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Migraines, Strokes and Chiropractic

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In contrast to recent reports by medical doctors that certain types of chiropractic adjustments of the cervical spine may cause strokes, for the past 100 years chiropractors have reported good, if not excellent, results in the treatment of migraine headaches. This is important because migraine headaches may be associated with an increased incidence of ischemic strokes. Therefore, by decreasing the incidence and severity of migraine headaches, chiropractic care may be able to decrease the incidence of ischemic strokes. This must be compared to the risk for strokes from certain types of cervical treatments, which, according to even the worst estimates, is extremely low, as well as to the use of prescription and nonprescription drugs, which cause significant increases in the incidence of cerebrovascular and ischemic strokes. Chiropractic care may be the safest and most effective type of treatment for migraine headaches, including high-risk cases in which there are no other treatment options.

Migraine Headaches and Variants

Classical migraine headaches are a paroxysmal disorder characterized by recurrent attacks of headache, with or without associated visual and gastrointestinal disturbances. The cause is unknown, but most migraines are believed due to some type of functional disturbance in cranial circulation, especially the posterior circulation. Although it is still being debated, recent evidence suggests that migraine headaches may be associated with an increased incidence of ischemic strokes. While some of these strokes appear to be due to genetic vascular weakness, most are not. Regardless of the cause, most migraines appear to affect the areas of the brain supplied by the posterior circulation. Several types of migraine variants are interesting in light of this article.

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a newly recognized type of migraine, in combination with small strokes. CADASIL primarily involves the white matter and basal ganglia similar to Binswanger's subcortical arteriopathic encephalopathy

(multi-infarct dementia). This is characterized by recurrent attacks of ischemia, migraine headaches with aura, severe mood disorders, subcortical dementia and widespread leukoencephalopathy (changes in white matter). The cause is unknown, but CADASIL appears to be due to microangiopathy as a result of faulty genetics. There are no known treatments at this time.

Basilar artery migraine (BAM) is associated with visual disturbances; dysarthria; ataxia; vertigo; tinnitus; bilateral paresthesias; decreased hearing; decreased level of consciousness; and bilateral paresis. There may also be a relationship between BAM and epilepsy. BAM is believed to be due to ischemia of the vertebrobasilar circulation to the brain. What is particularly interesting about the BAM is that it is associated with reversible ischemia. Researchers are looking into whether BAM headaches are likewise associated with an increased risk for strokes. There are no treatments available for BAM headaches, except to avoid medications used to treat migraine headaches that reduce blood flow to the brain, and may therefore worsen the affects of ischemia and increase the risk for ischemic strokes.

There are two types of migraines associated with the optic and oculomotor nerves that are supplied primarily by the anterior circulation. They are ophthalmopelagic and retinal migraines. Ophthalmopelagic migraine headaches are associated with ocular palsy. The pathophysiology of ophthalmopelagic headaches remains unclear, but it has been suggested that they may be due to ischemia, compression or inflammation of the blood vessels. Retinal migraines, on the other hand, produce a "mosaic" or "jigsaw" pattern of scotoma that enlarges and merges together, producing total visual loss. This is in contrast to classical migraines, which are associated with auras more likely to be associated with involvement of the occipital and temporal lobes supplied by the posterior circulation. Again, the cause is unknown, but retinal migraines are attributed to vasospasm of the choroidal or retinal arteries and subsequent ischemia.

Coital migraine headaches can affect women, but they mostly affect men. They may be brought on by hemodynamic changes that accompany intercourse and orgasm. This is important because intercourse and orgasm are associated with Valsalva-like maneuvers that increase pressure in the vertebral venous plexus (VVP), the superior sagittal sinus, and reverse the CSF pressure gradient. Moreover, Valsalva maneuvers increase pressure in the suboccipital cavernous sinus. This could affect the posterior circulation to the brain. Craniocervical syndromes may similarly affect the VVP, CSF pressure gradient and posterior circulation similar, and will be discussed later in this article.

Lastly, there are transformed or rebound migraine headaches. These are caused by the excessive use of medications, such as analgesics and ergotamine tartrate, to treat migraines. Using these medications for more than two days a week can lead to a constant headache that won't go away until the medication is withdrawn. The cause is unknown, but it is most likely due to worsening of the effects of ischemia, and the hyperperfusion and edema as a result of the ischemia.

Migraines, MRIs and Hyperintensity Signals

CADASIL, BAM and classic migraines are associated with increased hyperintensity signals on MRI. Hyper-intensity signals show up as areas of increased whiteness. They are attributed to several causes, including demyelination, vasculitis, ischemia and inflammation. It is interesting to note that hyperintensity signals are seen in similar locations in several other important degenerative diseases of the brain, including multiple sclerosis; Alzheimer's disease; Parkinson's disease; manic depression; and schizophrenia.

In migraine headaches, the hyper-intensity signals sometimes appear diffuse and scattered similar to those seen in multi-infarct dementia (Binswanger's subcortical encephalopathy), which also has a predilection for the white matter and basal ganglia. The hyperintensity signals in migraines are attributed to chronic ischemia, but studies of brain blood flow during migraine headaches show increased blood flow during the initial attack. It is therefore currently maintained that hyperperfusion most likely results in cytotoxic edema, which is suspected of playing a role in demyelination and ischemia.

The cause of the hyperperfusion is unknown. It is possible that oxidative stress from a reduction in blood flow in the posterior circulation, such as from craniocervical syndromes, triggers the neurovascular myogenic autoregulatory reflex mechanism to deliver more blood to the brain. If the increase in blood flow encounters increased resistance to outflow through the VVP, such as from a craniocervical syndrome, it would further lead to interstitial and cytotoxic edema. In addition, venous congestion and hypertension in the VVP may likewise stimulate baroreceptors in the suboccipital cavernous sinus, which in turn stimulate the neurovascular myogenic autoregulatory reflex mechanism. Thus, craniocervical syndromes may play a role in the delayed ischemia-hyperperfusion-edema blood flow patterns seen in MRI studies of migrainous patients.

In addition to the diffuse type of hyperintensity signals seen in certain cases of migraine headaches, most migraines are associated with predilection for certain areas of the brain, especially the cerebellum and other structures supplied by the posterior circulation. This includes the deep white matter structures and

periventricular areas. This is interesting because Alzheimer's, Parkinson's, schizophrenia and manic depression are also associated with hyperintensity signals in the deep white matter, basal ganglia and periventricular areas of the brain stem, and may, according to this author, be similarly affected by craniocervical syndromes. Classical migraines tend to involve the cerebellum, the labyrinth, and the occipital and temporal lobes.

As stated above, unlike most migraines, ophthalmoplegic and retinal migraines affect the oculomotor and optic nerves, which are supplied by the anterior circulation and would not be directly affected by craniocervical syndromes. In certain cases, however, because of the redundant design of the circulatory system of the brain, the posterior circulation may steal blood from the anterior circulation.

Brain Circulation

The brain gets its blood supply from the two carotid arteries and the two vertebral arteries. The carotid arteries enter the cranium through the *foramen lacerum*, and then pass through the cavernous sinus inside the cranium before entering the brain. The vertebral arteries, on the other hand, first enter the suboccipital cavernous sinus outside the cranium and then pass upward through the *foramen magnum* to enter the brain. The suboccipital cavernous sinus is connected to and part of the VVP. It is therefore affected by venous congestion and hypertension in the VVP. As stated above, the suboccipital cavernous sinus also has baroreceptors that appear to assist the neurovascular myogenic autoregulatory reflex mechanism in maintaining steady blood flow in the brain.

After entering the cranium, the two vertebral arteries unite at the level of the pons and medulla to form the basilar artery, which passes upward along the ventral surface of the brain stem. The carotid and basilar arterial systems come together at the base of the brain to form the circle of Willis. This creates a redundant system that provides a measure of backup protection, should one of the other routes fail. Thus, as stated previously, the posterior supply can steal from the anterior supply, and vice-versa. Furthermore, both the carotid and vertebral arterial systems show a high degree of variability in design. Some designs may be more efficient than others. Some designs may be predisposed to problems.

Blood exits the brain through the dural sinuses. The dural sinuses are not true veins. Instead, they are tubes formed from dura matter and lined with endothelium from veins. This makes them much stronger than ordinary veins and protects the drainage routes from compression from the weight of the brain. More importantly, the cavernous sinus and suboccipital cavernous sinus also protect the carotid and vertebral

arteries from compression (they also enhance brain cooling). The dural sinuses drain into two systems. One is through the sigmoid sinus and into the jugular veins; the other is through the occipital marginal sinus system and into the vertebral venous plexus. Just like the circle of Willis, this creates a redundant system that provides alternative drainage routes during postural and respiratory constraints. In addition, the suboccipital cavernous sinus system appears to serve as a rerouting mechanism.

Brain Blood Flow

Brain blood flow remains fairly constant through all activities of daily living and sleep. It is determined by the cerebral perfusion pressure, which is the difference between systemic arterial pressure and superior sagittal sinus venous pressure (SSVP). Steady arterial blood flow is further maintained by a neurovascular myogenic autoregulatory reflex mechanism, which dilates and constricts the arteries of the brain according to need. High systemic pressure, for example, causes the blood vessels of the brain to constrict. It has been suggested that loss of control of the neurovascular myogenic autoregulatory reflex mechanism, such as from aging, may play a role in faulty hemodynamics leading to chronic interstitial and cytotoxic edema and the subsequent diffuse hyperintensity signals seen on MRI.

In contrast to arterial flow and pressure, SSVP fluctuates widely from inversion to the recumbent and upright positions. In the upright position, it becomes negative. This increases cerebral perfusion pressure and brain blood flow. In addition, the cavernous sinus and suboccipital cavernous sinuses have baroreceptors that appear to assist the neurovascular myogenic autoregulatory reflex mechanism of the arteries. Their exact role is unclear, but as stated above, it seems likely that their role is to signal the arteries to increase pressure when confronted with increased outflow resistance in the VVP, such as in the recumbent and inverted positions or during Valsalva maneuvers. (Giraffes also have baroreceptors in the suboccipital cavernous sinus; whales and bats probably do as well. These baroreceptors probably likewise assist in stabilizing brain blood flow and ICP during extreme hydrodynamic stresses such as inversion in giraffes and bats and diving in whales, which is similar to an extreme Valsalva maneuver).

The Accessory Drainage System and the Craniocervical Spine

Some anthropologists believe that the large size of the human brain is due to the increase in blood flow from upright posture. The increase in inflow, however, must be offset by an increase in outflow to maintain intracranial fluid volume and pressure. This is achieved by an accessory drainage system that evolved to accommodate prolonged upright posture. The accessory drainage system in humans includes the occipital

marginal sinus system, the emissary veins and foramen, the suboccipital cavernous sinus and the VVP.

The craniocervical spine is the critical link between the occipital marginal sinus system and the vertebral venous plexus. It has been suggested that venous congestion, or hypertension of the VVP from spondylosis, is one of the most overlooked causes of hemodynamic failure and ischemia of the spinal cord. Moreover, it is my opinion that venous congestion and hypertension of the VVP and suboccipital cavernous sinus of the accessory drainage system from cranio-cervical syndromes may be one of the most overlooked causes of chronic ischemia, NPH and edema of the brain.

The Posterior Circulation of the Brain

The posterior circulatory system of the brain comes from the vertebral arteries, which branch off of the subclavian artery. The vertebral arteries enter the fifth or sixth cervical vertebra and ascend through the intertransverse foramina to the first cervical vertebra, where they wind posteriorly around the condyles and then enter the suboccipital cavernous sinus. From here, they pass through the foramen magnum and join together to become the basilar artery. Before joining, each vertebral artery gives off branches, which include the spinal; muscular; anterior and posterior spinal; meningeal; posterior inferior cerebellar; and medullary (medullary branches supply the white matter).

The posterior inferior cerebellar artery supplies the posterior inferior part of the cerebellum, which includes the *vermis* and flocculonodular lobe. Problems with the vermis cause vertigo. Likewise, episodic vertigo frequently occurs in patients suffering from ischemia in the distribution of the vertebrobasilar circulation. Problems with the cerebellum are also associated with ataxic gait. Ataxic gait is often one of the earliest signs of dementia and Alzheimer's disease. Parkinson's disease, on the other hand, is associated with stooped posture and shuffling gait. The flocculonodular lobe is one of the oldest parts of the cerebellum, and is associated with truncal muscles in vertebrates. This may explain the truncal rigidity and stooped posture seen in Parkinson's disease.

The medullary branches are of special interest, because they supply the medulla, pons and the periventricular white matter. Hyperintensity signals are frequently seen in the periventricular area. This is a white matter area of the brain comprised of myelinated neurons. Oligodendrocytes require a lot of oxygen just to maintain myelin - never mind repair it. The periventricular area, however, lies in a border zone where circulation is weak. This makes it particularly sensitive to oxidative stress from decreases in circulation.

After the vertebral artery, the basilar artery ascends to the circle of Willis and gives off branches along the way, which include the pontine; labyrinthine; anterior inferior cerebellar; superior cerebellar; posterior cerebral; choroidal branches (medial surface of thalamus); and cortical branches. The cortical branches then divide into branches that supply the uncus; fusiform gyrus; inferior temporal gyrus; cuneus; precuneus; lingual gyrus; and posterior surface of the occipital lobe.

The labyrinthine and anterior inferior cerebellar and superior cerebellar arteries may play a role in nausea and dizziness in migraine headaches. The posterior cerebral and cortical branches, on the other hand, supply portions of the occipital and temporal lobes. The posterior cerebral and cortical branches may therefore play a role in visual and hallucinatory disturbances seen in migraines.

Craniocervical Syndromes

The Cervical Syndrome by Ruth Jackson was published in 1956. She described myriad symptoms associated with injuries to the cervical spine. The term "craniocervical syndrome" more specifically refers to cervical syndromes that affect the brain. For this reason, they have also been called "cervicoencephalic" syndromes. Craniocervical syndromes can be due to congenital craniocervical malformations and synostosis, or acquired from aging and injuries to the spine. They can adversely affect cerebral hemodynamics in two ways: One is by venous congestion in the VVP, causing a reduction in the cerebral perfusion pressure; the other is by venous congestion and hypertension in the suboccipital cavernous sinus, causing compression of the vertebral arteries, and thus decreasing the posterior blood supply to the brain.

In many cases, the cause of ischemia of the spinal cord is unknown, although recent studies suggest that a compromise in venous drainage can result in delayed ischemia of the spinal cord. Despite the fact that it is also a redundant system, the venous drainage system of the spinal cord is still a low-pressure system that is susceptible to mild compression, and even slight elevations in pressure. Research on the lumbar spine, for example, has shown that 25 percent stenosis is associated with moderate to severe venous congestion. Furthermore, it has been shown that abnormal curvatures of the spine can cause functional stenosis, and increase pressure in the VVP. Lastly, it has been shown that venous congestion in the VVP without compression of the cord can cause glove-distribution sensory loss and cauda equina syndrome.

Researchers have concluded that injuries to the spinal cord from arterial, venous and CSF obstruction are far more common than we think. Similarly, it is my opinion that injuries to the brain from arterial, venous and CSF obstruction in the craniocervical spine are also far more common than we think. That is, craniocervical

syndromes may lead to cranial hydrodynamic failure, chronic NPH, edema and ischemia of the brain. Craniocervical syndromes, therefore, may play a significant role in migraine headaches, as well as many other degenerative diseases of the brain.

Migraines, Strokes and Chiropractic Care

Drugs and surgery cause far more strokes each year than chiropractic care. The number of strokes from bypass surgery in the U.S. alone each year exceeds the total number of all the reported cases of strokes that may or may not have been due to chiropractic care since it first began in 1895. Add to that hormone replacement therapy, birth control pills, steroids and PPA (found in many OTC medications), which also cause strokes. In addition, when these drugs are prescribed to patients with health conditions such as hypertension, diabetes and smoking, the risk for strokes increases significantly. Lastly, the medications specifically developed for the treatment of migraine headaches may actually worsen the effects of ischemia and strokes. Thus, chiropractic care may be the safest and most effective type of treatment for all types of migraine headaches, including high-risk cases such as CADASIL and BAM, for which there are no other treatment options.

Aside from disease, drugs and surgery, there are other risk factors chiropractors need to be aware of. Acute onset of intense or the "worst-ever" type headaches in patients with no prior history of headaches is definitely a significant risk factor for stroke. On the other hand, it was once thought that chronic headaches were less likely to be associated with strokes. It appears that while chronic headaches may be less likely to be associated with cerebrovascular strokes, they may be associated with an increased incidence of ischemic strokes. A female in childbearing years is a risk factor; migraine headaches in children are also a risk factor. Surprisingly, seniors are less likely to have ischemic strokes as a result of migraine headaches.

While research needs to be conducted to determine the risks along with the safest and most effective methods of chiropractic care of the cervical spine, at the same time, research needs to be conducted to examine the benefits of chiropractic care for the treatment of migraine headaches compared to the risks of prescription and nonprescription drug therapy. Timely and appropriate care of craniocervical syndromes over the course of a lifetime may help to decrease the incidence and severity of migraine headaches and ischemic strokes and the incidence and severity of other degenerative diseases of the brain.

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