Macular Degeneration

R. Paul Lee, DO, FAAO, FCA

History:

A 53-year-old female presented with the complaint that a recent image of her right retina obtained by her optometrist revealed vascular changes consistent with early macular degeneration. She wanted to know if I could help before she went to her ophthalmologist the following week. Several years ago, the ophthalmologist had performed laser surgery to reattach this same retina. Her vision seemed to be degenerating recently, which motivated her to see the optometrist.

Generally, she has been healthy, aside from some minor depression and injuries to her fingers. Besides the retinal surgery, she has had no other hospitalizations. She relates no allergies and takes no medications. She recalled only one auto accident in her twenties in which she was struck from the right side by another vehicle. Her injuries were minor and she sought no medical attention. Otherwise her trauma history was negative.

Physical:

Palpatory examination revealed traction on the right optic nerve. Tracing posteriorly to find the origin of the traction, it became clear that the optic chiasm was also drawn posteriorly and to the left. Further back, along the optic tracts, the thalamus had been shifted to the left and the lateral geniculate ganglion where the optic tracts terminated was pulled to the left.

Treatment:

As I watched the thalamus, I could detect that the force that caused the shift to the left also seemed to spin it on a vertical axis clockwise creating a fulcrum for the displacement of the thalamus. As I observed the fulcrum, it shifted and the thalamus assumed a neutral position. The thalamus began to breathe with PRM and the fluctuation of the CSF became quite enhanced for a time.

Once the thalamus achieved full resolution of its distortion into a position of health, the PRM returned to a normal healthy rhythm. I then looked at the optic tracts and the optic nerves. There was now a balance of the positions of all these parts and the whole system was breathing naturally including the globe of the right eye. The fulcrum now shifted to the facial bones, especially the right zygoma and maxilla. Once these changed their positions and breathing functions, the whole seemed improved and there was a stillpoint. Once PRM returned to a normal healthy quality, the treatment ended.

Follow up:

I saw the patient the following week after she had visited the ophthalmologist. He carefully examined the eye and reported no sign of the early vascular changes seen in the previous image of her retina. I found no traction on the optic nerve and no shift of the thalamus. We concluded the visit by saying that if her vision became worse that she should come back but my expectation was that she would not have macular degeneration.

Discussion:

In my experience, the thalamus seems to be especially troublesome in cases of trauma. It is a dense collection of nuclei, heavier than the surrounding tissue of the internal capsule made up of axonal tracts heading to the cerebral cortex. The third ventricle lies between the two halves of the thalamus allowing a certain amount of independent mobility of the two parts. The thalamic adhesion, between the right and left thalamus transiting the slit that constitutes the third ventricle, stabilizes the two halves of the thalamus into a more unitary function, however. But the thalamic adhesion can act as a fulcrum for the two halves of the thalamus. I commonly see the entire thalamic structure with the third ventricle shifted en mass, as well. Or there can be a combination of shifting the whole thalamus with a twisting around the thalamic adhesion.

Because the optic tracts travel a relatively long distance to reach the geniculate ganglia in their posterior, inferior position on the thalamus, the tracts are susceptible to being stretched by the shifting and twisting thalamus. The softer density of the surrounding fiber tracts and the fluid of the third ventricle between the two halves of the thalamus and the fluid of the lateral ventricles above the thalamus permit these traumatic distortions of the normal position of the thalamus. The fluid bath within which the optic nerves reside within its dural sheath permits tractioning of the nerves. Cerebrospinal fluid surrounds the optic nerves inside a dural sheath that is continuous from the sclera on the posterior eyeball to the meningeal dura within the vault through the optic canal. CSF surrounds the optic nerve from the sclera to the chiasm. From there to the lateral geniculate body in the thalamus, CSF surrounds the optic tracts as well. This freedom to float in the bath of CSF allows the entire optic pathway from the retina to the lateral geniculate body to be subject to traction.

One could speculate that the retinal detachment years earlier could have been caused by the same trauma that caused the stretching of the optic nerve. The traction through the optic nerve on the posterior aspect of the eyeball could have contributed to the retinal detachment.

The blood supply to the retina and the optic nerve can also be compromised by malpositions of the diencephalon. The ophthalmic artery proceeds anteriorly from the internal carotid as it emerges from the cavernous sinus near the anterior clinoid process. The ophthalmic artery proceeds through the optic canal with the optic nerve. Immediately the central retinal artery branches off the ophthalmic artery as it enters the bony orbit beyond the optic canal. The central retinal artery penetrates the dura and enters the subarachnoid space within the optic nerve. It then enters the center of the optic nerve itself to emerge in the posterior eyeball within the optic disc. One diagnoses adverse vascular changes of the retina by visualizing the optic disc. Traction on the optic nerve inhibits the flow of blood within the central retinal artery, which is visible on retinal imagery at the optic disc.

Releasing the traction on this patent's optic nerve by allowing the thalamus to return to its original position restored the circulation to the optic disc and resolved the threat of macular degeneration. There are other causes of macular degeneration than mechanical ones, but osteopaths have the opportunity to help this condition if there is a mechanical aspect to the etiology. Osteopaths also have an opportunity to change the vascular blood flow if it is not accompanied by a mechanical distortion. That is another topic.

Dental Corner

Massage Table a Perpetuating Factor

Douglas Vrona, DMD

Over the past 30 years, I've been treating head/neck/TMJ pain with a combination of Dr. Travell's trigger point injections, physical therapy and cranial osteopathy.

While treating a 47-year-old female physician from India, I discovered a perpetuating factor which in retrospect caused multiple relapses prolonging her case. The offender was found to be weekly massage therapy for her low back and shoulders utilizing a facial support ring attached to or within the massage table. The deleterious effects were as follows:

1) Intra-osseous strains within the frontal bone.
2) Locking of the frontal/ethmoid complex
3) Compression of the sphenobasilar synchondrosis.
4) Fixation of the zygomatic/temporal suture.
5) Retrussion of the mandibular condyle into the glenoid fossa causing external rotation of the temporal bones resulting in limited jaw opening. This was especially prevalent when the TMJ disc was not in place allowing bone-bone contact.
6) Compression of the maxilla/palatine articulation as a result of a retruded occlusion.
7) OAA fixation (C_1 is the axis of rotation of the upper & lower jaws).

This patient was referred by a chiropractor whose treatment had been unsuccessful. Symptoms included limited, painful jaw and cervical ROM with 10+ left OAA/shoulder/arm pain.

TMJ corrected axial tomography revealed left TMJ degenerative joint disease but with normal translation.

Her limited jaw opening was therefore due to a deficient cervical component. There was total loss of upper cervical function secondary to major cranial distortion from a difficult birth including face forward presentation. This patient's cervical spine was nearly fused on the left, I assume a result of bleeding during the birthing process. (fibrous union)

After six months of manipulation of the cranium, TMJ, cervical spine, shoulder girdle & thoracic outlet on the left, this patient has a normal pain free jaw opening, with no head/neck/shoulder pain, and a 100% cervical range of motion. CRI improved from 6 to 12 cycles/minute.

Trigger point injections were done in the left trapezius, levator scapular, SCM, scalenes, temporalis, masseter, supra & infra spinatus to release osseous structures & eliminate referred pain. Chiropractic manipulation to the low back & pelvis were done monthly.

This physician has been returned to her chiropractor, who now successfully maintains her case with full spine adjustments as needed.

Although she is a severe Class II - Division II malocclusion (*editor's note*: retruded mandible due to upper anterior teeth tipped in toward the palate), the medical/dental necessity of a splint or restorative dentistry was unwarranted.

When I reviewed my patient base, I found several more stubborn prolonged cases unknowingly having full body massage or acupuncture using a face ring for support. Osteopathic treatment without this perpetuating factor has been far more successful with an end in sight.

If 5 grams of pressure is all that is needed to move a cranial bone, the deleterious effect of a 10-15 lb. cranial vault lying on a massage table for 30-60 minutes is worth noting especially in chronic headache cases.

Any dentist who has a treatment case that involves cranial osteopathy and dental treatment please submit an article to me for the Dental Corner. I would be happy to help you with writing up your case.

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The Cranial Dental Proficiency Examinations are scheduled. Please check the website for upcoming events or for further information contact the Dento-Cranial Competency Board at 540-635-3610. Website: http://dentalcranial.org/home