16
Nervous System

Nervous System

Nervous sytem is the most important organisation which control and integrates the different body functions and maintains the constancy of internal environment despite of extreme change in external environment.

Importance of nervous system :

1. It is the first appearing control system of the body.
2. It controls the rapid and first activities of the body e.g. muscular activity, secretion of glands etc.
3. It has regulatory effect upon endocrine system.
4. It helps in adustment of the body with the external environment.

Organization of the nervous system

The nervous system is unique in the vast complexity of thought processes and control actions it can perform. It receives literally millions of bits of information from the different sensory organs and then integrates all these to determine the responses to be made by the body.

General design of the nervous system :

i. *The central nervous system neuron (the basic functional unit)* : The central nervous system contains more than *100 billion* neurons.

   a. The *incoming signals* enter the neuron through synapses mainly on the neuronal dendrites, but also on the cell body. For different types of neurons, there may be only a few hundred or as many as *200,000* such *synaptic* connections from the input fibers.
   b. The *output signal* travels by way of a single axon leaving the neuron, but this axon has many separate branches to other parts of the nervous system or peripheral body.

   A special feature of most synapses is that the signal normally passes only in the forward direction (from axon to dendrites). This allows the signal to be conducted in the required directions for performing necessary nervous functions. We shall also see that the neurons are arranged into a multitude of differently organized neural networks that determine the functions of the nervous system.

ii. *Sensory division of the nervous system (sensory receptors)* : Most activities of the nervous system are initiated by sensory experience emanating from sensory receptors, whether visual receptors in the eyes, auditory receptors in the ears, tactile receptors on the surface of the body, or other kinds of receptors. This sensory experience can cause an immediate reaction from the brain, or memory of the experience can be stored in the brain for minutes, weeks, or years and determine bodily reactions at some future date.

   This information *enters the central nervous system* through the peripheral nerves and is conducted immediately to multiple sensory areas in the -

   a. Spinal cord at all levels
   b. Reticular substance of the medulla, pons, and mesencephalon
   c. Cerebellum
   d. Thalamus
   e. Areas of the cerebral cortex.

   Then, from the sensory areas, secondary signals are relayed to essentially all other parts of the nervous system.

iii. *Motor division (the effectors)* : The most important eventual role of the nervous system is to control the various bodily activities. This is achieved by controlling-

   a. Contraction of appropriate skeletal muscles throughout the body.
   b. Contraction of smooth muscle in the internal organs.
   c. Secretion by both exocrine and endocrine glands in many parts of the body.

   These activities are collectively called motor functions of the nervous system, and the muscles and glands are called effectors because they perform the functions dictated by the nerve signals.

   The skeletal muscles can be controlled from many levels of the central nervous system, including the-

   a. Spinal cord
   b. Reticular substance of the medulla, pons, and mesencephalon
   c. Basal ganglia
   d. Cerebellum
   e. Motor cortex.

   Each of these areas plays its own specific role, the lower regions being concerned primarily with automatic, instantaneous motor responses of the body to sensory stimuli, and the higher regions with deliberate movements controlled by the thought process of the cerebrum.

iv. *Processing of information- integrative; function of the nervous system* : One of the most important functions of the nervous system is to process incoming information in such a way that appropriate mental and motor responses will occur. More than 99 per cent of all sensory information is discarded by the brain as irrelevant and unimportant. For

instance, one is ordinarily unaware of the parts of the body that are in contact with clothing, as well as of the seat pressure when sitting. Likewise, attention is drawn only to an occasional object in one's field of vision, and even the perpetual noise of our surroundings is usually relegated to the subconscious.

v. *Storage of information-memory* : Only a small fraction of the important sensory information causes an immediate motor response. Much of the remainder is stored for future control of motor activities and for use in the thinking processes. Most of this information storage occurs in the *cerebral cortex*, but even the *basal regions of the brain* and the *spinal cord* can store small amounts of information. The storage of information is the process we call memory

vi. *Major levels of central nervous system function* : Three major levels of the central nervous system have specific functional attributes :

a. *Spinal cord level* : We often think of the spinal cord as being only a conduit for signals from the periphery of the body to the brain or in the opposite direction from the brain back to the body. This is far from the truth. Even after the spinal cord has been cut in the high neck legion many highly organized spinal cord functions still occur. For instance, neuronal circuits in the cord can cause -

1. Walking movements.
2. Reflexes that withdraw portions of the body from painful objects.
3. Reflexes that stiffen the legs to support the body against gravity.
4. Reflexes that control local blood vessels, gastrointestinal movements and reflexes that control urinary excretion.

In fact, the upper levels of the nervous system often operate not by sending signals directly to the periphery of the body but by sending signals to the control centers of the cord, simply *commanding* the cord centers to perform their functions.

b. *Lower brain or subcortical level* : Many, if not most, of what we call subconscious activities of the body are controlled in the lower areas of the brain-in the medulla, pons, mesencephalon, hypothalamus, thalamus, cerebellum, and basal ganglia.

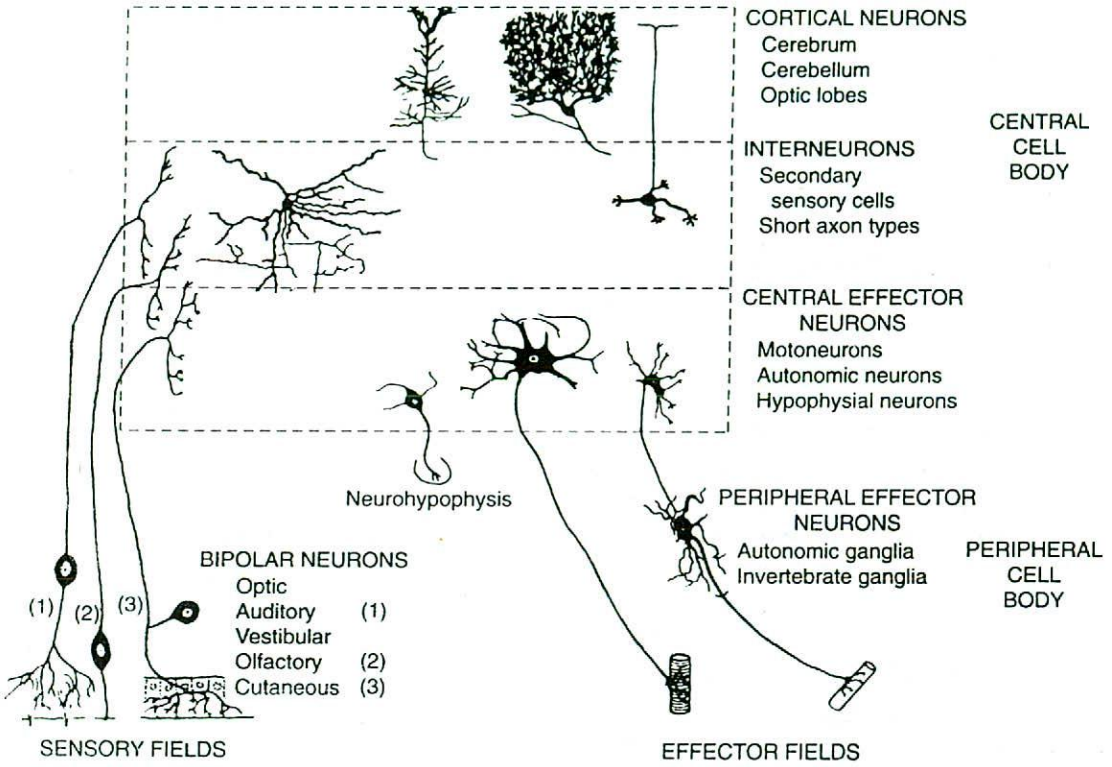

Fig. 16-1. *Some of the types of neurons in the mammalian nervous system.*

1. Subconscious control of arterial pressure and respiration is achieved mainly in the medulla and pons.
2. Control of equilibrium is a combined function of the older portions of the cerebellum and the reticular substance of the medulla, pons, and mesencephalon.
3. Feeding reflexes, such as salivation in response to the taste of food and the licking of the lips are controlled by areas in the medulla, pons, mesencephalon, amygdala, and hypothalamus.
4. Many emotional patterns, such as anger, excitement, sexual response, reaction to pain, and reaction to pleasure, can occur in animals after destruction of the cerebral cortex.

c. *Higher brain or cortical level* : Cerebral cortex is an extremely large memory storehouse. The cortex never functions alone but always in association with lower centers of the nervous system.

Without the cerebral cortex, the functions of the lower brain centers are often imprecise. The vast storehouse of cortical information usually converts these functions to determinative and precise operations.

Finally, the cerebral cortex is essential for most of our thought processes, but it cannot function by itself in this. In fact, it is the lower brain centers, not the cortex, that initiate *wakefulness* in the cerebral cortex, thus opening its bank of memories to the thinking machinery of the brain.

(Ref. Guyton & Hall 11th Edition; page 555-558)

Classification of nervous system

1. *Central nervous system* :
   a. Brain :
      i. Fore brain : cerebrum and diencephalons
      ii. Midbrain
      iii. Hind brain : pons, medulla oblongata, cerebellum
   b. Spinal cord :
      Cervical segments 8
      Thoracic segments 12
      Lumber segments 5
      Sacral segments 5
      Coccygeal segments 1.
2. *Perepheral nervous system* : (***somatic and autonomic***)
   i. Spinal nerves and their ganglia (31 pairs) :
      a. Cervical 8
      b. Thoracic 12
      c. Lumber 5
      d. Sacral 5
      e. Coccygeal 1.
   ii. Cranial nerves and their ganglia (12 pairs).

Autonomic nervous system

Introduction

The autonomic nervous system, like the somatic nervous system, is organized on the basis of the reflex arc. Impulses initiated in visceral receptors are relayed via afferent autonomic pathways to the CNS, integrated within it at various levels, and transmitted via efferent pathways to visceral effectors. This organization deserves emphasis because the functionally important afferent components have often been ignored.

(Ref. Ganong 22th edition; page 223)

General function of autonomic nervous system

I. *Functions of sympathetic system* :
   a. Generally prepares the body for action by increasing -
      i. Respiration
      ii. Blood pressure
      iii. Haert rate
      iv. Blood flow to the skeletal muscles.
   b. Dilating pupils of the eye.
   c. Generally slowing down the vsisceral function.

I. *Functions of parasympathetic system* :
   a. Functionally antagonistic to the sympathetic system by decreasing -
      i. Respiration
      ii. Blood pressure
      iii. Haert rate
      iv. Blood flow to the skeletal muscles.
   b. Constricting the pupils of the eye.
   c. Generally increasing the actions and functions of visceral system.

Thus the parasympathetic brings about the homeostasis.

Division of the autonomic nervous system :

a. *Anatomical division* :
   i. Sympathetic division
   ii. Parasympathetic division.
b. *Chemical divisions* :
   i. *Cholinergic division* :
   ii. *Noradrenergic division.*

(Ref. Ganong 22th edition; page 223, 224)

Anatomic organization of autonomic outflow

The peripheral motor portions of the autonomic nervous system are made up of

i. *Preganglionic neurons* :
   a. *Cell bodies* : The cell bodies of the preganglionic neurons are located in the visceral efferent intermediolateral gray column of the spinal cord on the homologous motor nuclei of the cranial nerves.
   b. *Axons* : Their axons are mostly mydinated, relatively slowly conducting B fibers.

ii. *Postganglionic* neurons : The *axons* synapse on the cell bodies of postganglionic neurons that are located in all cases outside the CNS. The axons of the postganglionic neurons, mostly unmyelinated C fibers, end on the visceral effectors.

Each *preganglionic* axon diverges to an average of eight or nine *postganglionic* neurons. In this way, autonomic output is diffused.

Anatomically, the autonomic outflow is divided into two components : the *sympathetic* and *parasympathetic* divisions of the autonomic nervous system. In the gastrointestinal tract, these both communicate with the *enteric nervous system*, and this is sometimes called a ***third division*** of the autonomic nervous system.

(Ref. Ganong 22th Edition; page 223)

Table 13-2. **Responses of effector organs to autonomic nerve impulses and circulating catecholamines.**

Effector Organs	*Cholinergic impulse response*	*Noradrenergic Impulses*	
		Receptor type[2]	*Response*
Eyes			
Radial muscle of iris	...	α_1	Contraction (mydriasis)
Sphincter muscle of iris	Contraction (miosis)	...	...
Ciliary muscle	Contraction for near vision	β_2	Relaxation for far vision
Heart			
S-A node	Decrease in heart rate, vagal arrest	β_1, β_2	Increase in heart rate
Atria	Decrease in contractility and (usually) increase in conduction velocity	β_1, β_2	Increase in contractility and conduction velocity
A-V node	Decrease in conduction velocity	β_1, β_2	Increase in conduction velocity
His-Purkinje system	Decrease in conduction velocity	β_1, β_2	Increase in conduction velocity
Ventricles	Decrease in contractility	β_1, β_2	Increase in contractility
Arterioles			
Coronary	Dilation	α_1, α_2	Constriction
		β_2	Dilation
Skin and mucosa	Dilation	α_1, α_2	Constriction
Skeletal muscle	Dilation	α_1	Constriction
		β_2	Dilation
Cerebral	Dilation	α_1	Constriction
Pulmonary	Dilation	α_1	Constriction
		β_2	Dilation
Abdominal viscera	...	α_1	Constriction
		β_2	Dilation
Salivary glands	Dilation	α_1, α_2	Constriction
Renal		α_1, α_2	Constriction
		β_1, β_2	Dilation
Systemic veins	...	α_1, α_2	Constriction
		β_2	Dilation
Lungs			
Bronchial muscle	Contraction	β_2	Relaxation
Bronchial glands	Stimulation	α_1	Inhibition
		β_2	Stimulation

(Ref. Ganong 21th Edition; page-229)

Table 13-2. **Responses of effector organs to autonomic nerve impulses and circulating catecholamines.**

Effector Organs	*Cholinergic impulse response*	*Noradrenergic impulses*	
		Receptor type[2]	*Response*
Stomach			
Motility and tone	Increase	$\alpha_1, \alpha_2, \beta_2$	Decrease (usually)
Sphincters	Relaxation (usually)	α_1	Contraction (usually)
Secretion	Stimulation	α_2	Inhibition
Intestine			
Motility and tone	Increase	$\alpha_1, \alpha_2, \beta_1, \beta_2$	Decrease (usually)
Sphincters	Relaxation (usually)	α_1	Contraction (usually)
Secretion	Stimulation	α_2	Inhibition
Gallbladder and ducts	Contraction	β_2	Relaxation
Urinary bladder			
Detrusor	Contraction	β_2	Relaxation (usually)
Trigone and sphincter	Relaxation	α_1	Contraction
Ureters			
Motility and tone	Increase (?)	α_1	Increase (usually)
Uterus	Variable[3]	α_1	Contraction (pregnant)
		β_2	Relaxation (pregnant andnonpregnant)
Male sex organs	Erection	α_1	Ejaculation
Skin			
Pilomotor muscles	...	α_1	Contraction
Sweat glands	Generalized secretion	α_1	Slight, localized secretion
Spleen capsule	...	α_1	Contraction
		β_2	Relaxation
Adrenal medulla	Secretion of epinephrine and norepinephrine	...	...
Liver	...	α_1, β_2	Glycogenolysis
Pancreas			
Acini	Increased secretion	α	Decreased secretion
Islets	Increased insulin and glucagon	α_2	Decreased insulin and glucagon secretion
		β_2	Increased insulin and glucagon secretion
Salivary glands	Profuse, watery secretion	α_1	Thick, viscous secretion
		β	Amylase secretion
Lacrimal glands	Secretion	α	Secretion
Nasopharyngeal glands	Secretion		...
Adipose tissue	...	$\alpha_1, \beta_1, \beta_3$	Lipolysis
Juxtaglomerular cells	...	β_1	Increased renin secretion
Pineal gland	...	β	Increased melatonin synthesis and secretion

(Ref. Ganong 21th Edition; page-230, 231)

Q. 02. **Give the difference between preganglionic and postganglionic neurons.**

Ans. Differences between preganglionic and postganglionic neurons :

Preganglionic neuron	*Postganglionic neuron*
a. Cell body is situated in the lateral gray column of the spinal cord and in the cranial nerve nuclei.	a. Cell body is situated in a ganglion (peripheral ganglion) outside the CNS.
b. The nerve fibres are myelinated.	b. The nerve fibres are unmyelinated.
c. Usually the fibres are long.	c. Usually the fibres are short.
d. Nerve endings act by liberating acetylcholine.	d. Nerve endings act by liberating adrenaline, noradrenaline or acetylcholine.

Table -2. ***Fast and slow responses of postganglionic neurons in sympathetic ganglia.***

Potential	*Duration*	*Mediator*	*Receptor*
Fast EPSP	30 ms	Acetylcholine	Nicotinic cholinergic
Slow IPSP	2 s	Dopamine	D_2
Slow EPSP	30 s	Acetylcholine	M_2 cholinergic
Late slow EPSP	4 mm	GnRH	GnRH

(Ref. Ganong 22th Edition; page226)

Anatomical autonomic outflow

Anatomically, the autonomic outflow is divided into two components :

i. Sympathetic
ii. Parasympathetic.

In the gastrointestinal tract, these both communicate with the *enteric nervous system*, and this is sometimes called a third division of the autonomic nervous system.

(Ref. Ganong 22th edition; page 223)

Sympathetic division : The axons of the sympathetic preganglionic neurons leave the spinal cord with the ventral roots of the first thoracic to the third or fourth lumbar spinal nerves.

i. They pass via the ***white rami communicantes*** to the ***paravertebral sympathetic ganglion chain***, where most of them end on the cell bodies of the postganglionic neurons.
ii. The axons of some of the postganglionic neurons pass to the viscera in the various sympathetic nerves. Others reenter the spinal nerves via the ***gray rami communicantes*** from the chain ganglia and are distributed to autonomic effectors in the areas supplied by these spinal nerves.
iii. The postganglionic sympathetic nerves to the head originate in the ***superior***, ***middle***, and ***stellate ganglia*** in the cranial extension of the sympathetic ganglion chain and travel to the effectors with the blood vessels.
iv. Some preganglionic neurons pass through the paravertebral ganglion chain and end on postganglionic neurons located in ***collateral ganglia*** close to the viscera.
v. Parts of the uterus and the male genital tract are innervated by a special system of ***short noradrenergic neurons*** with cell bodies in ganglia in or near these organs, and the preganglionic fibers to these postganglionic neurons presumably go all the way to the organs.

(Ref. Ganong 22th edition; page 223)

Transmission in sympathetic ganglia

At least in experimental animals, the responses produced in postganglionic neurons by stimulation of their preganglionic innervation include-

i. A rapid depolarization (fast EPSP) that generates action potentials
ii. A prolonged inhibitory postsynaptic potential (slow IPSP)
iii. A prolonged excitatory postsynaptic potential (slow EPSP)
iv. A late slow EPSP.

The late slow EPSP is very prolonged, lasting minutes rather than milliseconds. These slow responses apparently modulate and regulate transmission through the sympathetic ganglia. The initial depolarization is produced by acetylcholine via a nicotinic receptor. The slow IPSP is probably produced by dopamine, which is secreted by an *interneuron* within the ganglion. The interneuron is excited by activation of an M_2 muscarinic receptor. The interneurons that secrete dopamine are the small, intensely *fluorescent cells (SIF cells)* in the ganglia. The production of the slow IPSP does not appear to be mediated via cAMP, suggesting that a D_2 receptor is involved. The slow EPSP is produced by acetylcholine acting on a muscarinic receptor on the membrane of the postganglionic neuron. The late slow EPSP is produced by GnRH or a peptide closely resembling it.

(Ref. Ganong 22th Edition; page224)

Parasympathetic division :

i. *Cranial outflow* : The cranial outflow of the parasympathetic division supplies the visceral structures in the head via the oculomotor, facial, and glossôpharyngeal nerves, and those in the thorax and upper abdomen via the vagus nerves.

ii. *Sacral outflow* : The sacral outflow supplies the pelvic viscera via the pelvic branches of the second to fourth sacral spinal nerves.

The preganglionic fibers in both outflows end on short postganglionic neurons located on or near the visceral structures.

(Ref. Ganong 22th Edition; page223)

Q. 02. **Give the location of parasympathetic preganglionic and postganglionic neurons.**

Nerve	*Parasympathetic preganglionic neurons*	*Parasympathetic postganglionic neurons*
i. ***Cranial*** :		
Ocolomotor nerve	Edinger-Westphal nucleus in the mid brain	Cilliary ganglion
Facial nerve	Superior salivatory nucleus in the medulla oblongata	Sub-mandibular and pterygopalatine ganglia.
Glossopharyngial nerve	Inferior salivatory nucleus in the medulla oblongata	Otic ganglia
Vagus nerve	Dorsal nucleus of vagus in the medulla oblongata	Thoraeic and abdominal autonomic plexuses, close to or within the viscera supplied.
ii. ***Sacral*** : S-2, 3, 4	Anterior horn cells of the gray column of sacral segment of spinal cord.	Minute ganglia in the walls of pelvic viscera supplied.

Q. 02. **How do the sympathetic and parasympa- thetic divisions of the autonomic nervous system differ functionally?**

Ans. Functional differences between sympathetic and parasympathetic system :

Sympathetic	*Parasympathetic*
a. Prepares the body for defence in emergency.	a. Serves to comfort the body by promoting the orderly processes of the body.
b. Widespread function due to many postganglionic fibres and liberation of epinephrine and norepinephrine from suprarenal medulla.	b. Discrete action with few postganglionic fibres.
c. Catabolic function, mobi lizes body energies for dealing with increase in activity.	c. Anabolic function, conserves body energies.
d. Generally vasoconstrictor and raises blood pressure	d. Generally vasodilator, and lowers blood pressure
e. Accelerates heart rate, increases blood pressure, general constriction of cutaneous arteries thus causing increased blood supply to heart, muscle and brain.	e. Slows the heart(decreased heart rate decreases blood pressure). General vasodilator.
f. Provides a wider field of vision by dilating the pupil of the eye.	f. Constricts the pupil
g. Slows down peristalsis	g. Increases glandular and peristaltic activities.
h. Causes constriction of the sphincter except the sphincter pupillae.	h. Causes dilatation of the sphincter except the sphincter pupillae.

Chemical autonomic division

Chemical divisions of the autonomic nervous system : On the basis of the chemical mediator released, the autonomic nervous system can be divided into-

i. ***Cholinergic division*** : The neurons that are cholinergic are-
   a. All preganglionic neuron
   b. Anatomically parasympathetic postganglionic neurons
   c. Anatomically sympathetic postganglionic neurons which innervate sweat glands
   d. Anatomically sympathetic neurons which end on blood vessels in skeletal muscles and produce vasodilation when stimulated (sympathetic vasodilator nerves).

ii. ***Noradrenergic division*** : The remaining postganglionic sympathetic neurons are noradrenergic or, apparently, adrenergic in the case of the ICA cells.

The adrenal medulla is essentially a sympathetic ganglion in which the postganglionic cells have lost their axons and secrete norepinephrine, epinephrine, and some dopamine directly into the bloodstream.

The cholenergic preganglionic neurons to these cells have consequently become the secretomotor nerve supply of this gland. *(Ref. Ganong 22th Edition; page224)*

Chemical transmission of autonomic nervous system

Transmission at the synaptic junctions between pre and postganglionic neurons and between the postganglionic neurons and the autonomic effectors is chemically mediated the principal transmitter agents involved are *acetylcholine* and *norepinephrine*, although dopamine is also secreted by interneurons in the sympathetic ganglia and *GnRH* is secreted by some of the preganglionic neurons. GnRH mediates a slow excitatory response. In addition, there are cotransmitters in autonomic neurons; for example, VIP is released with acetylcholine, and ATP and neuropeptide Y with norepinephrine. VIP causes bronchodilation, and there may be a separate VIP secreting nonadrenergic noncholinergic nervous system innervating bronchial smooth muscle.

(Ref. Ganong 22th Edition; page223)

Neuron

A nerve cell with all its processes is called neuron.

Neuron is the structural and functional unit of nervous system. It consists of cell body or soma and two types of processes- axon and dendrite.

Zones of the neurons

Neurons generally have four important zones :

1. *Receptor or dendritic zone* : Where multiple local potential charges generated by synaptic connections are integrated.
2. *Action potential generated zone* : A site where propagated action potentials are generated i.e.
   a. The initial segment in spinal motor neurons.
   b. The initial node of Ranvier in cutaneous sensory neurons.
3. *Impulse propagation zone* : An axonal process that trnsmits propagated impulses to the nerve endings.
4. *Nerve endings* : Where action potential causes the release of synaptic transmitters.

The cell body is often located at the dendritic zone end of the axon, but it can be within the axon (i.e auditory neurons) or attatched to the side of the axon i.e cutaneous neurons. Its location makes no difference as far as the receptor function of the dendritic zone and transmission function of the axon are concerned.

It should be noted that integration of passive electrical activity is not the only function of dendrities, and flactuations in the sensitivity of individual dendrites alter their responses. Some nearons in the CNS have no axons, and local potentials pass from one dendrite to another. In other situations in the cortex, there is evidence for active as well as passive electrical responses to stimulation of dendrites.

(Ref. Ganong 22th edition)

Total number of neuron : 100 billion (about)

Dimensions of neurons : Spinal neuron supplying the muscles of foot, for example it has been calculated that if the cell body were the size of a tennis ball, the dendrities of the cell would fill an average sized living room and the axon would be upto 1.6 km (almost a mile) long although only 13 mm (0.5 inch) in diameter.

(Ref. Ganong 22th edition)

Function of neuron : Neuron is responsible for reception, conduction, integration and transmission of nerve impulse.

Classification of neuron

A. *According to number of processes :*
   i. *Unipolar :* Posseses a single process.
      Example : Only in foetus and lower vertebrae.
   ii. *Bipolar :* Here two processes present - One is axon and other is dendrite.
      Example : Inner nuclear layer of the retina and VIIIth cranial nerve nucleus.
   iii. *Pseudounipolar :* Pseudo means false. Originally these are bipolar but inturn two process fused into one which further divides into two. Ex : Present in dorsal root ganglia.
   iv. *Multipolar :* Posseses one axon and several dendrites.
      Ex : Pyramidal cell of cerebral cortex.

B. *According to function :*
   i. *Motor or efferent :* It carry impulse away from the cell body.

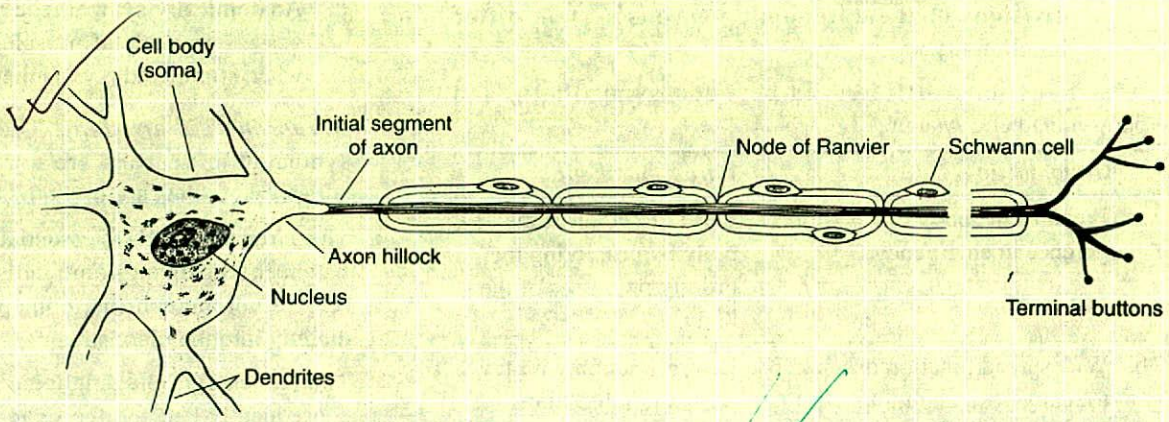

Fig. 16-2. *Motor neuron with myelinated axon*

ii. *Sensory or afferent :* It carry impules towards the cell body.

C. *According to position :*

i. *Upper Motor neuron :* Pyramidal cells and its axon is called upper motor neuron.

ii. *Lower motor neuron :* Anterior horn cells and its axon is called lower motor neuron.

D. *According to the length of the axon :*

i. *Golgi type I :* Contains very long axon that passes out the side of the grey matter and enters into the white matter.

ii. *Golgi type II :* Axon is short that does not leave the grey matter.

Resting membrane potential :

1. Large nerve fibers : -90 millivolts
2. Neural soma : -65 millivolts
3. Neurons : -70 millivolts.

Difference between axon and dendrite :

Axon	*Dendrite*
1. It carries impulse away from cell body.	1. It carries impulse toward the cell body.
2. It is single in each neuron.	2. Several in number from nill to many.
3. Generally long.	3. Generally short.
4. Usually unbranched.	4. Usually branched.
5. Histologically Nissle granules are absent.	5. Nissle granules are present.
6. Arises from axon hillock.	6. Arises from any part of the soma of the neuron.

Motor neuron

a. *Definition* : Motor neuron carries motor impulses from the CNS to the effector organs.

b. *Function* : Motor neurons are involved in :

i. Glandular secretion

ii. Muscular contraction

iii. Cardiac rates and respiratory movements.

c. *Types* : Two sets of motor neurons are involved to execute a motor function that are planned in motor cortex :

i. ***Upper motor neurons*** : The neurons in the brain and spinal cord that activate the motor neurons.

Effect of lesions :

a. Spastic paralysis

b. Hyperactive stretch reflexes

c. Absence of muscle atrophy.

However, there are three types of upper motor neurons to consider. Lesions in many of the posture-regulating pathways cause spastic paralysis, but lesions limited to the corticospinal and corticobulbar tracts produce weakness (paresis) rather than paralysis, and the affected musculature is generally hypotonic. Cerebellar lesions produce incoordination. The unmodified term upper motor neuron is therefore confusing.

ii. ***Lower motor neurons*** : The spinal and cranial motor neurons that directly innervate the muscles.

Effect of lesions :

a. Flaccid paralysis

b. Muscular atrophy

c. Absence of reflex responses.

(Ref. Ganong 22th edition; page 203)

Q. 00. **What are the differences between upper and lower motor neuron lesion?**

Ans. Differences between upper and lower motor neuron lesion :

Upper motor lesion :	*Lower motor lesion*
1. In upper motor neuron lesion paralized muscles are rigid due to increase muscle tone.	1. In upper motor neuron lesion paralized muscles become flaccid due to loss of muscle tone.
2. In upper motor neuron lesion deep reflexes are exaggerated & superficial reflexes lost.	2. In lower motor neuron lesion both superficial and deep reflexes lost.
3. In upper motor neuron lesion muscles are not wasted.	3. In lower motor neuron lesion muscles are degenerated & undergo wasting.
4. In upper motor neuron lesion Babinski sign positive.	4. In lower motor neuron lesion Babinski sign negative.

(Ref. Ganong 22th edition; page-203)

Q. 01. **Briefly describe the action potential of a neuron?**

Ans. Resting membrane potential of a neuron is usually about -70 mV.

A slight decrease in resting membrane potential leads to increased K^+ efflux and Cl^- influx restoring the resting membrane potential. However, when depolarization exceeds 7 mV, the voltage-gated Na^+ channels start to open at an increased rate (Na^+ channel activation), and when the firing level is reached, the influx of Na^+ along its inwardly directed concentration and electrical gradients is so great that it temporarily swamps the repolarizing forces.

The increase in Na^+ conductance is short-lived. The Na^+ channels rapidly enter a closed state called the inactivated state and remain in this state for a few milliseconds before returning

to the resting state. In addition, the direction of the electrical gradient for Na^+ is reversed during the over shoot because the membrane potential is reversed, and this limits Na^+ influx. A third factor producing repolarization is the opening of voltage-gated K^+ channels. This opening is slower and more prolonged than the opening of the Na^+ conductance. The net movement of positive charge out of the cell due to K^+ efflux at this time helps complete the process of repolarization. The slow return of the K^+ channels to the closed state also explains the after-hyperpolarization.

Althogh Na^+ enters the nerve cell and K^+ leaves it during the action potential, the number of ions involved is minute relative to the total numbers present. The fact that the nerve gains Na^+ and loses K^+ during activity has been demonstrated experimentally, but significant differences in ion concentrations can be measured only after prolonged, repeated stimulation.

The slower opening and delayed closing of the voltage-gated K^+ channels also explain accommodation. If depolarization occurs rapidly, the opening of the Na^+ channels overwhelms the repolarizing forces, but if the induced depolarization is produced slowly, the opening of K^+ channels balances the gradual opening of Na^+ channels, and an action potential does not occur.

A decrease in extracellular Ca^{++} concentration increases the excitability of nerve and muscle cells by decreasing the amount of depolarization necessary to initiate the changes in the Na^+ and K^+ conductance that produce the action potential. Conversely an increase in extracellular Ca^{++} concentration stabilizes the membrane by decreasing excitability.

(Ref. Ganong 22th Edition; Page 59)

Degenaration of neuron

a. *Definition :* The sum total destructive changes that happen to a nerve fibre after its injury or complete transection is called degeneration.

b. *Types of degeneration :* Degeneration are of two types-
   i. Retrograde degeneration : The degeneration that affect the cell body and proximal part of the cut fibre.
   ii. Wallerian degeneration : Degenerative changes that affect the distal part of the cut fibre.

c. *Causes of degeneration :*
   i. Transection of nerve fibre.
   ii. Crushing of nerve fibre.
   iii. Local injection of toxic substance.
   iv. Interference with blood supply.

(Ref. Wright's)

Changes take place after transection of a neuron

After transection nerve fibre degeneration takes place at two levels-

A. ***Retrograde degeneration*** :
   1. *Early change : Within 24 hours :*
      a. Nissle granules begins to disintegrate.
      b. Fragmentation and disintegration of Golgi apparatus & mitochondria.
      c. Cell body becomes spherical (due to imbibation of water.)
      d. Neurofibrin becomes tortuous and fragmented.
      e. Nucleus pushed to the periphery.
      f. Cell membrane shows degenerative change.
   2. *Late change :*
      a. Disappearance of Nissle granules, Golgi apparatus, neurofibril and endoplasmic reticulum.
      b. Chromatolysis : The above changes in the cell body and extrusion of the nucleus is known as chromatolysis.

B. ***Wallerian degeneration :***
   1. *Early changes : Within 24 hours :*
      a. Axon become swollen, neurofibrils become wavy, tortuous, granular and ultimately fragments out.
      b. Axon divides into numerous twisted segments.
      c. Myelin sheath degenerates.
      d. Schwann cells of neurolemal sheath shows proliferative change.
   2. *Late changes :*
      a. All the histological changes are intensified.
      b. The endoplasmic reticulum and plasma membrane disappear.
      c. Myelin sheath breaks into myelin drops and disappear.
      d. Invasion of macrophages that removes cellular debris.

Neuroglia

a. *Definition :* These are the non-excitable supporting connective tissues present in nervous system, called neuroglia.

b. *Classification of neuroglia :*
   1. Microglia
   2. Macroglia
      a. Astrocyte
      b. Oligodendrocyte
         i. Fibrous
         ii. Protoplasmic
      c. Glioblast.

c. *Functions of neuroglia :*
   i. Supporting
   ii. Insulation
   iii. Phagocytosis
   iv. Formation of myelin sheath.

Myelinogenesis

The process of formation and deposition of myelin sheath around a nerve fibre is called myelinogenesis.

Process of myelination :

i. *In peripheral nerve fibre :* Myelinogenesis begins with the growing of Schwann cell around the axon. The axon is first enveloped completely by the Schwann cell membrane. The Schwann cell membrane then gives several turns leaving the axon surrounded by many concentric layers. The cytoplasm disappear from this concentric layer. The compact myelin sheath consist of several concentric layer of Schwann cell membrane. The Schwann cell membrane consist of outer and inner layers of protein separated by a layer of lipid - sphingomyelin that act as a insulator. The myelin sheath over the axon are interrupted by constriction called node of Ranvier.

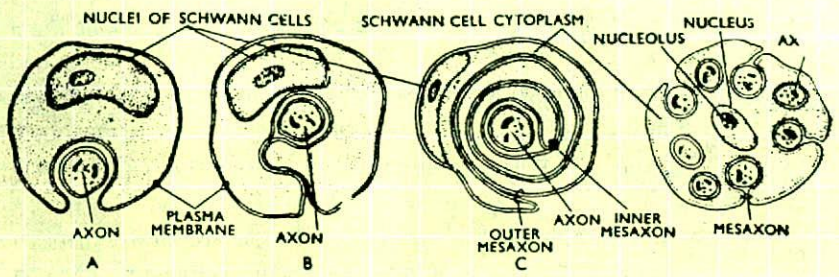

Fig. 16-3.Diagramatic representation of the process of myelination.

ii. *In CNS :* Myelinogenesis occured by the oligodendroglia similar to that of Schwann cell.

Nerve fibre

Definition : A nerve fibre is simply the long process of neuron which conduct nerve impulse.

Each nerve fibre has a central core of cytoplasm called axoplasm, surrounded by an outer membrane axolema. Axoplasm and axolema are collectively called axon cylinder.

Structure of a myelinated nerve fibre

Nerve fibre which is covered by myelin sheath is called myelinated fibre. A myelinated nerve fibre consist of following structures from within outwards-

i. *Axoplasm :* A central core of semifluid which flows from the cell body to the periphery. Axoplasm contains fibrilar components which run parallel to the axis of the fibre. Mitochondria and endoplasmic reticulum also present in it.

ii. *Axolema :* A membrane surrounding the axoplasm and separates it from surrounding structures.

iii. *Myelin sheath :*A sheath of phospholipid material outer to axolema which is a specialized set of Schwann cell membrane.

4. *Neurolema :* The outer most layer of a myelinated nerve.

(Ref. Wright's)

Properties of nerve fibre

1. *Excitability :*The nerve fibre can be stimulated by a stimulus of electrical, mechanical, chemical or thermal stimulus.
2. *Conductivity :* Nerve fibre can conduct impulse along both direction.
3. *Adaptation :* The nerve fibre quickly adapts itself. Due to this adaptation there is no excitation during the pasage of a constant current.
4. *Accomodation :* If a stronger stimulus is applied to a nerve very slowly there is no response due to lack of attaining threshold strength. This is accomodation.
5. *Infatiguability :* If a nerve fibre stimulated repeatedly, after a certain period it fails to give any response.
6. *All or none low :* If adequate stimulus is applied to a single nerve it will respone to its maximum.
7. *Summation :* In a nerve fibre summation of two submaximal stimuli is possible.

Calssification of nerve fibre

I. *According to the diameter and conduction velocity.*

Fiber type	*Diameter* mm	*Velocity of conduction* m/second	*Spike Duration* (ms)	*Absolute Refractory* peroid (ms)
A				
α	12-20	70-100		
β	5-12	30-70		
γ	3-6	15-30	0.4-0.5	0.4-1
δ	2-5	12-30		
B	<3	3-15	1.2	1.2
C				
Dorsal root	0.4-1.2	0.5-2	2	2
Sympathetic	0.3-1.3	0.7-2.3	2	2

N.B. *A and B fibers are myelinated; C fibers are unmyelinated.*

(Ref. Ganong 22th edition; page 61)

II. *Functionally :*
- i. Motor
- ii. Sensory.

III. *Histologically :*
- i. Myelinated
- ii. Un-myelinated.

IV. *Chemically :*
   i. Cholenergic : Producing acetylcholine
   ii. Adrenergic : Producing norepinephrin.

Numerical classification of sensory neuron

Numerical classification sometimes used for sensory neurons :

Number	*Origin*	*Fiber type*
Ia	Muscle spindle, annulo-spiral ending	Aα
Ib	Golgi tendon organ	Aα
II	Muscle spindle, flower spray ending touch, pressure	Aβ
III	Pain, and cold receptors; some touch receptors.	Aδ
IV.	Pain, temperature, and other receptors.	Dorsal root C

(Ref. Ganong 22th edition; page 61)

Relative susceptibility of mammalian A, B, C nerve fibers to conduction block produced by various agents.

Susceptibility to	*Most susceptible*	*Intermediate susceptible*	*Least susceptible*
Hypoxia	B	A	C
Pressure	A	B	C
Local anesthesia	c	B	A

(Ref. Ganong 22th edition; page 61)

Function of nerve fibre

I. A fiber :
   a. Aα fiber :
      i. Proprioception
      ii. Somatic motor.
   b. Aβ fiber :
      i. Touch
      ii. Pressure
      iii. motor.
   c. Aγ fiber :
      i. Motor to muscle spindle
   d. Aδ fiber :
      i. Pain
      ii. Cold
      iii. touch.

II. B fiber :
   i. Preganglionic autonomic.

III. C fiber :
   a. Dorsal root :
      i. Pain
      ii. Temperature
      iii. Some mechanoreception
      iv. Reflex responses.
   b. Sympathetic :
      i. Postgaglionic sympathetic.

(Ref. Ganong 22th edition; page 61)

Degree of neve injury

Sunderland's degree of injury :

a. *First degree injures* : These are pressure being applied to a nerve for a limited time thus occluding its blood supply and resulting in a local anoxia which impair the nerve function.

   In it, axon is not destroyed but merely losses its functional properties for a short time which returns within a few hours to few weeks.

b. *Second degree of injuries* : These are the results of prolonged and or severe pressure being exerted on some part of the nerve.

   Death of the axon at the site of pressure followed by the death of the axon distal to it, because the axon is separated from the cell body. Chromatolysis occurs in the cell body.

   Regeneration of axon is facilitated by the presence of uninterrupted endoneurial tubes.

c. *Third, fourth, and fifth degree injuries* : *Third degree* injuries are characterized by the endoneurial tubes becoming interrupted, *fourth degree* injuries by the fascicles becoming disorganized; and *fifth degree* injuries by the nerve trunk being severed.

(Ref. Wright's)

Spinal cord

1. *Anatomy* : The spinal cord is that part of CNS which is contained within the vertebral canal & is the prolongation of the brain.

2. *Development* : It developes from that portion of neural tube which lies caudal to the level of the 4th pair of somites.

3. *Lengths* : It is about 45 cm (18 inch) long & about 1.25 cm (0.5 inch) wide.

4. *Extention* : From the horizontal plane passing between the middle of the odontoid process of the 2nd cervical vertebra & the upper border of the posterior arch of the atlas & terminates below in a conical extremity, the *conus medullaris* at the lower border of the body of 1st lumbar vertebra.

5. *Enlargements* : It presents two enlargements - *cervical & lumber*. The *cervical enlargement* extends from the level of 3rd cervical to the 2nd thoracic segment of the spinal cord. From this enlargements nerves for the upper extremities emerge out.

   The lumbar enlargement extends from the ninth thoracic

vertebra to the twelfth thoracic vertebra. From it nerves for lower extremities emerge out.

6. *Filum terminale* : A delicate, thread like non-nervous filament, about 20 cms long. Extends from the lower end of conus medullaries and terminates by being attached to the dorsal aspect of first coccygeal vertebra.
7. *Coverings* : It has three coverings; from without inwards- dura mater, arachnoid mater & pia mater. The space between the arachnoid and pia mater is subarachnoid space which contain CSF and extends as down as the 2nd sacral vertebra.

Parts of spinal cord

On cross section, the spinal cord has two parts :

1. *Central- grey mater :* It is composed of a mass of nerve cells, nerve fibres consisting of both axons & dendrons together with a framework of neuroglial cells. It is divided into two symetrical halves which are connected by a transverse band, resemble the letter H. It has three column (horn)-
   a. Anterior horn
   b. Lateral horn (only in thoracic horn)
   c. Posterior horn.
2. *Peripheral- white matter :* It surrounds the grey matter and consists of nerve fibres & neuroglia. It appear as white because most of the fibre are myelinated.
3. *Central canal :* It exists through out the spinal cord and is continuous above with the inferior angle of 4th ventricle and below it expands as far down as 2" within filum terminale.

Functions of spinal cord

1. It is the main pathway for all incoming and outgoing impulses from the higher centre to the periphery & vice versa.
2. It is the main centre of reflex activites.
3. It exerts traphic control over the muscular system.

Spinal cord transection

a. *Causes of complete transection of spinal cord*
   i. Gun shut injury
   ii. Fracture, dislocation
   iii. Acute transverse myelitis.

b. ***Effects of complete transection of spinal cord***

The effects depend on the site of section.

I. *Above the 5th cervical segment* : The subject dies due to respirtatory failure or due to involvement of phrenic & intercostal nerves.

II. *Section between C_5-T_1* : The subject may survive with quadriplegia & the effects may be summarized in the following stages-

1. *Stage of spinal shock :* This stage follows immediately after the section & lasts for a variable time-
   a. Muscles are completely flaccid at and below the level of section and all the four limbs are paralized.
   b. Lack of responsiveness to stimulus below the section.
   c. All reflexes are lost except anal reflex.
   d. Sphincters are paralyzd.
   e. Retention of urine and faeces is present.
   f. Priapism (Persistent erection of penis) occurs, immediately after ingury.
   g. BP may or may not be effected.
2. *Stage of recovery or reflex activity :* After a variable time as the stage of shock passes off, depeding on the condition of the patient, functional activity appears; that is-
   a. Return of reflex tone,
   b. Involuntary movement,
   c. Return of reflex movement.
3. *Stage of failure of reflex activity.*

III. *Section at lumbar region :*
   i. Flaccid paralysis of lower limbs
   ii. Loss of all reflexes.

Causes of spinal shock : It is neither due to trauma nor due to fall of blood pressure. It is believed to be due to cutting off of the impulses from the higher centres which are excitatory to the spinal centres.

Effects of section of anterior root

1. *Functional changes* :
   a. Flaccid paralysis of the affected muscles.
   b. Loss of reflexes.
   c. Muscualr wasting.
   d. Reaction of degenaration present.
   e. Sympathetic paralysis.
2. *Degenarative changes* :
   a. Degenaration of peripheral portion.
   b. Degenaration of white rami communication upto sympathetic ganglion.
   c. Chromatolysis of anterior and lateral horn cells.

Effects of section of posterior root

1. *Functional changes :*
   a. Loss of all sensation.
   b. Incordinated movements of the muscles due to loss of kinaesthetic sensation.
   c. Loss of muscle tone and reflexes.
   d. Trophic ulcers due to loss of sensation.

e. Vasomotor disturbances due to degenaration of the antidromic vasodilator fibres.

2. *Degenarative changes :*
   a. If distal to the ganglion-
      i. Degenaration of the peripheral fibres upto the receptor organs.
      ii. Degenaration of the fibres of the recurrent sensibility.
      iii. Degenaration of the antidromic vasodilator fibres.
      iv. Chromatolysis of the ganglion cells.
   b. *If proximal to the ganglion* :
      i. Degenaration of tract of Gall & Burdach and of Lissauer & Comma (descending) tract upto the next neuron.
      ii. Chromatolysis of the ganglion cells.

Effects of hemisection of the spinal cord

Hemisection means section or lesion which involves one lateral half of spinal cord.

Effects :

i. *Degenartive change :*
   a. Cellular damage,
   b. Damage to fibres

ii. *Functional change :*
   a. Above the level of section
   b. At the level of section
   c. Below the level of section.

Degenarative change :

1. *Neural cell damage :*
   a. Damage to anterior horn cells lead to their destruction including the motor end plate and no regenaration occurs.
   b. Damage to posterior horn cells lead to degenaration of second neurons in the sensory pathway for pain and temperature sensation of same side.
   c. Damage to lateral horn cells lead to degenaration of the preganglionic viceromotor, pilomotor & vasomotor nerves.
2. *Damge to fibres :*
   a. Distal part of the divided fibres undergoes degenaration and involving the end-organs in connection with it.
   b. Proximal part of the fibre & the related nerve cells also degenarates for a variable extent.
   c. There may be associated transneuronal degenaration.

Functional change :

1. *Below the level of section :*
   a. *Same side :*
      i. Sensory change : Loss of fine touch, tactile localization, tactile discrimination and kinesthetic sensation due to the damage of tract of Gall and Burdach. But crude touch, pain and thermal sensation remain unchange.
      ii. Motor change : Extensive paralysis of upper motor type due to destruction of pyramidal tract.
   b. *Opposite side :*
      i. Sensory Change : Loss of touch, pain and thermal sensation due to damage of spinothalamic tract. But fine touch, tactile localization, tactile discrimination and Kinesthetic sensation remain unchanged.
      ii. Motor change : A few paralysis may occur.
2. *At the level of section :*
   a. *Same side :*
      i. Sensory change : Complete anesthetia due to destruction of posterior nerve root, posterior horn cell.
      ii. Motor change : Lower motor type of paralysis due to destruction of anterior horn cell.
   b. *Oppoisite side :*
      i. Sensory change : Some loss of pain sensation due to destruction of crossed horizontal spinothalamic tract.
      ii. Motor change : Nill or very slight because some direct fibres may injuried.
3. *Above the level of section :*
   a. *Same side* : Hyperaesthesia due to irritation of the upper cut end of the damaged fibre (possibly).
   b. *Opposite side* : Hyperasthesia may be referred.

Brown sequard syndrom

It is found that below the level of hemisection of spinal cord there is extensive sensory loss but little motor loss of opposite side and where in the same side there is extensive motor loss but little sensory loss. This phenomenon is known as Brown sequard syndrom.

Babinski sign

Normally scratching of the skin of the sole causes planter flexion of great toe. But in case of infants and pyramidal tract lesion at any level above the 1st sacral segment, this normal response is changed into dorsiflexion of great toe, with faning of other toes is called Babinski sign.

Synapse

Definition : Synapse is the junction between two neurons where one neuron ends and other neuron begins. It is the functional continuty between two neurons.

Functional anatomy of synapse : Before synapsing with a cell body or process the axon terminals of the presynaptic neuron enlarge called synaptic knob, terminal knobs, butons, or end-

feet. The membrane of the synaptic knob is called presynaptic membrane and that of the cell body or process of the post synaptic neuron is called postsynaptic membrane. Both these membrane are separated by synaptic cleft having a width

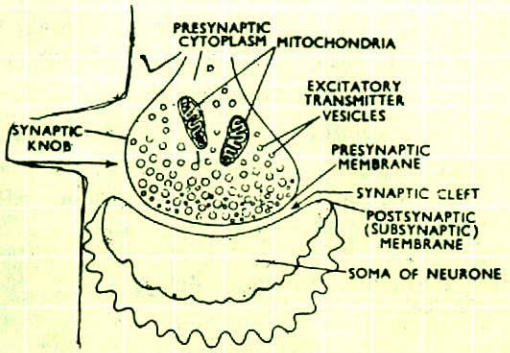

Fig. 16-4. Diagram of Synapse.

usually of 200-300 angstrom. The presynaptic knob contains numerous mitochondria and vesicles which contain neurotransmitter substances. The mitochondria supplies energy that is needed for the synthesis of the neurotransmitter substances.

Function of synapse :

1. Conduction or transmission of impulse.
2. Integration of impulse.
3. Formation of reflex arc.
4. Acts as a connection spot in neural chain.
5. Modulation of neural activity.

Classification of synapse

a. *According to structural organization :*
   i. *Axosomatic :* Axon of one neuron synapses with the cell body of other neuron.
   Example : Between the basket cell and purkinjee cell in the cerebellum.
   ii. *Axodendritic :* Axon of one neuron synapses with dendrite of other neuron. Ex. between the climbing fibres and the dendrite of the purkinjee cell.
   iii. *Axoaxonic :* Axon of one neuron synapses with axon of other neuron.

b. *According to the nature of neurotransmitter releases* :
   i. *Cholenergic synapse :* Releases acetyle choline.
   ii. *Adrenergic synapse :* Releases norepinephrine and epinephrine.

Properties of synapse

1. *One way conduction :* Synapse always conduct impulse one way that is from presynaptic neuron to the post synaptic neuron. It is due to present of abundant neurotransmitter in the presynaptic neuron.
2. *Synaptic delay :* It is the time taken by an impulse to cross the synapse. It is about 0.5 milli second.
   Cause :
   a. Delay in the release of neurotransmitters.
   b. Passing of neurotransmitters through the synaptic cleft.
   c. Delay in the combination of neurotransmitter with post synaptic receptors.
   d. Delay in the set up of action potential.
3. *Fatigue :* When a synapse is rapidly and repeatedly stimulated, the power of conduction of impulse will progressively become less, called fatigue of synapse. It is due to-
   i. Exhausness of neurotransmitter substances
   ii. Progressive inactivation of the receptor of the post synaptic neuron.
4. *Synaptic response :* Response to the impulse received by synapse may be similar or dissimilar. Sometime one neuron receives many impulse via synapse but it integrates and discharge its own.
5. *Inhibition :* It is an active process which either prevents the onset of activity in a structure or stops the activity already present. It may be direct, indirect, presynaptic, post synaptic and negative feed back inhibition.

Synaptic transmission : When an action potential arrives at the presynaptic terminal, it will depolarize the presynaptic membrane, causing opening of the Ca^{++} chanel and Ca^{++} from synaptic cleft enters into synaptic knob binds with receptor of presynaptic membrane. This Ca^{++} receptor binding causes the

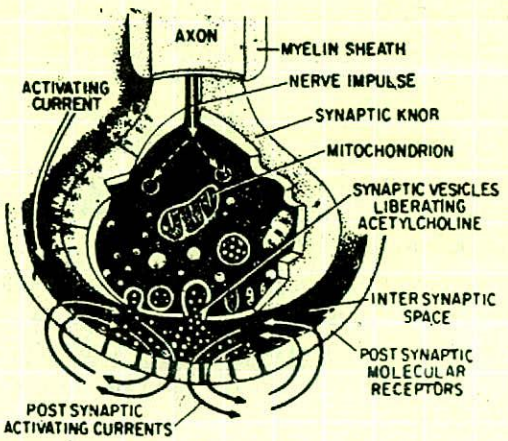

Fig. 16-5. Synaptic transmission.

vesicles bind with membrane, fuse with it and finally burst and neurotransmitter releases into synaptic cleft, passes through this cleft and binds with the specific receptors of the post synaptic membrane. The post synaptic membrane has Na^+, K^+ and CI^- chanel. The transmitter which open the Na^+ chanel are called excitatory and those which open the K^+ or CI^- chanel are called inhibitory impulse.

Q. 04. **What are the types of synaptic transmission?**

Ans. Types of synaptic transmission :

i. Chemical transmission
ii. Electircal transmission
iii. Both chemical and electrical transmission.

Characteristics of synaptic transmission : Some special characteristics of synaptic transmission are :

i. Summation :
   a. Spatial suntmation
   b. Temporal summation
ii. Synaptic delay
iii. Synaptic inhibition
   a. Direct inhibition
   b. Indirect inhibition
   c. Presynaptic inhibition
   d. Postsynaptic inhibition.
   e. Negative feed back inhibition.

Presynaptic neuron : Neuron which release neurotransmitters.

Postsynaptic neuron : Neuron on which neurotransmitters act.

Chemical synapse

i. *Definition* : Chemical synapses are the most common types of synapses present in human body. The neurons communicate via chemical transmitter i.e. neurotransmitters such as acetylecholine, serotonin.

   This chemical mediators binds to receptors on the surface of the post synaptic cell, and this triggers events that open or close channels in the membrane of the post synaptic cells.

   The chemical synapses transmit signals in one way direction i.e. unidirectional.

ii. *Morphology of a chemical synapse* : (same as the funtional anatomy of a synapse).

Electrical synapses :

i. *Definition* : In electrical synapses, the membranes of the presynaptic and postsynaptic neurons come close together, and gap junctions form between the cells.

   Like the intercellular junctions in other tissues, these junctions form low-resistance bridges through which ions pass with relative ease. There is electrical coupling, for example, between some of the neurons in the lateral vestibular nucleus.

   The electrical synapses transmit signals in two way direction i.e. bidirectional.

Q. 02. **Briefly describe the summation of synaptic transmission.**

Ans. *Summation of synaptic transmisssion* :

i. *Definition* : In synaptic transmission multiple excitatory or inhibitory stimuli can be summated and thus produce EPSP or IPSP respectively.

ii. Types : Summation is of two types spatial summation and temporal summation-
   a. *Spatial suntmation* : The effects of simultaneous discharge of many synaptic knobs over a wide area of the postsynaptic terminal can summate i.e. activity in one synaptic knob is said to facilitate activity in another to approach the firing level and produce FPSPs or IPSPs. This is called spatial summation.
   b. *Temporal summation* : The effect of successive discharge of a presynaptic terminal on the postsynaptic membrane can also summate and produce EPSPs or IPSPs. This is called temporal summation.

Q. 02. **Briefly describe the synaptic delay.**

Ans. *Synaptic delay* :

i. *Definition* : A certain amount of time is consumed in the transmission of an action potential from a presynaptic neuron to a postsynaptic neuron. This is called synaptic delay.
ii. *Duration* : About 0.5 millisecond.
iii. *Causes* :
   a. Delay in the release of neurotransmitters.
   b. Passing of neurotransmitters through the synaptic cleft.
   c. Delay in the combination of neurotransmitter with post synaptic receptors.
   d. Delay in the set up of action potential.
iv. *Importance* : To determine the synaptic pathway whether it is monosynaptic or polysynaptic. Minimum delay across one synapse is 0.5 ms. Delay > 0.5 ms indicates numbers of series of neurons in the circuits :
   * In monosynaptic pathway : delay 0.5 milli second.
   * In polysynaptic pathways : delay will be> 0.5 milli second.

Q. 02. **Briefly describe the synaptic inhibition.**

Ans. *Synaptic inhibition* :

i. *Definition* : Inhibition of synapses.
ii. Types : Synaptic inhibition may be of following types :
   a. *Direct inhibition* : Inhibition during the course of an IPSP is direct inhibition.
   b. *Indirect inhibition* : Inhibition during refractory period is called indirect inhibition.
   c. *Presynaptic inhibition* : The inhibition caused by *presynaptic synapse (axoaxonal)* that lies on the terminal nerve fibrils before they themselves terminate on the following neuron i.e. postsynaptic neuron.

      Mechanisms : Activation of these synapse on the presynaptic terminal causes :
      * Increase Cl^- influx in the presynaptic terminal and thus reduces amplitude of the action potential in the presynaptic terminal that causes reduced Ca^{++} influx

in the presynaptic terminal for neurotrailsmitter release.

* Activation of these synapse also cause voltage gated K^+ channels to open and the resulting K^+ efflux also decreases Ca^{++} influx.
* Activation of these synapses cause direct inhibition of transmitter release indepen- dent of Ca^{++} influx into the excitatory ending.

d. *Postsynaptic inhibition* : The inhibitions caused by inhibitory interneuron that make synapse with presynaptic terminal and ends on postsynaptic membrane and secrete inhibitory transmitter i.e glycin.

Excitatory post synaptic potential (EPSP) : When an excitatory impulse excites a motor neuron, depolarization of the membrane occurs. This is called EPSP. When the stimulus is stronger, EPSP reaches the threshold level and the nerve impulse is set up.

Cause : The excitatory impulse increases the Na^+ permeability to the post synaptic membrane and causes EPSP.

Inhibitory post synaptic Potential (IPSP) : When a motor neuron receives an inhibitory impulse, hyperpolarization of the cell membrane occurs. This is called IPSP. The hyperpolarization exerts an inhibitory effect on EPSP and depolarization of the cell membrane and causes inhibition in setting up the nerve impulse.

Cause : IPSP is due to the increased permeability of post synaptic membrane to K^+ & CI^- instead of Na^+.

Neurotransmiter

These are highly active chemical agents releases at the nerve ending and transmits impulses from nerve to nerve or from nerve to effector tissue.

Some neurotransmitter :

1. *Excitatory*
   a. Acctylcholine
   b. Adrenaline
   c. Noradrenaline
   d. Glutamate.
2. *Inhibitory*
   a. Dopamine
   b. Glycin
   c. Taurine
   d. Alanine.
   e. GABA
   f. Serotonin.
3. *Both excitatory and inhibitory*
   a. 5-HT (hydroxytryptamine)
   b. Histamine
   c. Prostaglandine.
   d. Nor-epinephrine.

(Ref. Guyton & Hall 11th edition; page 562)

Small molecule, rapidly acting transmitters

Class I : *Acetylecholile*

Class II : *The amines* :
Norepinephrine
Epinephrine
Dopamine
Serotonin
Histamine.

Class III : *Amino acids* :
Gama Aminobutyric acid (GABA)
Glutamate
Asparate.

Class IV : Nitric Oxide (NO).

(Ref. Guyton & Hall 11th edition; page 562)

Neuropeptides, slowly acting transmitters

a. *Hypothalamic releasing hormones :*
Thyrotropin releasing hormone
Luteinizing hormone-releasing hormone
Somatostatin (growth hormone inhibitory factor).

b. *Pituitary peptides :*
ACTH
Beta Endorphin
Alpha melanocyte-stimulating hormone
Prolactin
Luteinizing hormone
Thyrotropin
Growth hormone
Vasopressin
Oxytocin.

c. *Peptides that acts on gut and brain :*
Leucine enkephalin
Methionine enkephalin
Substance P
Gastrin
Cholecystokinin
Vasoactive intestinal polypeptide (VIP)
Neurotensin
Insulin
Glucagon.

d. *From other tissues :*
Angiotensin II
Bradykinin
Carnosine

Ref. Guyton & Hall 11th edition.

Sleep peptides
Calcitonin.

(Ref. Guyton & Hall 11th edition; page 563)

Site of acetylcholine secretion : *Acetylcholine is secreted by* :

i. The preganglionic neuron of autonomic nervous system.
ii. The post ganglionic neuron of parasympathetic system.
iii. The motor neurons innervate the skeletal muscle.
iv. The postganglionic neuron of sympathetic system that innervate the sweat and sebaceous gland.
v. Also from brain, cortex and basal ganglia.

Site of Adrenaline secretion : *Adrenaline is secreted by-*

i. The post ganglionic neuron of sympathetic system, except those innervate the sweat and sebaceous gland.
ii. The adrenal mdulla.

Q. 00. **Briefly discuss the sites of synthesis of the neurotransmitters.**

Ans. Sites of synthesis of neurotransmitters :

a. *Cytosol of the presynaptic terminal* : Principal neurotransmitters (small molecular types of transmitters) are synthesized in the cytosol of the presynaptic terminal.
b. *Ribosome in the neuronal cell body* : Neuro- peptides.
c. *Cell organelles* : That take part in the synthesis of neurotransmitter are :
   * Nisslgranules
   * Golgi complex
   * Mitochondria.

Q. 00. **Briefly discuss the storage of the neuro-transmitters.**

Ans. *Storage of neurotransmitters* : Neurotrans- mitters are stored in the synaptic vesicles. The neurotransmitter vesicles are distributed throughout the presynaptic nerve terminal. There are three kinds of synaptic vesicles :

a. *Small and clear synaptic vesicles* : Contain acetylcholine, GABA and glycin.
b. *Small with a dense vesicle* : Contain catecholamines.
c. *Large vesicles with a dense core* : Contain neuropeptides.

Q. 00. **Briefly discuss the mechanism of neuro-transmitter release.**

Ans. *Mechanism of neurotransmitter release* : Neuro-transmitter vesicles discharge their contents into the synaptic cleft by the process of exocytosis which requires :

a. Ca^{++} ions :
b. Energy
c. Docking proteins : synaptobrevin and syntaxin.

Ca^{++} ions are the key to synaptic vesicle fusion and discharge. The active zone (area of membrane thickening) of the presynaptic terminal contains rows of Ca^{++} channels. The quantity of transmitter release is directly proportional to the number of Ca^{++} influx.

Thus the processes of release are :

a. Action potential in the presynaptic nerve terminal opens voltage gated Ca^{++} channels and thus Ca^{++} influx occurs in the presynaptic terminal.
b. Ca^{++} influx triggers the release of neurotransmitter by binding and fusion of synaptic vesicle with the presynaptic memhrane. Protein that interact to produce synaptic vesicle docking and fusion with the presynaptic membrane are synaptobrevin in synaptic vesicle and syntaxin in the presynaptic membrane.
c. The area of fusion breaks down and transmitters are released into the synaptic cleft.

Small clear vesicles and the small dense core vesicles recycle in the ending.

Large dense core vesicles are autolysed and are not reused.

The Ca^{++} concentration is restored to the resting level by rapid sequestration and removal from the cell, primarily by a Ca^{++}-Na^{+} antiport.

Q. 00. **Briefly discuss the fate of neurotransmitter.**

Ans. Fate of neurotransmitter : After a transmitter is released at a nerve ending, it is either destroyed or removed from the synaptic cleft. The functional significance of this is that it prevents an undesirable sustained effect of neurotransmitter on the postsynap- tic neuron.

This removal is achieved by-

a. By *diffusion* of the transmitter out of the cleft into the surrounding fluids.
b. By enzymatic destruction within the cleft itself.

$$\text{Acetylcholine} \xrightarrow{\textit{Cholinesterase}} \text{Acetate} + \text{choline}$$

Catecholamines (adrenaline, noradrenaline) by monoamine oxidase (MAO).

c. By reuptake of transmitter into the presynaptic terminal by active transport.

Q. 00. **What is neuromodulators** (co-transmitter)?

i. *Definition* : Neuromodulators or neuropeptides are the chemical substances that can co-exist with the principal transmitter at a single synapse i.e in separate synaptic vesicle or in the same vesicle and are ejected from the presynaptic terminal into the synaptic cleft having the capability of modulating and modifying the activity of the principal transmitter.

ii. *Examples* :
   a. Substance - P
   b. Neurotensin
   c. Bradykinin
   d. Adenosine
   e. Neuropeptide-Y.

Q. 03. **Compare the principle neurotransmitters and neuromodulators.**

Ans. Comparison between the principle neurotrans- mitters and neuromodulators :

Points	*Principle neurotransmitter*	*Neuromodulator*
Action	Rapid excitation or inhibition of post synaptic membrane activity	Modulation and modification of principal neurotrans-mitter.
Site of action	Directly acts on postsynaptic membrane	Have no direct action on postsynaptic membrane
Duration	Short	Prolonged
Sites of synthesis	Cytosol of presynaptic nerve	In the soma by ribosomes
Location	Main sensory and motor system	Systems that control homeostasis
Size	Small molecule	Large molecule
Amount released	Large quantity	Small quantity
Potency	Less	> 1000 time potent
Receptor	Ion channels	G protein coupled receptor
Types of action.	Acute response	Delayed response

Neuromuscular junction

i. *Definition* : The junction between muscle fibre and motor nerve ending is called neuromuscular junction.

ii. *Description of a neuromuscular junction* :

a. *Axon terminal or end feet :* At the junction the nerve fibre losses myelin sheath and ends by dividing into many branches called axon terminal or end feet.
Content of axon terminals : It contain mito- chondria which provides energy and vesicles which contain neurotransmitter.

b. *Synaptic gutter* : The axon terminal lie within the synaptic gutter which is formed by the invagination of *sarcolema into sarcoplasm.*

c. *Synaptic cleft* : The gap between sarcolema and end feet is called synaptic cleft which is filled with ECF.

d. *Motor end plate* : The thickening portion of muscle fibre adjacent to end feet is called motor end plate.

Neuromuscular transmission

Sequence of event of neuromuscular transmission are given below :

i. Arrival of motor impulse at motor nerve terminal.

ii. Enter of Ca^{++} into the vesicles of end feet.

iii. Release of acetylcholine from prejunctional nerve terminal vesicle.

iv. Diffusion of acetylcholine across the synaptic cleft.

v. Acetylcholine binds to the receptor of motor end plate and increases the permeability of Na^{+} and production of local end plate potential (depolarization).

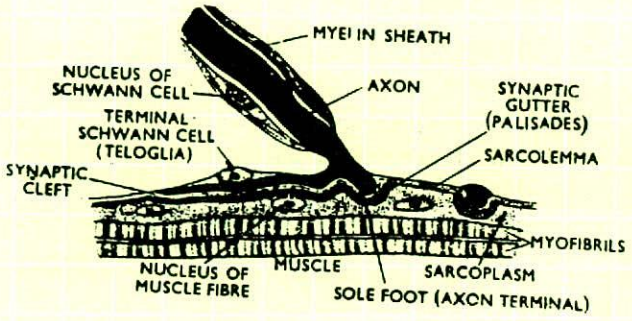

Fig. 16-6. Neuromuscular junction.

vi. When the end plate potential reaches the certain critical magnitude then the muscular membrane will be depolarized and set up a propagated action potential in muscle causes to the spike potential.

vii. Then the spike potential preceed and mechanical tension of muscle takes place.

(Ref. Wright's 13th page-257)

Receptor

Definition : Receptors are the specialised tissue that can be stimulated by any changes of both external or internal environment.

Classification of sensory receptors

I. ***Mechanoreceptors*** :

Skin tactile sensibilities
(epidermis and dermis)
Free nerve endings
Expanded tip endings
Merkel's discs
Plus several other variants
Spray endings
Ruffini's endings
Encapsulated endings
Meissner's corpuscles
Krause's corpuscles
Hair end organs

Deep tissue sensibilities
- Free nerve endings
- Expanded tip endings
- Spray endings
  - Ruffini's endings
- Encapsulated endings
  - Pacinian corpuscles
  - Plus a few other variants
- Muscle endings
  - Muscle spindles
  - Golgi tendon receptors
- Hearing
  - Sound receptors of cochlea
- Equilibrium
  - Vestibular receptors
- Arterial pressure
  - Baroreceptors of carotid sinuses and aorta

II. ***Thermoreceptors***
- Cold : Cold receptors
- Warmth : Warm receptors

III. ***Nociceptors***
- Pain : Free nerve endings

IV. ***Electromagnetic receptors***
- Vision
  - Rods
  - Cones

V. ***Chemoreceptors***
- Taste : Receptors of taste buds
- Smell : Receptors of olfactory epitheliurn
- Arterial oxygen
  - Receptors of aortic and carotid bodies
- Osmolality
  - Probably neurons in or near supraoptic nuclei
- Blood CO_2
  - Receptors in or on surface of medulla and in aortic and carotid bodies
- Blood glucose, amino acids, fatty acids
  - Receptors in hypothalamus.

(Ref. Guyton & Hall10th Edition; page-529)

Classification of receptor *(In Short)* : It is difficult to classify receptors. Different in structure, location & function they are of various types. Few of them are postullated below :

A. ***According to sensitivity to a particular form of energy :***

1. *Mechanoreceptor :* Which concern with mechanical deformation of receptor.
   Example : Baroreceptors of carotid and aortic sinuses.
2. *Thermoreceptor :* Which detect the change of temperature.
   Example : End bulb of Krause, End organ of Ruffini.
3. *Naciceptor :* Which detect the damage of tissue.
   Example : Free nerve ending (pain receptor).
4. *Electromagnatic receptor :* Which detect light on the retina of the eye.
   Example : Rod, cone.
5. *Chemoreceptor :* Detect any chemical change of the body.
   Example : Carotid and aortic bodies.

(Ref. Guyton & Hall 11th edition; page 529)

B. ***Accroding to distribution*** :

1. *Exteroceptor :* These are distributed to body surface and are stimulated by changes in external environment.
2. *Proprioceptor :* These are distributed to the muscle, tendons, joints etc.
3. *Interoceptor :* These are distributed to the viscera, blood vessels etc.

(Ref. Wright's 13th page-285)

C. ***Broadly, receptors are classified into*** :

1. *Exteroceptors :* Those which collect information from out side of the body. It is subdivided into-
   a. Cutaneous receptors :
      Example : Receptor for pain, touch, temperature, cold, pressure, tickling, itches.
   b. Tele receptors (Distance receptor) :
      Example : Receptor for vision, audition, olfaction.
2. *Interoceptors :* Those which collect information from inside of the body. These are-
   a. Proprioceptors : These are carrying information from muscles, tendons, joints etc.
   b. Visceroceptors :
      Example : Chemoreceptors, pain, baroreceptors, stretch receptors, osmoreceptors etc.

Properties of receptor

1. *Excitability :* Receptors are excited by stimulus of electrical, mechanical, thermal or chemical stimuli.
2. *Specificity :* Specific receptor is only stimulated by specific stimulus.
3. *Adaptaion :* When a maintained and constant stimulation is applied to receptor the frequency of discharge from that of receptor will gradually decline. This is called adaptation.
4. *Intensity discrimination :* Several receptors acting

Some receptors with their function & location

Receptor	*Function/sensation*	*Location*
Cutaneous receptor :		
1. Free nerve ending	Pain (mainly), touch, pressure.	Mainly dermis, cornea, also in muscle tendon, vessels etc.
2. Meissner's corpuscles	Touch (tactile localisation)	Superficial aspect of the dermis of hand, foot, nipple, lips, tip of the tongue & in the dermal papillae.
3. Markel's disc	Light touch.	Highly sensative areas (lips, fingers, genitalia).
4. End bulbs of Krause	Cold.	Dermis of skin (Conjunctiva, papillae of skin, lip, tonuge, genitalia), epineurium of nerve.
5. End organ of Ruffini	Heat.	Dermis of skin.
6. Pacinian corpuscles (Largest end organ)	Pressure.	Subcutaneous tissue, tendons of muscles, capsular ligament of joints, in the wall of the blood vessels, mesen teries & periosteum.
7. Golgi-Mazzoni bodies	Pressure.	Subcutaneous tissue, tendons of muscles, capsular ligament of joints, in the wall of the blood vessels, mesen teries & periosteum.
Proprioceptors *(Muscular receptor) :*		
1. Muscle spindle	Carries kinesthetic sensation. Maintains position, Maintains muscle tone, Mediate protective reflexes when muscle vigorously contract.	Between the muscle fibre
2. Tendon end organ of Golgi	Carries kinesthetic impulse. Send inhibitory impulse to inhibit the hypercontraction of muscle & tendon.	Tendons near the junction with muscle
3. Joint receptors a. Pacinian b. Golgi type I & II c. Ruffini's end organ.	Maintains the position & Movement of joint	Ligaments & Capsule of joint.

conjointly and help us to give information about the shape, size, texture and weight of an object.

5. *Doctrine of specific nerve energies :* The sensation evoke by any stimulation, the impulse generated in the receptor depends upon specific part of the brain which is ultimately activated.
6. *Projection :* When any part of the sensory pathway is stimulated, conscious sensation is referred to the location of receptor, this is called law of projection.

Q. 02. **Give the list of proprioceptors.**

Ans. Proprioceptors :

i. Muscle spindle
ii. Tendon end organ of Golgi
iii. Joint receptors :

a. Pacinian
b. Golgi type I & II
c. Ruffini's end organ.

Q. 02. **Give the list of cutaneous receptors.**

Ans. Cutenious receptors i.e. skin tactile sensibilities (epidermis and dermis) are :

a. Free nerve ending
b. Meissner's corpuscles
c. Markel's disc
d. End bulbs of Krause
e. End organ of Ruffini
f. Hair end organs.

(Ref. Guyton & Hall 11th edition)

Q. 05. **Discuss the location and functions of the cutaneous receptors.**

Ans. *Cutaneous receptors* :

Receptor	*Location*	*Function*
Free nerve endin	Mainly dermis, cornea, also in muscle, tendon vessels etc	Pain (mainly), touch and pressure.
Meissner's corpuscles	Superficial aspect of the dermis of hand, foot, nipple, lips, tip of the tongue & in the dermal papillae.	Touch (tactile localisation)
Markel's disc	Highly sensative areas i.e lips, fingers, genitalia.	Light touch
End bulbs of Krause	Dermis of skin (Conjunc tiva, papillae of skin, lip, tonuge, genitalia), epineurium of nerve.	Cold sensation
End organ of Ruffini.	Dermis of skin	Heat

Sensation

It is the feelings evoked by stimulus.

Sensory unit : The area of skin supplied by single nerve fibre is called sensory unit.

Modality of sensation

Sensory receptors are specialized to respond to one particular form of sensation or energy. Thus each main type of sensation that can be experienced is called a *modality of sensation.* Sensory modalities can be divided into two groups :

i. ***Conscious sensations*** :
   a. Pain
   b. Temperature : heat or cold
   c. Touch
   d. Pressure
   e. Joint position and movement
   f. Vision
   g. Hearing
   h. Smell
   i. Taste.

ii. ***Unconscious sensations*** :
   a. Arterial PO_2
   b. pH of CSF
   c. Arterial blood pressure
   d. Central venous pressure
   e. Muscle tension and length
   f. Osmotic pressure of plasma
   g. Arteriovenous blood glucose difference.

(Ref. Guyton & Hall 11th edition; page 572)

Low of specific nerve energies

The specificity of nerve fibre for transmitting only one modalities of sensation is called the low of specific nerve energies. Example-Pain is transmitted by a specific nerve fibre, temperature by another types of fibre etc.

End organ

These are specially organized body at the end of either a motor nerve or at the peripheral end of a sensory nerve, which transmit impulses to the efector organ or receive a particular stimuli from the periphery.

Receptor potential

i. *Definition* : Whatever the type of stimulus that excites the receptor, its immediate effect is to change the potential across the receptor membrane. This change in potential is called *receptor potential.*
   When the receptor potential rises above the threshold for eliciting action potentials in the nerve fiber attached to the receptor, then action potentials begin to appear.

ii. *Characteristics* :
   a. Non-propagated
   b. Have no refractory perroid
   c. Graded.
   d. Does not follow all-or- law.

(Ref. Guyton & Hall 11th edition; page 573-574)

Q. 02. **Distiguish between the receptor potentials and action potentia.**

Ans. Differences between the receptor potentials and action potential :

Action potential	*Receptor potential*
a. Propagated	a. Non-propagated
b. Have refractory peroid.	b. Have no refractory perroid

Action potential	*Receptor potential*
c. Non-graded	c. Graded.
d. Follows all -or-none law	d. Does not follow all-or-none law.

Q. 02. **What is adaption? Compare rapidly and slowly adapting receptors.**

Ans. *Adaptaion* :

i. *Definition* : When a maintained and constant stimulation is applied to receptor the frequency of discharge from that of receptor will gradually decline. This is called adaptation.

Comparison between rapidly and slowly adapting receptors :

Rapidly adapting receptors	*Slowly adapting receptors*
a. Receptors adapt rapidly	a. Recetors adapt slowly and incompletely.
b. Example : touch receptors	b. Example : Carotid sinus, muscle spindles, free nerve ending, end bulb of krause etc.
c. Rapidly adapting receptors detect change in stimulus strength.	c. Slowly adapting receptors detect continous stimulus strength.
d. Also called phasic recptor	d. Also called tonic receptor.

Reflex

Definition : Reflex is the involuntary motor response due to any type of sensory stimulation which is mediated through the CNS.

Importances of examination of reflexes

i. Skeletal muscles receives a segmental innervation. Most of these muscles are innervated by more than one spinal nerve and, therefore, by the same number of segments of the spinal cord. Thus, to paralyze a muscles completely it would be necessary to section several spinal nerves or destroy several segments of the spinal cord.

ii. To learn the segmental innervation of all the muscles of the body is an impossible task. Nevertheless, the segmental innervation of the muscles should be known, because it is possible to test them by eliciting simple muscle reflexes in the patient.

Classification of reflex

Reflex are classified principally of two types :

a. *Uncondition or inborn* : These are inheritent, fixed and cannot altered normally. *Example* : Knee jerk.

It is further classified into three types :

i. *Superficial reflex :* Here the stimuli are received by the skin or mucous membrane. *Example* : Plantar, abdominal and corneal reflex.

ii. *Deep reflex :* Here the stimuli are applied on the tendons. *Example* : Knee jerk, Ankle jerk, Biceps jerk etc.

iii. *Visceral reflex :* These are obtained from deep structures such as viscera. Ex. Micturation, defecation, vomitting reflex.

b. *Condition reflex* : Condition reflex is a reflex response to a stimulus which never produce previously but has been acquired by pairing the said stimulus repeatedly with one uncondition stimulus which normally produces the response. Example- Pavlov's experiment.

Properties of reflex

1. *Localization :* If a specific stimulation is given to a particular locus, then specific response will be evoked.
2. *Delay :* It is the interval in between the time of giving stimulation and getting response. It is about 18-25 milisec.
3. *Summation :* If a subminimal stimulus is applied to a receptor or a sensory neuron, the motorneuron will not discharge impulse but application of a number of subminimal stimuli will evoke reflex action, called summation.

   Cause : Application of a subminimal stimulus will produce insufficient EPSP where as a number of stimuli evoke sufficient EPSP-will cause reflex action.

   Types : It is of two types :

   a. *Spatial summation :* If two afferent nerve stimulated separately, action will not produce. But if they are stimulated repeatedly, reflex action will evoke.

   b. *Temporal summation :* Application of a number of subminimal stimuli on the same nerve will evoke reflex action but a individual stimulus will not produce reflex action.

4. *Occlution :* When a reflex action is produced by the simultaneous stimulation of two afferent nerve, the amount of tension in the muscle is less than the sum total tension set up in the same muscle by stimulating the two nerve separately, This is due to the some neuron which are common for both nerve.
5. *Subliminal fringe :* It is opposite the occlution. The total tension in the muscle due to separate stimulation of two nerve is less than the tension obtained with simultaneous stimulation. Here stimulus is sub-threshold.

   Explanation : During separate stimulation the impulse become adequate for some neurons and inadequate for others. But during simultaneous stimulation the impulse summated and become stronger.

6. *Facilation :* If a reflex elicited repeatedly at proper intervals,

Methods of eliciting, center and response of some superficial and deep reflex :

Name	*Method of eliciting*	*Response*	*Centre*
Superficial reflex			
1. Plantar	Scratching of the skin of sole.	Normally plantar flexion of great toe in adult but incase of infant it becomes dorsiflexion of great toe with fanning of other toes.	Lumber 5 to sacral 2 probably S_1.
2. Anal	Scratching of the skin of the perineum.	Contraction of external anal sphincter.	S_4 & S_5
3. Gluteal	Scratching of the skin of buttock.	Contraction of the gluteal muscles.	L_4, L_5 and upper sacral segments.
4. Pupillary	Fall of light on eye.	Contraction of pupil.	3rd cranial nerve nuclei.
5. Abdominal	Stroking abdominal wall below costal margin	Contraction of abdominal muscles	T_7 - T_{12}
6. Cremesteric	Stroking skin at upper and inner part of thigh	Upward movement of testicle	L_1 - L_2.
Deep reflex or tendon reflex :			
1. *Knee jerk*	Tapping of patellar tendon on semiflexed knee.	Forward jerking of leg due to contraction of the quadriceps femoris muscle.	L_2 to L_4
2. *Ankle jerk*	Tapping of tendo-calcaneous.	Plantar flexion of foot due to contraction of the gastrocnemius	L_5 to S_2
3. Supinator jerk	Tapping on the styloid process of the radius	Flexion and supination of the forearm	C_5 - C_6
4. Biceps jerk	Tapping of biceps tendon.	Flexion of forearm.	C_5 & C_6
5. *Triceps jerk*	Tapping of triceps tendon.	Extension of forearm due to contraction of triceps	C_6 to C_8

the response become progressively higher for the Ist few occasions and the reflex delay decreases.

7. *Reciprocal innervation :* If an afferent nerve is stimulated the protogonist contracts and the antagonist relaxes. This phenomenon is called reciprocal innervation.
8. *Fatigue :* If the reflex is excited repeatedly the response become gradually weaken and ultimately ceases. It is called fatigue.
9. *Recruitment :* If we stimulate a neuron reflexly, the tension develope gradually to the pick. As because after repeated stimulation of the afferent nerve, more and more internuntial neurons are activated; then they activates motor nerve. This phenomenon is called recruitment, as because more and more neurons are recruited.
10. *After discharge :* After reflex contraction if the stimulus is discontinued, the muscle does not completely relaxed at once but it relax gradually. As because the motor neuron stop discharge successivly.

Reflex arc

Definition : Reflex arc is the complete pathway of reflex action.

It consist of the following parts :

1. Receptor
2. Afferent nerve
3. Center
4. Synapse
5. Efferent nerve
6. Effector organ.

Classifications of reflex arc :

1. *Monosynaptic reflex arc :* Has two neuron or only one synapse.
   Example : Stretch reflex.
2. *Disynaptic reflex arc :* Has two synapse or three neurons.
   Example : Extension and crossed extension reflex.
3. *Polysynaptic reflex arc :* Here the synapse is more than two

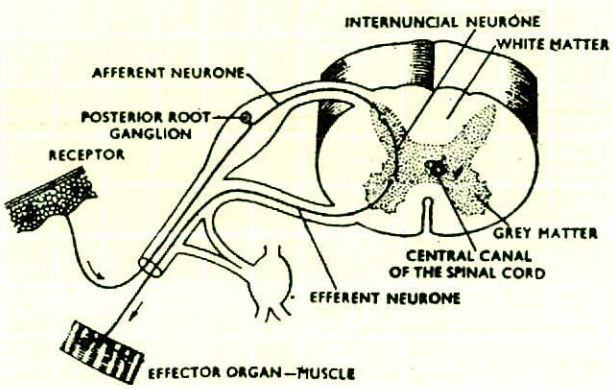

Fig. 16-7. *Reflex arc*

and more than one internuncial neuron exists.

Example : Withdrawl reflex.

Knee jerk

Definition : Knee jerk is one of the deep reflex. Tapping on patellar tendon in semiflex knee causes contraction of quadriceps muscle and forward jerking of leg is called knee jerk

1. *Stimuli* : Sharp tapping on patellar tendon of semiflexed knee.
2. *Response* : Forward jerking of leg.
3. *Centre* : L_2 to L_4 of spinal cord.
4. *Pathway of knee jerk* : Tapping on patellar tendon → Stretched of quadriceps muscle spindle → Stimulation of afferent nerve → Afferent impulse to the center of spinal

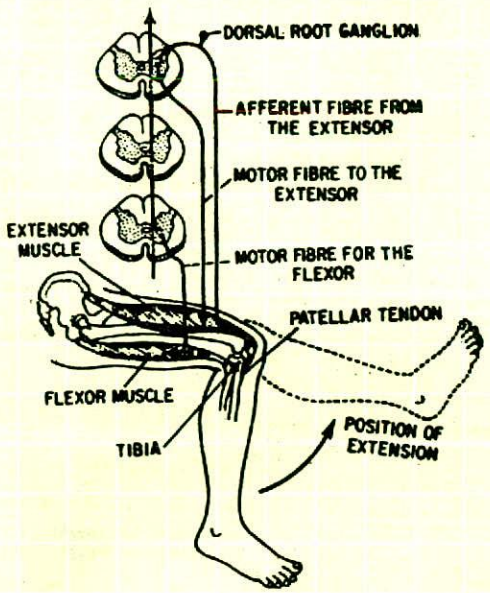

Fig. 16-8. *Knee jerk.*

cord via dorsal root ganglion → Synapse with alpha motor neuron → Motor impulse → Motor nerve to the quadriceps muscle → Contraction of quadriceps and relaxation of flexor muscle → Causes jerking forward of leg.

5. *Importance of knee jerk* :
   1. It is abolishes in lower motor neuron paralysis.
   2. Exaggerated in upper motor neuron paralysis.
   3. Pendular in cerebellar lession.

Stretch Reflex

i. *Definition* : Whenever a muscle is stretched, excitation of the spindles cause reflex contraction of the muscle and this is called stretch reflex.

ii. *Example* : i.e monosynaptic reflexes-
   a. *Knee jerk* : Forward jerking of leg due to reflex contraction of the quadriceps femoris muscle.
   b. *Ankle jerk* : Plantar flexion of foot due to reflex contraction of the gastrocnemius muscle.
   c. *Tricep jerk* : Extension of forearm due to reflex contraction of triceps etc.

iii. *Types* : It can be divided into two separate components called static reflex and a dynamic reflex.
   a. *The dynamic stretch reflex :* When a muscle is suddenly stretched, a strong signal is transmitted to the spinal cord, and this causes an instantaneous, very strong reflex contraction of the same muscle from which the signal originated- this is called dynamic stretch reflex. Thus, the reflex functions to oppose sudden changes in the length of the muscle, because the muscle contraction opposes the stretch.
   b. *The static reflex :* The dynamic stretch reflex is over within a fraction of a second after the muscle has been stretched to its new length but then a much weaker reflex continues for a prolonged period of time thereafter which- is called static reflex. The importance of this reflex is that it continues to cause muscle contraction as long as the muscle is maintained at an excessive length. The muscle contraction inturn opposes the force that is causing the excess length.

iv. *Importance* : The importance of the stretch reflex is that it continues to cause muscle contraction as long as the muscle is maintained at an excessive length. The muscle contraction inturn opposes the force that is causing the excess length.

(Ref. Guyton & Hall 11th edition)

Negative stretch reflex : If a muscle is already taut, any sudden release of the load on the muscle that allows it to shorten will elicit both dynamic and static reflex muscle inhibition rather than reflex excitation, this is called negative stretch reflex.

(Ref. Guyton & Hall 11th edition)

Withdrawal reflex : Any type of cutaneous sensory stimulus on a limb to cause the flexor mucles of the limb contract, thereby withdrawing the limb from the stimulus. This is called the withdrawal reflex or flexor reflex.

(Ref. Guyton & Hall 11th edition)

Crossed extensor reflex : Approximately 0.2 to 0.5 second after a stimulus elicits a flexor reflex in one limb, the opposite limb begins to extend. This is called the crossed extensor reflex.

(Ref. Guyton & Hall 11th edition)

Tracts

Tracts may be defined as a bundle of nerve fibres carring one or a group of motor or sensory impulses to and from the brain.

Types :

Tracts are of two types-

a. Ascending tracts or sensory tract
b. Descending tracts or motor tract

Tracts of spinal cord

i. ***Ascending tract***
   a. *Posterior funiculus :*
      1. Tract of Gall
      2. Tract of Burdach
      3. Inter segmental tract.
   b. *Anterior funiculus :*
      1. Anterior spinothalamic tract
   c. *Lateral funiculus :*
      1. Lateral spinothalamic
      2. Dorsal spino cerebellar
      3. Ventral spinocerebellar
      4. Spino-tectal
      5. Spino-olivary
      6. Spino-reticular
      7. Spino-vestibular.

ii. ***Descending tract***
   a. *Pyramidal tract :*
      1. Lateral corticospinal or crossed pyramidal tract.
      2. Anterior corticospinal or uncrossed pyramidal tract.
      3. Uncrossed lateral corticospinal tract.
   b. *Extrapyramidal tract :*
      1. Rubrospinal tract.
      2. Tectospinal tract.
      3. Olivo-spinal tract.
      4. Recticulo-spinal tract.
      5. Vestibulo-spinal tract.
   c. *Cortico-bulbar tract.*

Tract of gall & tract of Burdach

Extension : These tract lies in the posterior funsiculus. Tract of Gall extend through out the cord. Below the mid thoracic level, it occupies the whole breadth of posterior funiculus but above this it lies medial to the tract of Burdach which extend only above the mid thoracic level.

Origin : These tracts are made up of axons of the bipolar cells of the dorsal root ganglia. Tract of Gall receiving afferent fibre from lower half and Burdach receiving from the upper half of the body.

Course :

a. *1st order neuron :* These are the axon of the dorsal root ganglia. After entering into the spinal cord the fibres end in the following ways :
   1. Majority of fibres ascend and terminate in the nucleus gracil (GALL) & nucleus cuneatus (Burdach).
   2. Some fibres make reflex connection at different segment of spinal cord.
   3. Some fibres descend and make the comma tract of Schultze.

b. *2nd order neuron :* These are the axons of the cells of gracils and cuneatus nucleus and divide into two group :
   i. External arcuate
   ii. Internal arcuate fibre.

   External arcuate fibres are redivided into dorsal and ventral groups. Dorsal group goes to the same side cerebellum but ventral group goes to opposite cerebellum. The internal arcuate fibre crosses and goes to opposite side enters the

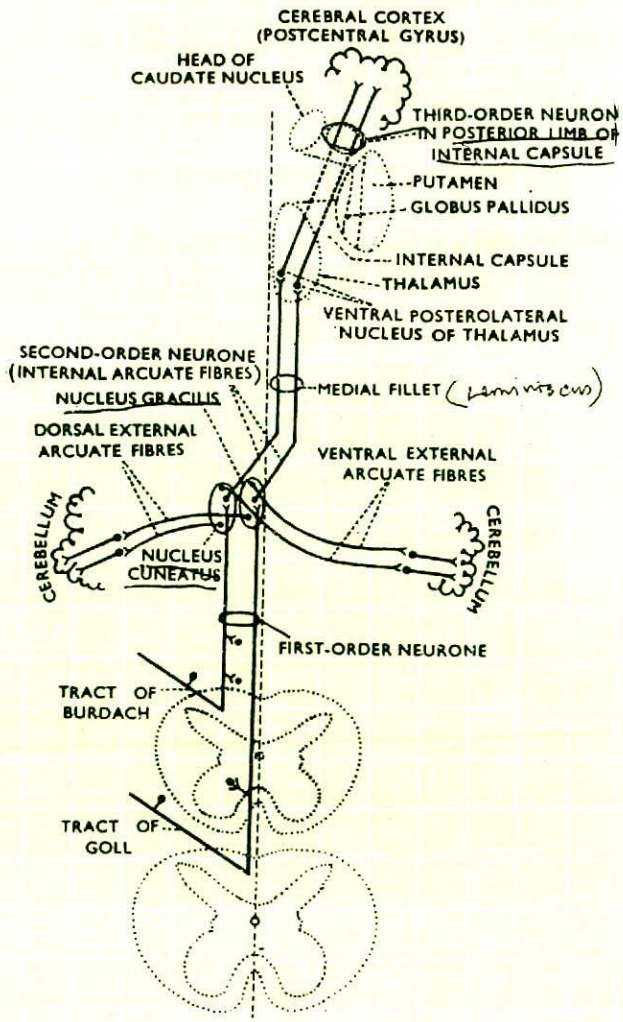

Fig 16-9. *Diagramatic reprentation of tracts of Gall and tract of Burdach.*

medial lemniscus and terminate into the ventral posterolateral nucleus of the thalamus.

c. *3rd order neuron :* Passes through the posterior limb of internal capsule and end into the somatostatic area or post central gyrus of cerebral cortex.

Functions :

1. Carries kinesthetic sensation.
2. Carries fine touch sensation.
3. Carries tactile localization.
4. Carries tactile discrimination.
5. Carries sense of vibration.
6. Sensory pathway for superficial reflex.

Spinothalamic tact

1. Anterior spinothalamic tract.
2. Lateral spinothalamic tract.

Extension : From the spinal cord to the thalamus and take position into opposite anterior and lateral funiculus respectively.

Origin & course :

a. *1st. order neuron :* Formed by the axons of the cells of dorsal root ganglia in the spinal cord. These fibres ends in the posterior horn cell of same side.

b. *2nd order neuron :* These are the axons of the nucleus centrodorsalis of posterior horn, crosses the white comissure goes to the anterior and lateral funiculus of opposite side and ascend as anterior and lateral spinothalamic tract respectively and finally terminate into the ventrobasal nucleus of thalamus and 3rd order neuron arises.

c. *3rd order neuron :* These fibre terminate into the somatosensory area of cortex.

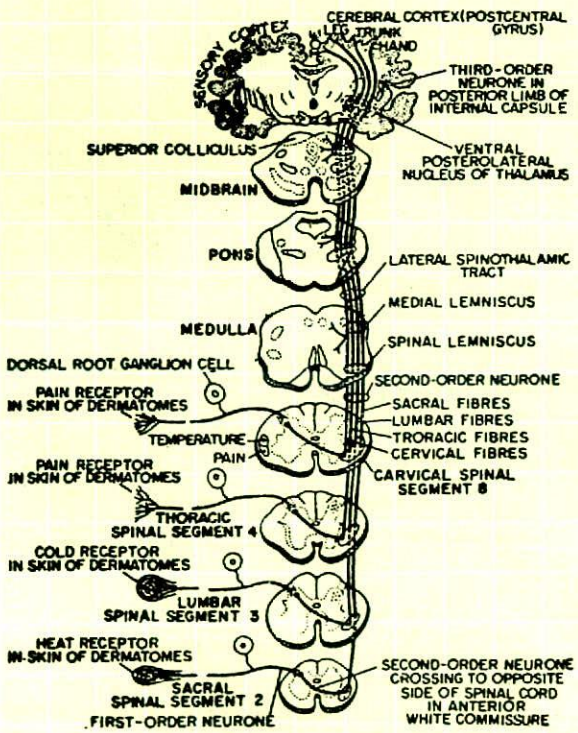

Fig. 16-10. *Spinothalalic tract*

Functions :

1. *Anterior spinothalamic :*
   a. Carries impulse of crude touch of opposite side.
   b. Carries pressure sensibility of opptsite side.
2. *Lateral spinothalamic :*
   a. Carries painful sensation of opposite half.
   b. Carries both hot and cold sensation of opposite side.
   c. Carries complex sensation (tickling & itching).

Cortico-spinal tract

Pyramidal system and extrapyramidal system

i. *Pyramidal system* : Because the fibers of the lateral corticospinal tract form the pyramids in the medulla, the corticospinal pathways have often been referred to as the *pyramidal system.*

ii. *Extrapyramidal system* : The rest of the descending brain stem and spinal pathways that do not pass through the pyramids and are concerned with postural control have been called the extrapyramidal system.

However, the ventral corticospinal pathway does not go through the pyramids, many pyramidal fibers are concerned with other functions, and the system that used to be called extrapyramidal is made up of many different pathways with multiple functions. Consequently, the terms pyramidal and extrapyramidal are misleading, and *it seems wise to drop them.*

Pathway of cortico-spinal tract : As because, in the medulla it passes through the pyramid; so, it is called pyramidal tract.

Origin : The fibres arise from the giant pyramidal cell of Betz of the precentral gyrus. At the cortex, the fibres remain as upside down position, that is toes fibre remain at the top, trunk fibres middle and head fibres in the below.

Course : From cortex the fibres passes through corona radiata then their course are given below-

1. *In the Internal capsule :* Passes through genu and anterior 2/3 of posterior limb of internal capsule.
2. *In the mid brain :* Passes through the middle 2/3 of the crus ceriberi.
3. *In the pons :* This tract occupies the ventral aspect passing through the pontine nuclei where this tract forms scattered bundle.
4. *In the medulla :* Occupies in the pyramid in the lower part of the medulla, majority fibres crosses the midline and some fibre remains in the same side.

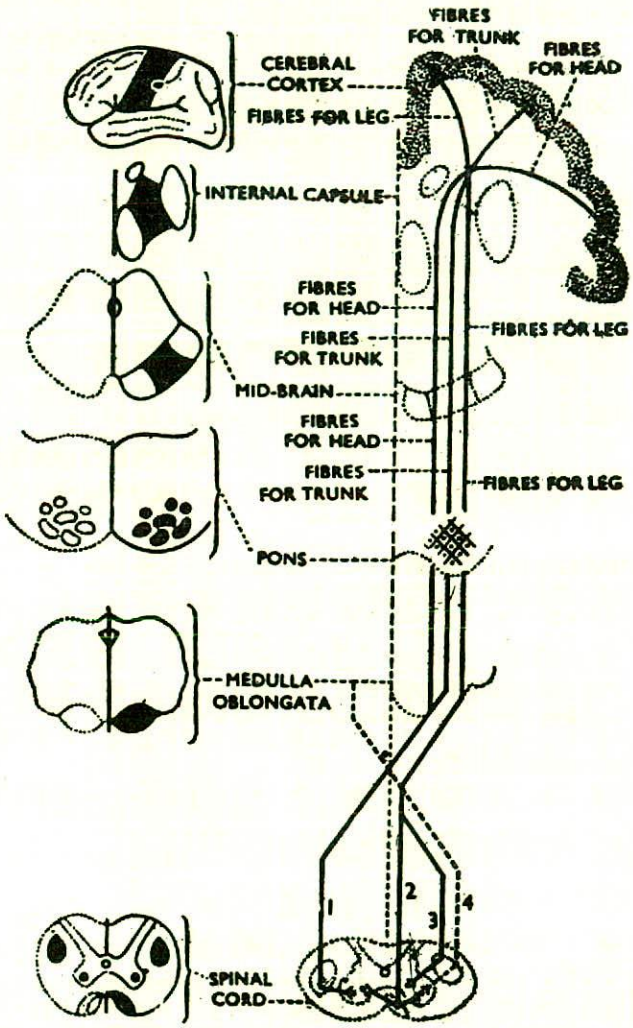

Fig. 16-11. *Cortico-spinal tract.*

5. *In the spinal cord :* The fibres terminate in the following way :
   i. The crossed fibre passes through lateral funiculus as lateral corticospinal tract and end in the anterior horn cells directly or through the internuncial neuron.
   ii. Some uncrossed fibre passes through anterior funiculus as anterior corticospinal tract and end opposite anterior horn cells and some end in the same side anterior horn cells directly or through internuncial neuron.
   iii. Some uncrossed fibres descend through lateral funiculus and end as lateral corticospinal tract.

Function pyramidal tract :

1. Convey motor impulses to the spinal cord for controlling voluntary fine movement of opposite side specially skillfull, and dancing type.
2. Constitute the pathway of superficial reflexes.

Functions of extra-pyramidal tract :

1. Movement of eye ball.
2. Responsible for tone, posture and equilibrium.
3. Responsible for complex movement of body and limbs.
4. It can transmit some voluntary impulse in case of pyramidal trct lesions.

Effect of pyramidal tract lesion :

a. *Changes in voluntary movement :*
   i. Disturbance in the voluntary movement of opposite arm and leg.
   ii. The discrete movements of fingers, walking, grasping, scratching etc can not perform properly.

Give the differece between pyramidal & extrapyramidal systems :

Points	*Pyramidal system*	*Extrapyramidal system*
1. Development	1. New motor system	1. Old motor system
2. Myelination	2. Starts at birth & is completed by 2nd or 3rd year.	2. Starts before birth
3. Onset of function	3. After completion of myelination.	3. From before birth.
4. Mode of discharge of impulse	4. Directly into Lower motor neuron in the anterior grey column in spinal cord.	4. Through already existing path by short interupted neurons. It is a poly synaptic multichannel cortical path way.
5. Nature of muscular activity	5. Non-postural, fine, isolated skill full movement of small muscles.	5. Gross postural movements affecting large muscle group.
6. Effect of damage	6. Flaccid paralysis. Incapacity & disability are more.	6. Spastic paralysis. Incapacity & disabilily are less.
7. Rate of conduction.	7. Slower	7. Faster.
8. Control in relation to body part.	8. Upper limb shows more pyramidal control.	8. Lower limb shows more extrapyramidal control.
9. Completion of work.	9. It finalizes the work	9. It concerned with tone, posture & equilibrium & setting up the platform for the work of pyramidal tract.

b. *Muscle tone :* Increases due to release phenomenon.

c. *Reflexes :*
   1. Superficial reflexes abolishd.
   2. Deep reflexes are exaggerated.
   3. Babinsk's sign positive.
   4. Knee and ankle clonus present.

Upper and lower motor neurons

In addition, the motor system has often been divided into upper and lower motor neurons.

i. *Upper motor neurons* : The neurons in the brain and spinal cord that activate the motor neurons.

   Effect of lesions :
   a. Spastic paralysis
   b. Hyperactive stretch reflexes
   c. Absence of muscle atrophy.

ii. *Lower motor neurons* : The spinal and cranial motor neurons that directly innervate the muscles.

   Effect of lesions :
   a. Flaccid paralysis
   b. Muscular atrophy
   c. Absence of reflex responses.

However, there are three types of *upper motor neurons* to consider. Lesions in many of the posture-regulating pathways cause spastic paralysis, but lesions limited to the *corticospinal* and *corticobulbar tracts* produce *weakness* (*paresis*) rather than paralysis, and the affected musculature is generally hypotonic. Cerebellar lesions produce incoordination. The unmodified term upper motor neuron is therefore *confusing*.

(Ref. Ganong 21th edition; page-203)

Q. Give the differece between upper motor lesion and lower motor lesion.

Upper motor lesion :	*Lower motor lesion*
1. Paralized muscles are rigid due to increase muscle tone.	1. Paralized muscles become flaccid due to loss of muscle tone.
2. Deep reflexes exaggerated & superficial reflexes lost.	2. Both superficial and deep reflexes lost.
3. Muscles are not wasted.	3. Muscles are degenerated & undergo wasting.
4. Babinski sign positive.	4. Babinski sign negative.

Release Phenomenon

Escape of lower motor neuron from the inhibitory impulses of upper motor neuron is called release phenomenon.

Normally, the upper motor neuron exerts inhibitory impulse to lower motor neuron for normal action. But in upper motor lesion they can't send inhibitory impulse to the lower motor neuron. So, the lower motor neurons are released from the inhibitory action of upper motor neuron. Then the activity of lower neuron is increased and muscle tone is increased.

Pain

Difinition : Pain is the protective mechanism of the body when any tissue is being damaged.

Types of pain :

a. Acute pain.

b. Slow pain.

Acute pain : It occurs within 0.1 sec when a pain stimulus is applied and is transmitted through type A delta pain fibres, other name of it sharp pain, pricking pain, fast pain.

Slow pain : It begins only after a seond. or more and then increases slowly over a period of many second and even minutes. It is transmitted through type C pain fibres. Other name : Burning pain, throbbing pain, aching pain.

(Ref. Guyton & Hall 11th edition)

Pain receptor : Free nerve ending.

Cause of pain :

1. Tissue damage.
2. Tissue ischemia.
3. Muscle spasm.
4. Irritation or over distension of viscera.

(Ref. Guyton & Hall 11th edition)

Referred Pain

i. *Definition* : Irritation of a viscus frequently produces pain that is felt not in the viscus but in some somatic region sharing the same segmental innervation. Such pain is said to be referred to the somatic structure.

   Deep somatic pain may also be referred, but superficial pain does not.

ii. *Explanation* : The explanation for referred pain is unknown. One theory is that the nerve fibres from the viscus and the dermatome ascend to the CNS along a common pathway, and the cerebral cortex is incapable of distinguishing between the sites of origin.

iii. *Mechanism (cause) of referred pain* :

   a. *Convergence theory* : Both somatic and visceral afferents converge on the same second order neuron that project to the brain. So when the same pathway is stimulated by visceral afferents the signal reaching the brain is no different and the pain is projected to the dermatome.

   b. *Facilitation theory* : Branches of the visceral pain fibres make synapse with the same spinothalamic neuron

receiving afferents from the somatic area. So, stimulation of visceral pain fibres causes sensation of pain in that somatic region.

iv. ***Examples of referred pain*** :

Pain originate	*Pain felt*
a. Cardiac (myocardial ischemia)	a. Left shoulder and upper arm Middle of the sternum.
b. Diaphragm	b. Shoulder tip.
c. Acute appendicitis	c. Periumblical region
d. Ureteric pain	d. Testicle
e. Renal pain	e. Directly behind the kidney
f. Peptic ulcere disease	f. Epigastrium
g. Pancreatic	g. Posterior abdominal wall directly behind the pancreas.

Q. 09. **Give the difference between visceral pain and somatic pain.**

Ans. Difference between visceral pain and somatic pain :

Visceral pain	*Somatic pain*
a. Poorly localized	a. Highly localized
b. Slow pain, unpleasant	b. Fast pain
c. Pass through C fibre Aδ & C fibre	c. Pass through
d. Asseciated with autonomic symptoms	d. Not associated with autonomic symptoms.
e. Often radiates or referred to other areas.	e. Do not referred

Pathway of pain and temperature

(also tickling & itching) :

i. *Receptor* :
   a. Pain receptor : Free nerve endings
   b. Temperature receptor :
      1. Cold receptor : End bulbs of krause
      2. Hot receptor : End organ of Ruffini's

ii. *Pathway* : Lateral spinothalamic tract.

iii. *Extension* : From the spinal cord to the thalamus and take position into opposite lateral funiculus.

iv. *Origin & course* :
   a. *1st order neuron :* Formed by the axons of the cells of dorsal root ganglia in the spinal cord. These fibres ends in the posterior horn cell of same side.
   b. *2nd order neuron :* These are the axons of the *nucleus centrodorsalis* of posterior horn, crosses the white comissure goes to the *lateral funiculus* of *opposite* side and ascend as *lateral spinothalamic* tract and finally terminate into the *ventrobasal nucleus* of thalamus and 3rd order neuron arises.
   c. *3rd order neuron :* These fibre terminate into the somatosensory area of cortex (Fig : 16-10).

Pathway of Crude touch

i. *Receptor* : Free nerve endings; Meissner's corpuscles

ii. *Pathway* : Anterior spinothalamic tract.

iii. *Extension* : From the spinal cord to the thalamus and take position into opposite anterior funiculus.

iv. *Origin & course* :
   a. *1st order neuron :* Formed by the axons of the cells of dorsal root ganglia in the spinal cord. These fibres ends in the posterior horn cell of same side.
   b. *2nd order neuron :* These are the axons of the *nucleus centrodorsalis* of posterior horn, crosses the white comissure goes to the *anterior funiculus* of *opposite* side and ascend as *anterior spinothalamic tract* and finally terminate into the *ventrobasal nucleus* of thalamus and 3rd order neuron arises.
   c. *3rd order neuron :* These fibre terminate into the somatosensory area of cortex (Fig : 16-10).

Pathway of Fine touch &

(sense of vibration, Kinesthetic sensation, tactile localization)

i. *Receptors* :
   a. Fine touch : Merkel's discs, expanded tip endings
   b. Tactile localization, sense of vibation, kinesthetic sensation : Free nerve endings, Expanded tip endings, (Merkel's discs, several other varients), Spray endings (Ruffini's endings), encapsulated endings (Meissner's corpuscles, krause's corpuscles.

ii. *Pathway* : Tract of Gall and tract of Burdach.

iii. *Origin and course* : Pl follow the tract of Gall and tract of Burdach (page 16.16).

Muscle tone

Muscle tone is a state of partial tetanus (tension) of the muscle during resting condition, maintained by continuous asynchronous discharge of impulses in the motor nerves suppling the muscle.

(Ref. Wright's 13th 297)

Importance of muscle tone :

1. It sets the bias for efficient willed movement

2. It sets the bias for the efficiencies of the reflex action.
3. It is also responsible for maintenance of posture.
4. It also helps in maintenance of form, texture and appearance, e.g look of subject.

Factors affecting muscle tone & posture :

1. Genesis of tone in the muscles at the segmental level.
2. Nociceptive and other factors influencing distribution of tone.
3. Supra spinal control-
   a. Role of cerebral cortex
   b. Role of cerebellum
   c. Role of hypothalamus.
   d. Role of corpus striatum
   e. Role of brain stem.
4. Role of labyrinthine and wrighting reflexes,
5. Role of drugs.
6. Role of sleep.

Mechanism of maintenance of muscle tone : The segmental spinal reflex is responsible for the tone in the muscle which develops following way-

1. *Firstly :* There is a fusiform or Gamma-efferent activity which is tonically discharging impulse to intrafusal fibres of muscle spindle. This causes contraction of intrafusal fibres leading to stretching of nuclear leaf, distorting the annulospiral endings and a receptor potential is set up.
2. *Secondly :* Receptor potential is changed to action potential and afferent discharges pass through annulospiral fibre as well as through flower spray fibres and are transmitted through posterior nerve root to spinal cord.
3. *Thirdly :* Afferent discharges from annulospiral fibre via monosynaptic path to lower motor neurons which results in muscular contraction a phasic reflex response. This release the tension of intrafusal fibres, abolishing the annulospiral discharges.
4. *Fourthly :* The annulospiral afferent discharge is followed by afferent discharges through Golgi-tendon fibres e. g. slow conducting discharges which slowly stimulate the alphamotor neuron for a more static reflex response. So, the contraction of extrafusal fibre is proportional to gamma-efferent activity and lasts as long as the activity lasts.

Causes of abolish of muscle tone :

1. By destroying the centre in the spinal cord.
2. By destroying the posterior root.
3. By destroying the anterior root.
4. By division of the pripheral nerve entering a muscle.
5. By transection of spinal cord.

Motor Unit

A motor neuron together with the group of muscle fibres which it innervates is called a motor unit. A ventral horn cell & its efferent fibre is a motor neuron. The size of the motor unit varies inversely with the precision of the movements performed by the part; e.g. in the limb muscles the unit may contain up to 2000 muscle fibres, in the extrinsic eye muscles less than 5.

(Ref. Wright's, 13th page- 255)

Motor neuron pool : Several anterior horn cells of spinal cord or their homologus, join together to carry out the order for a particular action. They constitute the motor neuron pool.

Muscle spindle

It is a special type of receptor present within the muscle fibre. Each muscle spindle consist of two to ten bundle of muscle

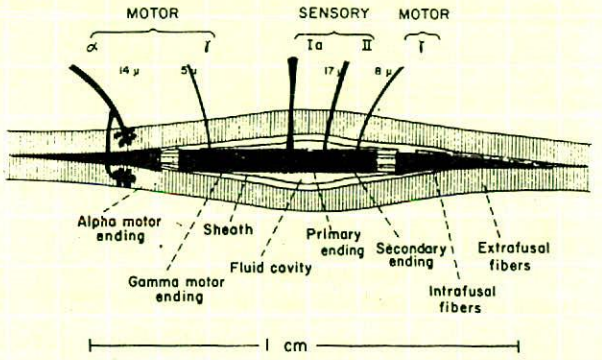

Fig. 16-14. *Muscle Spindle.*

fibres enclosed within a capsule. The capsule is tapered at both ends. The modified muscle fibre within this capsule is known as interfusal muscle fibre which are arranged in two groups-

i. *Nuclear bag fibres :* Displaying an aggregation of nuclei at the equatorial plane.

ii. *Nuclear chain fibres :* Displaying a single row of nuclei in a chain of muscle fibre.

Each muscle spindle is innervated by sensory and motor nerve fibre. The sensory innervation is of two types-

a. Primary or Annulospiral ending placed at centre.
b. Secondary or flowerspray ending on both side of the primary ending.

The motor terminal are of three types-two gamma efferent fibre and one β-efferent fibre.

Innervation of muscle spindle : Each muscle spindle is innervated by sensory and motor nerve fibre.

1. *Sensory innervation* :
   a. Primary or annulospiral ending placed at centre.
   b. Secondary or flowerspray ending on both side of the primary ending..
2. *Motor innervation* : The motor terminal are of three types :

a. Two gamma-efferent fibres.
b. One beta-efferent fibre.

Functions of muscle spindle :

1. Responsible for stretch reflex during muscle contraction.
2. Maintains muscle tone
3. Maintains posture
4. Carries kinesthetic sensation
5. Mediates protective reflex when muscle vigorously contract.

Golgi tendon organs

(Neurotendinous spindles)

i. *Definition* : Neurotendinous spindles are specialized sense organs present in muscle tendon providing information about muscle tension.

ii. *Location* : At the junction of tendon with muscle.

iii. *Structure* : Golgi tendon organs consists of a net-like collection of knobby nerve endings among the fascicles of a tendon.

iv. *Innerevation* : Ib group of myelinated rapidly conducting sensory nerve fibres.

v. *Mechenism of action* : Stimulation of these Ib fibers leads to the production of IPSPs on the motor neurons that supply the muscle from which the fibers arise. The nerve endings are activated when tension develops in the tendon- thus neurotendinous spindle detects change in muscle tension.

vi. *Function* : Provide information about muscle tension to the CNS and thus influence voluntary muscle activity.

(Ref. Ganong 22th Edition: page 133)

Q. 00. **Compare between the muscle spindle and Golgi tendon organ.**

Ans. Comparison between muscle spindle and golgi tendon organ :

Muscle spindle	*Golgi tendon organ*
a. Present within muscle fibres	a. Present in muscle-tendon junction.
b. Sensory fibres are both I (alpha) and II (gamma) type.	b. Sensory fibres are Ib group type.
c. Regulates velocity of muscle contraction and muscle length.	c. Regulates muscle tension
d. Muscle spindle reflexes are excitatory	d. Golgi tendon reflexes are inhibitory.

Cerebral cortex

Definition : Cerebral cortex forms a complete covering of the cerebral hemisphere and is composed of gray matter.

Total number of neuron : The cerebral cortex contain approximately 10 billions neurons.

Types of nerve cells in cerebral cortex :

a. Pyramidal cells
b. Stellate cells
c. Fusiform cells
d. Horizontal cells of Cajal
e. Cells of Martinotti.

Layers of the cerebral cortex : There are six layers in the cerebral cortex :

i. *Molecular layer (plexiforrn layer)* : This is the most superficial layer; it consists rnainly of a dense network of tangentially oriented *nerve fibers*. These fibers are derived from the apical dendrites of the pyramidal cells and fusiform cells, the axons of the stellate cells, and the cells of Martinotti. Afferent fibers originating in the thalamus and in association with commissural fibers also are present. Scattered among these nerve fibers are occasional horizontal cells of Cajal.

This most superficial layer of the cortex clearly is where large numbers of synapses between different neurons occur.

2. *External granular layer* : This layer contains large numbers of *small pyramidal cells* and *stellate cells*. The dendrites of these cells terminate in the molecular layer, and the axons enter deeper layers where they terminate or pass on to enter the white matter of the cerebral hemisphere.

3. *External pyramidal layer* : This layer is composed of *pyramidal cells*, whose cell body size increases from the superficial to the deeper borders of the layer. The apical dendrites pass into the molecular layer and the axons enter the white matter as projection, association, or commissural fibers.

4. *Internal granular layer* : This layer is composed of closely packed stellate cells. There is a high concentration of horizontally arranged fibers known collectively as the *external band of Baillarger.*

5. *Ganglionic layer (internal pyramidal layer)* : This layer contains very *large and medium-size pyramidal cells*. Scattered among the pyramidal cells are *stellate cells* and cells of *Martinotti*. In addition, there are a large number of horizontally arranged fibers that form the *inner band of Baillarger*. In the motor cortex of the precentral gyrus, the pyramidal cells of this layer are very large and are known as Betz cells. These cells account for about 3 percent of the projection fibers of the *corticospinal* or *pyramidal tracts*.

The cerebellum and its connections :

The afferent cerebellar pathways :

Pathway	*Function*	*Origin*	*Destination*
Corticopontocerebellar	Conveys control from cerebral cortex	Frontal, parietol, temporal, and occipital lobes	Via pontine nuclei and mussy fibers to cerebellar cortex
Cerebro-olivocerebellar	Conveys control from cerebral cortex	Frontal, parietal. temporal, and occipital lobes	Via inferior olivary nuclei and climbing fibers to cerebelar cortex
Cerebroreticuloeerebellar	Conveys control from cerebral cortex	Sensorimotor areas	Via reticular forniatmon
Anterior spinocerebellar	Conveys information from muscles and joints	Muscle spindles, tendon organs, and joint receptors	Via mossy fibers to cerebe]lar cortex
Posterior spinocerebellar	Conveys information from muscles and joints	Muscle spindles, tendon organs, and joint receptors	Via mossy fibers to cerebellar cortex
Coneocerebellar	Conveys information from muscles and joints of upper limb	Muscle spindles, tendon organs, and joint receptors	via mossy fibers to cerebellar cortex
vestibu!ar nerve	Conveys information of head position and movement	Ulricle, saccole, and semicrcufar canals	Via mossy fibers to cortex of flocculonodular lobe
Other afferents	Conveys information from midbrain.	Red nucleus, teclum	Cerehellar cortex

(Clinical Neuroanatomy 5th Edition; page 236)

The efferent cerebellar pathways :

Pathway	*Function*	*Origin*	*Destination*
Globose-emboliform-rubral	Influences ipsilateral motor activity	Globose and emboli-form nuclei	To contralateral red nucleus, then via crossed rubrospinal tract to ipsilat-eral motor neurons in spinal cord.
Dentothalamic	Influences ipsilateral motor activity	Dentate nucleus	To contralateral ventrolateral nucleus of thalamus, then to contralateral motor cerebral cortex; corti-cospinal tract crosses midline and controls ipsilateral motor neurons in spinal cord.
Fastigial vestibular	Influences ipsilateral extensor muscle tone	Fastigial nucleus	Mainly to ipsilateral and to.contralat-eral lateral vestibular nuclei; vestibulospinal tract to ipsilateral motor neurons in spinal gord.
Fastigial reticular	Influences ipsilateral muscle tone	Fastigial nucleus	To neurons of reticular formation; reticulospinal tract to ipsilateral motor neurons to spinal cord.

Note that each cerehellar hemisphere influences the voluntary muscle tone on the same side of he body.

(Clinical Neuroanatomy 5th Edition; page 238)

6. *Multiform layer (layer of polymorphic cells)* : Although the majority of the cells are *fusiform*, many of the cells are *modified pyramidal cells*, whose cell bodies are triangular or ovoid. The cells of *Martinotti* also are conspicuous in this layer. Many nerve fibers are present that are entering or are leaving the underlying white matter.

(Clinical Neuroanatomy 5th Edition; page 284)

Motor system

Name (list) the motor systems :

The motor system comprises-

i. Cerebral cortex (cortical motor area)
ii. Pyramidal system
iii. Extrapyramidal system
iv. Cerebellum
v. Spinal motor neurons i.e. anterior horn cells and homogenous neurons in the motor nuclei of the cranial nerves (these neurons are the final common paths to motor system).

Function of motor system

The function of the motor system are :

a. Control of posture and movement
b. Reflex activities i.e. swallowing, chewing, micturation etc.

General motor control scheme

a. *Planning of movement* occurs in the cortex, basal ganglia, and lateral portion of the cerebellar hemisphere (neocerebellum).
b. *Commands* for voluntary movement originate in cortical association area.
c. *Relay* of command to final common paths via corticospinal and corticobulbar system.
d. *Execution* of commands by the motor neurons in the brain stem and spinal cord i.e. anterior horn cells.
e. *Adjustment* of movement by the medial and intermediate portions of the cerebellum.

Motor area of the cerebral cortex

Cortical motor areas : Brodmann described the division of the cerebral cortex into different area of specialization. The motor cortical area include :

a. Primary motor area (Brodmann's area 4) : Responsible for motor execution.
b. Premotor cortex (Brodmann's area 6 and part of 8, 44, 45 : It programs the activity of the primary cortex i.e. design the pattern of movement.
c. Supplementary motor area : Planning the movement.

Cerebellum

Nuclei of cerebellum : The cerebellum has an external cerebellar cortex (gray matter) and inner white matter within which lie deep intracerebellar nuclei.

a. *The cerebellar cortrical cells* :
   i. Stellate cells and basket cells in external molecular layers.
   ii. Purkinje cells : are large Golgi type I neurons in middle layer i.e purkinjee cell layer.
   iii. Golgi cells : In inner granular layer.

   The Purkinjee cells are among the biggest neurons in the body. Their axons constitute the only output from the cerebellar cortex (make synapse with deep cerebellar nuclei)- exert inhibitory effects on the intracerebellar nuclei. The stellate cells, basket cells and Golgi cells serve as inhibitory interneurons.

b. *Deep intracerebellar nuclei* : They are the functional unit of cerebellum. These are-
   i. Dentate nucleus (most laterally) : largest ccrebellar nucleus
   ii. Emboliform nucleus
   iii. Nucleus globose
   iv. Fastigial nucleus.

Vestibulocerebellum

i. *Structure* : Composed of flocculonodular lobe and nodule of the vermis :
ii. *Connection* :
   a. *Afferent connection* : From the vestibular nerve and vestibular nuclei.
   b. *Efferent pathway* : Transmitted through the vestibulospinal, and reticulospinal tracts.
iii. *Function* : Assists in maintaining of equilibrium by bringing about modification in muscle tone.
iv. *Lesion* : produces disorder in equilibrium.

Spinocerebellum

i. *Structure* : Composed of pyramid of the vermis and adjacent medial portions of the hemisphere.
ii. *Connection* :
   a. *Afferent connection* : Receives input from the proprioceptive endings in muscles and tendons, and from touch and pressure receptors (spinocerebellar tracts, cuneocerebellar fibres, and reticulocerebellar tracts).
   b. *Efferent pathway* : Transmitted through the vestibulospinal and reticulospinal tracts.
iii. *Function* : Play an active role in the maintenance of posture and the performance of voluntary movements.
iv. *Lesion* : Produces disorder in gait.

Neocerebellum

i. Structure : Composed of lateral portion of the cerebellar hemisphere.

ii. *Connection* :
   a. *Afferent connection* : Receives a very large input through the corticopontocerebellar tracts from the cerebral cortex of the opposite side.
   b. *Efferent pathway* : Transmitted through the thalamus to the motor area of the cerebral cortex, then through the corticospinal and corticonuclear fibres to the lower motor neurons.

iii. *Function* : Facilitates a smooth, coordinated voluntary movement and ensure that the force, direction, and extent of the movement are accurate i.e. planning and programming of the movements.

iv. *Lesion* : Hypotonia, intention tremor.

Q. 00. **Briefly describe the cerebelllar afferent and efferent fibres.**

Ans. Cerebellar afferent and efferent fibres :

Cerebellar afferent fibres :
   a. *Types* :
      i. Climbing fibres : The climbing fibres are the terminal fibres of the olivocerebellar tracts.
      ii. Mossy fibres : The mossy fibres are the terminal fibres of all other cerebellar afferent tracts.
   b. Cerebellum receives afferent fibres from the :
      i. Cerebral cortex
      ii. Spinal cord
      iii. Vestihular nuclei
      iv. Red nucleus, tectum.

Cerebellar efferent fibres : The entire output of the cerebellar cortex is through the axons of the purkinjee cells. The efferent outflow of the cerebellum destined to :
   i. Globose and emboliform nucleus →Red nucleus → ipsilateral anterior horn cells of the spinal cord (reticulospinal tract).
   ii. Dentate nucleus → Thalamus → motor cortex → ipsilateral motor neurons in spinal cord.
   iii. Fastigial nucleus → Vestibular nucleus → ipsilateral motor neurons in spinal cord (vestibulospinal tract).
   iv. Fastigial nucleus → Reticular formation → ipsilateral motor neurons in spinal cord (reticulospinal tract).

Function of cerebellum

1. It maintains the tone, posture and equilibrium.
   a. It maintains tone through higher cortical and lower spinal centre by excitatory and inhibitory control.
   b. By adjusting the distribution of tone in postural muscle, it maintains the errect posture.
   c. It maintains the equilibrium of the body in standing, jumping, running by adjusting the muscle which are responsible for this purpose.
2. It co-ordinates the voluntary movements.
3. It makes the movement smooth and nonauscilatory.
4. It contributes the range, force, speed and direction of movement for the purpose.

Q. 06. **What is cerebro-cerebellar feed back mechanism**?

Ans. Cerebro-cerebellar feed back mechanism :

i. The mechanism by which the cerebellum controls the activity of motor cortex is called cerebro-cerebellar feed back mechanism or error control mechanism.

ii. During voluntary movements impulse from the cerebral cortex to the muscle spindle comes via corticospinal tract.

iii. At the same time a collateral branceh goes to the cerebellum via fronto-pontocerebellar tract.

iv. The cerebllum also gets impulse from the muscle spindle through the spinocerebellar tract. Then from the cerebellum impulse goes to the thalamus and then goes through the dento-rubro-corticothalamic tract to the cerebral cortex.

Generally motor cortex send impulse to the muscle more than required. Then cerebellum automatically inhibit the cerebral cortex after calculating appropriate time and movement required to reach to the point of intention.

(Q. What is error control by cerebellum?)

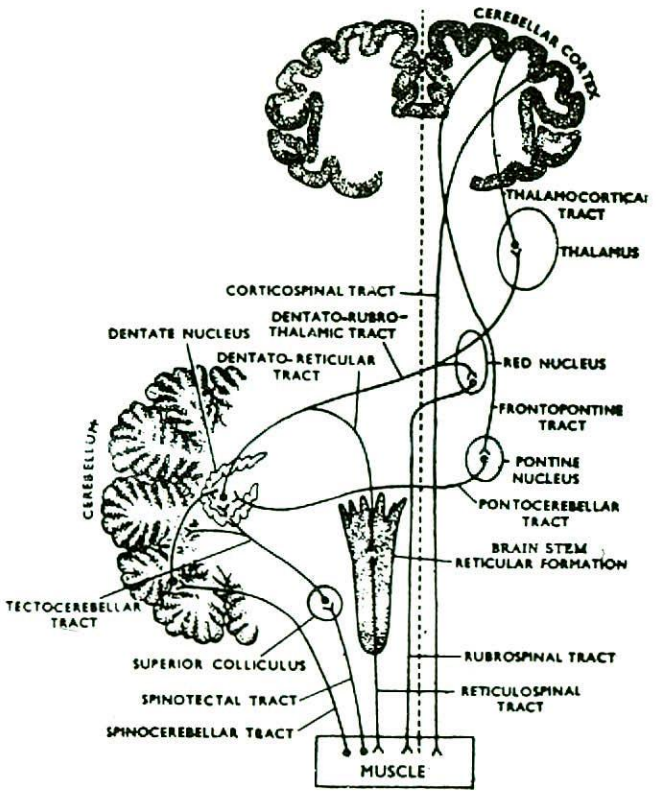

Fig. 16-15. *Schematic reprensentation of the cerebellar feadback system.*

Q. 04. ***Describe how cerebellum controls equilibrium.**

Ans. Control of equilibrium by cerebellum :

i. During the control of equilibrium, it is presumed that the information from both the body periphery and the vestibular apparatus is used in a typical feedback control circuit to provide *anticipatory correction* of postural motor signals as necessary for maintaining equilibrium even during extremely rapid motion, including rapidly changing directions of motion.

ii. The feedback signals from the peripheral areas of the body help in this process.

iii. Their help is mediated mainly through the *cerebellar vermis* that functions in association with the axial and girdle muscles of the body.

iv. It is the role of the vestibulocerebellum to help the brain stem vestibular and reticular nuclei compute the required positions of the respective parts of the body at any given time, despite the long delay time for passage of sensory signals from the periphery to the cerebellum.

(Ref. Guyton & Hall 11th Edition; page 704)

Posture

Q. 02. **What is posture**?

Ans. Posture :

i. *Definition* : Posture may be defined as the position adopted by the individual within his or her environment.

In standing posture the line of gravity passes through the odontoid process of the axis, behind the centers of the hip joints, and in front of the knee and ankle joints.

Q. 02. **What are the posture-regulating systems**?

Ans. Posture-regulating systems are : The posture of an individual depends on the degree and distribution of muscle tone.

Posture is maintained by the :

i. Integrity of the reflex arc

ii. Nervous input from higher levels of the nervous system received by the lower motor neuron i.e
   a. Vestibular apparatus
   b. Cerebral centres, midbrain and cerebellum
   c. Visual centres
   d. Reticular activating system.

iii. General information arising from other muscle groups and joints (proprioception).

Signs of cerebellar lesions

In cerebellar lesions two types of disturbance occur :

1. Disturbance of posture.
2. Disturbance of voluntary movement.

1. *Disturbance of posture* :
   a. *Atonia* : Loss of muscle tone.
   b. *Nystagmus* : Tremor of eye ball when viewing towards a same place.
   c. *Deep reflexes* : Characterstically pendular.

2. *Disturbance in voluntary movement* :
   a. *Ashthenic movement* : Movement will be weak.
   b. *Ataxia* : It means incoordination of movement.
      i. Decomposition of movement : Movement will take place in obvious stage.
      ii. Asynergia : Means lack of cordination between protogonist, antagonist and synergist.
      iii. Dysmetria : Movement will overshoot the intended mark.
   c. *Intention tremor* : Jerking of organ or tremor during voluntary work.
   d. *Gait* : Gait will be deviated towards the affected side then again bring himself towards the previous side.
   e. *Speech* : It will be scanning and sluggish.
   f. *Adiadochokinesis* : Inability to perform rapid pronation and supination.

Test for cerebellar lesion

i. Finger nose test
ii. Adiadochokinesis
iii. Gait test
iv. Speech test.

Paralysis

Temporary suspension or permanent loss of function, specially loss of sensation or voluntary motion is called paralysis,

Types :

1. *Spastic paralysis* : The paralysis due to the lesions of upper motor neurons is called spastic paralysis.
2. *Flaccid paralysis* :The paralysis due to the lesions of lower motor neurons of spinal cord is called flaccid paralysis.

Difference between spastic and flaccid paralysis

Spastic paralysis	*Flaccid paralysis*
1. Paralized muscles are rigid due to increase muscle tone (spastic paralysis).	1. Paralized muscles become flaccid due to loss of muscle tone. (flaccid paralysis)
2. Deep reflexes exaggerated & superficial reflexes lost.	2. Both superficial and deep reflexes lost.
3. Muscles are not wasted.	3. Muscles are degenerated and under go wasting.
4. Babinski sign positive.	4. Babinski sign negative.

Thalamus

Function of the thalamus

Although an enormous amount of research has been devoted to this area, we still know very little about the functional significance of many of the nuclei.

The following basic principles should be committed to memory :

1. The thalamus is made up of complicated collections of nerve cells that are centrally placed in the brain and are interconnected
2. A vast amount of sensory information of all types (except smell) converges on the thalamus and presumably is presumably integrated through the interconnections between the nuclei. The resulting information pattern is distributed to other parts of the central nervous system. It is probable that olfactory information is first integrated at a lower level with taste and other sensations and is relayed to the thalamus from the amygdaloid complex and hippocampus through the marnmillothalamic tract.
3. Anatomically and functionally, the thalamus and the cerebral cortex are closely linked. The fiber connections have been established, and it is known that following

Table : **The various thalamic nuclei, their nervous connections, and their functions**

Thalamlc nucleus	*Afferent neuronal loop*	*Efferent Neuronal loop*	*Function*
Anterior	Mammillothalamic tract, cingulate gyrus, hypothalamus	Cingulate gyrus, hypothalamus	Emotional tone, mechanisms of recent memory
Dorsomedial	Prefrontal cortex, hypothalamus, other thalamic nuclei	Prefrontal cortex, hypothalamus, other thalamic nuclei	Integration of somatic, visceral, and olfactory information and relation to emotional feelings and subjective states
Lateral dorsal, lateral posterior, pulvinar	Cerebral cortex, other thalamic nuclei	Cerebralcortex, other thalamic nuclei	Unknown
Ventral anterior	Reticular formation, substantia nigra, corpus striatum, premotor cortex, other thalamic nuclei	Reticular formation, substantia nigra, corpus striatum, premotor cortex, other thalamic nuclei	Influences activity of motor cortex
Ventral lateral	As in ventral anterior nucleus but also major input from cerebellum and minor input from red nucleus		Influences motor activity of motor cortex
Ventral posteromedial(VPM)	Trigeminal leminiscus, gustatory fibers	Primary somatic sensory (areas 3, 1, and 2) cortex	Relays common sensations to consciousness
Ventral posterolateral (VPL)	Medial and spinal lemnisci	Primary somatic sensors' (areas 3, 1, and 2) cortex	Relays common sensations to consciousness
Intralaminar	Reticular formation, spinothalamic and trigeminothalamic tracts	To cerebral cortex via other thalamic nuclei, corpus striatum	Influences levels of consciousness and alertness
Midline	Reticular formation	Unknown	Unknown
Reticular	Cerebral cortex, reticular fornation	Other thalamic nuclei	? Cerebral cortex regulates thalamus
Medial geniculate body	Inferior colliculus, lateral lemniscus from both ears but predominantly the contralateral ear	Auditory radiation to superior temporal gyrus	Hearing
Lateral geniculate body	Optic tract	Optic radiation to visual cortex of occipital lobe	Visual information from opposite field of vision

(Clinical Neuroanatomy 5th Edition; page374)

removal of the cortex the thalamus can appreciate crude sensations. However, the cerebral cortex is required for the interpretation of sensations based on past experiences. For example, if the sensory cortex is destroyed, one can still appreciate the presence of a hot object in the hand; however, appreciation of the shape, weight, and exact temperature of the object would be impaired.

4. The thalamus possesses certain very important nuclei whose connections have been clearly established. These include the ventral posteromedial nucleus, the ventral posterolateral nucleus, the medial geniculate body and the lateral geniculate body. Their positions and connections should be learned.
5. The ventroanterior and the ventrolateral nuclei of the thalamus form part of the basal nuclei circuit and thus are involved in the performance of voluntary movements. These nuclei receive input from the globus pallidus and send fibers to the prefrontal, supplemental, and premotor areas of the cerebral cortex.
6. The large dorsomedial nucleus has extensive connections with the frontal lobe cortex and hypothalamus. There is considerable evidence that this nucleus lies on the pathway that is concerned with subjective feeling states and the personality of the individual.
7. The initralaminar nuclei are closely connected with the activities of the reticular formation and they receive much of their information from this source. Their strategic position enables them to control the level of overall activity of the cerebral cortex. The intralaminar nuclei are thus able to influence the levels of consciousness and alertness in an individual.

(Clinical Neuroanatomy 5th Edition; page 375, 376)

Thalamic syndrom

i. Disturbance in sensation
ii. Loss of tactile and thermal sensation
iii. Sensory ataxia
iv. Hemiplegia with hyperaesthesia
v. Severe spontaneous pain.

Hypothalamus

Nucleus of hypothalamus : Hypothalamus is divides into three zones.

1. *Periventicular zone :*
   i. Part of preoptic.
   ii. Suprachiasmatic.
   iii. Paraventricular.
   iv. Infundibular.
   v. Posterior group.
2. *Intermediate zone :*
   i. Part of preoptic.
   ii. Anterior group.
   iii. Dorsomedial.
   iv. Ventromedial.
   v. Premamillary.
3. *Lateral zone :*
   i. Part of preoptic.
   ii. Supraoptic.
   iii. Lateral group.
   iv. Tuberomamillary.
   v. Lateral tuberal.

(Ref. Gray's Anatomy)

Function of Hypothalamus

1. *Endocrine control :* With the help of releasing or inhibitory factors, it regulates the secretion of different endocrine gland.
2. *Neurosecretion :* Supraoptic and paraventicular nucleus secrete ADH and oxytocin.
3. *General autonomic effect :* Anterior hypothalamus mediates the parasympathetic activity and the posterior part mediate the sympathetic activity. Thus it regulates the CVS, respiratory and alimentary functions.
4. *Temperature regulation :* Cold sensative area exists in anterior part and heat sensative area exist in posterior part-by which it regulates body temperature.
5. *Regulation of food and water intake :* Hunger center is placed laterally, the satiety center is medially by which it regulates food and water intake.
6. *Sexual behaviour and reproduction :* It control sexual function and reproductive cycle through gonadotropin production.

Afferent & efferent connections of the hypothalamus

The principal afferent and efferent neural pathways to and from the hypothalamus are mostly unmyelinated. Many connect the hypothalamus to the limbic system. There are also important connections between the hypothalamus and nuclei in the midbrain tegmentum, pons, and hindbrain.

Norepinephrine-secreting neurons with their cell bodies in the hindbrain end in many different parts of the hypothalamus. Paraventricular neurons that probably secrete oxytocin and vasopressin project in turn to the hindbrain and the spinal cord. Neurons that secrete epinephrine have their cell bodies in the hindbrain and end in the ventral hypothalamus.

There is an intrahypothalamic system of dopamine-secreting neurons which have their cell bodies in the arcuate nucleus and end on or near the capillaries that form the portal vessels in the median eminence. Serotonin-secreting neurons project to the hypothalamus from the raphe nuclei.

(Ref. Ganong 22th Edition; page233)

Table. **Summary of principal hypothalamic regulatory mechanisms** :

Function	*Afferents from*	*Intearating areas*
Temperature regulation	Temperature receptors in the skin, deep tissues, spinal cord, hypothalamus, and other parts of the brain	Anterior hypothalamus, response to heat; posterior hypothalamus, response to cold
Neuroendocrine control of :		
Catecholamines	Limbic areas concerned with emotion	Dorsal and posterior hypothalamus
Vasopressin	Osmoreceptors, *volume receptors*, others	Supraoptic and paraventricular nuclei
Oxytocin	Touch receptors in breast, uterus, genitalia	Supraoptic and paraventricular nuclei
Thyroid-stimulating hormone (thyrotropin,TSH) via TRH	Temperature receptors in infants, perhaps others	Paraventricular nuclei and neighboring areas
Adrenocorticotropic hormone (ACTH) and β-lipotropin (β-LPH) via CRH	Limbic system (emotional stimuli); reticular formation ('systemic stimuli); hypothalamic and anterior pituitary cells sensitive to circulating blood cortisol level; suprachiasmatic nuclei (diurnal rhythm)	Paraventricular nuclei
Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) via GnRH	Hypothalamic cells sensitive to estrogens, eyes, touch receptors in skin and genitalia of reflex ovulating species	Preoptic area; other areas
Prolactin via PIH and PRH	Touch receptors in breasts, other unknown receptors	Arcuate nucleus; other areas (hypothalamus inhibits secretion)
Growth hormone via somatostatin and GRH	Unknown receptors	Periventricular nucleus, arcuate nucleus
Appetitive behavior		
Thirst	Osmoreceptors, probably located in the organum vasculosum of the lamina terminalis; angiotensin II uptake in the subfornical organ	Lateral superior hypothalamus
Hunger	Glucostat cells sensitive to rate of glucose utilization; leptin receptors; receptors for other polypeptides	Ventromedial, arcuate, and paraven - tricular nuclei; lateral hypothalamus
Sexual behavior	Cells sensitive to circulating estrogen and androgen, others	Anterior ventral hypothalamus plus, in the male, piriform cortex
Defensive reactions (fear, rage)	Sense organs and neocortex, paths unknown	Diffuse, in limbic system and hypothalamus
Control ot body rhythms	Retina via retinohypothalamic fibers	Suprachiasmatic nuclei.

(Ref. Ganong 22th Edition; page 236)

Q. 02. **Mention the endocrine functions of hypothalamus.**

Ans. *Endocrine control :* With the help of releasing or inhibitory factors, it regulates the secretion of different endocrine gland.

It secretes :

i. Catecholamines
ii. Vasopressin
iii. Oxytocin
iv. Thyroid-stimulating hormone (thyrotropin,TSH) via TRH
v. Adrenocorticotropic hormone (ACTH) and β-lipotropin (β-LPH) via CRH
vi. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) via GnRH
vii. Prolactin via PIH and PRH
viii. Growth hormone via somatostatin and GRH.

Basal ganglia

Anatomic considerations : The term basal ganglia is generally applied to five structures on each side of the brain :

a. Three large nuclear masses underlying the cortical mantle :
   i. Caudate nucleus
   ii. Putamen
   iii. Globus pallidus : The globus pallidus is divided into an external and an internal segment.
b. Others :
   iv. Functionally related subthalamic nucleus (Body of Luys)
   v. Substantia nigra : The substantia nigra is divided into a pars compacta and a pars reticulata.

The caudate nucleus and the putamen are frequently called the *striaturn*; the putamen and the globus pallidus are sometimes called the *lenticular nucleus*.

Parts of the thalamus are intimately related to the basal ganglia.

(Ref. Ganong 22th Edition; page213)

Connections of the basal ganglia :

I. *Afferent connections* :
   i. The main afferent connections to the basal ganglia terminate in the striatum. They include the-
      a. Corticostriate projection from all parts of the cerebral cortex.
      b. There is also a projection from the centromedian nucleus of the thalamus to the striatum.
   ii. *The connections between the parts of the basal ganglia* : They include-
      a. A dopaminergic nigrostriatal projection from the pars compacta of the substantia nigra to the striatum.
      b. A GABAergic projection from the striatum to the pars reticulata of the substantia nigra.

   The caudate nucleus and the putamen project to both segments of the globus pallidus. The external segment of the globus pallidus projects to the subthalamic nucleus, which in turn projects to both segments of the globus pallidus and the substantia nigra.

II. *Efferent connections* : The principal output from the basal ganglia is from the-
   i. Internal segment of the globus pallidus via the *thalamic fasciculus* to the ventral lateral, ventral anterior and centromedian nuclei of the thalamus.
   ii. From the thalamic nuclei, fibers project to the prefrontal and premotor cortex.
   iii. The substantia nigra also projects to the thalamus.
   iv. There are a few additional projections to the habenula and the superior colliculus.

However, the main feature of the connections of the basal ganglia is that the cerebral cortex projects to the striatum, the striatum to the internal segment of the globus pallidus, the internal segment of the globus pallidus to the thalamus, and the thalamus back to the cortex, completing a loop.

The output from the internal segment of the globus pallidus to the thalamus is *inhibitory*, whereas the output from the thalamus to the cerebral cortex is *excitatory*.

The striatum is made up of a unique mosaic of patches or striosomes composed of nerve endings in a matrix that receives other endings. The neurons of the corticostriate projection that originate in the deep portion of layer 5 of the cortex terminate in the patches, whereas the neurons that originate in layers 2 and 3 and the superficial part of layer 5 end primarily in the matrix. Neurons with their cell bodies in patches project in large part to dopaminergic neurons in the pars compacta of the substantia nigra, whereas many of the neurons with their cell bodies in the matrix project to GABAergic neurons in the pars reticulata of the substantia nigra.

However, the physiologic significance of these connections is uncertain.

(Ref. Ganong 22th Edition; page 213, 214)

Metabolic considerations of basal ganglia : The metabolism of the basal ganglia is unique in a number of ways. These structures have a high O_2 consumption. The copper content of the substantia nigra and the nearby locus ceruleus is particularly high. In Wilson's disease, a genetic autosomal recessive disorder of copper metabolism in which the plasma level of the copper-binding protein cerulo- plasmin is usually low, there is chronic copper intoxication and severe degeneration of the lenticular nucleus.

(Ref. Ganong 22th Edition; page 214)

Function of basal ganglia : Our knowledge of the precise functions of the basal ganglia is still rudimentary.

Basal ganglia are involved in the *planning* and *programming* of movement or, more broadly, in the processes by which an *abstract thought* is converted into *voluntary action*.

They discharge via the thalamus to areas related to the motor cortex, and the corticospinal pathways provide the final common pathway to the motor neurons. In addition, the field potentials in the basal ganglia oscillate, and it has been suggested that the oscillations may have functions like the putative functions of the oscillations of the thalamocortical circuits.

The basal ganglia also play a role in sonic cognitive processes, and these are particularly the province of the caudate nucleus.

(Ref. Ganong 22th Edition; page 214)

Diseases of the basal ganglia in humans : Disorders of movement associated with diseases of the basal ganglia in humans are of two general types :

i. *Hyperkinetic* : The hyperkinetic conditions, those in which there is excessive and abnormal movement, include-
   a. Chorea
   b. Athetosis
   c. Hallism.

ii. *Hypokinetic* : Hypokinetic abnormalities include-
   a. Akinesia
   b. Bradykinesia.

Chorea is characterized by rapid, involuntary *dancing* movements.

Athetosis is characterized by continuous, slow writhing movements.

Choreiform and atherotic movements have been likened to the start of voluntary movements occurring in an involuntary, disorganized way. In ballism, there are involuntary movements that are flailing, intense, and violent.

Akinesia is difficulty in initiating movement and decreased spontaneous movement.

Bradykinesia is slowness of movement.

(Ref. Ganong 22th Edition; page214, 215)

Parkinsons disease

i. *Pathophysiology* : The disease is associated with progressive neuronal degeneration in the substantia nigra. The degeneration of the neurons of the substantia nigra that send their axons to the corpus striatum results in a reduction in the release of dopamine within the corpus striatum. This lead to hypersensitivity of the dopamine receptors in the postsynaptic neurons in the striatum. Clinical appearance of patients with the *shaking palsy*.

ii. *Age group* : 45 to 55.

iii. *Characteristics* :
   a. Reduction in movement, i.e. bradykinesia or akinesia
   b. Involuntary movements i.e. tremor, chorea, dystonia
   c. Rigidity.

iv. *Causes* :
   a. Idiopathic
   b. Drug e.g. phenothiazine
   c. MPTP can destroy substantia nigra
   d. Postencephalitic parkinsonism.

v. *Symptoms* :
   a. Tremor (resting); disappears during sleep.
   b. Bradykinesia i.e. difficulty in rising from a chair or getting into or out of bed.
   c. Micrographia i.e. small writing
   d. Limbs feel stiff
   e. Difficulties in fine movements.

vi. *Signs* :
   a. *Tremor* (4-7 Hz rest tremor)
   b. *Pill-rolling* movements of the fingers
   c. *Rigidity (lead-pipe rigidity and Cogwheeling)*
   d. Akinesia
   e. Expressionless face
   f. Postural changes i.e patient stands with a stoop
   g. Short, shuffling, and festinating gait
   h. Speech is monotonous, slurring dysarthria.

vii. *Treatment* :
   a. Dopamine agonist
   b. Anticholinergic drugs.

(Q. State the clinical features of perkinsonism)

Wilson's disease

i. *Pathophysiology* : This is a disease of abnormal copper metabolism. There is deposition of copper in the brain particularly in the basal ganglia that leads to degeneration of basal ganglia, in the cornea and in the liver.

ii. *Causes* :
   a. Failure to excrete copper
   b. Low serum ceruloplasmin (copper binding protein).

iii. *Neurological problems* : The neurological problems associated with Wilson's disease are :
   a. Tremor
   b. Dysarthria
   c. Involuntary movements
   d. Dementia.

iii. *Signs* :

Kayser-Fleischer ring : It is a specific sign for wilson's disease and which is due to copper deposition in Descemet's membrane in the cornea.

It appears as a ***greenish brown pigment*** at the selerocorneal junction just within the cornea.

Hunger

Feeding & satiety : Body weight depends on the balance between caloric intake and utilization of calories. Obesity results when the former exceeds the latter.

Food intake is regulated not only on a meal-to-meal basis but also in a way that generally maintains weight at a given set point. Dieters can lose weight when caloric intake is reduced but when they discontinue their diets, 95% of them regain the weight they lost. Similarly, during recovery from illness, food intake is increased in a *catch-up* fashion until lost weight is regained.

Role of the hypothalamus : Hypothalamic regulation of the appetite for food depends primarily upon the interaction of two areas :

i. A lateral *feeding center* in the bed nucleus of the medial forebrain bundle at its junction with the pallidohypothalamic fibers. Stimulation of the feeding center evokes eating behavior in conscious animals, and its destruction causes severe, fatal anorexia in otherwise healthy animals.

ii. A medial *satiety center* in the ventrornedial nucleus. Stimulation of the ventromedial nucleus causes cessation of eating, whereas lesions in this region cause hyperphagia and, if the food supply is abundant, the syndrome of *hypothalamic obesity*.

(Ref. Ganong 22th Edition; page 235, 237)

Other factors affecting food intake

i. *Temperature* : Food intake is increased in cold weather and decreased in warm weather. However, there is little evidence that body temperature is a major regulator of food intake.

ii. *Gastrointestinal tract* : Distention of the gastrointestinal tract inhibits appetite, and contractions of an empty stomach (*hunger contractions*) stimulate appetite, but denervation of the stomach and intestines does not affect the amount of food eaten.

iii. Cultural factors

iv. Environment

v. Past experiences related to the sight, smell, and taste of food also affect food intake.

Brown fat, a special form of body fat that has an extensive sympathetic innervation, may also contribute to the regulation of body weight.

Long term regulation of Appetite : The net effect of all the appetite-regulating mechanisms in normal adult animals and humans is an adjustment of food intake to the point where calorie intake balances energy expenditures, with the result that body weight is maintained. Children are notorious for their uneven food intake, their appetite for certain foods, and their unwillingness to eat others. However, over time they balance food intake with energy expenditure for immediate needs and growth and they grow and develop at a normal pace. Humans gain weight with advancing age, but this is normally a slow, carefully regulated process. One investigator calculated that the average woman gains 11 kg between the ages of 25 and 65. Considering that the total food intake of a woman over the 40 year period is more than 18 metric tons, the error in food intake over energy expenditure that produces the weight gain is less than 0.03%.

(Ref. Ganong 22th Edition; page 240)

Thirst

Thirst mechanism is under hypothalamic control. Drinking is regulated by plasma osmolality and ECF volume in much the same fashion as *vasopressin secretion*. Water intake is increased by increased effective osmotic pressure of the plasma, by decreases in ECF volume, and by psychologic and other factors. Osmolality acts via *osmoreceptors*, receptors that sense the osmolality of the body fluids. These osmoreceptors are located in the anterior hypothalamus.

Decreases in ECF volume also stimulate thirst by a pathway independent of that mediating thirst in response to increased plasma osniolality. Thus, *hemorrhage* causes increased drinking even though there is no change in the osmolality of the plasma. The effect of ECF volume depletion on thirst is mediated in part via the *renin-angiotensin system*. Renin secretion is increased by hypovolemia and results in an increase in circulating angiotensin II. The angiotensin II acts on the subfornical organ, a specialized receptor area in the diencephalon, to stimulate the neural areas concerned with thirst. There is some evidence that it acts on the organum vasculosum of the lamina terminalis (OVLT) as well. These areas are highly permeable and are two of the circumventricular organs located *outside the blood-brain barrier*. However, drugs which block the action of angiotensin II do not completely block the thirst response to hypovolemia, and it appears that the baroreceptors in the heart and blood vessels are also involved.

The intake of liquids is increased during eating (prandial drinking). The increase has been called a learned or habit response, but it has not been investigated in detail. One factor is any increase in plasma osmolality that occurs as the food is absorbed. Another may be an action of one or more gastrointestinal hormones directly on the subfornical organ. There is now some evidence that gastrointestinal hormones stimulate the same subfornical neurons that respond to angiotensin II.

Whenever the sensation of thirst is obtunded, either by direct

damage to the diencephalon or by depressed or altered states of consciousness, patients stop drinking adequate amounts of fluid. Dehydration results if appropriate measures are not instituted to maintain water balance. If the protein intake is high, the products of protein metabolism cause an osmotic diuresis, and the amounts of water required to maintain hydration are large. Most cases of *hypernatremia* are actually due to simple dehydration in patients with psychoses or cerebral disease who do not or cannot increase their water intake when their thirst mechanism is stimulated.

(Ref. Ganong 22th Edition; page 240)

Other factors-regulating water intake

A number of other well-established factors contribute to the regulation of water intake. Psychologic and social factors are important. Dryness of the pharyngeal mucous membrane causes a sensation of thirst. Patients in whom fluid intake must be restricted sometimes get appreciable relief of thirst by sucking ice chips or a wet cloth.

Some kind of pharyngeal gastrointestinal *metering* must be involved, though it is not well developed.

(Ref. Ganong 22th Edition; page 241)

Neural basis of instinctual behavior and emotions- *Limbic systems*

i. ***Introduction*** : Emotions have both mental and physical components. They involve *cognition*, an awareness of the sensation and usually its cause; *affect*, the feeling itself; *conation*, the urge to take action; and *physical changes* such as hypertension, tachycardia, and sweating.

The *hypothalamus* and *limbic systems* are intimately concerned with emotional expression and with the genesis of emotions.

ii. ***Anatomic considerations*** : The term *limbic lobe* or ***limbic system*** is applied to the part of the brain that consists of a rim of cortical tissue around the hilum of the cerebral hemisphere and a group of associated deep structures- amygdala, the hippocampus, and the septal nuclei. The region was formerly called the rhinencephalon because of its relation to olfaction, but only a small part of it is actually concerned with smell.

Histology : The limbic cortex is phylogenetically the oldest part of the cerebral cortex. Histologically, it is made up of-

a. *Allocortex* : a primitive type of cortical tissue, which in most regions has only three layers and surrounds the hilum of the hemisphere.

b. *Juxtallocortex* : A second ring of transitional cortex between the allocortex and the neocortex. It has three to six layers and is found in regions such as the cingulate gyrus and the insula.

The cortical tissue of the remaining nonlimbic portions of the hemisphere is called ***neocortex***. It generally has six layers and is the most highly developed type. The actual extent of the allocortical and juxtallocortical areas has changed little as mammals have evolved but these regions have been overshadowed by the immense growth of the neocortex, which reaches its greatest development in humans.

Correlations between structure & function : One characteristic of the limbic system is the paucity of the connections between it and the neocortex. However, from a functional point of view, neocortical activity does modify emotional behavior and vice versa. On the other hand, one of the characteristics of emotion is that it cannot be turned on and off at will.

Another characteristic of limbic circuits is their prolonged after-discharge following stimulation. This may explain in part the fact that emotional responses are generally prolonged rather than evanescent and outlast the stimuli that initiate them.

iii. ***Limbic functions*** :

a. Olfaction

b. The limbic system is concerned with autonomic responses, particularly changes in blood pressure and respiration.

c. Along with the hypothalamus, it is also concerned with-

1. Sexual behavior
2. Emotions of rage and fear
3. Motivation.

These responses are elicited from many limbic structures, and there is little evidence of localization of autonomic responses. This suggests that the autonomic effects are part of more complex phenomena, particularly emotional and behavioral responses.

(Ref. Ganong 22th Edition; page 256)

Sexual behavior

Mating is a basic but complex phenomenon in which many parts of the nervous system are involved. *Copulation* itself is made up of a series of reflexes integrated in spinal and lower brain stem centers, but the behavioral components that accompany it, the urge to copulate, and the coordinated sequence of events in the male and female that lead to *pregnancy* are regulated to a large degree in the limbic system and hypothalamus. Learning plays a part in the development of mating behavior, particularly in primates, but in nonprimate mammals, *courtship* and *successful mating* can occur with no previous sexual experience. The basic responses are therefore innate and are undoubtedly present in all mammals.

However, in humans, the sexual functions have become

extensively encephalized and conditioned by social and psychic factors.

i. ***Relation to endocrine function*** :

a. In nonprimate mammals, removal of the gonads leads eventually to decreased or absent sexual activity in both the male and the female-although the loss is slow to develop in the males of some species.

b. Injections of gonadal hormones in castrated animals revive sexual activity. Testosterone in the male and estrogen in the female have the most marked effect. Large doses of testosterone and other androgens in castrated females initiate female behavior, and large doses of estrogens in castrated males trigger male mating responses.

c. It is unsettled why responses appropriate to the sex of the animal occur when the hormones of the opposite sex are injected.

d. In women, *ovariectomy* does not necessarily reduce *libido* (defined in this context as sexual interest and drive) or sexual ability. Postmenopausal women continue to have sexual relations, often without much change in frequency from their premenopausal pattern. However, adrenal androgens are still present in these women.

e. Testosterone, for example, increases libido in males, and so does estrogen used to treat diseases such as carcinoma of the prostate. The behavioral pattern that was present before treatment is stimulated but not redirected. Thus, administration of testosterone to homosexuals intensifies their homosexual drive but does not convert it to a heterosexual drive.

ii. ***Neural control in the female*** : In mammals, the sexual activity of the female is cyclic. Most of the time, the female avoids the male and repulses his sexual advances. Periodically, however, there is an abrupt change in behavior and the female seeks out the male, attempting to mate. These short episodes of *heat* or *estrus* are so characteristic that the sexual cycle in mammalian species that do not menstruate is named the *estrous cycle.*

In women, sexual activity occurs throughout the menstrual cycle.

iii. ***Neural control in the male*** : In male animals, removal of the neocortex generally inhibits sexual behavior. However, cats and monkeys with bilateral limbic lesions localized to the piriform cortex overlying the amygdala develop a marked intensification of sexual activity. They not only mount adult females; they also mount immature females and other males and attempt to copulate with animals of other specics and with inanimate objects. The extent to which these animal studies are applicable to men are uncertain, though there have been a few reports of hypersexuality in men with bilateral amygdaloid lesions.

The hypothalamus also involved in the control of sexual activity in males. Simulation along the medial forebrain bundle and in neighboring hypothalamic areas causes penile erection with considerable emotional display in monkeys. In castrated rats, intrahypothalamic implants of testosterone restore the complete pattern of sexual behavior.

(Ref. Ganong 22th Edition; page 257, 258)

The reticular formation and the reticular activating system

The *reticular formation*, the phylogenetically old reticular core of the brain, occupies the midventral portion of the medulla and midbrain. It is primarily an anatomic area made up of various neural clusters and fibers with discrete functions. For example, it contains the cell bodies and fibers of many of the serotonergic, noradrenergic, and adrenergic systems. It also contains many of the areas concerned with regulation of heart rate, blood pressure, and respiration.

Some of the decending fibers in it inhibit transmission in sensory pathways in the spinal cord. Various reticular areas and the pathways from them are concerned with spasticity and adjustment of stretch reflexes.

The RAS is a complex polysynaptic pathway. Collaterals funnel into it not only from the long ascending sensory tracts but also from the trigeminal, auditory, and visual systems and the olfactory system. The complexity of the neuron net and the degree of convergence in it abolish modality specificity, and most reticular neurons are activated with equal facility by different sensory stimuli. The system is therefore *nonspecific*, whereas the classic sensory pathways are *specific* in that the fibers in them are activated by only one type of sensory stimulation. Part of the RAS bypasses the thalamus to project diffusely to the cortex. Another part ends in the intralaminar and related thalamic nuclei and, from them, is projected diffusely and nunspecificallv to the whole neocortex.

(Ref. Ganong 22th Edition; page 192)

Conciousness

A conscious person is awake and aware of himself or herself and the surroundings. For normal consciousness, active functioning of two main parts of the nervous system- the *reticulum formation* (in the brainstem) and the *cerebral cortex*- is necessary. The reticular formation is responsible for the state of wakefulness. The cerebral cortex is necessary for the state of awareness, that is, the state in which the individual can respond to stimuli and interact with the environment. *Eye opening* is a brainstem function: *speech* is a corearal cortex function. Drugs that produce unconsciousness, such as anesthetics, selectively depress the *reticular alerting mechanism*, while those that cause

wakefulness have a stimulating effect on this mechanism.

A physician should be able to recognize the different signs and symptoms associated with different stages of consciousness, namely, ***lethargy***, ***stupor***, and ***coma*** (*unconciousness*).

In a *lethargic individual* the speech is slow and the voluntary movement is diminished and slow. The movement of the eyes is slow.

A *stupored patient* will speak only stimulated with painful stimuli. The voluntary movements are nearly absent, the eyes are closed, and there is very litte spontaneous eye movement. A deeply stupored patient will not speak; there will be mass movements of different parts of the body in response to severe pain. The eyes will show even less spontaneous movement.

An unconscious patient will not speak and will respond only reflexly to painful stimuli, or not at all; the eyes are closed and do not move.

Clinically, it is not uncommon to observe a patient with, for example, intracranial bleeding pass progressively from conciousness to lethargy, stupor, and coma, and then, if recovery occurs, pass in the reverse direction. For these altered states of unconciouness to occur, the thalamocortical system and the reticular formation must be either directly involved bilaterally or indirectly affected by distortion or pressure.

(Clinical Neuroanatomy 5th Edition; page 295)

Sleep

Sleep is a changed state of consciousness.

Changes that occur in sleep :

i. Pulse rate decrease
ii. Respiratory rate decrease
iii. Blood pressure fall
iv. Eyes deviated upward
v. Pupils contract but react to light
vi. Tendon reflexes are lost
vii. Plantar reflex may become extensor.

A sleeping person is not, however, unconscious, because he or she may be awakened quickly by the cry of a child, for example, even though he or she has slept through the background noise of an air-conditioner.

Sleep is facilitated by reducing the sensory input and by fatigue. This results in decreased activity of the reticular formation and the thalamocortical activating mechanism. Whether this decreased activity is a passive phenomenon or whether the reticular formation is actively inhibited, is not known.

(Clinical Neuroanatomy 5th Edition; page 296)

Q. 00. **Describe how sleep and wakefulness are related to the reticular activating system (RAS).**

Ans. A sleeping person is not, however, unconscious, because he or she may be awakened quickly by the cry of a child, for example, even though he or she has slept through the background noise of an air-conditioner.

Sleep is facilitated by reducing the sensory input and by fatigue. This results in decreased activity of the reticular formation and the thalamocortical activating mechanism. Whether this decreased activity is a passive phenomenon or whether the reticular formation is actively inhibited, is not known.

(Clinical Neuroanatomy 5th Edition; page 296)

Q. 00. **How is non-REM sleep distinguished from rapid eye movement (REM) sleep?**

Ans. *Characteristics of non-REM sleep and REM sleep* :

non-REM	*REM sleep*
a. Originate in midline pontine and medullary nuclei raphe nuclei.	a. Originate in pontine reticular formation.
b. Mediated by serotonin	b. Mediated by noradrenaline.
c. Absence of eye movement	c. Rapid conjugate eye movement.
d. Stability of temperature, blood pressure, heart rate and respiration.	d. Fluctuation of temperature, blood pressure, heart rate and respiration.
e. Absence of muscle twisting.	e. Muscle twitchting present
f. Absence of dream.	f. Presence of dreams.

(Q. Mention the characteristics of rapid eye movement (REM) sleep.

Q. Mention the characteristics of non-REM sleep?

Q. Write short notes on- REKM).

Q. 00. **What is Bell-Magendie law?**

Ans. The principle that in the spinal cord the dorsal roots are sensory and the ventral roots are motor is known as the *Bell-Magendie law*.

(Ref Ganong 22th Edition, page-129)

Q. 00. **Write short notes on- Weber-Fechner law.**

Ans. Weber-Fechner law : There are two ways in which information about intensity of stimuli is transmitted to the brain by :

i. Variation in the frequency of the action potentials generated by the activity in a given receptor, and
ii. Variation in the number of receptors activated.

It has long been taught that the magnitude of the sensation felt is proportionate to the log of the intensity of the stimulus (Weber-Fechner law).

(Ref Ganong 22th Edition, page-126)

Cerebro-spinal fluid (CSF)

1. *Definition* : The fluid contained in the central canal of spinal cord, sub-arachnoid space and cerebral ventricles is known as CSF.
2. *Site of formation* : Most of the cerebrospinal fluid is elaborated by the *choroid plexuses* of lateral, third and fourth ventricles. A small amount of fluid is, however, formed by *diffusion from the brain* itself through the extra-choroidal, ependyma and from pial vessels.
3. *Rate of formation* : Average rate is about 20 ml/hour or 500 ml/day.
4. *Mechanism of formation* : Two theories :
   i. Filtration : Plays a minor role.
   ii. Secretion : This is the main process.
5. *Character* : It is a clear, colourless, transparent fluid, does not coagulate on standing & alkaline in reaction.

Specific gravity	:	1.004-1.006
Volume	:	About 150 ml in adults.
Pressure	:	110-130 mm of H_2O (1 drop/second thorugh lumber puncture needle).

6. *Composition* : CSF resembles colloid free plasma with certain variations of crystalloid content.
   a. *Water :* 99.13%
   b. *Solids :* 0.87%

i. Protein	:	20-30	mg%
ii. Glucose	:	50-80	mg%
iii. Chloride	:	700-750	mg%
iv. Cholesterole	:	0.06-0.22	mg%
v. Cells (leukocyte)	:	5	/cu. mm.
vi. Electrolytes	:	Na, K, Ca, Mg, HCO_3, urea, creatinine.	

7. *Function of C S F* :
   i. It acts as a fluid buffer.
   ii. It regulates the volume of cranial contents.
   iii. It is the medium through which nutritive materials as well as waste products are interchanged.

Formation of CSF

Most of the cerebrospinal fluid is elaborated by the *choroid plexuses* of lateral, third and fourth ventricles. A small amount of fluid is, however, formed by *diffusion from the brain* itself through the extra-choroidal, ependyma and from pial vessels.

Procedure : The rich vascular choroid plexuses present a surface area ranging from 150 to 300 sq.cm. The simple cuboidal epithelium of ependymal cells that line the choroidal villi (choroidal epithelium) are connected by tight junctions close to the apical portions of cells. Such tight junctions act as barriers and prevent the passage of proteins and other large molecules from the blood to the ventricular CSF. The CSF is mainly secreted by the choroidal epithelium by *active sodium pump* with expenditure of energy through the microvilli of the choroidal membrane. The net transfer of positive charge by the movement of Na^+ into the CSF is associated with the movement of Cl^- and HCO_3^- into, and the reciprocal movement of H^+ and K^+ out of the CSF. The movement of water into CSF takes place passively to maintain osmotic equilibrium. Thus, the choroidal epithelium acts bi-directionally and is specialised to secrete, to dialyse and to absorb. As the cerebrospinal fluid circulates, its composition is further modified by exchanges of solutes with the brain across the ventricular ependyma and with the plasma across the pia-arachnoid of the subarachnoid space.

Circulation of CSF

After its formation by the choroid plexuses of the lateral ventricles, the fluid appears in the third ventricle through the two interventricular forarnina, and thence into the fourth ventricle through the *aqueduct of Sylvius*. From the fourth ventricle the CSF leaves the ventricular system and enters into the cerebello-medullary cisern through the foramen of *Magendie* and foramina of *Luschka* in the roof of the fourth ventricle.

Thereafter, the CSF circulates in two directions :

a. A part of the fluid undergoes sluggish movement caudally along the spinal subarachnoid space, depending in part upon the movement of the vertebral column.
b. The bulk flow of the fluid from the cerebellomedullary cistern, however, takes place in rostral direction through the tentorial notch. The fluid passes sucessively along the inferior surface and the supero-lateral surface of the cerebral hemisphere until the fluid reaches the arachnoid villi associated with the superior sagital sinus for its final absorption into the venous blood.

Absorption of CSF

Most of the fluid is drained into the venous blood of the superior sagittal sinus through the arachnoid granulations which are composed of numerous arachnoid villi. The arachnoid villi are permeated by a network of minute channels (vide supra) which act as one-way valves and permit bulk flow of the CSF including macromolecules of proteins from the subarachnoid space to the dural venous sinus, preventing at the same time the reverse flow. Such unidirectional flow is regulated by the pressure CSF. Higher colloid osmotic pressure of the venous blood may facilitate the absorption of CSF. A small amount of the fluid may be absorbed into the cervical lymphatics along the sheaths of the cranial nerves.

Thus, the cycles of formation., circulation and absorption of CSF follow a pressure gradient from the high pressure capillary bed of choroid plexuses to the low pressure fluid-filled ventricles and subarachnoid space, and thence to the lowest pressure of the venous blood of the dural sinus (Noback).

Directions Write rue& 'F' for false against each of the following statement.

Introduction

Q. 01. **Brain stem consists of**
- T a. mid brain
- T b. medulla oblongata
- T c. pons
- F d. fore brain
- F e. hind brain.

Q. 02. **Nervous system**
- T a. is the unique system of thought and control action
- T b. receives information
- T c. integrates all the information
- T d. consists of sensory division
- F e. is the only control system of the body.

Q. 03. **The nervous system controls**
- T c. the rapid activities of the body
- T b. the rate of secretion of some endocrine glands
- T d. the rapidly changing visceral events
- T e. the muscle contraction.
- F a. the metabolic activities of the body

Q. 04. **In the postnatal period the greatest growth in the grey matter of the CNS is of**
- T a. Dendritic tree
- F b. Neuron cell number
- F c. Length of axon
- F d. Size of Perikaryon
- F e. Length of nerve fiber.

Q. 05. **phagocyttosis in the CNS is done by**
- T a. Microglia
- F b. Astrocytes
- F c. Schwann cells
- F d. Oligocytes
- F e. Macverophages.

Q. 06. **Blood brain barrier is deficient at**
- T a. Area postrema
- F b. Thalamus
- F c. Meta thalamus
- F d. Cerebellum
- F b. Hypothalamus.

Q. 07. **Blood brain barrier is made up of**
- T a. Astrocytes
- F b. Oligodendrocytes
- F c. Oligodendroglia
- F d. Microglia
- F e. Schwann cells.

Q. 08. **The blood brain barrier**
- T a. slows equilibration of solutes between blood & brain tissue fluid.
- T b. is highly permeable to water
- F c. is more effective barrier for fat soluble substances than water soluble substance
- F d. is highly permeable to plasma protein
- F e. is more effective barrier in infants than in adults.

Q. 09. **The blood brain barrier**
- T a. permits CO_2 to pass easily
- T b. is more permeable in infants than in adults
- T c. allows bilirubin to cross in infants.
- F d. is more permeant to water soluble substances
- F e. permits H^+ to pass freely

Q. 10. **Regarding brain metabolism**
- T a. it accounts for about 15% of the total metabolism in the body
- T b. per unit mass of tissue is about 7.5 times the metabolism in non nervous tissue
- T c. it occurs in the neutrons
- F d. it accounts for about 50% of the total body metabolism
- F e. it occurs in glial supportive tissue.

Q. 11. **Oxygen consumption of whole human brain in ml/minute is about**
- T a. 49
- F b. 29

F c. 35
F d. 6
F e. 40.

Q. 12. **Repolarization state of the action potential includes**
T a. essential diffusion of K^+ ions outward through the membrane
T b. return of membrane potential to the resting level.
F c. important action of Na^+-K^+ pump
F d. increase in Na^+ conductance to 20 times its normal.
F e. no change in K^+ conductance.

Q. 13. **The motor function are**
T a. contraction of skeletal muscles
T b. contraction of smooth muscles in the internal organs
T c. secretion by exocrine glands
F d. activation of intracellular enzymes
F e. intracellular protein synthesis.

Q. 14. **Skilled motor function is**
T a. dependent on pyramidal system
T b. largely influenced by slowly acting feedback from proprioceptors.
T c. dependent on visual feedback
F d. initiated only in the cerebral cortex
F e. greatly affected by transection of extrapyramidal tracts.

Spinal cord

Q. 15. **Spinal cord**
T a. receives sensory signal through posterior root
T b. has alpha motor neuron
T c. has gamma motor reuron.
F d. receives sensory signal through anterior root
F e. has delta motor neuron

Q. 16. **phagocyttosis in the CNS is done by**
T a. Microglia
F b. Astrocytes
F c. Schwann cells
F d. Oligocytes
F d. Macrophages.

Q. 17. **Lowest most level of integration of stretch reflex is at**
T a. Spinal cord
F b. Cerebral cortex
F c. Medulla
F d. Lower medulla
F e. All of the above.

Q. 18. **Inhibition of the spinal cord may be brought about by**
T a. Glycine
F b. Glutamic acid
F c. Aspartic acid
F d. Strychiniae
F e. None of the above.

Q. 19. **Which of the following reflexes disappear in the absence of functional connections between the spinal cord and the brain?**
T a. Swallowing reflex
F b. Seating reflex
F c. Withdrawal reflex
F d. Erection of penis
F e. All of the above

Q. 20. **Hemisection of the spinal cord at cervical level causes**
T a. loss of motor activity in same side
T b. no loss of micturation reflex
F c. loss of pain sensation in same side
F d. greater loss of conscious proprioception in the opposite side
F e. respiratory failure

Q. 21. **If we divide the spinal cord above medulla, what happens to respiration?**
T a. Normal respiration.
F b. It becomes slower and deeper
F c. Apneustic breathing
F d. Breathing ceases
F e. Abormal respiration.

Q. 22. **Autonomic segmental reflexes occurring in the spinal cord are**
T a. emptying reflexes of the urinary bladder
T b. sweating
T c. vascular response to heat and cold
F d. dynamic stretch reflex
F e. papillary reflex

Q. 23. **Neuronal circuits in spinal cord cause**
T a. walking movement
T b. reflexes that withdraw body from painful object
T c. reflex that control blood vessels
F d. reflex that attract body towards painful object
F d. steady state condition.

Q. 24. **Below the level of spinal hemisection supports the following features**
T a. extensive sensory loss on opposite side
T b. extensive motor loss on same side
T c. little motor loss on opposite side
F d. extensive motor loss on opposite side
F e. extensive sensory loss on same side

CSF

Q. 25. **CSF is principally secreted by**

T a. Choroid plexus
F b. Arachnoid granulation
F c. Floor of fourth ventricle
F d. Periaqueductal grey
F e. 1 ml/min.

Q. 26. **CSF production per minute**
T a. 0.30 - 0.35 ml/min
F b. 0.5 ml/min
F c. 2 ml/min
F d. 1 ml/min
F e. 1 L/min

Q. 27. **CSF pressure (lumbar)**
T a. 70 - 180 mm CSF
F b. 50 - 100 mm CSF
F c. > 200 mm CSF
F d. 150 - 200 mm CSF
F d. 90 - 120 mm CSF.

Q. 28. **CSF pressure in the lying down posture is**
T a. 50-150 mm of Hg
F b. 20-50 mm of Hg
F c. 150-200 mm of Hg
F d. 200-300 mm of Hg
F d. 40-70 mm of Hg.

Q. 29. **Below..... pressure CSF absorption stops**
T a. 68 mm CSF
F b. 60 mm CSF
F c. 80 mm CSF
F d. 50 mm CSF
F d. 95 mm CSF.

Q. 30. **CSF in comparison to blood contains**
T a. Lower calcium
T b. Lower sodium
T c. Lower cells
F d. Lower chloride
F e. Lower macrophages.

Q. 31. **Cerebrospinal fluid**
T a. is secreted by choroid plexus
T b. is absorbed by arachnoidal villi.
T c. has got same osmotic pressure as plasma
F d. is formed at a rate of 100 ml/day
F e. has got same concentration of glucose as plasma.

Q. 32. **Regarding cerebrospinal fluid**
T a. it fills the subarachnoid space
T b. the rate of production is about 550 ml/day
T c. it is produced by choroid plexus.
F d. the composition differ from brain extracellular fluid.
F e. it does not protect the brain.

Q. 33. **P^{H} of CSF is**
T a. 7.33
F b. 7.13
F c. 7.23
F d. 7.40
F e. 7.38

Q. 34. **Volume of CSF is about**
T a. 150 ml
F b. 50 ml
F c. 100 ml
F d. 200 ml
F e. 250 ml.

Q. 35. **CSF does not pass through**
T a. Epidural spaces
F b. Ventricles
F c. Venous sinuses
F d. Subarachnoid space
F e. 4th cventricles.

Q. 36. **All the following are more in CSF compared to plasma except**
T a. Glucose
F b. Mg
F c. Cl
F d. HCO_3
F e. All of the above.

Q. 37. **True statement regarding CSF is**
T a. It flows fromIII ventricle to the IV ventricle
T b. Produced mainly by choroid plexus.
F c. Produced only by choroid plexus.
F d. Daily production < 700 ml
F e. CSF analysis rules out active secretion as a cause of formation of CSF

Q. 38. **Indications of lumbar puncture**
T a. to obtain CSF sample
T b. to inject diagnostic agent
F c. elevated intracranial pressure
F d. severe degenerative joint disease
F e. coagulation abnormalities.

Autonomic nervous system

Q. 39. **Autonomic nervous system controls**
T a. contraction of smooth muscle
T b. relaxation of cardiac muscle
T c. secretion of gland
T d. different internal bodily system.
F e. contraction and relaxation of skeletal muscles

Q. 40. **Sympathetic nervous system stimulation causes**
T a. increased arterial pressure

T b. increased mental activity.
T c. increased blood glucose concentration
F d. decreased muscle strength
F e. decreased rate of blood coagulation

Q. 41. **Sympathetic stimulation cause**
T a. increased heart rate
T b. dilatation of bronchi.
T c. dilatation of the pupil
F d. constriction of the pupil
F e. decreased heart rate

Q. 42. **Sympathetic stimulation causes**
T a. glycogenolysis
T b. increased sweat secretion.
F c. bronchial muscle contraction
F d. papillary constriction
F e. skeletal muscle vasoconstriction

Q. 43. **Stress response of sympathetic nervous system are**
T a. increased blood pressure
T b. increased glycolysis
T c. increased rate of blood coagulation.
F d. decreased blood glucose concentration
F e. decreased muscle strength

Q. 44. **Sympathomimetic drugs are**
T a. also called adrenergic drugs
T b. epinephrine and nor epinephrine
T c. act by stimulating sympathetic nerve
F d. also called cholinergic drugs
F e. acetylcholine and pilocarpine

Q. 45. **Parasympathetic nerves**
T a. if stimulated cause papillary constriction
T b. have preganglionic fibers longer than those of post ganglionic
F c. if stimulated cause decreased peristalsis and tone of the gut lumen
F d. When stimulated causes copious sweating
F e. cause vasodilation in skin

Q. 46. **Parasympathetic stimulation causes**
T a. constriction of pupil
T b. constriction of bronchi
T c. contraction of gall bladder
F d. increased heart rate
F e. increased force of contraction of heart muscle.

Q. 47. **Parasympathomimetic drugs**
T a. are also called cholinergic drugs
T b. act by stimulating parasympathetic nerve
T c. are acetylcholine and pilocarpine
F d. act by inhibiting parasympathetic nerve
F e. are epinephrine and norepinephrine.

Q. 48. **Autonomic ganglion is mainly**
T a. Cholinergic
F b. Adrenergic
F c. Dopamergic
F d. Serotonergic
F e. All of the above.

Q. 49. **Vagal stimulation causes the following effects**
T a. Intestinal secretion
T b. Consticton of intestinal musculature
T d. Fall in blood pressure
F c. Relaxation of bronchial musculature
F e. All of the above.

Q. 50. **Right and left vagus respectively go to:**
T a. SA node, AV node
F b. AV node, SA node
F c. AV node, bundle of His
F d. SA node, bundle of His
F e. Inter nodal pathway.

Q. 51. **Bathmotropic effect is produced by**
T a. Stimulation of vagus
F b. Nerves other than vagus
F c. Atropine
F d. Section of vagus
F e. Homatropin.

Q. 52. **Gag reflex is mediated by cranial nerve**
T a. IX
F b. VII
F c. X
F d. XII
F e. II

Neuron

Q. 53. **In a neuron**
T a. axon is the motor part
T b. dendrite is the sensory part
F c. axon is the sensory part
F d dendrite is the motor part
F e soma is the motor part.

Q. 54. **A neuron**
T a. is the structural and functional unit of nervous system
T b. transmit impulses from one part of the body to another
T c. contains dendrite and axon
F d. has no nucleus
F e. has ability for division.

Q. 55. **Internuncial neurons are**
F a. Essential part of stretch reflex

F b. Essential part of all reflexes
F c. Always excitatory
F d. Always inhibitory
T e. None of the above

Q. 56. **Neuronal soma contains more**
T b. more Na^+ ion outside
T c. more K^+ ion inside
T e. less Cl^- ion inside.
F d. more K^+ ion outside
F a. ion inside

Q. 57. **What is true regarding the gamma efferent neuron?**
T a. An *A group motor neuron* with a smaller diameter than that of alpha etferent neurons
T b. Innervates intrafusal fibres
T c. Innervates muscle fibres that stretch annulospiral endings
T d. All of the above
F d. None of the above.

Q. 58. **Increased Gamma efferent discharge is seen in**
T a. Jendrassik's maneuvre
T b. Anxiety
T c. Stimulation of skin
F d. Rapid shallow breathing
F er. Rapid heraert breat.

Q. 59. **Dendrite**
T a. are branching processes
T b. extend from soma
T c. contain presynaptic terminals
F d. are non-branching processes
F e. extend from axon

Q. 60. **An axon**
T a. is single in number.
T b. conducts impulses away from the cell body
T c. is the beginner of action potential by EPSP
F d. conducts impulses away from CONS
F e. is poor in voltage gated Na^+ channel

Q. 61. **Non myelinated axons differ from myelinated in that they**
T a. Lack nodes of Ranvier
F b. Are more excitable
F c. Are not capable of regeneration
F d. Are not capable of dfegeneration
F e. Are not associated with Schwann cells

Q. 62. **Changes in nerve cell body due to degeneration are**
T a. broken up golgi apparatus.
T b. chromatolysis
T c. nucleus pushed to the periphery
F d. nucleus pushed to the centre
F e. shrinking of cell

Q. 63. **The action potential of a neuron**
T a. is associated with net movement of Na^+-K^+ across the membrane
T b. is initiated by rapid influx of Na^+
F c. is maintained by slow influx of Ca^{++}
F d. shows plataue potential before repolarization
F e. lasts more than that of cardiac muscle.

Q. 64. **Effects of upper motor neuron lesion are**
T a. loss of superficial reflex
T b. Spastic paralysis
F c. muscle wasting
F d. loss of deep reflex
F e. decrease muscle tone.

Q. 65. **Upper motor neuron lesion produces**
T a. hyper-reflexia
T b. disuse atrophy of the muscle
F c. flaccid paralysis
F d. extensive sensory loss
F e. plantar flexion.

Q. 66. **A unilateral upper motor neuron lesion in the internal capsule is best characterized by**
T a. Diminished use of contralateral appendages below the lesion
F b. Rapid shallow breathing
F c. Ipsilateral hypotonicity
F d. Flexion of the leg
F e. Rapid shallow breathing.

Q.67. **Lower motor neuron lesion produces**
T a. flaccid paralysis
T b. atrophy of the muscle
T c. hyper-reflexia.
F d. extensor plantar response
F e. fasciculation

Q. 68. **Neuronal activity is**
T a. increased by theophyline
T b. increased by caffeine
T c. decreased by glycine.
F d. inhibited by caffeine
F e. decreased by theophyline

Nerve fibre

Q. 69. **Group B fibre are**
T a. Sympathetic preganglionic
T b. Parasympathetic preganglionic
F c. Sympathetic post ganglionic
F d. Parasympathetic post ganglionic
F e. All of the abocve.

Q. 70. **Wallenberg degeneration is seen in**
- T a. Proximal cut end of nerve with cell body
- F b. Distal cut end of nerve without cell body
- F c. Both the free ends of the cut nerve
- F d. Only the cell body
- F d. All are true.

Q. 71. **First change to occur after nerve cut is**
- T a. Myelin sheath degeneration
- F b. Schwan cell mitosis
- F c. Axonal sprouting
- F d. Myelin sheath regeneration
- F e. Nuclear disintegration

Q. 72. **Which of the following nerve fibres is affected by local anaesthetics first**
- T a. C
- F b. Type II
- F c. A
- F d. B
- F e. Aβ.

Q. 73. **What are some of the modalities of sensation that are detected by free nerve endings?**
- T a. Crude touch
- T b. Pain
- T c. Tickle snesations
- F d. Muscle stretch
- F e. Isometric contraction

Q. 74. **The purkinje fibres**
- T a. Have a conduction velocity of about fives time that seen in head muscle
- F b. Have action potentials about a tenth as long as those in heart muscle
- F c. Are myelinated axons
- F d. All of the above
- F e. None of the above.

Q. 75. **Spike duration is maximum in which nerve fibre :**
- T a. A-beta
- F b. A-alpha
- F c. A -delta
- F d. C
- F e. B.

Q. 76. **The percentage of pyramidal fibres which are unmyelinated**
- T a. 50
- F b. 20
- F c. 35
- F d. 75
- F e. 65

Q. 77. **The most susceptible fibre to hypoxia is**
- T a. B
- F b. A
- d c. C
- e d. Aδ
- F e. All are equally sensitive

Q. 78. **First change to occur in the distal segment of cut nerves**
- T a. Axonal degeneration
- F b. Myelin degeneration
- F c. Mitosis of schwann cell
- F d. Myelin regeneration
- F e. Axonal regeneration

Q. 79. **The fibre which is the thickest in human nerve is**
- T a. Proprioception
- F b. Touch
- F c. Pain
- F d. Temperature
- F e. Hot.

Q. 80. **Conduction in which type of nerve fibres is blocked maximally by pressure?**
- T a. A-beta
- F b. C
- F c. A-alpha
- F d. A-gamma
- F e. B.

Q. 81. **Percentage of sensory fibres in a pure motor nerve is**
- T a. 40 %
- F b. 0 %
- F c. 10 %
- F d. 2 %
- F e. 25 %

Q. 82. **Relation between nerve thickness and conduction velocity is**
- T a. Linear
- F b. Parabolic
- F c. Hyperbolic
- F d. No relation
- F e. All of the above.

Q. 83. **The intensity of sensory stimuli is determined by**
- T a. Frequency of action potential
- F b. Duration of latent period
- F c. Amplitude of action potential
- F d. Amplitude of generation potential
- F e. All of the above.

Q. 84. **Which is true about resting membrane potential of the nerve**
- T a. Potassium (extracellular) mainly
- F b. Can be measured by applying electrode over the nerve fibre contributes

F c. Equal to the resting membrane potential of the muscle.
F d. Reduction of RMP Inhibits a voltage dependent increase in sodium ion permeability
F e. None of the above.

Q. 85. **Which sensation is not lost on the side of lesion in Brown sequard syndrome**
T a. Temperature
F b. Touch
F c. Vibration sense
F d. Muscle sense
F e. None of the above.

Q. 86. **The myelin sheath**
T a. is interrupted by nodes of Ranvier
T b. provides saltatory conduction
F c. significantly decreases conduction velocity
F d. is found on C-fibers
F e. of the axons in central nervous system, is formed by schwann cells.

Q. 87. **The velocity of conduction of nerve nerve fibers is**
T a. increased with increase in fiber diameter
T b. as high as 130 meters/second
F c. decreased with increase in fiber diameter
F d. not related to diameter of the fiber
F e. faster in unmyelinated than in myelinated nerve fibers.

Q. 88. **C-fibers**
T a. conduct impulse at low velocity
T b. conduct pain sensation
F c. are myelinated nerve fibers
F d. possess saltatory nerve conduction
F e. conduct position sensation.

Q. 89. **Wallerian degeneration is characterized by**
T a. mitotic division of schwann cells
T b. invasion by microglial cells
T c. breaking of myelin sheath into fat droplets.
F d. chromatolysis
F e. dissolution of nissl granules

Q. 90. **Conductivity & excitability of a nerve fibre increases due to**
T a. increased temperature
T b. Na^+-K^+ (neuroexcitatory)
F c. mechanical pressure
F d. CO_2
F e. Ca^{++} (neuroselective).

Q. 91. **General classification of nerve fiber are**
T a. Aα
T b. Aγ
T c. C
F d. D
F e. E

Q. 92. **Properties of nerve fibers are**
T a. excitability
T b. conductivity
T c. refractory period.
F d. autorhythmicity
F e. contractility

Q. 93. **Myelinated nerve fibers are**
T a. type A fibers
T b. group III
T c. type Aδ fibers
F d. type C fibers
F e. group IV.

Q. 94. **Unmyelinated nerve fiber**
T a. constitutes more than one half in most peripheral sensory fibers.
T b. constitutes all of the post ganglionic fibers
F c. are Aα fibers
F d. conducts impulese at high velocity
F e. has diameter ranging from 1 to 20 micrometer.

Q. 95. **Unmyelinated nerve fibers are**
T a. type C fiber
T b. group IV fiber
F c. group II fiber
F d. type A fiber
F e. type B fiber

Q. 96. **Stimulation of vagus nerve causes**
T a. mucous secretion from bronchial mucosal cell
T b. contraction of gall bladder
F c. contraction of spleen
F d. secretion in the the salivary gland
F e. reduction in the strength of ventricular contraction.

Receptor

Q. 97. **Receptor which itself is a dendrite of a nerve**
T a. Olfactory
F b. Gustatory
F c. Visual
F d. Hearing
F e. All of the above.

Q. 98. **Paccinian corpuscles are major receptors for**
T a. Pressure
F b. Pain
F c. Touch
F d. Temperature
F e. All of the above.

Q. 99. **Which is caused by acetylcholine through nicotinic receptors**
- T a. Contraction of skeletal muscle
- F b. Descrease of heart rate
- F c. Secretion of saliva
- F d. Contraction of pupils
- F e. All of the above.

Q. 100. **Stimulants that cause excitation of receptors are**
- T a. mechanical deformation of receptors
- T b. application of chemicals to the membrane
- T c. changes of temperature of the membrane
- T d. effect of electromagnetic radiation on the receptor.
- F e. application of toxic substance to the membrane

Q. 101. **Slowly adapting receptors are**
- T a. receptors in the vestibular apparatus
- T b. pain receptors
- T c. baroreceptors
- T d. chemoreceptor.
- F e. thermoreceptors

Q. 102. **Mechanoreeeptors are**
- T a. free nerve endings
- T b. muscle spindles
- T c. golgi tendons
- F d. cold receptors
- F e. rods.

Q. 103. **Electromagnetic receptors**
- T a. are rods
- T b. are cones
- T c. detect light on retina
- F d. detect osmolality of body fluid
- F e. are present in vessel wall.

Q. 104. **Sensory receptors**
- T a. adapt partially
- T b. adapt completely
- T c. respond at a high impulse rate at first
- F d. respond at a low impulse rate at first
- F e. respond progressively at higher rate.

Q. 105. **Meissner's corpuscle found in**
- T a. finger tip
- T b. lips
- F c. joint capsule
- F d. deep facial tissue
- F e. hairy part of skin.

Q. 106. **Free nerve endings are present in**
- T a. joint surface
- T b. periosteum
- T c. arterial wall
- F d. retinal wall
- F e. wall of GIT.

Q. 107. **Pacinian corpuscles are**
- T a. rapidly adapting touch receptors
- T b. mechanoreceptors
- T c. adapted due to readjustments in the structures of the receptor itself
- F d. responsive to pain stimulation
- F e. types of temperature receptor

Q. 108. **Sensory receptors**
- T a. undergo rapid and slow adaptation.
- T b. act as transducers to produce receptor potential
- T c. are highly sensitive to one type of stimulus
- F d. show propagated type of action potential.
- F e. are specialized to respond to different forms of energy.

Q. 109. **The mechanoreceptors are**
- T a. merkel's disc
- T b. krause's corpuscles
- T c. muscle spindle.
- F d. receptors of taste bud
- F e. rods and cones

Q. 110. **Receptor for**
- T a. vision are rods and cones
- T b. pressure are pacinian corpuscles
- F c. proprioception are free nerve ending
- F d. temperature are nociceptors
- F e. pain sensation are stretch receptors.

Q. 111. **α -receptors cause**
- T a. vasoconstriction
- T b. iris dilatation
- T c. intestinal sphincter contraction
- F d. vasodilatation
- F e. intestinal sphincter relaxation.

Q. 112. **Beta receptors cause**
- T a. cardioacceleration
- T b. bronchodilatation
- T c. vasodilatation
- F d. vasoconstriction
- F e. bronchoconstriction.

Q. 113. **The following are the few examples of sensory receptors with their sense organs**
- T a. rods and cones : eye
- T b. baroreceptor : aortic arch
- F c. hair cells : olfactory epithelium
- F d. muscle spindles : tendon of the muscle
- F e. salty taste buds : posterior part of the tongue.

Q. 114. **The following are the sensory receptors with their respective sensations**
- T a. olfactory epithelium : smell
- T b. baroreceptors : blood pressure.
- T c. free nerve endings : pain
- F d. pacinian corpuscles : cold sensation
- F e. osmoreceptor : blood PCO_2

Neurotransmitters

Q. 115. **Receptor which itself is a dendrite of a nerve**
- T a. Olfactory
- F b. Gustatory
- F c. Visual
- F d. Hearing
- F e. All of the above.

Q. 116. **Paccinian corpuscles are major receptors for**
- T a. Pressure
- F b. Pain
- F c. Touch
- F d. Temperature
- F e. All of the above.

Q. 117. **Which is caused by acetylcholine through nicotinic receptors**
- T a. Contraction of skeletal muscle
- F b. Descrease of heart rate
- F c. Secretion of saliva
- F d. Contraction of pupils
- F e. All of the above.

Q. 118. **Alpha component of G protein act by**
- T a. activation of gene transcription
- T b. opening specific ion channel
- T c. activation of enzyme
- F d. inactivation of enzyme
- F e. inactivation of gene transcription.

Q. 119. **Acetylcholine**
- T a. is synthesized from acetic acid
- T b. releases from all post ganglionic parasympathetic neuron
- T c is destroyed by acetyl cholinesterase
- F d. acts as inhibitory neurotransmitter
- F e. releases from all post ganglionic sympathetic neuron

Q. 120. **Norepinephrine is**
- T a. secreted from adrenal medulla
- T b. synthesized from tyrosine
- T c. destructed by monoamino-oxidase
- F d. destructed in spleen
- F e. the parasympathetic transmitter.

Q. 121. **Of the neurotransmitter**
- T a. serotonin is the sleep inducing neurotransmitter.
- T b. dopamine is rapidly acting transmitter
- T c. gastrin acts on brain
- F d. GABA is slowly acting transmitter
- F e. glutamate always cause inhibition

Q. 122. **The following neurotransmitter are peptides**
- T a. substance-P
- T b. met-enkephalin
- T c. β-endorphin
- F d. serotonin
- F e. acetylcholine.

Q. 123. **The following neurotransmitter are synthesized in the preganglionic sympathetic neutrons**
- T a. norepinephrine
- T b. acetylcholine
- T c. L-Dopa
- F d. histamine
- F e. glutamine.

Q. 124. **The following are the few examples of excitatory and rapidly acting neurotransmitter**
- T a. nitric oxide
- T b. epinephrine
- F c. GABA
- F d. dopamine
- F e. substance P

Q. 125. **Acetylcholine is secreted at the endings of**
- T a. preganglionic fibers to the adrenal medulla
- T b. motor nerves at the myoneural junction.
- T c. preganglionic sympathetic
- T d. preganglionic parasympathetic
- F e. preganglionic sympathetic fibers supplying skin blood vessels

Q. 126. **IPSP produced by the neurotransmitter are**
- T a. GABA
- T b. dopamine
- T c. glycine
- F d. adrenaline
- F e. acetylcholine.

Q. 127. **Rapidly acting neurotransmitter are**
- T a. glutamate
- T b. glycine
- F c. gastrin
- F d. cholecystokinin
- F e. angiotensin II.

Q. 128. **Slowly acting neurotransmitter are**
- T a. thyrotropin
- T b. cholecystokinin
- F c. dopamine

F d. histacine
F e. serotonin.

Q. 129. **Neurotransmitter acting on basal ganglia are**
T a. norepinephrine.
T b. dopamine
T v. acehylcholine
T d. gamma aminobutyric acid
F e. epinephrine

Q. 130. **Neuropeptides**
T a. are produced in endoplasmic reticulum
T b. are more potent than small molecule transmitters
T c. usually cause prolonged actions
F d. are removed within milli-seconds
F e. are GABA, nitric oxide, dopamine etc.

Synapse

Q. 131. **IPSP is due to**
T a. Cl^- influx
F b. K^+ influx
F c. Na^+ influx
F d. Ca^{++} influx
F e. Mg^{++}.

Q. 132. **EPSP is due to**
T a. Na^+ influx
F b. K^+ influx
F c. Na^+ eflux
F d. Ca^{++} influx
F e. Mg^{++}.

Q. 133. **Synapse**
T a. determines the direction of nervous signals
T b. is the junction of two neutrons
T c. in CONS are almost chemical in nature
F d. in CONS are almost electrical in nature
F e. blocks strong signal but allows weak signal to pass.

Q. 134. **Chemical synapse**
T a. acts by releasing neurotransmitter
T b. transmits signals in one direction
T c. is mainly present in CNS
F d. transmits signals in both direction
F e. is mainly present in PNS.

Q. 135. **A chemical synapse exhibits following properties**
T a. delay
T b. summation
T c. presynaptic inhibition
F d. two way conduction
F e. infatigability

Q. 136. **Presynaptic terminals**
T a. contain mitochondria.
T b. are called synaptic knob
T c. are separated from post synaptic terminal by synaptic cleft
T d. contain transmitter substance
F e. contain no mitochondria

Q. 137. **Presynaptic membrane contains**
T a. release site
T b. voltage gated calcium channel
F c. no calcium channel
F d. sodium channel
F e. no release site.

Q. 138. **Post synaptic membrane contains**
T a. excitatory receptors.
T b. cation channel
T c. anion channel
F d. no anion channel
F e. no cation channel.

Q. 139. **Excitatory post synaptic potential (EPSP) are due to**
T a. depression of K^+ channel
T b. opening of Na^+ channel
F c. opening of Cl^- channel
F d. opening of K^+ channel
F e. increased conduction through Cl^- channel

Q. 140. **Inhibitory post synaptic potential (IPSP) are due to**
T a. increased conductance of K^+
T b. activation of receptor enzyme.
T c. opening of $C1^-$ channel
F d. closing of $C1^-$ channel
F f. opening of Na^+ channel

Q. 141. **Excitatory post synaptic potential is due to**
T a. depressed conduction through chloride channel
T b. decreased conductance of K^+ channel
T c. opening of Na^+ channels
F d. opening of chloride ion (Cl^-) channel
F e. increased conductance of K^+ channel.

Q. 142. **Synaptic transmission**
T a. is decreased in acidosis.
T b. become fatigue due to repetitive stimulation
T c. is increased in alkalosis
F d. become over stimulated due to repetitive stimulation
F e. is decreased in alkalosis

Q. 143. **Transmission of impulse across a chemical synapse needs**
T a. spreading of action potential over a presynaptic terminal

T b. activation of post-synaptic ion channel by neurotransmitter
T c. activation of second messenger systems within the neuron.
F d. Ca^{++} influx through the post-synaptic neuron to release neurotransmitter.
F e. release of excitatory neurotransmitter only.

Q. 144. **Excitation of the post-synaptic neuron occurs by**
T a. decreasing Cl^- influx
T b. activation of Na^+ channels
F c. opening of anion channels
F d. releasing GABA from pre-synaptic terminal.
F e. increasing the K^+ conductance to the exterior.

Q. 145. **Some post synaptic receptors, when activated cause excitation of the post synaptic neuron by**
T a. an increase in the number of excitatory membrane receptors.
T b. opening of sodium channels
F c. opening of calcuim channels
F d. opening of chloride channels
F e. an increase in the conductance of potassium ions through the receptor

Q. 146. **Saltatory conduction**
T a. conserves energy for the axon because only the nodes depolarize.
T b. is faster than nonsaltatory conduction
T c. transmits impulses with a velocity proportional to fibre diameter
F d. is accomplished almost entirely by the sequential changes in the voltage gated potassium channel
F e. occurs only in unmyelinated nerve fibers

Reflex

Q. 147. **Crossed extensor reflex is a**
T a. Withdrawal reflex
F b. Postural reflex
F c. Monosynaptic reflex
F d. Sympathetic reflex
F e. parasympathetic reflex.

Q. 148. **All these are seen in a spinal reflex**
T a. Summation
T c. Memory
T d. Adaptation
F b. Fatigue
F e. All of the above.

Q. 149. **Unidirectional transport is seen across**
T a. Synapse
F b. Axon
F c. Both of above
F d. None of above
F e. All of the above.

Q. 150. **Lowest most level of integration of stretch reflex is at**
T a. Spinal cord
F b. Cerebral cortex
F c. Medulla
F d. Lower medulla
T e. Pons.

Q. 151. **Superficial reflexes are**
T a. Corneal
T b. Plantar
F c. Ankle
F d. Hoffmans
F e. Jaw

Q. 152. **The basic postural reflex is**
T a. Flexor reflex
F b. Crossed extensor reflex
F c. Golgi tendo reflex
F d. Positive supporting reflex
F e. Jaw.

Q. 153. **A reflex action**
T a. is initiated at a sensory receptor organ
T b. may result in endocrine secretion
T c. is excitatory
T d. is inhibitory.
F e. is independent of higher centers in the brain

'Q. 154. **Superficial reflexes are**
T a. papillary reflex
T b. plantar reflex
T c. cremasteric reflex
F d. knee jerk
F e. ankle jerk.

Q. 155. **Deep reflexes are**
T a. biceps jerk
T b. ankle jerk
T c. knee jerk
F d. papillary reflex
F e. conjunctival reflex.

Q. 156. **Reflex arc**
T a. has afferent nerve fibers
T b. has receptor
T c. is a complete pathway for a reflex action
F d. is a partial pathway for a reflex action
F e. has no receptor

Q. 157. **Conditioned reflexes**
T a. are acquired
T b. can be established.
T c. can be abolished

F d. are inherent
F e. can not be abolished

Q. 158. **Uncondition reflexes**
T a. can not be altered
T b. are also called inborn reflex.
T c. are inherent
F d. are acquired
F e. can be altered.

Q. 159. **Withdrawal reflex**
T a. response is inhibition of extensor muscle
T b. occurs in response to noxious stimuli
T c. response is flexor muscle contraction
F d. response is flexor muscle relaxation
F e. is a typical monosynaptic reflex.

Q. 160. **Regarding knee jerk**
T a. its receptor is muscle spindle
T b. it is a stretch reflex
T c. it is monosynaptic reflex
F d. it is polysynaptic reflex
F e. its efferent nerve is femoral nerve.

Q. 161. **A reflex action**
T a. may be carried out by all muscles and secretory glands
F b. is not influenced by higher center in the brain
F c. is a voluntary response to a stimulation.
F d. is usually initiated by higher centre
F e. is always inborn in nature.

Q. 162. **The flexor withdrawal reflex is a**
T a. spinal cord reflex
T b. polysynaptic reflex
T c. protective reflex
F d. monosynaptic reflex
F e. muscle stretch reflex

Q. 163. **Maintenance of upright posture and balance depends upon**
T a. inputs from the eyes, vestibular apparatus and somatic proprioceptors
T b. the body's center of gravity which must be maintained over the body base
F c. contraction of the flexor group of skeletal muscle
F d. only on the vestibular apparatus
F e. only on the proprioceptors.

Q. 164. **Postural reflexes are**
T a. optical weighting reflexes
T b. positive supporting reaction
T c. stretch reflexes
F d. superficial reflexes
F e. autonomic reflexes

Muscle tone & Motor unit

Q. 165. **Muscle spindle is**
T a. Receptor for myotatic or stretch receptor reflex
F b. Receptor for a variety of multisynaptic reflexes
F c. Occurs only in antigravity extensor muscles
F d. Excited by both stretch and contraction of the muscles in which it is located
F e. All of the above.

Q. 166. **A twitch of motor unit is called**
T a. Fasciculation
F b. Myoclonic jerk
F c. Tremor
F d. Chorea
F e. Golgi tendo reflex.

Q. 167. **Curare in therapeutic doses**
T a. Decreases the amplitude of skeletal muscle potential
F b. Prevents propagation of action potential in skeletal muscle
F c. Enhances the action of choline esterase
F d. Enhances the action of catecholamines
F e. Incereases propagation of action potential in skeletal muscle

Q. 168. **Muscle tone is increased by**
T a. gamma efferent impulese to muscle spindle
T b. extrapyramidal lesion
T c. upper motor neuron lesion
F d. lower motor neuron lesion
F e. cerebellar lesions

Q. 169. **Motor units**
T a. in large postural muscles have high innervation ratio
T b. consist of a single motor neuron supplying many muscle fibers
F c. consist of a single muscle fiber innervated by many motor neutrons
F d. can increase force of contraction
F e. contain gamma motor neutrons.

Q. 170. **Intrafusal muscle fiber**
T a. has no or few actin and myosin filament in central region
T b. is innervated by γ efferent fiber
T c. does not contract in its central portion
F d. has more actin and myosin filament in central region
F e. is innervated by α efferent fiber.

Q. 171. **Muscle spindle**
T a. has both intrafusal and extrafusal muscle fiber
T b. is the sensory receptor of muscle
T c. has intrafusal muscle fiber

F d. has no intrafusal muscle fiber
F e. has no extrafusal muscle fiber.

Q. 172. **Golgi tendon organs**
T a. are the receptors of muscle tendon
T b. transmit information about tendon tension
F c. are the receptors of muscle
F d. transmit information about change of muscle fiber length
F e. transmit no information about tendon tension

Q. 173. **Regarding muscle stretch reflex**
T a. reciprocal inhibition relaxes muscles
T b. it is antagonistic to the stretched muscle
T c. the muscle spindles ate sensitive to changes in muscle length
F d. the sensitivity of the muscle spindle can be reduced by increasing the activity of gamma motor neutrons
F e. the reflex arc is polysynaptic.

Q. 174. **Muscle tone is diminished or lost when**
T a. all peripheral nerves innervating a particular muscle are cut
T b. α-motor neutrons are destroyed.
T c. spinal cord ventral root efferent are cut
F d. corticospinal tracts are damaged
F e. basal ganglial structures are damaged

Q. 175. **In a highly trained distance runner, muscle tissue increases in**
T a. enzymes for oxidative phosphorylation
T b. myoglobin content
T c. mass due to hypertrophy of individual fibers
F d. mass due to increase in number of muscle fibers
F e. mass due to an increase in blood supply.

Q. 176. **Rapid eye movement**
T a. is usually associated with active bodily muscle movement
T b. is characterized by irregular heart rate
T c. is characterized by irregular respiratory rate
F d. is usually not associated with muscle movement
F e. causes decreased brain metabolism.

Tract

Q. 177. **Injection of hypertonic saline into which area causes diuresis**
T a. Supraoptic nucleus
F b. Paraventricular nucleus
F c. Preoptic nucleus
F d. Posterior pituitary
F d. Anterior pituitary.

Q. 178. **In the neurohypophysis, secretory granules accumulate in**
T a. Nerve endings
F b. Pituicytes
F c. Intercellular spaces
F d. Capillary endothelium
F e. Axon.

Q. 179. **Decreased hypothalamic function casues depressed levels of the following homones**
T a. Growth hormone
T b. TSH
T c. ACTH
F d. Prolactin
F e. Oxytocin.

Q. 180. **One of the following is a function of hypothalamus**
T a. Homeostasis of temperature
F b. Swallowing
F c. Vomiting
F d. Respiration
F e. Diglutation.

Q. 181. **Profound hypothermic signs include**
T a. Slow breathing
T b. Bradycardia
T c. Hypotension
F d. Hyperactivity
F e. Hypoactivity

Q. 182. **Which of the following are true of median eminence**
T a. Portion of ventral hypothalamus
T b. Hypothalmo-hypophyseai vessels arise here
T c. Outside the blood-brain barrier
F a. Portion of dorsal hypothalamus
F d. All are correct

Q. 183. **What is the mechanism by which infection causes fever**
T a. Pyrogens acting independent of hypothalamus
T a. Effects of inflammatory mediator.
F b. Increased heat production and decreased heat loss
F c. Decreased heat production and decreased heat loss
F d. None of the above

Q. 184. **Ascending tracts are**
T a. spino-olivary tract
T b. fasciculus gracilis
T c. fascicutus cuneatus
F d. rubrospinal tract
F e. vestibulospinal tract.

Q. 185. **Descending tracts are**
T a. extra pyramidal tract
T b. pyramidal tract
T c. corticobulbar tract

F d. spinocortical tract
F e. spin-olivary tract.

Q. 186. **Extrapyramidal tracts are**
T a. reticulospinal tract
T b. tectospinal tract
T c. rubrospinal tract
F d. fasciculus tract
F e. lateral spinothalamic tract.

Q. 187. **Functions of tract of gall are to carry**
T a. kinesthetic sensation from lower part of the body
T b. senses of vibration.
T c. fine touch from lower half of the body
F d. fine touch from upper half of the body
F e. pain sensation

Q. 188. **Sensations transmitted through dorsal column medial leminical system are**
T a. pressure sensation.s
T b. touch sensation
T c. position senses from joints
F d. thermal sensation
F e. pain sensation.

Q. 189. **Spinothalamic tracts transmit**
T a. pain sensation
T b. thermal sensation
T c. sexual sensation.
F d. vibration
F e. proprioception

Q. 190. **Antero-lateral column carries the following sensation**
T a. cold
T b. itching.
T c. crude touch
F d. pressure
F e. position senses.

Q. 191. **Tract of gall**
T a. has a higher degree of spatial orientation of the nerve fibers in respect to their origin
T b. carries position senses from the lower part of the body
T c. carries rapidly transmitted sensations
F d. carries pain sensation
F e. is formed by C-fibers.

Q. 192. **Lateral spinothalamic tract**
T a. transmit thermal sensation
T b. is a cruder type of transmission system
F c. formed by A α fibers
F d. carries proprioception
F e. transmit sensory information that must be transmitted rapidly

Q. 193. **Regarding dorsal spino-thalamic tract**
T a. the second order neuron start from tract of lissauer
T b. it terminates in the thalamus
T c. it occupies the lateral column of spinal cord
F d. the second order neuron start from posterior horn cell.
F e. it terminates in precentral gyrus.

Q. 194. **Functions of extrapyramidal tracts are to maintain**
T a. movement of eye ball
T b. posture
T c. muscle tone
T d. equilibrium
F e. kinesthetic sensation.

Q. 195. **Regarding corticospinal tract**
T a. the fibers originate from giant pyramidal cells
T b. it transmits motor signals directly from cortex to spinal cord
T c. it contains more than 1 million fibers.
F d. the fibers are mostly unmyelinated.
F e. the fibers mostly cross in lower part of pons.

Q. 196. **Pain stimulants are**
T a. bradykinin
T b. prostaglandin
T v. histamine
F d. epinephrine
F e. oxytocin.

Q. 197. **Pain is**
T a. unpleasant sensation due to tissue damage
T b. mainly a protective mechanism for body
T c. produced by histamine
F d. produced by epinephrine
F e. produced by sodium.

Q. 198. **Alternative name of fast pain includes**
T a. pricking pain
T b. acute pain
T c. sharp pain
F d. aching pain
F e. throbbing pain.

Q. 199. **Alternative name of slow pain are**
T a. nauseous pain
T b. throbbing pain
T c. chronic pain
F d. electric pain
F e. pricking pain.

Q. 200. **Tracts through which pain sensation transmitted are**
T a. paleospinothalamic
T b. neospinothalamic
F c. rubrospinal

F d. vestibulospinal
F e. corticospinal.

Q. 201. **Fast pain is**
T a. elicited by thermal stimuli
T b. felt within 0.1 second
T c. elicited by mechanical stimuli
F d. elicited by chemical pain stimuli
F e. felt within 0.6 second.

Q. 202. **Fast pain is**
T a. transmitted through neospinothalamic tract
T b. stimulated by glutamate
T c. Transmitted through Aδ fiber
F d. transmitted through C fiber
F e. transmitted through paleospinothalamic tract.

Q. 203. **Brown-Sequard's syndrome is characterized by the loss of**
T a. voluntary movements on the same side
T b. pain sensation on the opposite side
F c. thermal sensation on the same side
F d. position sensation on the opposite side
F e. two-point discrimination on the opposite side.

Q. 204. **Extrapyramidal motor system lesion causes**
T a. lead pipeparalysis
T b. an abnormal gait with small fast regular step
T c. tremor at rest
F d. muscle paralysis
F e. muscle wasting.

Q. 205. **Anterolateral system carries following**
T a. pressure
T b. pain
T c. temperature
F d. vibration
F e. position sensation.

Q. 206. **Pyramidal tract**
T a. conveys impulse for movement of finger and hand
T b. is related to superficial reflexes
F c. controls movement of eye ball
F d. maintains equilibrium
F e. carries pain sensation.

Cerebral cortex

Q. 207. **Skilled voluntary movement is initiated at**
T a. Cerebral cortex (motor cortex)
F b. Basal ganglia
F c. Cortical association areas
F d. Cerebellum
F d. Medulla.

Q. 208. **All of the following manifestations are seen in cases of cerebellar damage in human beings except**
T a. Ataxia, atonia and asthenia.
T b. Loss of non-declarative/reflexive memory
T c. Loss of adjustment of vestibulo-ocular reflex
F d. Static tremour and rigidity.

Q. 209. **Destruction of sensory area 1 of brain leads to loss of which sensations?**
T a. Sterognosis & 2 point discrimination
F b. Pain
F c. Touch
F d. Sterognosis gone but 2 point discrimination retained
F e. Temperature.

Q. 210. **The following relay in sensory cortex**
T a. Pain
T b. Touch
T c. Temperature
F d. Olfaction
F e. Vision.

Q. 211. **Pyramidal fibers are**
T a. Projection fibres
F b. Association fibres
F c. Commissural fibres
F d. Association fibres and commissural fibres
F e. All of the above.

Q. 212. **Broca's area : which is true**
T a. Supplied by MCA (middle cerebral artery)
F b. Present bilaterally in brain
F c. Lesion causes Laryngeal palsy
F d. Present in the temporal lobe
T e. Supplied by ACA (anterior cerebral artery).

Q. 213. **For each 1° F rise of temperature, cerebral blood flow falls by**
T a. 4%
F b. 1%
F c. 2%
F d. 7%
F d. 6%.

Q. 214. **Temopral lobe lesion causes**
T a. Homonymus upper quadrantinopia
F b. Homonymus lower quadrantinopia
F c. Bi-temporal hemianopia
F d. Bi-nasal hemianopia
F e. Protanopia.

Q. 215. **Damage to sensory area 1 of the cerebral cortex results in**
T a. Loss of tactile and two point discrimintion
F b. Loss of perception of pain

F c. Loss of perception of touch
F d. Loss of only tactile discrimintion
F d. Loss of only discrimintion.

Q. 216. **Vomiting centre is situated in the**
T a. Amygdala
F b. Hypothalamus
F c. Pons
F d. Medulla
F e. Thalamus.

Q. 217. **Broca's area is present in**
T a. Inferior frontal gyrus
F b. Superior temporal Gyru
F c. Precentral gyrus
F d. Post central gyrus
T a. Inferior temporal gyrus.

Q. 218. **Motor area of Bradman is area**
T a. 4
F b. 1
F c. 5
F d. 7
F e. 17

Q. 219. **Layers of cerebral cortex are**
T a. external pyramidal layer
T b. internal granular layer
T c. molecular layer
T d. external granular layer
F e. layers of rods and cones.

Q. 220. **Storage of information**
T a. is located in cerebral cortex
T b. causes future thinking process with its large fraction
T c. causes response with its small fraction
F d. causes response with its large fraction
F e. causes future thinking process with its small fraction.

Q. 221. **Study of living brain have been dramatically enhanced by the development of the following new imaging techniques**
T a. computerized tomography
T b. positron emission tomography
T c. magnetic resonance imaging
T d. angiography
F e. electroencephalography.

Cerebellum

Q. 222. In parkinsonism, tremor is :
T a. 6-8 per second
F b. 2 per second
F c. 2-4 per second
F d. uncountable
F e. 1 per second

Q.223. **Vestibular fibres relay in**
T a. Floculonodular lobe of cerebellum
F b. Vermis
F c. Lateral geniculate body
F d. Auditory cortex
F e. Medial geniculate body.

Q. 224. **Floculonodular lobe of cerebellum is concerned with**
T a. Equilibrium
F b. Co-ordination
F c. Baroreception
F d. Chemoreception
F e. Olfaction.

Q. 225. **The cerebellum**
T a. Has a totally inhibitory output from its cortex
F b. Has only excitatory signal output from its deep nuclear layers
F c. Has a conscious interpretation of motor activity
F d. Has inhibitory influence on muscle tone in humans
F e. All of the above.

Q. 226. **Purkinjee cell is a**
T a. Output cell
F b. Input cell
F c. Inter neuron
F d. Connector neuron
F e Ganlion cell.

Q. 227. **In cerebellar disease, the correct statements are**
T a. The Rombergs sign is positive
T b. There is Adiodokokinesis
T c. There is pendular knee jerk
F d. There is involuntary tremor response.

Q. 228. **Cerebellum is important in controlling of**
T a. movement maintenance
T b. balance maintenance
T c. accuracy of muscle movement
T d. smooth muscle movement
F e. initiating voluntary muscle movement

Q. 229. **Disease of one cerebellar hemisphere would likely results in**
T a. past pointing
T b. a tendency to fall toward the side of the lesion.
F c. involuntary tremor
F d. spastic paralysis
F e. bradykinesia

Q. 230. **Efferent fibers connecting the cerebellum are**
T a. fastigio-vestibular tract
T b. fastigio-bulbar tract

T c. cerebello-olivary tract
F d. dorsal spino-cerebellar tract
F e. vestibulo-cerebellar tract

Q. 231. **Disorders of the cerebellum cause**
T a. symptoms ipsilateral to the defect
T b. weakness of movements so that movements are carried out slowly
T c. ataxia
F d. increased muscle tone
F e. exaggerated deep reflexes.

Q. 232. **Cerebellum**
T a. responds to vestibular stimuli and assists in maintaining equilibrium
T b. plays important role in progression from one movement to the next
T c. facilitates a smooth, co-ordinated voluntary movements
F d. does not play an active role in maintenance of posture
F e. receives sensory information.

Q. 233. **Effect of cerebellar lesion are**
T a. asthenia
T b. ataxia
T c. hypotonia
F d. paralysis
F e. perkinsonism.

Q. 234. **Afferent fibers connecting the cerebellum are**
T a. vestibulo-cerebellar tract
T b. olivo-cerebellar tract
T c. dorsal spino-cerebellar tract
F d. fastigio-vestibular tract
F e. cerebello-olivary tract.

Q. 235. **Functions of the cerebellum are**
T a. prediction of future position of moving parts of the body
T b. prediction of rates of progression of visual phenomenon
T c. error control of movement
F d. initiation of motor activity
F e. appreciation of sensation.

Q. 236. **Equilibrium is controlled by**
T a. cerebellum
T b. reticular substance
T c. mesencephalon
F d. limbic system
F e. basal ganglia.

Hypothalamus

Q. 237. **Injection of hypertonic saline into which area causes diuresis**
T a. Supraoptic nucleus
F b. Paraventricular nucleus
F c. Preoptic nucleus
F d. Posterior pituitary
F d. Anterior pituitary.

Q. 238. **In the neurohypophysis, secretory granules accumulate in**
T a. Nerve endings
F b. Pituicytes
F c. Intercellular spaces
F d. Capillary endothelium
F e. Axon.

Q. 239. **Decreased hypothalamic function casues depressed levels of the following homones**
T a. Growth hormone
T b. TSH
T c. ACTH
F d. Prolactin
F e. Oxytocin.

Q. 240. **One of the following is a function of hypothalamus**
T a. Homeostasis of temperature
F b. Swallowing
F c. Vomiting
F d. Respiration
F e. Diglutation.

Q. 241. **Profound hypothermic signs include**
T a. Slow breathing
T b. Bradycardia
T c. Hypotension
F d. Hyperactivity
F e. Hypoactivity

Q. 242. **Which of the following are true of median eminence**
T a. Portion of ventral hypothalamus
T b. Hypothalmo-hypophyseai vessels arise here
T c. Outside the blood-brain barrier
F a. Portion of dorsal hypothalamus
F d. All are correct

Q. 243. **What is the mechanism by which infection causes fever**
T a. Pyrogens acting independent of hypothalamus
T a. Effects of inflammatory mediator.
F b. Increased heat production and decreased heat loss
F c. Decreased heat production and decreased heat loss
F d. None of the above

Q. 244. **Hypothalamus**
T a. inhibits sympathetic nerve in hot weather
T b. causes vasodilatation in hot weather
T c. inhibits parasympathetic nerve in cold environment

F d. stimulates parasympathetic nerve in cold environment
F e. stimulates sympathetic nerve in hot weather.

Q. 245. **Hypothalamus**
T a. regulates the activity of anterior pituitary gland
T b. regulates body temperature
T c. participates in the integrated control of CVS
F d. maintains rhythmic breathing
F e. controls the voluntary movements of the body.

Q. 246. **Hypothalamus**
T a. is the centre for emotion and personality
T b. regulate body temperature
T c. is the centre for autonomic nervous system.
F d. is the centr for somatic nervous system
F e. has no endocrine function.

Q. 247. **Thalamus**
T a. is situated in the midbrain and part of fore brain
T b. is an important reflex center
T c. is a collection of gray matter containing nerve cells
F d. is situated in the hind brain
F e. regulates body temperature.

Q. 248. **Functions of different nuclei of hypothalamuse include**
T a. ventro-medial nucleus causes satiety
T b. lateral nucleus causes thirst and eating
F c. posterior nucleus causes increased heat loss
F d. anterior nucleus causes decreased heat loss
F e. supra-optic nucleus causes secretion of oxytocin.

Q. 249. **Posterior nucleus of hypothalamus**
T a. causes vasoconstriction
T b. inhibits parasympathetic nerve
T c. stimulates sympathetic nerve
F d. inhibits sympathetic nerve
F e. stimulates parasympathetic nerve.

Q. 250. **Posterior nucleus of hypothalamus**
T a. decreases heat loss
T b. is activated in cold weather
F c. causes vasodilatation
F d. is activated in hot weather
F e. increases heat loss.

Q. 251. **Anterior nucleus of hypothalamus**
T a. inhibits sympathetic nerve
T b. causes vasodilatation
T c. stimulates parasympathetic nerve
F d. stimulates sympathetic nerve
F e. inhibits parasympathetic nerve.

Q. 252. **Anterior nucleus of thalamus**
T a. increases heat loss
T b. is activated in hot environment
F c. causes vasoconstriction
F d. is activated in cold environment
F e. decreases heat loss.

Basal ganglia/limbic system

Q. 253. **Stereoanesthesia is due to lesion of**
T a. Nucleus cuneatus
F b. Nucleus Gracilis
F c. Spinoreticular tract
F d. Spinothalamic tract
F e. All of the above.

Q. 254. **Where is motor activity probably initiated in the brain?**
T a. Basal ganglia
F b. Motor cortex
F c. Premotor cortex
F d. Cerebellum
F e. Pons.

Q. 255. **Vomiting centre is situated in the**
T a. Amygdala
F b. Hypothalamus
F c. Pons
F d. Medulla
F e. Mid brain.

Q. 256. **Hyperkinetic syndromes such as chorea and athetosis are usually associated with pathological changes in**
T a. Basal ganglia complex
F b. Motor areas of cerebral cortex
F c. Anterior hypothalamus
F d. Pathways for recurrent collateral inhibition in the spinal cord
F e. Pons

Q. 257. **Arousal response is mediated by**
T a. Reticulo activating system
F b. Dorsal column
F c. Spinothalamic tract
F d. Vestibule cerebellar tract
F e. Spinothalamic tract.

Q. 258. **During light sleep, the sleep spindles that appear have the frequency of**
T c. 14-16 /second
F a. 1-2 /second
F b. 6-12 /second

F d. 21-26 /second
F c. 6-8 /second.

Q. 259. **Headache can be produced by**
T a. Dilatation of intracranial blood vessels
T b. Presence of blood in CSF
T c. Loss of CSF following lumbar puncture
F d. Mechanical damage to parietal cortex
F e. Mechanical damage to temporal cortex.

Q. 260. **The condition known as REM sleep is**
T a. Referred to as paradoxcal sleep
F b. That point at which the indivudual becomes aware and alert
F c. Characterized by total lack of all muscular activity
F d. Charactrized by slow high voltage regular EEG activity
F e. All of the above.

Q. 261. **In a person with a history of alcohol abuse which of the following aspects of memory is most likely to be impaired?**
T a. Recall of events occuring a few weeks previously
F b. Accurate perception of stimuli
F c. Immediate recall of new information
F d. Recall of events in the remote past
F e. Recognition of familiar objects

Q. 262. **The basal ganglia controls**
T a. simple gross movement
T b. complex pattern of movement
T c. body equilibrium
F d. relative intensity of movement
F e. reflex movement.

Q. 263. **Basal ganglia dysfunction is characterized by**
T a. lead-pipe rigidity
T b. bradykinesia
F c. hypotonia
F d. intention tremor
F e. dysmetria.

Q. 264. **Basal ganglia**
T a. lesion produces tremor in the distal joints
T b. helps to prepare the body for movements.
T c. enables the trunk and limbs to be placed in appropri -ate positions before the motor cortex activates discrete movements
F d. lesion usually reduces muscle tone
F e. lesion produces incordination of movements.

Q. 265. **Important structures comprising the limbic system are**
T a. amygdala
T b. hippocampus
T c. hypothalamus
F d. cerebellum
F e. cerebral cortex.

Q. 266. **Slow wave sleep**
T a. is characterized by vegetative functions of the body
T b. is broken periodically by rapid eye movement sleep
T c. occurs during the first hour of sleep
F d. is exceedingly unrestful
F e. is associated with 10 to 30% increase of blood pressure.

Q. 267. **During deep sleep there is a fall in**
T a. metabolic rate
T b. urine formation
F c. hand skin temperature
F d. arterial PCO_2
F e. growth hormone and cortisol ratio.

Q. 268. **Effects of lesion in basal ganglia are**
T a. hemibalismus
T b. Perkinsonism
T c. chorea
F d. insomnia
F e. nystigmus.

Q. 269. **Basal ganglia consists of**
T a. putamen
T b. subthalamic nucleus.
T c. caudate nucleus
F d. hypothalamus
F e. thalamus.

Q. 270. **Limbic system consist of**
T a. amygdala
T b. hypothalamus
T c. orbitofrontal gyrus
F d. thalamus
F e. cerebellum.

EEG

Q. 271. **The frequency of alpha waves in EEG is**
T a. 8 to 12 cycles/second
F b. 15-25 cycles/second
F c. 2-5 cycles/second
F d. None of the above

Q. 272. **The electroencephalographic (EEG) curves are called**
T a. Berger's rhythm
F b. ABCDE curves
F c. Neurogenic rhythm
F d. REM rhythm

Q. 273. **EEG rhythm recorded from the surface of the scalp during REM sleep:**

T a. Beta
F b. Alpha
F c. Delta
F d. Theta

Q. 274. **The frequency of Beta waves (per sec) in EEG is** :

T a. 13-30
F b. 0-4
F c. 4-7
F d. 7-13
F e. 2-5.

Q. 275. **Which of the following is wrongly matched about EGG waves and their duration :**

T a. Q wave- 0.10 second
F b. P wave - 0.10 second
F c. S wave - 0.04 second
F d. QRS complex- 0.10 second.

Q. 276. **Buerger waves (alpha waves) of EEG have the rhythm per second of**

T a. 8-13
F b. 0-4
F c. 4-7
F d. 13-30

Q. 277. **An EEG**

T a. Is bilaterally symmetrical
F b. Provides indication of intelligence
F d. Tends to show waves of smaller amplitude during deep sleep than of alert state
F e. Show waves with a lower frquency during intense thought than during sleep.