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Anatomy and Physiology of Neurological Compensatory Mechanisms

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The skull is a unique part of anatomy. It exists in a state of dynamic equilibrium. The components of the skull and their role in maintaining integrity and normal intracranial pressure will be reviewed in this article. Compensatory mechanisms that attempt to prevent the destructive effects of cerebral ischemia and injury will be discussed.
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The components of the skull exist in a state of dynamic equilibrium. If one component of the skull is altered, the other components either need to compensate for the change or will be altered themselves. In this article, the components of the skull and their role in maintaining integrity and normal intracranial pressure (ICP), and finally, how innate compensatory mechanisms attempt to prevent the destructive effects of cerebral ischemia and injury will be reviewed.

THE CRANIUM

The cranium is made of an inner and outer table of bone separated by spongy tissue, which helps to maximize strength but allows it to be lightweight as well.[1] The only opening in the skull is the foramen Magnum, "big hole," located at the base of the skull, through which the spinal cord emerges. The cerebral cortex has a right and a left hemisphere, sitting above the tentorium. "Like a tent," the tentorium is made of the inner foldings of the dura (outer covering of the brain) which separate the cerebral cortex from each other and the cerebellum. The tentorium helps to absorb downward pressure from the cerebral cortex on the cerebellum and brainstem[2] and provides a physical barrier to the movement of intracerebral structures.[3] The brainstem consists of the midbrain, pons, and medulla, all lying above the foramen Magnum in the skull, making them vulnerable to compression if increased ICP exists.

Cerebral volume consists of 3 components: cerebral tissue, blood, and cerebral spinal fluid (CSF). Monroe and Kellie theorized that for intracranial pressure to remain normal, the components must compensate for increased volume or ICP will increase.[4] Normally, their capacity for compensation is large, accomplished via numerous avenues. These mechanisms will be reviewed while looking at cerebral anatomy and physiology. Remember, once neuronal cells become injured, the brains ability to compensate is finite, decompensation can occur rapidly, and damage to the vulnerable adjacent tissues (known as secondary injury) will ensue.[1]

COMPONENTS OF THE SKULL: THE BRAIN

The brain tissue occupies 80% of intracerebral volume, including brain tissue and interstitial and intracellular water.[1] The outermost covering of the brain and the spinal cord is the meninges, "membranes." The 3 layers of the meninges are the dura mater, the arachnoid membrane, and the pia mater.

The dura mater lines the inner table on the skull and is very inelastic. In fact, this strong dural layer has been

described by Barbara McLean as the box (dura mater) within a box (the skull), protecting the delicate structures within. The dura mater consists of 2 layers, the periosteal lining and the meningeal lining. Between these 2 layers are sinuses or drainage canals where CSF and venous blood can drain into before exiting the skull.

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The middle layer of the meninges is called the arachnoid membrane, a translucent, thin, web-like structure, loosely covering the brain and housing circulating CSF. The third and inner most layer of the meninges is the pia mater, adhering to the brain and following all of the gyri (folds) of the brain surface. Between each of the layers of the meninges are spaces or potential spaces. The epidural space lies above the dura and the subdural space is below the dura. Both of these are only potential spaces, and fluid or blood within these space areas would be abnormal. The subarachnoid space (SAS) is below the arachnoid layer, and normally, only 150 mL of CSF at one time circulates in this actual space around the brain and spinal cord.[5] Within the SAS are small structures called arachnoid villi necessary for reabsorbing CSF into the drainage sinuses or canals. Cerebral spinal fluid will be discussed in greater depth later in this article.

The white and gray matter of the brain, or the central nervous system, consists of 2 types of cerebral cells: the neurons responsible for neurotransmission and the neuroglial cells responsible for providing support and structure to the brain. The neuroglial cells are diverse and complex and were originally described by Dr Virchow, a German physiologist in 1846, as the "glue of the brain." The 2 main categories of neuroglial cells will be addressed here: the microglial cells and the macroglial cells. The microglial cells are the smallest of the glial cells and primarily act as macrophages eliminating debris and serving as the brain's weapon against invading microorganisms (see Table 1). Lying dormant until called into action, these chemical arsenals have the potential to support and/or destroy neuronal tissue.

Subtypes of the macroglial cells include oligodendrocytes, astrocytes, and the ependyma cells.[6] The oligodendrocytes are responsible for myelin sheath formation, replacing myelin already destroyed. A second type of macroglial cell is the star-shaped astrocyte. Astrocytes relay nutrients through the neuronal network, reuptake neurotransmitters such as glutamate, and its unique morphology helps to create the blood-brain barrier (BBB).

Together, the microglial and macroglial cells act to maintain the structure of the brain tissue and aid in the removal of old and dead cells by acting as chemical protectors. Cleaning up the environment as a way to protect uninjured tissue from waste products, they have the ability to disintegrate old cells and ultimately lead to replacement with connective tissue. Interestingly, the neuroglial cells use more than half of the delivered energy just to perform normal functioning and the remaining energy to maintain their integrity. Another type of macroglial cell is the ependyma cell located in the ventricles of the brain and is responsible for CSF secretion.

The ability of these highly specialized cells to perform is dependent on a constant energy supply. The brain uses essentially all the oxygen delivered to it. Even though the brain is 2% of ideal body weight, it requires 20% to 25% of the body's oxygen and glucose to form the cells fuel, adenosine triphosphate.[3]

A cascade of events initiated by ischemia and cellular response to injury known as secondary injury drive neuronal damage after brain injury and/or ischemia (see Figure 1). Ischemia results from inadequate cerebral perfusion or increased cerebral oxygen consumption.[7]

When there is a lack of oxygen to fuel the production of adenosine triphosphate, cellular and inflammatory events are initiated, including the release of the neuroexcitatory amino acid, glutamate. Normally, small

TABLE 1 Neuroglial Cells

	Subtypes	Action	Other
Microglial		Acts as macrophages, cleaning up debris	Lie dormant till needed
Macroglial	Oligodendrocyte	Myelin sheath formation	
	Astrocyte	Reuptake of neurotransmitters such as glutamate and relay nutrients through neuronal network	Star-shaped, foot processes form part of the BBB
	Ependyma	Secrete CSF	Located in the cerebral ventricles

BBB indicates blood-brain barrier; CSF, cerebral spinal fluid.

amounts of glutamate allow neurons to communicate with one another, but the excessive release of glutamate after injury and ischemia may contribute to an accumulation of intracellular calcium. In addition, the disruption of the sodium/potassium pump causes calcium to be pumped into the cell.[3] Calcium, when not tightly regulated, can trigger the release of proteolytic enzymes and potentiate the destruction of the cells cytoskeleton (structure of the cell). These 3 processes—excessive release of glutamate, dysfunction of the sodium/potassium pump, and the release of proteolytic enzymes—all contribute to the increasing intracellular calcium and ultimately cell death.

Injured or destroyed cells are edematous (cytotoxic edema), and capillaries can become "leaky" (vasogenic edema), contributing to cerebral edema and increased cerebral volume. A vicious cycle ensues with increased cerebral edema compressing healthy tissues further perpetuating secondary injury and subsequently neuronal cell death. Brain tissue cannot decrease its mass as a way to compensate for increased intracerebral volume. The brain will need to rely on the other two components of the skull to alter their physical properties to compensate for increasing cerebral volume to prevent increased ICP.[8]

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THE COMPONENTS OF THE SKULL: THE BLOOD

The second component in the skull, blood, occupies 10% of intracranial space.[5] The brain receives a constant supply of blood at a rate of 750 mL/min via 2 internal carotid arteries and 2 vertebral arteries.[1] The internal carotid arteries take blood to the interior of the skull via the right and left middle cerebral arteries and the right and left anterior cerebral arteries. The right and left external carotid arteries take blood to the external portions: the skull, face, and scalp via the right and left maxillary artery and the right and left meningeal arteries. The anterior cerebral arteries and middle cerebral arteries are appropriately named for the portions of the brain for which they feed: anterior/frontal portions and lateral/middle portions, respectively.

The posterior portion of the brain, including the occipital lobe and the cerebellum, are logically fed by the left and right posterior cerebral arteries. Their blood volume originates from the left and right subclavian arteries, flowing into the right and left vertebral arteries (interestingly protected in the spinal column), and finally, entering the skull through the foramen Magnum. The 2 vertebral arteries meet to form the basilar artery, bifurcating into the left and right posterior cerebral arteries. The posterior cerebral arteries, middle cerebral arteries, and anterior cerebral arteries, along with several other vessels, meet on the floor of the skull to form the Circle of Willis, a super highway of blood vessels dedicated to taking blood to the furthest regions of the brain. If a blockage would form in one of these

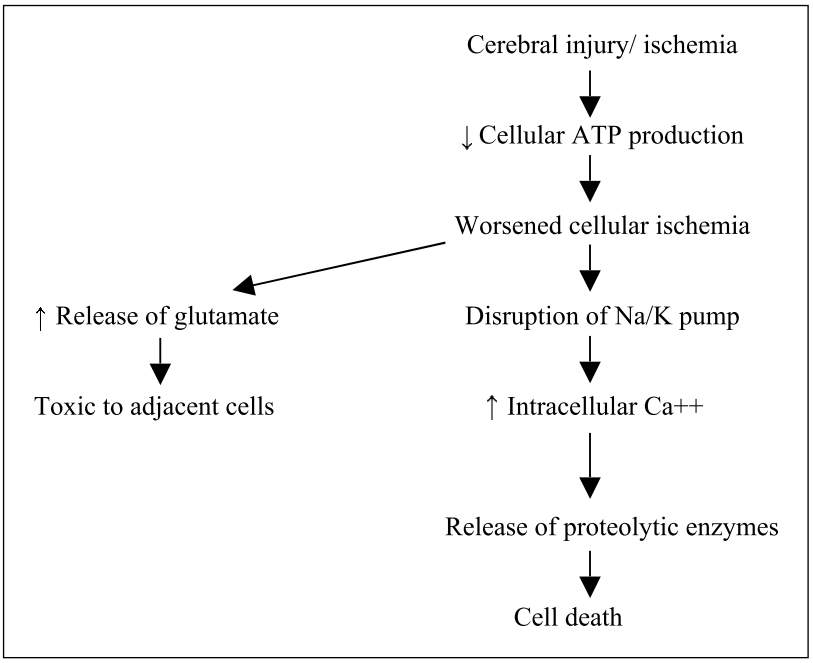

Figure 1. Increased intracellular calcium.

1. Decrease production rate of CSF, secondary to decreased cerebral blood volume.
2. Shunt more CSF out of skull into spinal SAS.
3. Increase rate of reabsorption of CSF into dural sinuses by opening of arachnoid villi
4. Decrease cerebral blood volume via autoregulation and vasoconstriction.
5. Spontaneous hyperventilation to vasoconstrict cerebral vessels and limit cerebral blood volume.

Figure 2. Compensatory mechanisms.

vessels, blood may still be able to be delivered to the affected area via another route.[5]

Compared to other arteries in the body of the same size, the cerebral arterial walls are thinner due to their lack of smooth muscle and decreased media.[2] Normally, these vessels do not need to accommodate high pressures like the rest of the body, nor do they have the ability to develop collateral circulation in response to ischemia. The cerebral arterioles possess the ability to autoregulate their size, dilating and constricting, to increase or decrease cerebral blood flow to meet tissue demands.[2] The cerebral vessels deliver a constant supply of blood even when there are wide fluctuations in systemic blood pressure. Autoregulation is an automatic compensatory mechanism used to either limit the amount of cerebral volume through vasoconstriction and therefore limit pressure or increase volume through vasodilation when demands for oxygen and blood are greater (see Figure 2). Autoregulation is impaired, however, when the patient's mean arterial pressure (MAP) is <70 or >170 mm Hg, if ICP is >40 mm Hg, or from localized or global cerebral injury.[4]

The body also responds to increasing cerebral mass by causing spontaneous hyperventilation. The cerebral vasculature responds by vasoconstricting, limiting cerebral blood flow, and ultimately, negatively impacting the delivery of oxygen and glucose to the tissues. This innate compensatory mechanism can worsen ischemia further,[8] but intervening early with mechanical ventilation may decrease the damaging effects of spontaneous hyperventilation and improve cerebral blood flow.

The patient suffering from a neurological insult is especially vulnerable to the increased metabolic demands of the injured tissue when supply is low. For example, cerebral blood flow drops by 50% after traumatic brain injury in the first 24 to 48 hours after injury.[9] This, in addition to the potential loss of autoregulation, can seriously compromise cerebral perfusion. Cerebral perfusion pressure (CPP) grossly reveals information on cerebral oxygen supply and demand. The measurement of MAP represents flow to the cerebrum and ICP represents the resistance to the flow. The end product of these 2 forces is CPP (MAP − ICP = CPP). When CPP falls below 60 mm Hg, cerebral blood flow drops, the ability of the cerebral arteries to autoregulate is impaired,[1] and cerebral blood flow becomes passive.[7] Hypotension has a dramatic effect on mortality related to traumatic brain injury and should be aggressively avoided.[10] Critical care nurses need to intervene when the patient's own compensatory mechanisms fail by supporting hemodynamics and optimizing cerebral perfusion.

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Interestingly, the venous system does not "mirror" the arterial system. Rather, venous blood flows through its own system of canals and drainage sinuses to excrete venous blood from the skull.[5] These large canals lack valves, normally found in peripheral veins, contributing to increased cerebral blood volume if there is a physical obstruction to venous outflow from the skull.[1] For example, anything increasing intrathoracic or intraabdominal pressure or obstructing jugular venous drainage via increased right atrial pressures, malposition of the head, or tight trach ties can increase intracerebral volume.[8] Conversely, any intervention promoting venous outflow from the head could, in turn, decrease cerebral volume and help limit increasing intracranial pressure. For example, keeping the head in alignment with the patient's trunk, having the head of the bed elevated, limiting positive end expiratory pressure if the patient is on a ventilator, decreasing intraabdominal pressure with the use of laxatives, and keeping right atrial pressures in check with meticulous volume management will encourage venous outflow and may

help decrease cerebral blood volume and intracranial pressure.[8]

Existing between the arterial and the venous network is the BBB, a unique capillary system consisting of an endothelial layer whose cells are tightly abutted against one another. In close proximity are the specialized neuroglial cells called astrocytes. The bottom of the astrocyte, or "glial feet,"[2(p785)] form tight junctions or connections supporting all sides of the capillary and creating the link between blood and the neuron (see Figure 3).

The intimate relationship between the tightly packed endothelial cells and glial feet results in a restrictive barrier normally difficult for neurotoxic substances to pass through. If the BBB is injured from toxins or ischemia, it can become quite porous and the BBB may become permeable to substances harmful to the neurons. The loss of this tight junction is, in part, due to the sensitivity of the astrocyte to decreased perfusion. Like all cerebral cells, the astrocyte needs a constant supply of oxygen and glucose to perform normal functions. Ischemia leads to a disruption of the sodium and potassium pump, accumulation of intracellular fluid, and swelling of the cells. The swollen glial feet create a breach in the tight junction, leaving the neurons unprotected from the porous BBB.

THE COMPONENTS OF THE BRAIN: CSF

The last 10% of what fills the skull is the CSF surrounding the brain and spinal cord in the SAS. Like all fluids, CSF is noncompressible; therefore, it acts as a cushion and support for the brain and spinal cord. Cerebral spinal fluid is normally produced at a rate of 20 mL/h, but the rate of production depends largely on cerebral blood flow.[1] For example, if cerebral blood flow is decreased because of vasoconstriction via autoregulation, this compensatory response will result in decreased ICP directly due to decreased cerebral blood volume and indirectly due to decreased production of CSF. Choroid plexi are capillary tufts primarily located in the 2 lateral ventricles responsible for the production of 95% of the CSF. The ependyma cells secrete the CSF into the ventricles. The last 5% of CSF is formed in the 3rd and 4th ventricles, and a negligible amount is produced in the vessels around the spinal column.[1]

As mentioned earlier, tiny finger-like projections in the SAS, called arachnoid villi, act as 1-way valves to absorb CSF into the dural sinuses to be carried out of the skull via the jugular veins. The arachnoid villi have the ability to open a lot or a little to shunt more or less CSF from the skull. The SAS around the spinal column has the ability to expand and accommodate increased volume if necessary, making this another great compensatory mechanism for decreasing overall cerebral volume.[11] In addition, the SAS acts as the brains lymphatic system to carry dead cells and debris out of the skull and can be an accessory pathway for interstitial fluid to be slowly reabsorbed and drained from the skull.[1] Unfortunately, the arachnoid villi may become clogged by

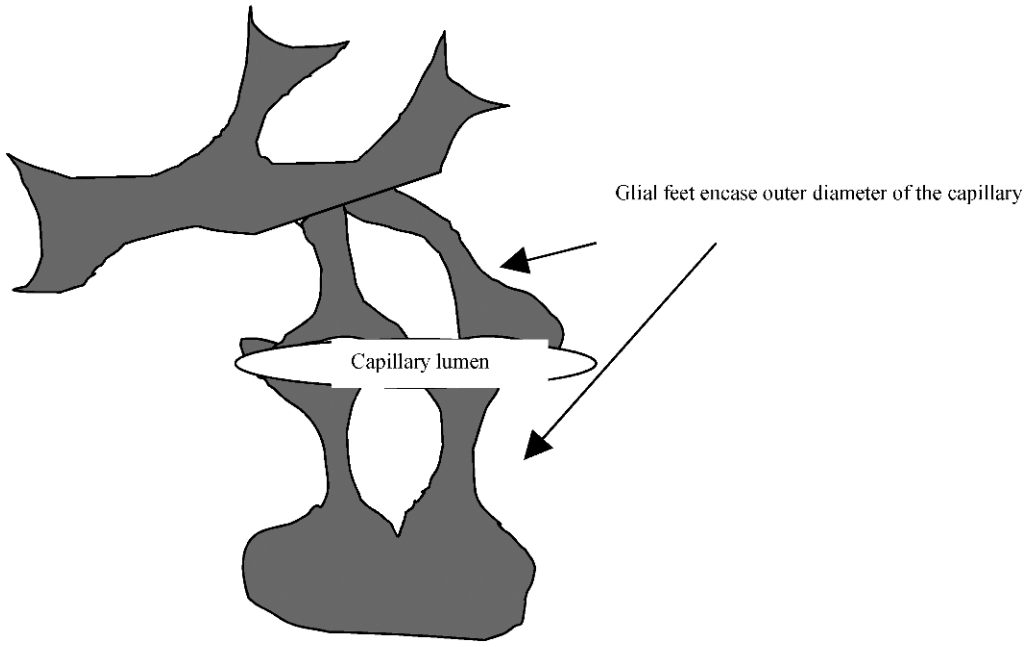

Figure 3. Glial feet and capillary wall.

infectious debris or blood and their ability to reabsorb CSF may be impaired or overwhelmed.

CONCLUSION

The ability of the components of the skull to compensate for increasing mass from bleeding, inflammation, or edema is finite. Once the compensatory mechanisms are exhausted, ICP will rise and neurological compromise will be evident.[1] Compression of the vital structures inside the closed box can result in secondary injuries. Clearly, the brain has some superb and complex physical characteristics which aid in protection and preservation: (1) the strength of the skull, (2) the tough meninges covering the brain, (3) the millions of neurons which "serve and protect," (4) the super highway of blood vessels, (5) autoregulation of vascular size, (6) the restrictiveness of the BBB, and (7) the cushion and support of the CSF. All the brain requires, in return, is a constant supply of blood delivering oxygen and glucose. If the brain is deprived of these 2 ingredients for energy production because of injury or ischemia, the innate compensatory mechanisms will attempt to optimize perfusion to the brain.

The brain tissue, CSF, and blood have an intimate and dynamic relationship with each other. Under normal circumstances, the brain has a large capacity for self-protection and for compensating in the initial stages of an injury. This can be accomplished through autoregulation, augmenting or restricting blood flow, the shunting of CSF and venous blood from the skull, and decreasing the production of CSF. After compensatory mechanisms have been exhausted, intracranial pressure will rise depriving vulnerable tissue the oxygen and glucose it needs.

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