

LECTURE 16

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NEUROPATHOPHYSIOLOGY (HEAD INJURY)

With the advent of the automobile, head injuries are becoming increasingly more prevalent in the clinical environment and deserve inclusion here. Head injuries may be either of two types: (1) **Closed head injury** in which the cranium is intact but may be broken and depressed with bone fragments; and (2) **Open head injury** in which a break opens the cranium to the outside. In either case damage to the brain is likely, but in closed head injuries the likelihood of further damage from increased **intracranial pressure** from bleeding or swelling must be considered. Being confined within the cranium, the brain is susceptible to increased intracranial pressures. Increased intracranial pressure can compress blood vessels obstructing blood supply to other parts of the brain and increasing the extent of injury. With open head injuries, the risk of infection is always a concern.

Because of the high density of neuron cell bodies contained within the CNS, injury to these neurons is considered irreversible. Recall that mature, postmitotic cells, such as neurons, don't divide to replace injured or lost cells. So, any losses due to injury result in permanent deficits unless some other CNS area can assume the lost function. Consequently, the CNS must be protected from injury. Injury to the CNS is likely from two sources: (1) **chemical injury** from changes in blood chemistry affecting CNS tissues; (2) **mechanical injury** from trauma to the brain or cord.

C.N.S. PROTECTION

Review the material on CSF formation, circulation and function and the blood brain barrier (pp. 958-962). Also, read about head trauma (1030-1051) before proceeding.

Blood-Brain Barrier

The blood-brain barrier is just as the name implies, a barrier against potential chemical injury to the CNS from uncommon materials in circulating blood. Blood capillaries of both the brain and spinal cord show a specialized cell layer outside their walls that controls the materials that pass into the interstitial spaces from blood. Since transport across these specialized capillaries is mostly by active transport requiring membrane carrier molecules, molecules without carrier proteins can not cross, thereby protecting the CNS from transient changes in blood chemistry. However, lipid soluble

molecules can cross directly through the phospholipid membrane layers and enter CNS interstitial spaces. Alcohol, THC, and other drugs cross by this means. The blood-brain barrier also blocks certain chemotherapy agents from passing across making treatment of CNS neoplasms more complicated.

Cerebrospinal Fluid

Simply encasing the brain and spinal cord within bone of the cranium and vertebral column does not afford the CNS complete protection from mechanical injury. CNS structures can become injured by impacting the inside of their bony enclosures. To minimize this possibility, the brain and spinal cord are surrounded by a thin layer of fluid, the **cerebrospinal fluid (CSF)**. This fluid has the same density as brain and cord tissue so that they are suspended, **neutrally buoyant**, within it and are completely surrounded by it. Since incompressible fluids dissipate force readily, the CSF serves to “cushion” the CNS organs it surrounds.

CSF is formed as a secretion of specialized capillaries within the hollow spaces of the brain (**ventricles**) and is reabsorbed by capillary “villi” of the arachnoid meningeal layer. This system of protection is dependent upon the entire CNS being surrounded by three membrane layers, termed **meninges**. The outer meningeal layer is the **dura mater** and defines two spaces: (1) the **epidural space** just outside the dura; and (2) the **subdural space** just beneath it. Arterial blood vessels occur in the epidural space and venous return vessels, especially the **sagittal sinus**, occur in the subdural space. Beneath the subdural space is the middle meningeal membrane, the **arachnoid**, named for its web-like network of blood vessels. The arachnoid layer has capillary villi that project into the **subarachnoid space** beneath it and serve to reabsorb CSF back into venous blood returning from the brain and cord. Thus, CSF is formed in the brain’s interior ventricles, passes into the subarachnoid space, and is reabsorbed back into blood from which it came. Impairment of this CSF circulation can cause problems. A common problem occurs when the **cerebral aqueduct** between the third and fourth ventricle is obstructed. Fluid and pressures build up in the lateral ventricles of the two hemispheres pressing them out against the inner cranium (**hydrocephalus**). This increased intracranial pressure can compress other blood vessels and cause damage to brain tissue. Surgical shunts can bypass the blocked aqueduct and return CSF directly to venous blood vessels to alleviate the problem. The third meningeal layer, **pia mater**, lies directly on the surface of the brain and cord and is the innermost layer.

Although effective, the protection afforded by the meninges and CSF is not absolute. Impacts to the head can cause the suspended brain to shift within its CSF environment and strike the inner cranium to cause damage. Damage caused by such head impacts is often characteristically in two directly opposite locations. For example, impact to the forehead causes the brain to shift forward striking the frontal lobes against the inner frontal bone (**coup injury**-at the point of impact). But, as the brain rebounds, the occipital lobes, directly opposite the frontals, strike the inner occipital bone (**contrecoup injury**-directly opposite the point of impact). Consequently, head injured clients should be assessed for both potential injury sites.

REVIEW QUESTION:

1. Compare and contrast the effects of cerebral aqueduct blockage in infants and adults.

Concussion

A concussion results from sudden movement or “shaking” of the brain within the cranium and can cause multiple sites of injury varying in the degree of severity with the causative force. Mild concussions may go unnoticed (subclinical) while severe ones can cause temporary loss of consciousness with pre- and/or post-traumatic **amnesia** surrounding the time of the injury. Since concussions damage soft tissue of the brain, they do not appear on diagnostic scans.

One unfortunate consequence of multiple concussions is their “additive” effect. This situation is most familiar when it occurs in athletes. Minor impacts to the head, as might occur with contact sports, can cause imperceptible, subclinical concussions whose effects can be additive even though the effect of each event was not noticeable. Such events can lead to post-traumatic Parkinsonism in later life. Often a concussion can break blood vessels and lead to a more serious hematoma.

Hematomas

A ruptured aneurysm, a broken blood vessel from head trauma, or a postoperative bleed can result in bleeding into tissue spaces (**hematoma**) of the brain or cord. Should such an event occur in the brain, two dangers exist: (1) tissues downstream of the broken vessel are deprived of blood supply; and (2) bleeding into the cranium generates increased intracranial pressures. Severity of the intracranial bleed depends upon the source. A **subdural hematoma** suggests a broken venous vessel with bleeding into the subdural space. Since venous pressures are lower than arterial, this is less serious than an epidural bleed. An **epidural hematoma** results from a high pressure, arterial bleed into the epidural space and can cause intracranial pressures to rise higher and faster. Both are surgical emergencies because of the need to relieve the pressure and stop the bleeding, but an epidural hematoma is more serious. **Intracerebral hematomas** also exist, when the bleeding is in brain tissue itself, and can be quite serious depending upon the extent of bleeding.

Herniation

An unfortunate consequence of increased intracranial pressure, is displacement of the brain away from the area of highest pressure. Localized increases in pressure, as might occur with hematomas, can compress the brain against the skull and force portions of it through any available openings. The **tentorium** of the cerebellum provides one such opening through which the brainstem normally passes. As pressure displaces the brain to one side, the temporal lobe on the side of the bleed is forced downward below the

tentorium to exert pressure on the brainstem. Should the pons or medulla be affected, death can result.

REVIEW QUESTIONS:

1. Which cranial nerve is likely to be affected by temporal lobe herniation?
2. What signs would the above situation produce in a client?

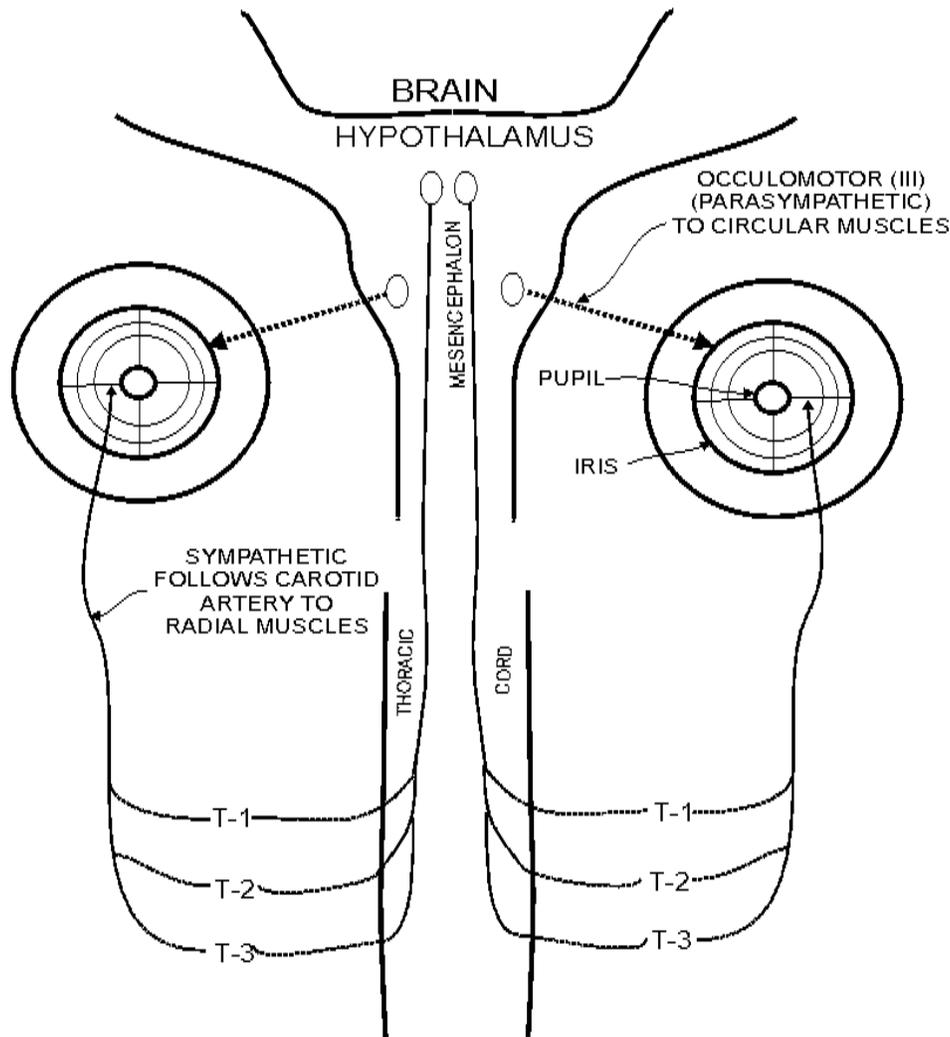
PHYSIOLOGY OF HEAD INJURY ASSESSMENT

All head traumas can exist with varying levels of severity and can shift unexpectedly toward improvement or deterioration. Consequently, accurate assessment and careful monitoring is essential, especially in the early post-trauma period. This assessment necessitates application of basic physiological principles.

Pupillary Reflex

Monitoring the pupil's extent of dilation or constriction as well as their reactivity to light can be a valuable assessment tool if the underlying physiology is understood. The iris contains both circular smooth muscles, which constrict the pupil when active, and radial smooth muscles, which dilate the pupil when active. **Parasympathetic** activation constricts the pupil and **sympathetic** activity dilates it. Examine the illustration below comparing the courses taken by the parasympathetic, **oculomotor nerve** (Cranial Nerve III) and the sympathetic nerves. Parasympathetic supply to the iris is from the midbrain where the third nerve originates out to the circular muscles of the iris. Sympathetic innervation originates in the hypothalamus and courses down the brainstem and cord emerging within the first three thoracic nerves. Sympathetic fibers then follow the carotid artery back up to the head and to the radial muscles of the eye iris.

PUPILLARY REFLEX



Because of the different paths these two nerve supplies take, brain and brainstem trauma interrupt the sympathetic and parasympathetic tracts in different patterns. Consequently, the pupillary reflex can be a valuable assessment tool. For example, damage to the hypothalamus destroys only the sympathetic branch allowing the parasympathetic to predominate. Parasympathetic nerve supply causes constriction with reaction to light. In the lower brainstem (pons and medulla), damage causes a similar response, but more exaggerated. In this case, the pupils are tightly constricted (“pinpoint”) and unreactive or “fixed.” Notice that midbrain, mesencephalon, damage disrupts both the sympathetic and the parasympathetic pathways resulting in pupils being midposition and “fixed.”

In usual situations, both pupils respond similarly (bilaterally). However, if the parasympathetic oculomotor nerve is damaged outside the brain and at some point along its course to the eye, parasympathetic supply is disrupted only to that one eye. In the

affected eye sympathetic predominates and the pupil dilates while the other eye remains normal. This condition is common with temporal lobe herniation as the protruding lobe of the brain presses on the oculomotor nerve on the herniated side. Thus, the dilated (“blown”) pupil indicates the side of herniation.

Altered Respiratory Patterns

Since the respiratory centers occur primarily in the medullary portion of the brainstem, damage to this area causes disrupted breathing patterns. Damage to the upper brainstem and above results in a “**Cheyne-Stokes**” respiratory pattern characterized by alternating bouts of shallow and deep breathing. However, lower brainstem injury causes a pattern of breathing termed “**central neurogenic**” that is characterized by rapid shallow breaths. This pattern of respiration is serious and may appear just prior to death.

Posturing

Other motor activities, or the lack thereof, can be helpful in assessing a head-injured patient especially in the absence of consciousness. One such case is “posturing” in which flexor or extensor muscles become rigidly contracted in particular patterns. **Decorticate** posturing is the less severe of the two possibilities and involves flexion of the upper extremities and extension of the lower extremities. Decorticate posturing indicates damage to the cortex or to the internal capsule pathways leading to it. **Decerebrate** posturing is more serious and indicates damage to the lower brainstem in the pontine area. With decerebrate posturing, head extension, back arching, and extension of the extremities are characteristic. A major clinical problem results when posturing is confused with seizures.

Glasgow Coma Scale

Since head trauma patients may be subjected to multiple assessments in the course of their treatment, it is essential to be able to record and to communicate their progress accurately to different individuals involved in their care management. The Glasgow Coma Scale was developed to standardize neurological assessments based upon a series of relatively simple tests. Subjects are given points based upon their responsiveness in three categories of activities: (1) motor responsiveness; (2) verbal responses; and (3) eye opening. For example, a normal person can display any number of fingers when asked, can carry on conversation about who s/he is, the location, date and time, etc., and eyes are normally open and following objects in their environment. In contrast, a comatose patient may be incapable of any motor response, may make no sound even in response to noxious stimulation, and may not open the eyes even when stimulated. The normal person gets the highest point score of 15, while the comatose patient may get the lowest possible score of 3. Scores of 7 and below constitute coma.

The Glasgow coma scale allows an emergency medical technician to score a patient at the scene of an accident and for emergency room physicians to rescore the

same patient upon examination to determine if improvement or deterioration is occurring. Floor nurses can also reassess at intervals to record the patient's progress. Since everyone uses the same scale, subjectivity is reduced and accurate communication about patient progress is possible.

DISCUSSION QUESTIONS: (Post answers to the "Patho Discussion Group")

1. A side-impact auto accident victim with a potential head injury was assessed on the scene to have a GSC score of 9. Upon arrival at an emergency room, the score had dropped to a 6 and a scan showed a subdural hematoma in the left temporal area. After surgical correction of the hematoma, the score improved to an 13. Then, without warning, the patient lost consciousness and showed a "blown" left pupil with increasingly more rapid and shallow breathing and extensor posturing. Speculate in detail what may have been happening to cause this deterioration.
2. After a fall from a ladder, a homeowner experienced nausea, lethargy, visual impairment with memory failures and was taken to the emergency room for examination. A superficial contusion was seen at the base of the skull at the back of the head and on the left side and a precautionary CT scan was ordered. Assume a "worst case scenario" and discuss what might be expected on the scan.