Lecture 19- Bien 500 2023:
Guyton Chapter -- :

The portion of the nervous system that controls most visceral functions of the body is called the autonomic nervous system

General organization of the autonomic nervous system. The autonomic nervous system is activated mainly by centers located in the spinal cord, brain stem, and hypothalamus.

The autonomic nervous system also often operates by means of visceral reflexes. That is, ***subconscious sensory signals*** from a visceral organ can enter the autonomic ganglia, the brain stem, or the hypothalamus and then return ***subconscious reflex responses*** directly back to the visceral organ to control its activities.

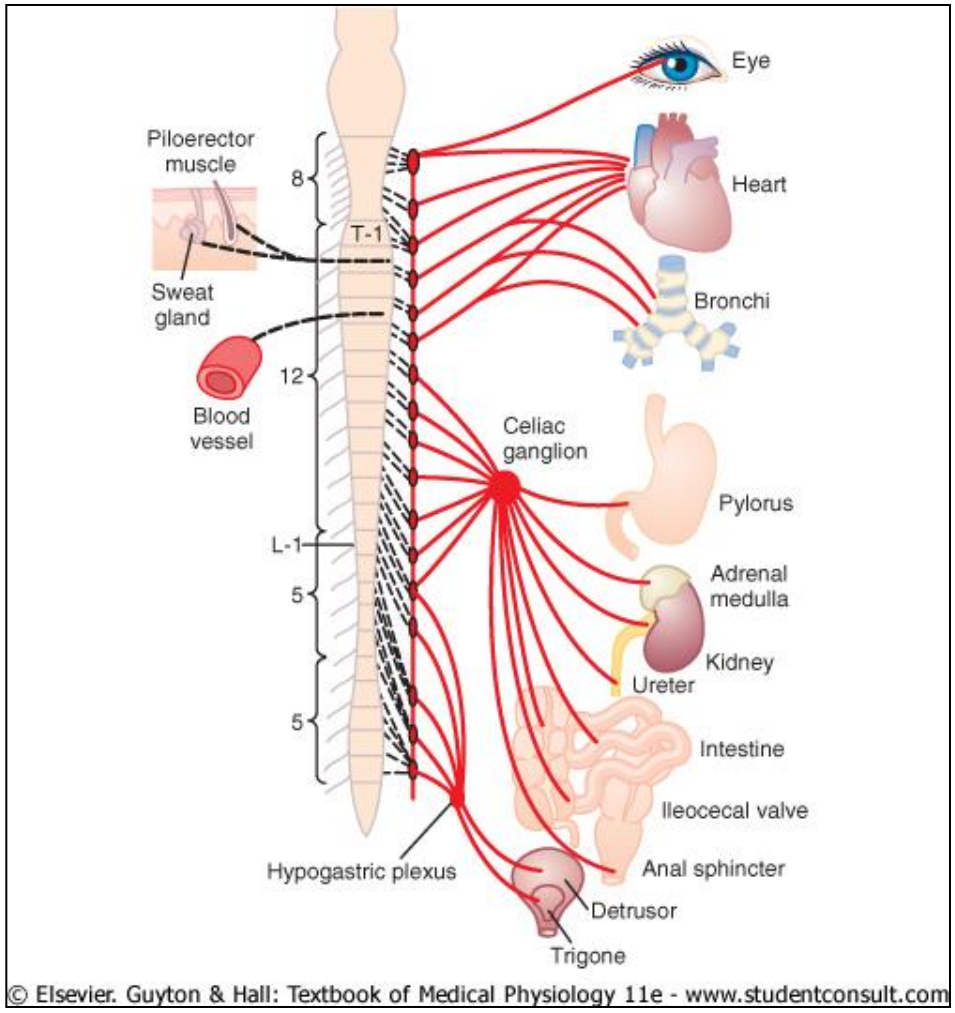

Figure 60-1 Sympathetic nervous system. The black dashed lines represent postganglionic fibers in the gray rami leading from the sympathetic chains into spinal nerves for distribution to blood vessels, sweat glands, and piloerector muscles.

- The ***efferent*** autonomic signals are transmitted to the various organs of the body through two major subdivisions called the sympathetic nervous system and the parasympathetic nervous system.
- Physiologic anatomy of the ***sympathetic nervous system:*** Fig. 60-1. The sympathetic nerve fibers originate in the spinal cord along with spinal nerves between cord segments T-1 and L-2 and pass *first into the sympathetic chain* and then to the tissues and organs that are stimulated by the sympathetic nerves.

Preganglionic and postganglionic sympathetic neurons

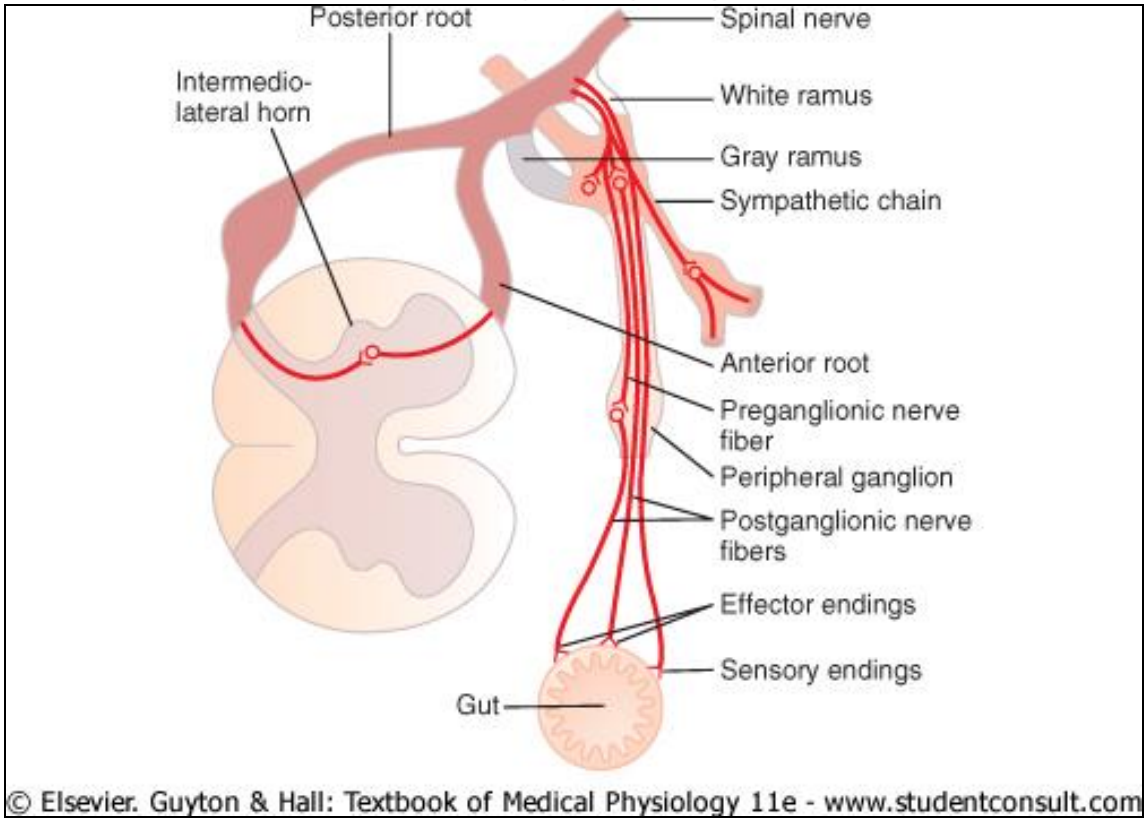

Figure 60-2 Nerve connections between the spinal cord, spinal nerves, sympathetic chain, and peripheral sympathetic nerves.

- The sympathetic nerves are different from skeletal motor nerves in the following way:
- each sympathetic pathway from the cord to the stimulated tissue is composed of two neurons, a preganglionic neuron and a postganglionic neuron, in contrast to only a single neuron in the skeletal motor pathway.
- The cell body of each preganglionic neuron lies in the intermediolateral horn of the spinal cord; its fiber passes, as shown in fig. 60-2, through an anterior root of the cord into the corresponding spinal nerve.

- Immediately after the spinal nerve leaves the spinal canal, the preganglionic **sympathetic** fibers leave the spinal nerve and connect to one of the ganglia of the sympathetic chain. The fibers can then do one of the following
- 1)synapse with postganglionic sympathetic neurons in the ganglion that it enters;
- 2) it can pass upward or downward in the chain and synapse in one of the other ganglia of the chain; or
- 3) it can pass for variable distances through the chain and then through one of the sympathetic nerves radiating outward from the chain, finally synapsing in a peripheral sympathetic ganglion.

- The postganglionic sympathetic neuron thus originates either in one of the sympathetic chain ganglia or in one of the peripheral sympathetic ganglia.
- From either of these two sources, the postganglionic fibers then travel to their destinations in the various organs.

Special nature of the **sympathetic** nerve endings in the adrenal medullae

- Preganglionic sympathetic nerve fibers pass, without ***synapsing***, all the way from the intermediolateral horn cells of the spinal cord, through the sympathetic chains, and finally into the two adrenal medullae.
- There they end directly on modified neuronal cells that secrete epinephrine and norepinephrine into the blood stream.
- These secretory cells embryologically are derived from nervous tissue and are actually themselves postganglionic neurons.
- They even have rudimentary nerve fibers, and it is the endings of these fibers that secrete the adrenal hormones epinephrine and norepinephrine.

Physiologic anatomy of the **parasympathetic** nervous system

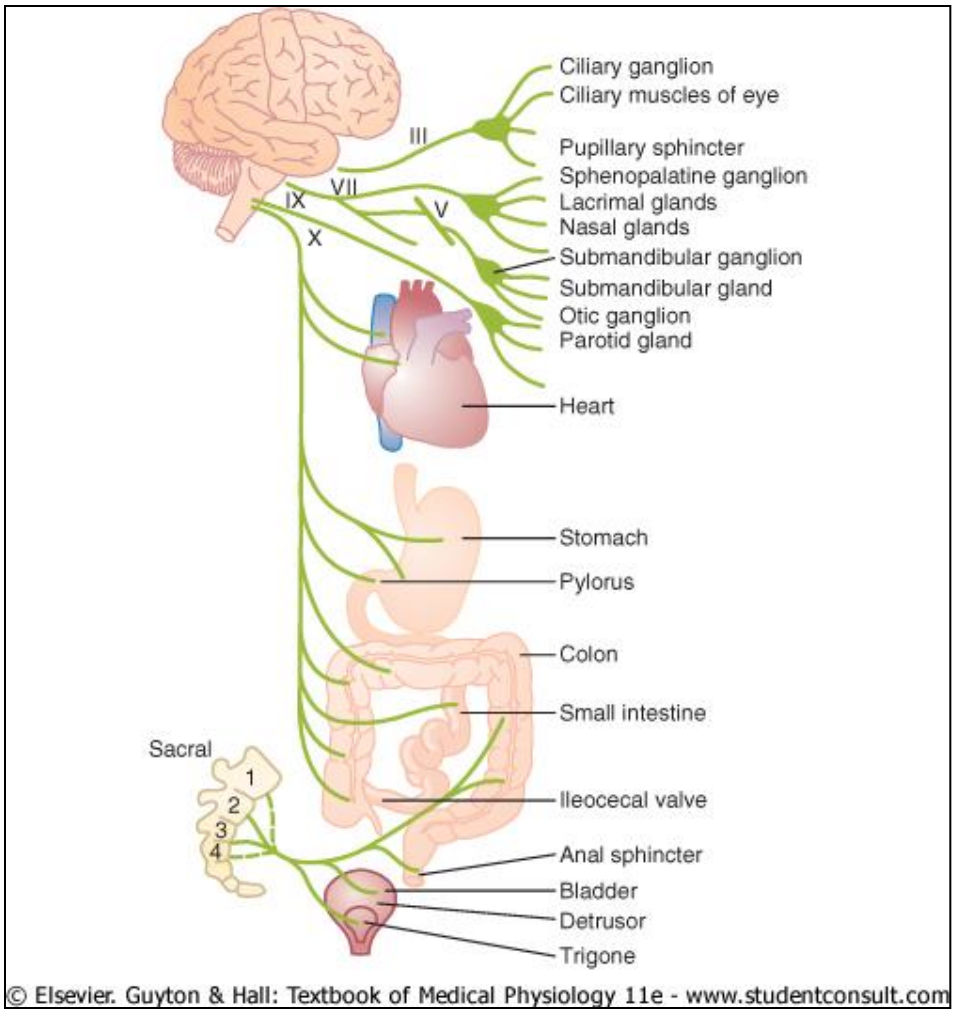

Figure 60-3 Parasympathetic nervous system.

Shown in fig. 60-3. Parasympathetic fibers leave the CNS through cranial nerves III, VII, IX, and X. About 75% of all parasympathetic nerve fibers are in the vagus nerve (cranial nerve X)

Preganglionic and postganglionic **parasympathetic** neurons.

- The parasympathetic system, like the sympathetic, has both preganglionic and postganglionic neurons.
- However, except in a few cases, the preganglionic fibers pass uninterrupted all the way to the organ that is to be controlled.
- In the wall of the organ are located postganglionic neurons.
- The preganglionic fibers synapse with these, and very,very short postganglionic fibers leave the neurons to innervate the tissues of the organ.
- This is different than the arrangement of the sympathetic ganglia, because the cell bodies of the sympathetic postganglionic neurons are almost always located in the ganglia of the sympathetic chain or in various other discrete ganglia, rather than in the excited organ itself.

Basic characteristics of sympathetic and parasympathetic function

- The sympathetic and parasympathetic nerve fibers secrete mainly one or other of two synaptic transmitter substances, acetylcholine (Ach) or norepinephrine. Those fibers that secrete acetylcholine are said to by ***cholinergic***.
- Those that secrete norepinephrine are said to be ***adrenergic***, a term derived from adrenalin, which is an alternative name for epinephrine.
- All preganglionic neurons are cholinergic in both the sympathetic and the parasympathetic nervous systems.
- Ach or Ach-like substances, when applied to the ganglia, will excite both sympathetic and parasympathetic postganglionic neurons.
- Most of the postganglionic neurons of the parasympathetic system are also cholinergic. ***Conversely***, most of the postganglionic **sympathetic** neurons are adrenergic.

- Thus, the terminal nerve endings of the parasympathetic system mostly all secrete Ach. Almost all of the sympathetic nerve endings secrete norepinephrine, but few secrete Ach.
- These hormones (neurotransmitters) in turn act on the different organs to cause respective parasympathetic or sympathetic effects. Therefore, *Ach is called a parasympathetic* transmitter, and *norepinephrine a sympathetic* transmitter.

Mechanisms of transmitter secretion and subsequent removal of the transmitter at the postganglionic endings.

- Many of the parasympathetic and sympathetic nerve fibers touch the effector cells of the organs that they innervate as opposed to formal neuromuscular junctions as found in skeletal muscle innervation.

- In some cases, they terminate in the connective tissue located adjacent to the cells that are to be stimulated. Where these filaments touch or pass over or near the cells to be stimulated, they usually have bulbous enlargements called varicosities; it is in these varicosities that the transmitter vesicles of Ach or epinephrine are synthesized and stored.
- Also in these varicosities are ***large numbers of mitochondria*** that supply ATP, required to energize Ach or norepinephrine synthesis.

- When an action potential spreads over the terminal fibers, the depolarization process increases the permeability of the fiber membrane to calcium ions, allowing these ions to diffuse into the nerve terminals or nerve varicosities.
- The calcium ions in turn cause the terminals or varicosities to empty their contents to the exterior. Thus the transmitter substance is secreted.

Synthesis of Ach, its destruction after secretion, and duration of its action

- Ach is synthesized in the terminal endings and varicosities of the cholinergic nerve fibers where it is stored in vesicles in highly concentrated form until it is released. The basic chemical reaction of this synthesis is the following:
- Acetyl-CoA + choline via choline acetyltransferase---->>>> Ach
- **(Where does Acetyl-CoA come from?)**
- Once acetylcholine is secreted into a tissue by a cholinergic nerve ending, it persists in the tissue for a few seconds while it performs in nerve signal transmitter function.
- Then it is split into an acetate ion and choline, catalyzed by the enzyme ***acetylcholinesterase***. (same enzyme as used at neuromuscular junctions).
- The choline that is formed is then transported back into the terminal nerve ending, where it is used again and again for synthesis of new Ach.

Synthesis of norepinephrine, its removal, and its duration of action

- Synthesis of norepinephrine begins in the axoplasm of the terminal nerve endings of adrenergic nerve fibers but is completed inside the secretory vesicles. The basic steps are the following:
- 1. tyrosine-→ Dopa
- 2. Dopa-→ Dopamine
- 3. transport of dopamine into the vesicles
- 4. dopamine-→ norepinephrine
- in the adrenal medulla, this reaction goes still one step further to transform about 80 percent of the norepinephrine into epinephrine, as follows:

5. norepinephrine-→ epinephrine

[what kind of molecule is tyrosine?]

- norepinephrine is removed from the secretion site in one of 3 ways:
- 1) reuptake into the adrenergic nerve endings themselves by an active transport process (this process accounts for removal of 50-80 percent of the secreted norepinephrine);
- 2) diffusion away from the nerve endings into the surrounding body fluids and then into the blood; and
- 3) destruction of small amounts by tissue enzymes (such as monoamine oxidase , which is found in the nerve endings).

Receptors on the effector organs

- Before Ach, epineph, or norepi can stimulate an effector organ, it must first bind with ***specific receptors*** on the effector cells.
- Binding of the transmitter to the receptor causes a conformational change in the structure of the protein receptor molecule. This conformational change excites or inhibits the cell, most often by 1) causing a change in cell membrane permeability to one or more ions or
- 2) activating or inactivating an enzyme attached to the other end of the receptor protein where it protrudes into the interior of the cell.

Excitation or inhibition of the effector cell by changing its membrane permeability

- A conformational change in structure of the receptor often opens or closes an ion channel thus altering the permeability of the cell membrane to various ions.
- For instance, sodium and/or calcium ion channels frequently become opened and allow rapid influx of the respective ions into the cell, usually depolarizing the cell membrane and exciting the cell.
- At other times potassium channels are opened, allowing potassium ions to diffuse out of the cell, and this usually inhibits the cell because loss of electropositive K+ ions creates hypernegativity inside the cell.

Receptor action by altering intracellular ***second messenger*** enzymes

- Another way a receptor often functions is to activate or inactivate an enzyme (or other intracellular chemical) inside the cell.
- The enzyme often is attached to the receptor protein where the receptor protrudes into the interior of the cell.
- For instance, binding of norepi with its receptor on the outside of cells can increase the activity of the enzyme adenylyl cyclase on the inside of the cell, and this causes formation of cyclic adenosine monophosphate (cAMP).
- The cAMP in turn can initiate any one of many different intracellular actions, the exact effect depending on the chemical machinery of the effector cell. Thus an autonomic transmitter substance can cause inhibition in some organs or excitation in others.

Two principal types of acetylcholine receptors- muscarinic and nicotinic receptors

- Acetylcholine activates mainly two types of receptors: muscarinic and nicotinic receptors.
- Muscarinic receptors are found on all effector cells that are stimulated by the postganglionic cholinergic neurons of either the parasympathetic or the sympathetic nervous system.
- Nicotinic receptors are found in the autonomic ganglia at the synapses between the preganglionic and postganglionic neurons of both the sympathetic and parasympathetic systems.
- (nicotinic receptors are also found for example at neuromuscular junctions in skeletal muscle).

Adrenergic receptors- alpha and beta receptors

- There are two major types of adrenergic receptors: alpha receptors and beta receptors.
- Norepi. Excites mainly alphas but also some betas to a lesser extent; epinephrine excites alphas and betas approximately equally.
- Therefore the relative effect of epi and norepi on different effector organs are determined by the type of receptors in the organs (on the cells).
- Both alpha and beta receptors can have excitatory as well as inhibitory functions.
- Thus the receptors are not necessarily excitatory or inhibitory, but rather their final effect ultimately depends on the affinity of the hormone for the receptors in the given effector organ.

Excitatory and inhibitory actions of sympathetic and parasympathetic stimulation- examples

- Eyes. Two functions of the eyes are controlled by the autonomic nervous system: 1) pupil opening and 2) focus of the lens.
- Heart. In general, sympathetic stimulation increases the overall activity of the heart. This is accomplished by increasing both the rate and force of heart contraction.
- Parasympathetic stimulation causes mainly opposite effects-decreased heart rate and strength of contraction

Function of the adrenal medullae

- Stimulation of the sympathetic nerves to the adrenal medullae causes large quantities of epinephrine and norepinephrine to be released into the circulating blood, and these two hormones in turn are carried in the blood to all tissues of the body.
- The circulating epinephrine and norepi. Have almost the same effects on the different organs as the effects caused by direct sympathetic stimulation, except that the effects last 5 to 10 times as long because both of these hormones are removed from the blood slowly over a period of 2 to 4 minutes.

- Circulating norepi causes constriction of essentially all the blood vessels of the body; increases activity of the heart, inhibition of the gastrointestinal tract, and dilation of the pupils of the eyes.
- Epinephrine causes almost the same effects as those caused by norepi.
- It increases the rate of metabolic activities such as glycongenolysis in the liver and muscle, and glucose release into the blood.

Value of the adrenal medullae to the function of the sympathetic nervous system

- An important value of the adrenal medullae is the capability of epinephrine and norepi to stimulate structures of the body that are not innervated by direct sympathetic fibers.
- For instance, the metabolic rate of every cell of the body is increased by these hormones, especially by epinephrine, even though only a small proportion of all the cells in the body are innervated directly by sympathetic fibers.

Autonomic reflexes

- Many visceral functions of the body are regulated by autonomic reflexes.
- Cardiovascular autonomic reflexes.
- Several reflexes in the cardiovascular system help to control especially the arterial blood pressure and the heart rate.
- One of these is the baroreceptor reflex: stretch receptors called baroreceptors are located in the walls of several major arteries including the arch of the aorta.
- When these become stretched by high pressure, signals are transmitted to the brain stem, where they inhibit the sympathetic impulses to the heart and blood vessels and excite the parasympathetics; this allows the arterial pressure to fall back to normal.

Stimulation of discrete organs in some instances and mass stimulation in other instances by the sympathetic and parasympathetic systems.

- -Sympathetic system often responds by mass discharge.
- --Parasympathetic system usually causes specific localized response

Alarm or stress response of the sympathetic nervous system

- When large portions of the sympathetic nervous system discharge at the same time- that is a mass discharge- this increases in many ways the ability of the body to perform vigorous muscle activity as follows:
- 1. increase arterial pressure.
- 2. increased blood flow to active muscle concurrent with decreased blood flow to organs such as the gastrointestinal tract and the kidneys that are not needed for rapid motor activity.
- 3. increased rates of cellular metabolism throughout the body.
- 4. increased blood glucose concentration.
- 5. increased glycolysis in the liver and in the muscle
- 6. increased muscle strength
- 7. increased mental activity
- 8. increase rate of blood coagulation

- The sum of these effects permits a person to perform far more strenuous physical activity than would otherwise be possible.
- Because ***either mental or physical stress*** can excite the sympathetic system, it is frequently said that the purpose of the sympathetic system is to provide extra activation of the body in states of stress: this is called the sympathetic **stress response**.
- The sympathetic system is especially strongly activated in many emotional states.
- Most of the aforementioned sympathetic events ensue immediately—this is called the sympathetic alarm reaction.
- It is also called the fight or flight reaction because an animal in this state decides almost instantly whether to stand and fight or to run. In either event, the sympathetic alarm reaction makes the animal's subsequent activities vigorous.

Chapter 61: cerebral blood flow, cerebrospinal fluid, and brain metabolism

- Regulation of cerebral blood flow.
- At least 3 metabolic factors have potent effects in controlling cerebral blood flow:
- 1) CO_2 concentration;
- 2) hydrogen ion concentration; and
- 3) oxygen concentration.
- Increase of cerebral blood flow in response to excess CO_2 or excess hydrogen ion concentration.
- Increased CO_2 concentration in the arterial blood perfusing the brain greatly increases cerebral blood flow.
- Increasing the acidity of the brain tissue will likewise increase cerebral blood flow, including substances such as lactic acid, pyruvic acid and any other acidic material formed during the course of tissue metabolism.

Importance of cerebral blood flow control by CO_2 and hydrogen ions

- Increased acidity greatly ***depresses*** neuronal activity.
- Therefore, it is fortunate that an increase in acidity also causes an increase in blood flow, which in turn carries hydrogen ions, carbon dioxide, and other acid forming substances away from the brain tissues, thus helping to maintain a normal, constant level of neuronal activity.

Oxygen deficiency as a regulator of cerebral blood flow

- Experiments have shown that a decrease in cerebral tissue PO_2 below about 30 mm Hg (normal value is 35 to 40 mm Hg), immediately begins to increase cerebral blood flow.
- This is fortuitous because brain function becomes deranged at not much lower values of PO_2.
- thus, the oxygen mechanism for local regulation of cerebral blood flow is a very protective response against diminished cerebral neuronal activity and, therefore, against derangement of mental capability

Effect of brain activity on blood flow

- Using blood flow measurement techniques, it has been observed that blood flow in each individual segment of the brain changes as much as 100 to 150 per cent within seconds in response to changes in local neuronal activity.
- For instance, simply making a fist of the hand causes an immediate increase in blood flow in the motor cortex of the opposite side of the brain.
- Reading a book increases the blood flow, especially in the visual areas and language areas of the cortex.
- Measuring procedures can also be used for localizing the origin of epileptic attacks because local brain blood flow increases acutely and markedly at the focal point of each attack.
- Demonstrating the effect of local neuronal activity on cerebral blood flow, fig. 61-2 shows a typical increase in blood flow in a cat’s brain when intense light is shined into its eyes for one-half minute.

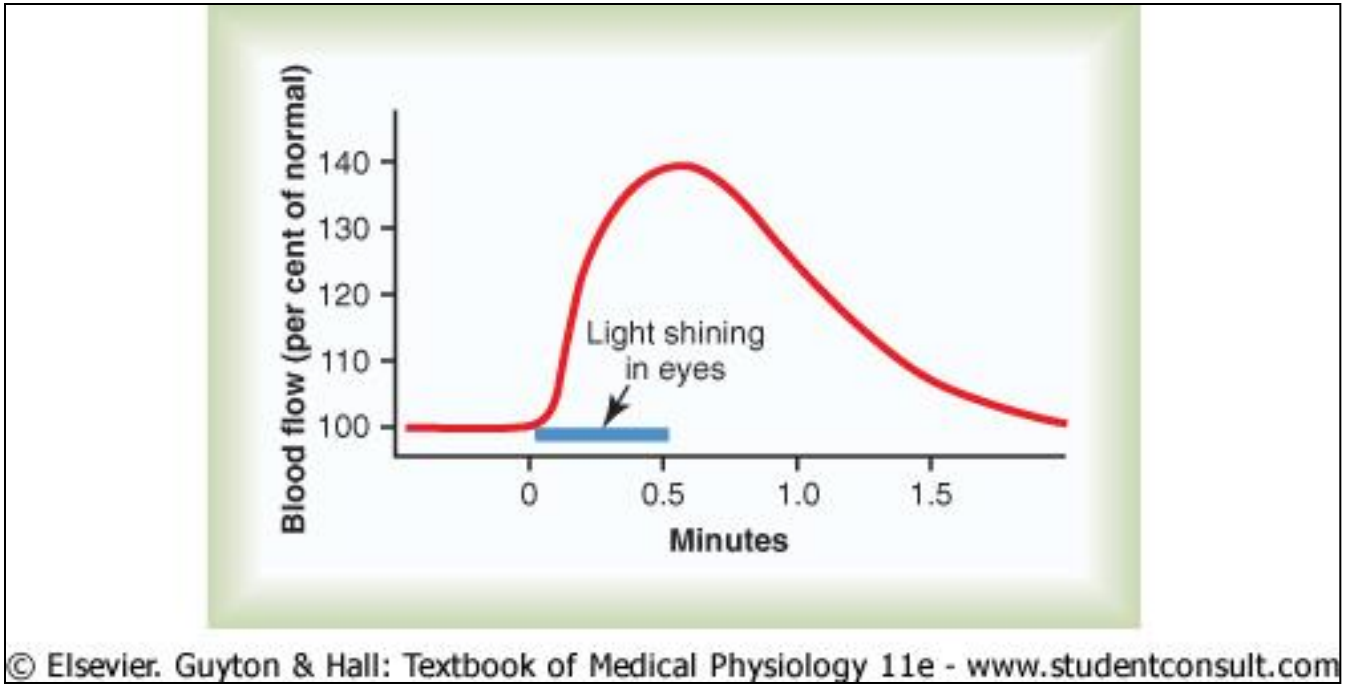

Figure 61-2 Increase in blood flow to the occipital regions of a cat's brain when light is shined into its eyes.

Role of the sympathetic nervous system in controlling cerebral blood flow

- When mean arterial pressure rises acutely to an exceptionally high level, such as during strenuous exercise or during other states of excessive circulatory activity, the sympathetic nervous system normally constricts the large- and intermediate-sized brain arteries enough to prevent the high pressure from reaching the smaller brain blood vessels.
- This is important in preventing vascular hemorrhages into the brain

Cerebral microcirculation

- An important structural characteristic of the brain capillaries is that they are much less leaky than the blood capillaries in almost any other tissue of the body.
- One reason for this is that the capillaries are supported on all sides by glial feet, which are small projections from the surrounding glial cells that abut against all surfaces of the capillaries and provide physical support to prevent overstretching of the capillaries in case of high capillary blood pressure.

Cerebral stroke occurs when cerebral blood vessels are blocked

- Almost all elderly people have blockage of some small arteries in the brain, and as many as 10 percent eventually have enough blockage to cause serious disturbance of brain function, a condition called a stroke.
- Most strokes are caused by arteriosclerotic plaques that occur in one or more of the feeder arteries to the brain.
- The plaques can activate the clotting mechanism of the blood, causing a blood clot to occur and block blood flow in the artery, thereby leading to acute loss of brain function in a localized area.
- In about one quarter of people who develop strokes, high blood pressure makes one of the blood vessels burst: hemorrhage then occurs, compressing the local brain tissue and further compromising its functions.
- The neurological effects of the stroke are determined by the brain area affected.

Cerebrospinal fluid (CSF) system

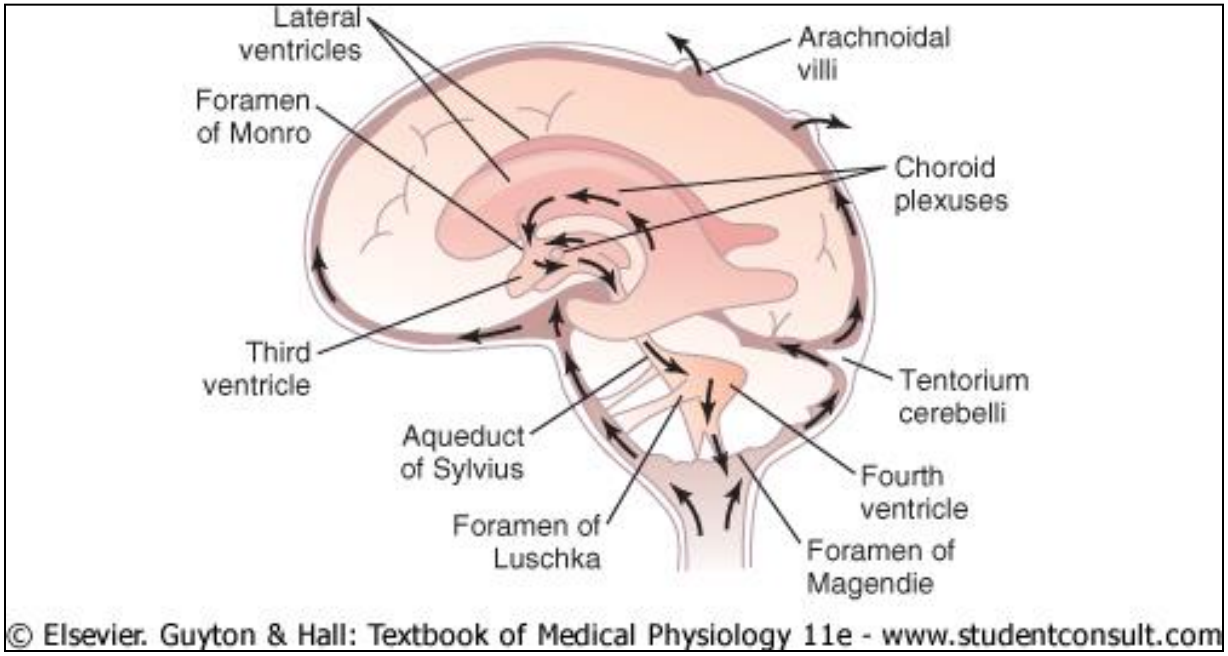

Figure 61-4 The arrows show the pathway of cerebrospinal fluid flow from the choroid plexuses in the lateral ventricles to the arachnoidal villi protruding into the dural sinuses.

The cerebrospinal fluid as shown in fig. 61-4 is present in the ventricles of the brain, in the cisterns around the outside of the brain, and in the subarachnoid space around both the brain and the spinal cord. All these chambers are connected with one another, and the pressure of the fluid is maintained at a surprisingly constant level.

Cushioning function of the CSF

- A major function of the CSF is to cushion the brain within its solid vault.
- The brain and the CSF have about the same specific gravity (only about 4 percent different), so that the brain simply floats in the fluid.
- Therefore, a blow to the head, if it is not too intense, moves the entire brain simultaneously with the skull, causing no one portion of the brain to be momentarily contorted by the blow.

Formation, flow, and absorption of CSF

- The arrows in Fig. 61-4 show that the main channels of fluid flow from the choroids plexuses and then through the CSF system.
- The fluid secreted in the lateral ventricles passes first into the third ventricle, then down into the 4th ventricle.
- The subarachnoid space surrounds the entire brain and spinal cord. CSF flows through the subarachnoid space to surround the cerebrum.

Secretion by the choroids plexus

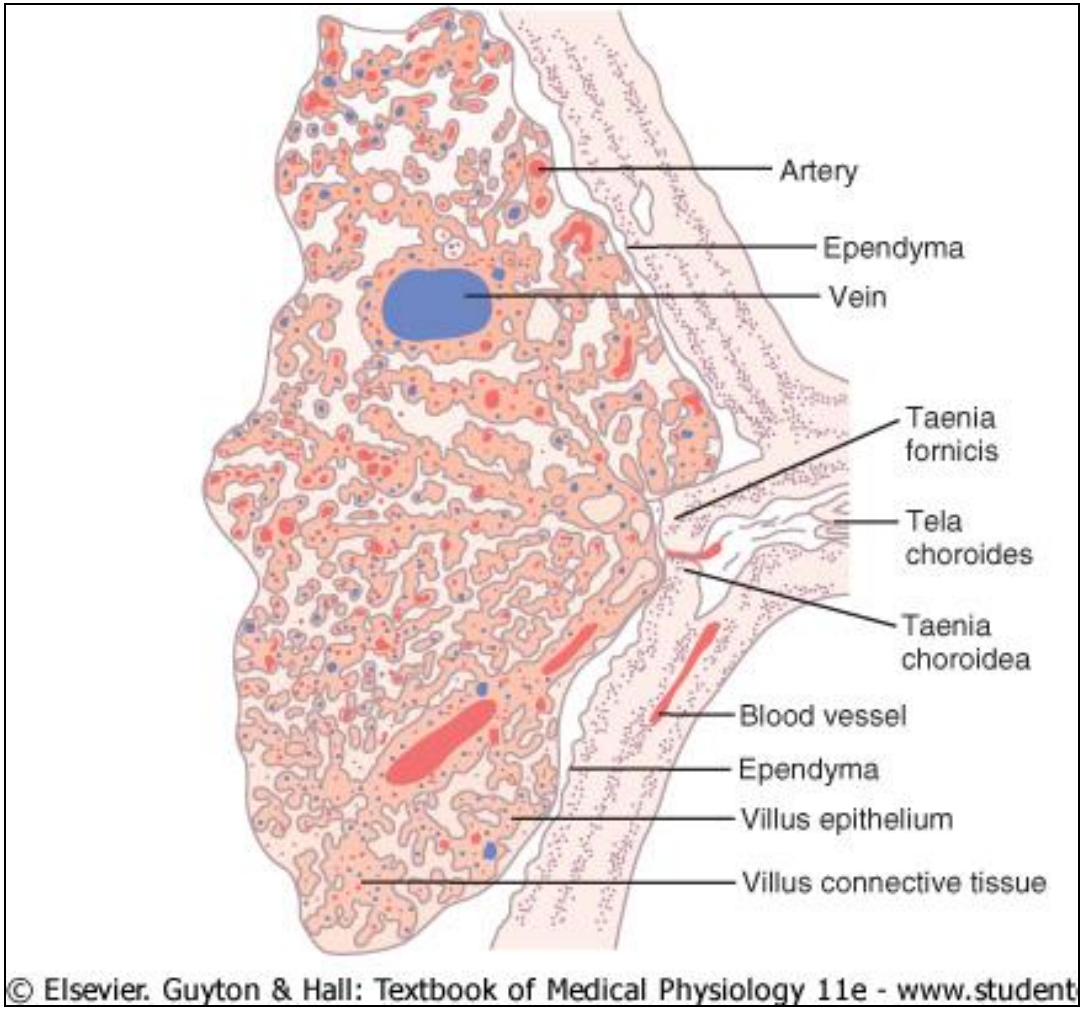

Figure 61-5 Choroid plexus in a lateral ventricle.

The choroids plexus, a section of which is shown in fig. 61-5, is a cauliflower like growth of blood vessels covered by a thin layer of epithelial cells. This plexus projects into lateral ventricles, and the 3rd and fourth ventricles.

Secretion of fluid into the ventricles by the choroids plexus depends mainly on active transport of sodium ions through the epithelial cells lining the outside of the plexus.

The sodium ions in turn pull along large amounts of chloride ions as well because of the positive charge of sodium ions.

The two of these together increase the quantity of osmotically active sodium chloride in the CSF, which then causes almost immediate osmosis of water through the membrane, thus providing the fluid of the secretion.

Perivascular spaces and CSF

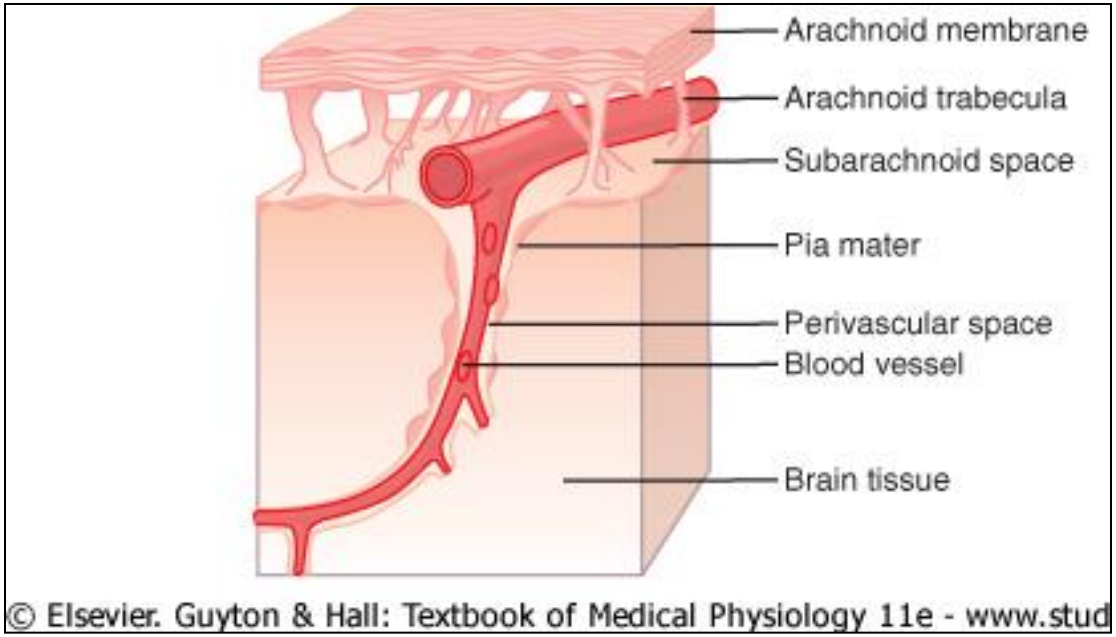

Figure 61-6 Drainage of a perivascular space into the subarachnoid space. (Redrawn from Ranson SW, Clark SL: Anatomy of the Nervous System. Philadelphia: WB Saunders Co, 1959.)

The large arteries and veins of the brain lie on the surface of the brain but their ends penetrate inward, carrying with them a layer of pia mater, the membrane that covers the brain, as shown in Fig. 61-6.

the pia is only loosely adherent to the vessels, so that a space, the perivascular space, exists between it and each vessel.

Lymphatic function of the perivascular spaces

- In addition to transporting fluid and proteins, the perivascular spaces transport extraneous particulate matter out of the brain.
- For instance, whenever infection occurs in the brain, dead white blood cells and other infectious debris are carried away through the perivascular spaces

Blood-CSF and Blood-brain barriers

- The concentrations of several important constituents of CSF are not the same as in extracellular fluid elsewhere in the body.
- Furthermore, many large molecular substances hardly pass at all from the blood into the CSF or into the interstitial fluids of the brain, even though these same substances pass readily into the usual interstitial fluids of the body.
- Therefore, it is said that barriers, called the blood-CSF barrier and the blood-brain barrier (BBB) exist between the blood and the CSF and brain fluid, respectively.

- In general, the blood-CSF barrier and BBB are highly permeable to water, CO_2, O_2, and most lipid-soluble substances such as alcohol and anesthetics; slightly permeable to electrolytes such as sodium, chloride, and potassium; and almost totally impermeable to plasma proteins and most non-lipid soluble large organic molecules.
- The cause of the low permeability of the blood-CSF barrier and the BBB is the manner in which the endothelial cells of the brain tissue capillaries are joined to one another.
- They are joined by so-called ***tight junctions***.
- That is, the membranes of the adjacent endothelial cells are tightly fused rather than having large pores between them, as is the case for most other capillaries of the body.

Brain edema

- One of the most serious complications of abnormal cerebral fluid dynamics is the development of brain edema.
- Because the brain is encased in a solid cranial vault, accumulation of extra edema fluid compresses the blood vessels, often causing seriously decreased blood flow and destruction of brain tissue.
- The usual cause of brain edema is either greatly increased capillary pressure or damage to the capillary wall that makes the wall leaky to fluid.
- A very common cause is a serious blow to the head, leading to brain concussion, in which the brain tissues and capillaries are traumatized so that capillary fluid leaks into the traumatized tissues.

- Once brain edema begins, it often initiates two vicious cycles because of the following positive feedbacks:
- 1) edema compresses the vasculature. This in turn decreases blood flow and causes brain ischemia.
- The ischemia in turn cause arteriolar dilation with still further increase in capillary pressure. The increase capillary pressure then causes more edema fluid, so that the edema becomes progressively worse.
- 2) the decreased cerebral blood flow also decreases oxygen delivery.
- This increases the permeability of the capillaries, allowing still more fluid leakage. It also turns off the sodium pumps of the neuronal tissue cells, thus allowing these cells to swell in addition.

- Once these two vicious circles have begun, heroic measures must be used to prevent total destruction of the brain.
- One such measure is to infuse intravenously a concentrated osmotic substance such as very concentrated mannitol solution.
- This pulls fluid by osmosis from the brain tissue and breaks up the vicious cycles.
- Another procedure is to remove fluid quickly from the lateral ventricles of the brain by means of ventricular needle puncture, thereby relieving the intracerebral pressure

Brain Metabolism

- Total brain metabolic rate and metabolic rate of neurons:
- Under resting but awake conditions, the metabolism of the brain accounts for about 15 percent of the total metabolism in the body, even thought the mass of the brain is only 2 percent of the total body mass.
- Therefore, under resting conditions, brain metabolism per unit mass of tissue is about 7.5 times the average metabolism in non-nervous system tissues.
- Much of the excess metabolism in the brain occurs in neurons, and is due to the movement of ions through the membranes.
- For example, each time a neuron conducts an action potential, ions such as sodium, potassium, and calcium move through the membranes, therefore increasing the need for additional membrane transport to restore proper ionic concentration differences across the neuron membranes after an action potential.

Special requirement of the brain for oxygen-lack of significant anaerobic metabolism

- Most tissues of the body can live w/o oxygen for several minutes and some for as long as 30 minutes.
- During this time, the tissue cells obtain their energy through processes of anaerobic metabolism, which means release of energy by partially breaking down glucose and glycogen but without combining with oxygen, delivering energy only at the expense of consuming tremendous amounts of glucose and glycogen. –however, it does keep the tissues alive

- The brain is not capable of much anaerobic metabolism.
- One of the reasons for this is the high metabolic rate of the neurons, so that most neuronal activity depends on second-by-second delivery of oxygen from the blood.
- Thus, cessation of blood flow to the brain or sudden total lack of oxygen in the blood can cause unconsciousness within 5 to 10 seconds.
- ***Under normal conditions most brain energy is supplied by glucose***.
- Almost all the energy used by the brain cells is supplied by glucose derived from the blood.
- As is true for oxygen, most of this is derived minute by minute and second by second from the capillary blood, with a total of only about a 2-minute supply of glucose normally stored as glycogen in the neurons at any given time.
- A special feature of glucose delivery to the neurons is that its transport into the neurons through the cell membrane is not dependent on insulin, while insulin is required for glucose transport into most other body cells.

End!