Dynamic Chiropractic

CHRONIC / ACUTE CONDITIONS

The Craniocervical Spine and Multiple Sclerosis, Part 2 of 2

Michael Flanagan, DC, DABCN

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The cause of MS remains unknown, despite decades of research. Recent MRI studies, however, suggest demyelination and axonal degeneration in MS may be caused by ischemia, edema and inflammation. This paper hypothesizes that craniocervical syndromes and degeneration of the cervical spine can cause obstruction of the vertebral arteries and accessory drainage system of the brain. Over time, this can lead to chronic ischemia, edema and normal pressure hydrocephalus in the brain and spinal cord, which may lead to oxidative stress reactions, excitotoxicity, demyelination and other neurodegenerative processes in the brain and spinal cord.

The Accessory Drainage System and Suboccipital Cavernous Sinus

The accessory drainage system is unique to humans and hominids; it serves to drain the brain during prolonged upright posture. It includes the occipital marginal sinuses and accessory veins, the suboccipital cavernous sinus and the vertebral *venous plexus* (VVP).

The occipital marginal sinus and accessory venous outlets drain to the suboccipital cavernous sinus and VVP. The craniocervical spine is the critical link between the cranial dural sinuses and the accessory drainage system. It is possible that congenital and acquired craniocervical syndromes, and degenerative conditions of the spine, can obstruct the accessory drainage system.

The blood-brain barrier is both an anatomical and a physiological sieve that screens blood to produce CSF and interstitial fluids. Its permeability can be affected by chemicals, hyperventilation and trauma. Its permeability also increases with upright posture, as a result of an increase in the CSF pressure gradient mitigated by a decrease in superior sagittal sinus venous pressure (SSVP). The increase in the CSF pressure gradient causes the passive production of CSF needed to compensate for the loss of CSF caused by increased outflow.

An inadequate amount of CSF in the cisterns and subarachnoid spaces in the upright position causes the brain stem to slide down the clivus toward the *foramen magnum*. A pressure conus occurs when the brainstem sinks into the foramen magnum and depresses all vital functions and consciousness. It is possible, however, that a decrease in CSF volume in the cisterns and subarachnoid spaces may be enough to cause obstruction of the occipital marginal sinuses and other venous outlets to the accessory drainage system, without compressing the brainstem and depressing all vital functions and consciousness. The VVP lies between the cord and nerve roots in the neural canal and intervertebral foramen. It has been shown that degeneration of the spine affects hydrodynamics in the VVP long before it compresses the cord, and that venous hypertension in the VVP can sufficiently decrease perfusion pressure and arterial blood flow as to cause ischemia of the cord and nerve roots. It can even cause a syrinx: an abnormal collection of cerebral fluid in the spine. Symptoms of hypertension in the VVP include glove distribution sensory loss, Lehermitte's sign and *cauda equina* syndrome.

The suboccipital cavernous sinus links the VVP to the accessory drainage outlets in the brain, and functions as the entrance point of the vertebral arteries into the brain. The suboccipital cavernous sinus cools and protects the vertebral arteries in the same way the cavernous sinus cools and protects the internal carotid arteries. While the suboccipital cavernous sinus protects the vertebral arteries from external pressures acting on the sinuses, it does not protect them from an increase in venous pressure inside the sinuses.

It is possible that venous hypertension in the VVP and suboccipital cavernous sinus can cause obstruction to blood flow through the vertebral-basilar arteries and the branches of the vertebral arteries that supply the cord. In either case, ischemia of the areas of the brain and cord supplied by the vertebral arteries, including the cervical cord; lower cerebrum; brain stem; cerebellum; and periventricular areas, can occur.

Cranial Hydrodynamics and the Cervical Spine

CSF volume is maintained by active and passive production. Active production uses energy from ATP to secrete salts and create an osmotic pressure gradient that draws CSF into the ventricles; passive production relies on an increase in the CSF pressure gradient from upright posture. In fact, SSVP becomes negative in the upright position. The negative pressure siphons CSF out of the *telea choroidea* and into the ventricles. The passive production of CSF production offsets the increase in outflow during prolonged upright posture.

The actual value of SSVP is affected by many factors, such as gravity, height above the heart, and resistance to flow. Resistance can be increased by inversion and Valsalva maneuvers. It is possible that craniocervical syndromes and degeneration of the cervical spine also can increase resistance to venous outflow during prolonged upright posture. If so, craniocervical syndromes and degeneration of the spine may decrease the CSF pressure gradient, and consequently, the passive production of CSF during prolonged upright posture.

Proper function of normal cranial hydrodynamics is essential to humans and upright posture. Prolonged upright posture causes an increase in CSF outflow that must be matched by an increase in CSF production, to prevent the brain from slipping downward toward the foramen magnum, possibly obstructing the occipital marginal sinuses and other venous outlets to the accessory drainage system.

Obstruction to CSF flow out of the ventricles in which it is produced, and into the cisterns and subarachnoid spaces where it is needed for support, causes a hydrocephalic condition and weakens brain support during prolonged upright posture. Furthermore, because CSF and interstitial fluids flow along the same perivascular pathways (Verchow-Robin spaces), interstitial edema also may occur. Therefore, passive production of CSF and the CSF pressure gradient are critical to upright posture. Craniocervical syndromes and degeneration of the cervical spine may interfere with normal cranial hydrodynamics; over time, this can cause chronic NPH and interstitial edema, leading to demyelination and other neurodegenerative processes.

Craniocervical Syndromes and MS

Injuries of the head and neck that result in acute injury to the brain and/or blood-brain barrier can cause inflammation, edema and ischemia. These conditions can cause chemical damage to myelin. In addition to the chemical effects of edema on demyelination, it also has been suggested that edema can damage myelin simply by stretching it.

Severe head and neck traumas most likely cause extreme hydrodynamic loads in the ventricles and

around them in the cisterns and periventricular spaces. These hydrodynamic stresses may be even greater around the acute angles of the anterior and posterior horns of the lateral ventricles, an area where hyperintensity signals are frequently found in many neurodegenerative diseases. If interstitial edema can stretch and damage myelin, then certainly much more extreme forces associated with head and neck trauma can likewise cause acute damage to myelin. Barring severe injuries, however, oligodendrocytes and neurons are capable of healing themselves more than previously thought, provided they have sufficient blood flow and oxygen.

The real challenge to healing following brain trauma occurs approximately two to four weeks after an initial injury, when a cascade of degenerative events often follows severe episodes of ischemia. The effects of ischemia on the glutamate cascade and excitotoxicity were discussed in the first part of this paper. However, while acute injuries can cause acute demyelination nerve damage, it doesn't explain the prolonged degenerative processes associated with MS and other diseases of the brain and cord.

In addition to the effects of head and neck trauma on the brain and cord, such trauma also can injure the bones, muscles, cartilage and connective tissues of the craniocervical spine. Over time, this can lead to craniocervical syndromes and degeneration of the spine, which can cause obstruction of the posterior blood supply and accessory drainage system of the brain. In addition to injuries, some people are born with craniocervical syndromes, and some acquire them simply through aging. Regardless of the cause, obstruction of the posterior blood supply can lead to chronic ischemia of the lower cerebrum, cerebellum, brain stem and cervical cord. Obstruction of the accessory drainage outlets, on the other hand, can lead to chronic NPH and interstitial edema. Moreover, these conditions may cause demyelination and other neurodegenerative processes.

Gender, Genetic, Racial and Environmental Factors in MS

The gender, genetic, racial and environmental factors described in the beginning of this article are difficult to explain in light of prevailing viral and immunological theories. They make more sense in light of craniocervical syndromes and degeneration of the cervical spine. There are numerous variations in the design of the accessory drainage system and craniocervical spine that may predispose humans to hydrodynamic failure resulting from craniocervical syndromes and degenerative conditions of the spine. Females, for example, typically have smaller features in the craniocervcial spine and accessory drainage system, which may affect the capacity of their drainage systems, thus making them more susceptible to the effects of craniocervical syndromes and degenerative conditions of the spine.

There are racial variations in the design of the craniocervical spine and accessory drainage system. For example, there are significant differences between the round, orthognathic Asian cranium and the protruding, prothognathic European cranium. Furthermore, Eskimo and most aboriginal crania are more closely related to Asian crania. The design of the cranium, especially the basicranium, may provide a clue as to why Asians, Eskimos and many aboriginal people have a much lower incidence of MS. It also may explain why when they do get MS, the hyperintensity signals show up in slightly different locations.

Relative to environmental factors, people who live in the northeast may have a higher incidence of weather-related trauma. Some injuries may be especially traumatic to young or developing brains and spines. Finally, the susceptibility to MS and other degenerative diseases of the brain from these types of injuries may be affected by many factors related to racial, genetic, gender and age variations in the design of the craniocervical spine and accessory drainage system.

Chiropractic Care and MS

Principles of Neurology, by Adams and Victor (1981), states: "A matter of considerable interest is whether blockage of the dural sinuses, into which CSF is absorbed can result in tension hydrocephalus. The problem is that blockages are rarely found."

So far, we have only looked for blockages in the drainage system of the brain in the dural sinuses inside the cranium. The most likely place for blockage to occur during prolonged upright posture, however, is outside the cranium in the craniocervical spine. The craniocervical spine contains both the VVP and suboccipital cavernous sinus of the accessory drainage system. Congenital and acquired craniocervical syndromes and degeneration of the cervical spine may cause venous hypertension in the VVP and suboccipital cavernous sinus, leading to obstruction of the accessory drainage system and vertebral arteries and eventual chronic NPH (tension hydrocephalus), edema and ischemia of the brain and cord.

The role of craniocervical syndromes and degeneration of the cervical spine in neurodegenerative diseases of the brain and cord requires further investigation. Studies also need to be done to determine the safest and most effective ways of maintaining the health and integrity of the spine, especially the craniocervical spine. Timely and appropriate spinal care over the course of a lifetime may help reduce the incidence and severity of craniocervical syndromes and degeneration of the cervical spine; more importantly, it also may help decrease the incidence and severity of MS and other neurodegenerative diseases of the brain and cord.

Michael Flanigan, DC
Old Tappan, New Jersey

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