pass! e.g. sodium ion channels for **Na^+ to pass.**
- when neurons are stimulated, ion channels open & ions diffuse across the memb., an *electrochemical gradient* is created & an electrical current flows. **Work is produced!**

1. Leakage/Passive channels - always open; cell membrane has a higher K^+ leakage than Na^+

2. Gated/Active channels - open & close in response to a stimulus.

4 Types of Channels Relative to Stimulus:

a. **voltage** - response to memb. voltage changes (Na^+ & K^+)
b. **chemical** - response to neurotransmitters/hormones (only found on the cell body & dendrites)!
c. **mechanical** - response to vibration/pressure
d. **light** - response to light

Question:
What is an adaptive advantage of the 4 types of gated channels in the membrane?

C. The Resting Membrane Potential: "The Polarized Cell"(**See fig 12.12; page 417)**
Resting membrane potential (RMP) – the membrane voltage in a resting neuron; it is said to be "polarized" because the inside(ICF) of a neuron is negatively charged w/ respect to the outside(ECF). ie, -40 mV to –90 mV

RMP – occurs because there is a small build up of neg.(-) ions inside the cell memb. (ICF), and an equal build up of pos. (+) ions outside the cell memb. (ECF).
* this difference sets up a gradient (electrochemical).
* the PE (potential energy) is measured in millivolts, (1/1000)V
* in neurons **RMP = -70 mV**; **"like voltage stored in a battery can do work"**
* Na^+ & K^+ gated-channels are closed during RMP (but they are "leaky" ion channels)
NOTE: ↑ electrical gradient ; ↑ PE to do work (voltage in mV)

* most body cells have a membrane potential which varies from
- 40 mV to -90 mV depending on the cell, ie. ***a polarized cell***
* when ion channels open or close in the cell membrane, ions flow & this changes the membrane potential (mV)

D. Two Factors that contribute to the RMP:
1. Distribution of *ions* on either side of the cell membrane.

- ECF (pos. charged) is rich in: Na^+ and Cl^-
- ICF (neg. charged) is rich in: K^+, PO_4^{2-} and *amino acids* (HUGE molecules)!

2. Relative Permeability of the Cell Membrane to Na^+ and K^+

- K^+ is 50 to 100 times more permeable than Na^+
- K^+ is the major ion being transported!
- K^+ ion channels are "leaky"
- Na^+ is slightly permeable which .....
  a. balances the K^+ outflow
  b. helps maintain the RMP (-70 mV)

E. Resting Memb. Gradient - the inside is negatively charged & the outside is positively charged.
3 reasons below:

a. more negative ions (proteins & PO_4^{2-}) inside the cell.

b. 100x more K^+ leave the cell by diffusion than Na^+ enters by *diffusion*; as a result the cell becomes more negative.

c. 3 Na^+ are ***actively pumped out*** for every 2 K^+ *pumped in*.

(ATP is used here, i.e. the **Na^+/K^+ Pump**)

ie. RMP is maintained by the Sodium/Potassium Pump!

VI. Membrane Potentials Act As Signals- "Nerve Impulse":

- Neurons use changes [relative to RMP] in their membrane potential as communication signals between neurons.

A. Changes in memb. potential can be produced by:

1) anything that changes the memb's. permeability to ions.
2) anything that alters the [ion] on either side of the membrane.

B. **Nerve impulse** - a flow of *electrical energy*; consists of many depolarizations and repolarizations. Made of many continuous action potentials.

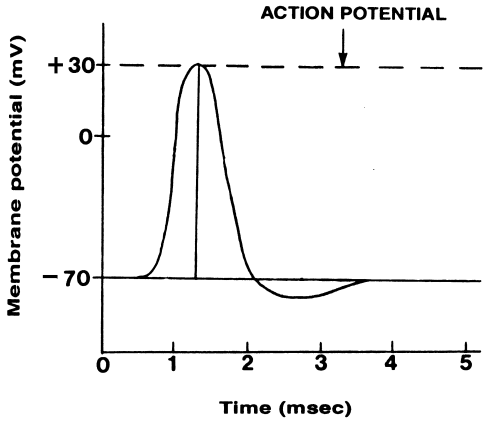
*** Lasts ~ 1 millisec (~ 10 – 1,000 impulses/sec)**
* During an act. pot. 2 voltage gated channels open and then close.
1st - Na^+ gated channels open & close
2nd - K^+ gated channels open & close
(See Fig. 12.14, page 419)

C. Characteristics of an Action Potential:
1. *Threshold stimulus* - any stimulus strong enough to initiate an action potential (from -70 mV towards -55mV); an unstable equilibrium state between Na^+ and K^+
WOW! It only takes 0.012% of Na^+ to influx(move into the cell).

2. *Depolarization* - rapid opening of voltage gated Na^+ channels (-65 mV towards 0 mV to + 30 mV); results in an action potential.

3. *Repolarization* - slower opening of voltage gated K^+ channels and closing of Na^+ channels. Membrane becomes more negative (from + 30 mV back to – 70mV).

4. *Hyperpolarization* - the outward flow of K^+ during repolarization causes the membrane potential to reach a more negative level (-90 mV) than that of the RMP (-70 mV); an action potential does not result.



D. Generation of Action Potentials (Nerve Impulses):
(See Fig. 12.14 and 12.15, pages 419-420)

[Remember: the the memb. potential is at RMP = -70 mV & the Na^+ & K^+ gated channels are closed!]

Depolarization:

1. A *stimulus* causes the nerve cell membrane to reach threshold (-55 mV); a few Na^+ gated channels are open.

2. The membrane becomes more permeable to Na^+

3. More Na^+ gated channels open and Na^+ rushes inward; driven by a conc. gradient *(diffusion)*, and an electrical gradient!

4. As Na^+ enters the cell, the inside of the membrane becomes more positively charged. The membrane potential will move towards -60mV.

5. The membrane depolarizes further; the continued inflow of Na^+ magnifies the initial stimulus.
Q??? What type of feedback mechanism is this????

6. The membrane is completely depolarized **at 0 mV → an action potential is now generated!**

7. Na^+ channels remain open until the inside of the membrane potential is reversed to + 30 mV, i.e. the inside is more positive than the outside!
8. The nerve impulse is "*like a wave of negativity traveling"* along the outside of the nerve cell membrane.

9. The impulse is propagated/transmitted along the nerve, as adjacent areas are depolarized, more Na^+ channels open, thus more Na^+ flow inward. *"like toppling a row of dominos"*

10. After fractions of second, the depolarization causes the K^+ channels to open at the site of the original stimulus.

11. K^+ diffuses outward with the conc. gradient; simultaneously, the Na^+ channels close.

Repolarization begins: [restores the RMP by Na^+ channels closing and K^+ channels opening]

1. the K^+ channels opening accelerates the K^+ outflow
2. the membrane potential changes from + 30 mV to 0 mV and then to **-70 mV (RMP).**

Hyperpolarization occurs:

1. the K^+ flowing outward causes the membrane potential to drift more negative. i.e. - 90 mV
2. the K^+ channels close; membrane potential returns to resting level.

Note: Na^+/K^+ Pump: maintains the $[Na^+]$ $[K^+]$ conc. gradient by actively pumping the Na^+ out & the K^+ back into the cell.

ClinicalApp: Neurotoxins (scorpion venom) and Local Anesthetics
e.g. Novocaine, Lanocaine used for pain blocking.

They prevent the Na^+ channels from opening.
??Q: How does this *physiologically* block pain?

Absolute refractory period – when a patch of neuron membrane is generating an action potential and it is depolarizing (Na^+ channels opening & closing), the neuron is incapable of responding to another stimulus. This enforces one-way transmission of a nerve impulse down the neuron.

Relative refractory period – follows the absolute refractory period (during a repolarization). The neuron can be re-stimulated at this time if the stimuli is strong enough.

E. Propagation of A Nerve Impulse - nerve impulses (depol. & repol.)
self-propagate/travel along the axonal membrane from cell body to the *end-buttons* of the neuron. "*A Domino Effect"*

F. All or None Principle: if stimulated, the neuron will generate an act. pot. with a constant/maximum strength unless fatigue or toxic material.
*like igniting a trail of gun powder; "a flash point when igniting gasoline"

??Q: If all action potentials are of the same amplitude(+30 mV), then how can your sensory & motor systems respond in varying manner? i.e. why does a light touch feel different from firmer pressure; and how can you control muscle action to be light & delicate or strong & vigorous?

3 explanations below:

1. Frequency of Nerve Impulse:
2. Stimulus Duration:
3. Receptive Fields:

1. Frequency of Nerve Impulse:

↑ intensity of stimulus; ↑ frequency of nerve impulses; traveling down axon ↑ in recruitment of sensory receptor firing

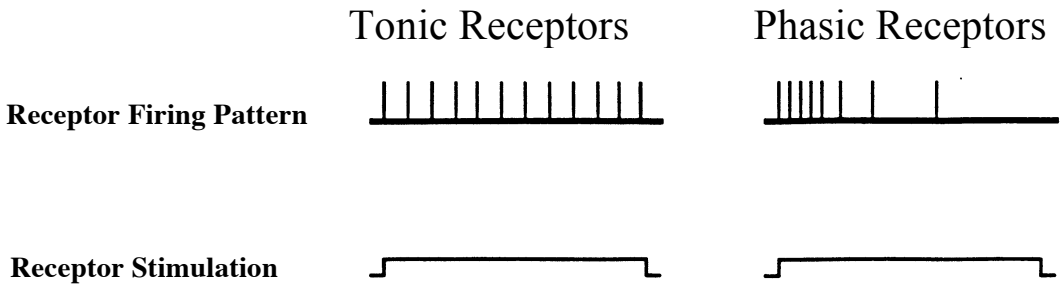
eg. ↑ pressure of touch w/ finger ; ↑ surface area ; ↑ "calling in" of additional sensory afferent neurons

2. Stimulus Duration:

- receptors differ in the way they respond to a constantly maintained stimulus.

??Q: Why is it that when you sit on a chair you feel the pressure initially, but after a few seconds the intensity of pressure is less?

* 2 types of receptor responses to stimulus :
a. Tonic receptors *"slowly adapting receptors"*– e.g. pain receptors.
b. Phasic receptors *"rapidly adapting receptors"*– e.g. smelling fish, sitting on a stool, acclimating to the Jacuzzi temp.



3. Receptive Fields: the size or area that the receptor covers in the body; ie. receptor density (# of receptors/area of body).

a. large receptor field – has **fewer receptors** w/in a particular region, but has the disadvantage of having **less discriminating** ability or sensitivity as to the exact location of the stimulus.
e.g. your back or buttock region(dorsal surface).

b. small receptor field - has many receptors w/in a particular region. Allows for **finer discrimination** in regards to stimulus input due to the **greater numbers of receptors** in the region.
e.g. the tips of your fingers can detect the slightest change in surface area (e.g. detecting surface changes for reading brail).

G. Speed(Velocity) of Impulse Conduction: nerve impulse speed is related to diameter of axon, temperature & presence of a myelin sheath around the axon.
a. ↑ diameter ; ↑ speed
b. ↑ temp. ; ↑ speed
c. presence of myelin sheath ; ↑ speed

??Q: Why is ice applied to an injured area to reduce the pain?
* The largest nerve fibers are those associated in relaying nerve impulses from sensory receptors to CNS. Smaller nerve fibers are associated w/impulses from organs (bladder for example) to CNS.

Q??: What might be the physiological advantage of this association mentioned above?

Clinical App: Multiple Sclerosis; (**See page 433)**
An autoimmune disease where the myelin sheath is destroyed. Leads to degeneration of the CNS; short-circuiting of nerve impulses.

VII. Signal Transmission at Synapses: *A chemical signal (NT's)*
(See figure 12.17; page 425)
* remember neuromuscular Jcts. from muscle physiology??
* *synaptic junction* - a tiny space between communicating neurons or between a neuron and an effector (a muscle or gland).
- *Presynaptic* neurons (info. sender) and
- *Postsynaptic* (info. receiver); an effector or neuron.

Chemical Synapses: **(See figure 12.17; page 425)**
- synapses are essential to homeostasis because info. is filtered here, i.e. info. is transmitted to the neighboring neuron, or it is blocked!
- site of drug action both therapeutic or addictive.
- some psychiatric disorders, e.g. *Tourettes Syndrome*;

(a super-sensitivity of dopamine receptors in the CNS has been suggested).
- nerve impulses **don't "jump" across** the synaptic junction; the signal crosses in the form of neurotransmitter(chemicals).

3 ways neurotransmitters are removed from the synaptic junction:
a) enzymes degrade the NT's e.g. AChase degrades ACh
b) diffuses away from the synaptic cleft – far out of reach of its receptors so it can no longer exert its effect.
c) neurotransporters or "up-take pumps" recycle the NT such as norepinephrine (NE) back into the vesicles of the presynaptic neuron

VIII. Neurotransmitters(NT): chemical messengers between neurons.
(See pages 428 – 430, and chapter 15, page 534)
- Found in CNS, PNS & Blood (some hormones are NT's)
- Neurotransmitters released from the pre-synaptic neuron can cause an excitatory or an inhibitory effect on the post-synaptic neuron.

A. Two categories of neurotransmitters: *excitatory* and *inhibitory*
1. Excitatory "gas pedal" - causes a depolarization (-55 mV) of the postsynaptic neuron when excitatory NT's bind to the receptor sites on Na^+ ion channels and depolarization occurs; (EPSP's) a nerve impulse is generated. eg. ACh binds to muscle tissue, glutamate, dopamine, serotonin.
i.e. excitatory effect > inhibitory effect
*EPSP = Excitatory PostSynaptic Potential

2. Inhibitory "brake pedal" - causes a hyperpolarization (-90 mV) of postsynaptic neuron when inhibitory NT's bind to the receptor sites for Cl^- & K^+ ion channels; (IPSP's) no nerve impulse can generate. eg. GABA, Glycine, and ACh binding to heart muscle tissue.
i.e. inhibitory effect > excitatory effect
*IPSP = Inhibitatory PostSynaptic Potential

NOTE: the hyperpolarization occurs because Cl^- & K^+ gates open and Cl^- moves into the cell and K^+ moves out of the cell. The inside becomes more negative (farther away from –70mV).
*sometimes the same neurotransmitter is excitatory in one location, but inhibitory in another location because of the type of neurotransmitters released or the receptors they bind to on the tissues (effector).

C. Synthesis of common neurotransmitters: **(See Table 12.3; page 430)**
many are proteins made from amino acids such as epinephrine, norepinephrine, serotonin & dopamine.

- some amino acids serve directly as neurotransmitters such glutamate & aspartate.

NOTE: some neurons have 2-3 diff. types of neurotransmitters that they release.

D. Disorders assoc. w/ neurotransmitters:
1. **Parkinson's** – hyposecretion (↓) of dopamine; causes jerky body movements. An inhibition of the motor drive.
Usually treated w/ drugs that enhance dopamine's effect. (See page 568)
2. **Schizophrenia** – hypersecretions (↑) of dopamine; causes mood disturbances.
3. **Alzheimer's -↓** ACh releasing neurons in the brain.
ACh plays a role in learning & memory in the cerebral cortex and hippocampus. Declining memory. **(See page 517)**
4. **Huntington's chorea** – a late-onset hereditary disorder that causes degeneration of cerebral cortex…. Death**. ↓** GABA; an over-stimulation of the motor drive; jerky movements.
Usually treated w/ drugs that block dopamine's effect. **(See page 564)**
5. **Depression** - "unipolar disorder"- assoc. w/ low levels of *serotonin* in the brain. eg, treated w/ Prozac & Elavil that deactivate the neurotransporters for serotonin; therefore, ↑ availability of serotonin in the synaptic cleft.

Clinical App: **Strychnine Poisoning (see page 427)**

- the drug binds to & blocks inhibitory receptors (Glycine receptors) on the post-synaptic neuron, thus there is no inhibition of skeletal muscle contraction. All muscles including the diaphragm can't relax; the person can't breathe.

E. Alteration of Nerve Impulse:
* a neuron's environment may be altered which has a direct influence on the nerve impulse conduction
1. ***Leg falling asleep*** - nerve conduction is blocked; lack of blood carrying O_2 to the neuron; it won't fire an impulse.

F. Effects of drugs:
2. ***Hypnotics, tranquilizers, anaesthetics*** - depresses impulse conduction by increasing the threshold for excitation of neurons, e.g. alcohol, depressants, barbiturates, and valium.
i.e. instead of - 55 mV threshold, it is - 30 mV; it is more difficult for the cell to be excited.

3. ***Nicotine (stimulant)*** - increase impulse conduction by decreasing the threshold for excitation of neurons. ie. instead of - 55 mV threshold, it is - 65 mV. This causes an ↑ dopamine release.

4. ***Cocaine/Crack/Amphetamines*** **–** act to block the reuptake pumps, so the neurotransmitter dopamine remains in the synaptic cleft longer than normal; thus, short-term euphoria.

5. ***Methamphetamine*** **– (2 ways)** causes the presynaptic neuron to release more dopamine, and it works by blocking reuptake pumps so the dopamine remains in the synaptic cleft longer than normal; thus, short-term euphoria.

6. ***Caffeine (stimulant)*** **–** blocks receptors for adenosine(a sleep inducer) from binding. Instead of sedation, the receptors w/ caffeine bound to them causes an increase in impulse conduction.
You stay AWAKE! Caffeine is also considered a stimulant because it decreases the threshold stimulus from –55mV to –65mV. The cell is more easily excited.

7. ***Ecstacy or MDMA***- interferes w/ the enzymes involved with the deactivation of the neurotransmitters serotonin; thus, it remains in the synapse longer; short-term euphoria. ↑ serotonin can cause brain damage. Also, MDMA can cause death by: ↑ body temp. & ↑ HR

8. ***LSD*** – most potent hallucinogen; inhibits serotonin by blocking serotonin receptors.

9. ***Morphine/Heroin* – "opioids"** mimic your NT's such as *endorphins* and *enkephalins*. Brings about euphoria and pain relief.

10. ***Alcohol*** – a depressant; produces mild euphoria due to
↑ dopamine released. Ion channels shrink, so ion movement is disrupted (causes an inhibitory response). One can create a tolerance; physical & psychological dependency.

11. ***Valium*** – a depressant; enhances the action of GABA.
Causes a hyperpolarization (-90mV) of the neuron.

Note: psychoactive drugs "mood altering drugs" like **THC, LSD** and morphine - are similar in chemical structure to natural neurotransmitters in the body "mimic" & then interact with receptors on the post-synaptic neurons. ie, **serotonin & dopamine are implicated**. ***(see page 430)**

12. ***Botulism*** - toxin from bacteria that inhibits the release of ACh from motor neuron; thus, weakens muscle contraction

13. ***Nerve gas/Mustard gas*** - inhibits the enzyme AChase from destroying ACh on the motor-end plate(muscle fiber). The muscles can't relax, ie. death from asphyxiation.

14. ***Curare*** – plant poison that binds to and blocks ACh receptor sites, thus preventing the binding of ACh. The muscles can't contract, i.e. death from asphyxiation

15. ***Tetanus*** **"Lockjaw"** - *bacteria* that interferes w/ inhibitory (brake) synaptic mechanisms to motor neurons. The excitatory (gas) inputs remain "unchecked"; results in excessive, involuntary skeletal muscle contraction. Spasms of the jaw muscles are early signs.

IX. Several Ways to Modify Chemical Synaptic Transmission:
- Stimulate or inhibit neurotransmitter synthesis.
- Block or enhance neurotransmitter release.
- Stimulate or inhibit neurotransmitter removal in synaptic jct. ie. neurotransporters or "up-take pumps"
- Deactivation of enzymes that break down NT's
- Block or activate (mimic neurotransmitters) receptor site.

A. Drug Dependency & Tolerance:
Dependency - 2 forms of "addiction**"**. Addiction acts on: thinking, emotions, and behavior.
a. psychological – "a craving for the drug".
"Gosh, 'id sure be cool to smoke some pot on Friday night!"

b. physical – requires one to take the drug to avoid *withdrawals* (unpleasant symptoms such as vomiting and abdominal cramps). eg. methadone is a substitute for heroine addiction.
Methadone Clinics for addicts.

Characterized by:

- Progressive increased consumption of drug.
- Persistent tendency to relapse even after abstinence and withdrawal symptoms are no longer evident.
- Drugs that lead to dependence act on the "pleasurable" neural pathways in the brain called the ***nucleus accumbens***.

Tolerance – to a drug occurs when increasing the dosage of the drug is required to achieve the effects that initially occurred in response to a smaller dose.

***Two theories below*:**
1) Based on previous drug use; the presence of the drug stimulates the synthesis of the enzymes that degrade the drug in the synaptic jct. As ↑[drug] ; ↑[enzymes that degrade drug], Thus, more of the drug must be administered for the same initial effect.
2) Tolerance can develop as a result of changes in the number and/or sensitivity of receptors that respond to the drug. For example, the drug's effects might ADD to those of normally occurring neurotransmitter, thereby producing an increased response that, by feedback inhibition, eventually decreases the release of the naturally occurring neurotransmitter.

Disorders:
1. *Epilepsy*-
   - 2nd most common neurological disorder to strokes.
   - brief attacks of motor, sensory, or psychological

   malfunction, ie. seizures caused by abnormal electrical discharges from excitatory neurons in the brain due to the inhibitory mechanism malfunctioning.
2. *Lou Gehrig's Disease* – rare degeneration of motor neurons (death).
3. *Tourettes Syndrome* – a hereditary neurobehavioral disorder; characterized by motor & vocal tics. Supersensitivity of dopamine receptors in the CNS has been suggested as the patho-physiology. Persons usually grow out of it.

X. Patterns of Neural Processing: **(See figure 13.13; page 461)**
Circuits – the patterns of synaptic connections, i.e. excitatory or inhibitory. They may act in *parallel or serial patterns*.

1. *Serial Processing* – the whole systems works in an all-or- nothing manner. One neuron stimulates the next in sequence, which stimulates the next neuron causing a specific response, e.g. Reflex Arc (spinal reflex)
* Reflexes – rapid, autonomic (ANS) response to a stimulus, in which the same stimulus *always* causes the *same* motor response. eg. jerking your arm away from a hot plate. Blinking your eye when an object approaches.
Reflex arcs have 5 essential components:

1) sensory receptor, eg. touch or pain
2) sensory afferent neuron (adds to CNS)
3) integration center (CNS)
4) motor efferent neuron (exits CNS)
5) effector (response)