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Alzheimer’s Disease: Can Chiropractic Make a Difference?

By Michael Flanagan, DC, DABCN

What is the cause of Alzheimer’s disease? After more than a decade of intensive research, the scientific community seems to be arriving at the consensus that there is no one cause of Alzheimer’s disease. Rather, many factors, both genetic and extrinsic, probably contribute to the disorder in varying degrees in different individuals. Some scientists, "suspect that the real heart of the disease lies beyond the brain and that neuronal death represents a secondary or even a tertiary ’side effect’ of a more fundamental dysfunction."[6]

Alzheimer’s disease has mushroomed into a global health crisis. According to T. Franklin Williams, director of the National Institute on Aging in Bethesda, Maryland, Alzheimer’s disease "is by far the most threatening epidemic that we have in our nation."[6] And while our nation continues to wrestle with the issue of abortion, the equally compelling issue of euthanasia looms on the horizon.

My research leads me to believe that a dysfunction in the fluid mechanics of the brain may be an overlooked cause of Alzheimer’s disease. Fact: Alzheimer’s patients often have "normal pressure hydrocephalus," meaning an increased volume of cerebrospinal fluid (CSF) in the brain but without a significant increase in pressure. Fact: On autopsy, the size of the cavities which produce CSF are often enlarged in Alzheimer brains as are the fissures and sulci (which means Alzheimer brains have shrunk in size).

Some investigators have interpreted these observations as indicating that Alzheimer’s disease causes brain degeneration and subsequently a decrease in brain size, causing an increase in CSF volume to fill the empty space inside the cranial vault. But, I believe the reverse is true: increased resistance to fluid outflow from the brain causes chronic normal pressure hydrocephalus and toxic metabolic edema, which leads to neuronal degeneration and eventually a decrease in brain size.[3,4]

The cause of the pathological increase in the brain fluid volume is an impairment in the CSF and venous drainage system. In addition to the jugular veins, the brain also drains via accessory veins into the vertebral venous plexus, an extensive system of valveless veins in the neural canal.[3,4] This accessory drainage route

is unique to humans. According to anthropologists, "the sprouting of these veins (accessory veins) and the reorganization of other venous channels may have set the groundwork for the dramatic increases in brain size observed in the Homo lineage that led to modern humans. Philip V. Tobias of University of the Witwatersrand in Johannesburg, South Africa, and Dean Falk of the State University of New York in Albany, propose that "early hominids developed an altered blood flow from the brain when they began walking upright. In some species, such as A. africanus, a network of veins was established to cool the brain and allow it to increase greatly in size. Other species, such as australopithecines, has no extensive cooling system and thus remained small-brained."[1]

The corollary to this hypothesis is that if the large size of the human brain is attributed to this accessory drainage system, then it seems likely that decreasing their effectiveness would lead to neuronal degeneration and a decrease in brain size.

Since there is no lymphatic drainage system in the brain, CSF and interstitial fluids from the brain mix and travel together through the perivascular and subarachnoid spaces to eventually be absorbed by arachnoid granulations of the superior saggital sinus. Movement of these fluids from the ventricles and interstitial spaces is dependant upon a slight pressure gradient between the ventricles and the superior sagittal sinus. Because the superior sagittal sinus and other dural sinuses have no valves, this pressure gradient is very much affected by pressure in the vertebral venous plexus. And pressure in the vertebral venous plexus is affected by things such as posture, Valsalva maneuvers, and degenerative conditions of the spine.[3,4]

Degenerative conditions of the spine, including stenosis, scoliosis, and kyphosis can decrease the effective size of the neural canal and thus impinge on vertebral venous plexus, compromising the effectiveness of the accessory drainage system of the brain. The impairment in cerebral drainage has the "double whammy" of inhibiting the removal of metabolic waste products, which may ultimately be responsible for the process of neuronal degeneration.[3,4]

Metabolic waste products as a cause of neuronal degeneration is not a new idea. "Early writers once considered retained toxic metabolites to be the cause of diabetic neuropathy which cause damage to the myelin sheath. Although questions can now be posed in more precise terms, surprisingly little advance has accrued concerning the causation of diabetic neuropathy."[2] Similarly, little is known about the cause of demyelination of the brain as in multiple sclerosis.

A clue to the effects of metabolic toxicity causing neuronal degeneration may come from studies done on stroke. While the core tissues involved in stroke (those tissues dependant upon the blocked artery) are probably overwhelmed by many destructive processes, the penumbral tissues (those tissues receiving blood from other arteries) are damaged by a cascade of events caused by altered cytochemistry.[7]

It is believed that the initial or induction stage of stroke is due to ischemia which causes neurons to release excessive glutamate. This causes overstimulation of glutamate receptors, which control calcium and sodium channels, causing those elements to rush in and disrupt the ability of the neurons to respond normally to signals from other nerves.[7]

In the next stage, called the amplification stage, another type of glutamate receptor triggers the production of intracellular messengers. These messengers cause the release of calcium from internal cellular stores. Thus, calcium levels within the cell continue to soar. The excess calcium then combines with the elevated level of one of the intracellular messengers to alter the activity of several families of enzymes. These enzymes modify membrane proteins, increasing the sensitivity of neurons to excitatory signals which increases the release of glutamate from nerve terminals. Thus, the events of this stage are associated with the buildup of calcium and a worsening of excitotoxicity in the adjacent neurons.[7]

The second stage ultimately gives way to the third and final stage, called the expression stage, during which irreversible damage occurs. Early in this stage calcium activates enzymes that attack several classes of molecules, including nucleic acids, proteins and lipids. The breakdown of phospholipids in the outer cell membrane (the myelin sheath is composed of phospholipids) and in membranes of internal organelles may be particularly devastating.[7]

When phospholipids are degraded, one by-product is arachidonic acid, the metabolism of which leads to formation of highly reactive compounds called oxygen-free radicals. These compounds, in turn, can initiate a chain reaction, called peroxidation, capable of destroying the cell membrane.[7]

Arachadonic acid can harm cells in other ways as well. It participates in the formation of eicosandoids, which increase the aggregation of blood cells and the constriction of blood vessels. Such changes are probably aggravated by the formation of platelet-activating factor, yet another consequence of phospholipid breakdown. Together eicosanoids and the platelet-activating factor can thus generate a viscious cycle in which ischemic injury leads to further ischemia.[7]

Interestingly, a 1983 study concluded that active hydrocephalus produces significant periventriucular demyelination which was attributed to mechanical stretching of nerves. Of the patients studied, elevated myelin basic protein was found in 80 percent of those with normal pressure hydrocephalus.[5] If this is so, then the breakdown of the myelin (phospholipid degradation) sheath may cause a similar cascade of events as seen in the expression stage of stroke.

So does this mean that all people with degenerative conditions of the spine such as scoliosis, kyphosis, or stenosis will get normal pressure hydrocephalus, toxic metabolic brain edema and later Alzheimer's disease? Probably not. Rather the configuration of the neural canal and the basicranium may predispose certain individuals to compression of the vertebral venous plexus and accessory veins, resulting in normal pressure hydrocephalus and toxic metabolic edema. This is much like an acute iridocorneal angle, which tends to impinge on the canal of Schlemn and inhibits the absorption of aqueous humor, resulting in acute angle glaucoma.

My hypothesis can have quite pragmatic results: Maintaining the health of the spine and early treatment of spinal injuries may prevent damage to the neural canal and subsequent degenerative conditions of the brain such as Alzheimer's disease.

I need help from our profession to test the validity of my hypothesis. I am proposing two initial pilot studies. Both are non-invasive, retrospective epidemiological studies. Both were overviewed by an epidemologist who said they were feasible. The first is to investigate the prevalence of spinal stenosis, scolosis, and kyphosis in Alzheimer's patients. The second is to compare the incidence of Alzheimer's disease in lifetime chiropractic patients versus non-chiropractic patients. Both require the expertise of a biostatistitian or epidemiologist. Later, if these studies are positive then more invasive, quantitative flow studies will have to be done by others.

If anyone has any suggestions or professional connections which can help this project started please call or write to me at the following address:

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The investment in this project would be relatively small, the potential gain tremendous. Our profession just might provide a clue to one of the most perplexing neurological problems of the day. Moreover, lifetime chiropractic care might help reduce the incidence of an extremely costly global epidemic.

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