Journal of the

Arizona Association of Chiropractic

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Sacral-Coccygeal Chordoma eroding the S3, S4, S5 segments and also the coccyx.

In this issue.....

Arizona Association of Chiropractic

Upcoming Events

June 9-11, 2023	**AAC Annual Convention** **Wildhorse Pass Casino & Resort**
1st Wednesday of each month	**AAC Dinner—6:00pm** **La Famiglia Pizza & Pasta** **1859 W. Guadalupe Rd.** **Mesa, AZ 85202**

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President's Message

Charles Dickens opened A Tale of Two Cities with the famous quote, "It was the best of times it was the worst of times." We all have those realizations and that probably applies to the last two years in the Arizona Legislature, especially as it relates to the chiropractic profession.

I would like to start off by welcoming our new team of the executive committee: Past president Dr. Trever Penny, VP Dr. James Bogash, Treasurer Dr. Rebecca Carson, Secretary Dr. Scott Tauber and our new Executive Assistant, Sandra O' Grady. We have an amazing team that has come together and we are on it…

Once again Senator Nancy Barto and Representative John Kavanagh led the way in supporting our efforts and they deserve our unswerving support and appreciation. We need to continue to help those that support us.

SB 2021 applied significant restraints on filing liens for care passed the Legislature in the waning hours of the session and the Governor signed it. But even that could be a short-lived loss as consideration for a statutory definition of reasonable and customary is likely to be introduced in the 2023 session. That proposed legislation could clarify what chiropractors can expect for services provided in personal injury cases. But we will be at the table when that legislation is being crafted.

The last two years at the Arizona Legislature:

In 2021 legislation passed that relates to diagnostic services. The attorney general said that we were not able to do a part of a basic exam that we learned in our schools. We took on that issue and passed language that enables chiropractors to test patients for things such as COVID virus, strep throat, and other diseases, utilizing diagnostic methodology that are included as they should be.

More importantly, after years of trying, the 2022 legislature finally authorized chiropractic coverage under Arizona's Medicaid system or AHCCCS. Funding for that coverage was included in the general appropriations legislation. The law took effect on October 1st (the first day of the Federal fiscal year.) We have already had insurance plans that underwrite the AHCCCS program contact us to work on the implementation process. We need to be very cautious as we work on the implementation to guarantee that this coverage is not administratively impeded in any way.

As we look forward to the 2023 session we need to address several insurance disparities as well as evaluate whether to move forward on such things as allowing chiropractors to administer and prescribe nutritional supplements. Again, it is something is taught in chiropractic schools it should be allowed for those chiropractors who want to provide it. At the AAC, our job is to represent all chiropractors, not a particular type, all wonderful, different types.

There are other professions that have expanded their scope that may have less education, yet were able to. I have to give them the credit for doing this and would like to increase our scope of practice. Now for me, I may not do any of the things I propose but, if we do not do a thing, I fear we will be absorbed. There are some that will say we are better at adjusting than any other professions. I believe that, but the public perception does not know that.

It is truly up to you, **"Together we move forward".** The above is just some of what the association has been doing for the Chiropractors in Arizona, So when asked, "Why should one join?… Well, tell them without membership none of the above would have happened. It costs money to make things happen. We have just under 2000 Chiropractors in Arizona. We could be one of the most powerful Associations in Arizona if we had complete membership. It takes 30 seconds on the website to join and be a part of making things happen. I am not going to beg those that don't want to move things forward and cost it has on others. Each of us should want to be a part of something special that has your back.

I personally want to thank our members, you made the above things happen.

Andrew Altman, DC

"Seeing" Instability

Mike Winberry DC

Prior to the invention of digital motion x-ray (DMX), plain films in flexion and extension were, and still are, the industry standard for evaluation of spinal instability. X-Ray radiation penetrates through all of the soft tissue, but not through the bony tissue, and it is the shadows of the bones which show up as images on the finished plain film radiograph. For decades now, we have known what happens at every joint in the human body when you move. This knowledge is out there, packed into textbooks and sitting on the shelves for anyone who cares to read about it. Researchers have done computer simulations, worked with cadaver spines, and made fiberoptic recordings in order to figure all of this out. Before DMX ever came along, unusual gapping at the end ranges between the bones on the static flexion and extension films was considered to be the medical industry's standard of proof that ligament damage had occurred, and since DMX is nothing more than x-ray (albeit highly specialized), the same principle is applied to its interpretation. While MRI gives you direct information as to the integrity of a tissue, the technology has limitations, such as need to have gaps between the individual slices which may be larger than the ligaments that need to be visualized, thereby leaving them out altogether. An alar ligament is only about 2 mm. wide, so if it happens to fall into the 3.3 mm. gap that your friendly neighborhood MRI tech set up on the study, you'll never see it. In current practice, spinal MRI studies are disc studies, and the status of the ligaments is seldom mentioned. DMX studies are complementary to MRI studies because they are ligament studies, albeit "indirect" ones, and not disc studies. While the ligaments are invisible, the gapping between the bones is obvious. But very little of what I flag on a report as an injury is done by "eye-balling;" the reason it takes me 4-5 hours to put a report together is because of all the measuring I do. I am a firm believer in measuring whatever I can; I have found that if you don't measure it, you will miss it.

Recently, I was having a conversation with an attorney who likes having Digital Motion X-Ray (DMX) studies as part of his personal injury cases, and he was frustrated because he had a client who had been injured several months before in an auto collision, had been receiving chiropractic care, but was still complaining about the neck pain and the headaches. When he suggested to the treating doctor that maybe a DMX could identify the source of the pain, he was rebuffed by the doctor, who said that he "wasn't seeing the instability." Well, I've heard this before, and I think it's time a statement like that needs to be unpacked and analyzed, to see if it has any validity.

Let's take "seeing the instability" at face value. Can the naked eye really "see" an unstable segment in a patient's neck? No, it can't. Gross visual inspection of a patient is pretty much limited to statements which describe the patient's posture, things like "anterior weight bearing head," "unleveling of the head," or "cock robin appearance," or to description of external injury, such as abrasions, lacerations, contusions, and edema. There isn't anything you can reliably point to as an outward sign that there is internal spinal derangement. In fact, that's one of the biggest problems with dealing with the auto collision patients: outwardly, they look fine. They look just like you and me. We call them the "walking wounded" because they look normal on the outside, but on the inside they are badly injured and the quality of their lives is seriously impaired. There is a tendency for this "miserable minority" created by motor vehicle collisions to be judged at face value by their peers. That's no different than judging a book by its cover.

Well, if you can't see the instability, can you feel it? We chiropractors have prided ourselves in our palpation skills from the very beginning. The Motion Palpation Institute taught us to feel the fixations, and then to adjust them. If we found anything which was moving better than the obvious fixation we had found, then we just called that either normal motion or hypermobility, and ignored that. Really, all we did was adjust fixations, without putting a lot of thought into it. The problem with this approach is that with whiplash injuries, the muscle guarding which is initiated when ligaments are injured is widespread, and locks down the whole neck, and not just the segments which have sustained ligamentous subfailure. This rigidity can literally go on for months or years. I've had patients come into my clinic for a cervical DMX study a full two years after the traumatic incident, and their range of motion was still substandard due to muscle guarding. And under these circumstances, it is difficult to sort out manually the injured joints from the non-injured joints. For instance, there is an orthopedic test for alar ligament damage in which the occiput is stabilized with one hand while the spinous process of C2 is palpated for motion as the occiput is passively laterally flexed. The C2 spinous process may also be braced to block movement in order to

stress the alar ligaments. This may be helpful in the chronic situation, but it is not helpful in the acute situation, when muscle guarding makes it painful to create any movement, much less any shear or rotation between the atlas and the axis. And, actually, I've seen this test done and deemed negative too many times in a physical therapist's notes, only to do a cervical DMX on the patient and find out that he/she definitely does have alar ligament damage.

Consider this case: a patient was injured ten months prior to the DMX study, and had been out of treatment for the previous three months. Cervical spine MRI had never been performed, and while stress x-rays were available, nothing in the notes indicated that they had ever been properly evaluated. He was still doing the physical therapy exercises he had been taught, and occasionally required an NSAID for relief. The PT notes indicated that the alar ligament test was negative. I asked him about the chiropractic treatment he had received, and he told me that he felt it really helped, but he rated his recovery to date at only 50% (with 100% meaning that pre-injury status had been reached). He was still having headaches and neck pain several days per week, and his work with computers was difficult for him to do. I asked him what the chiropractor told him when he was released from care, and he was told that "everything seems to be moving the way it's supposed to," so give it some time, and everything should continue to improve. Well, he was already figuring out that maybe that wasn't true, because he was miserable, and the results of the cervical DMX study categorically showed that he was not moving well, at all. Just take a look at the angular motion analysis chart from the case. **(Table 1).**

Understanding an angular motion analysis table is not rocket science. The normal total range of motion at each level is listed in the column on the far right. Interpretation of normal is easy- the total range of motion at each level should be represented by equal parts flexion and extension. In other words, at C2-C3, where the total range of motion is 10°, 5° of that should be in flexion, and the other 5° should be in extension, or something roughly equivalent to that. Let's say the upper limit of normal intersegmental angular motion is 11° (in one direction), even though the discussion is a little deeper than that, which I'll take up at a later date.

At C0-C1 and C1-C2, the rules for evaluating range of motion are different. Most of the motion at both levels should occur in extension, and, for the most part, flexion is merely the return movement from extension. At C0-C1 the superior articulating surfaces of the atlas converge to the anterior and present a bony block to further flexion. At C1-C2, the transverse ligament of the atlas wraps around the odontoid process of C2, which limits not only translation, but angular motion as well. In both the craniocervical junction and the subaxial spine, the formula for measuring total range of motion in flexion and extension designates flexion as the "positive" value and extension as the "negative" value. In normal studies, because of each individual's unique configuration at C0-C1 and C1-C2, most of the time flexion is measured in the negative range, at 0°, or maybe 1-3°, while the greater portion of the motion is measured as extension.

That being said, the functional abnormalities are highlighted in red on the chart. While a total range of motion of 15° at C1-C2 would ordinarily be considered to be within normal limits, the balance between flexion and extension is not. 10° is an abnormal amount of flexion and is suggestive of craniocervical ligament subfailure. In this case, that was corroborated by pathologically asymmetrical para-odontoid spaces as seen on the AP Open Mouth projection, as per Taniguchi's formula, which is indicative of alar ligament damage. Notice that in this table, some of these hypermobilities and hypomobilities "match up," in that at one level, hypermobility in one direction is accompanied by hypomobility in the opposite direction.

Level	Flexion	minus	(-Extension)	equals	Total ROM	Normal ROM
C1-C2	10 degrees	minus	(-5 degrees)	equals	15 degrees	<20 degrees
C2-C3	0 degrees	minus	(-13 degrees)	equals	13 degrees	10 degrees
C3-C4	7 degrees	minus	(-12 degrees)	equals	19 degrees	15 degrees
C4-C5	10 degrees	minus	(-11 degrees)	equals	21 degrees	20 degrees
C5-C6	12 degrees	minus	(-0 degrees)	equals	12 degrees	20 degrees
C6-C7	n/a	minus	(-0 degrees)	equals	n/a	17 degrees

Table 1. Angular motion analysis chart

When a segment can move excessively in one direction, and be fixated in the opposite direction, it is a sign of excessive hypersensitivity in a tissue, such as an intervertebral disc lesion or a torn ligament. C5-C6 gets 12° of flexion, and 0° of extension, while C2-C3 gets 13° in extension and 0° in flexion. While flexion at C6-C7 couldn't be measured, the extension at 0° would always be abnormal. Folks, none of this is normal, and furthermore, the patient's pain complaint can be explained by this obvious dysfunction.out disc herniation, it means that the chiropractor has more work to do, as he/she attempts to restore the flexion at C2-C3 and the extension at C5-C6 and C6-C7, with chiropractic manipulative therapy.

No one could look at this chart, and with a straight face tell a patient that everything is moving normally. But in order to tell a patient anything, you have to have this chart. To get this chart, you have to do plain film x-rays or a fluoroscopic motion study and run them through CRMA software so that you can measure the intersegmental motion. Even though numerous IME doctors claim on their reports that no evidence of spinal instability can be found, "motion of the individual spine segments cannot be determined by a physical examination but is determined with flexion and extension roentgenograms," so if they did not review the existing x-rays or order new ones, their examination is incomplete. While the authors of the AMA Guides specifically state that the x-rays have to be plain films and no other, their objections to using a fluoroscopic study are invalid because they use talking points which only apply to old radiographic technology (like dip tanks and acetate film) which have been phased out, and cannot be applied to the DICOM images produced by a hospital-grade fluoroscope. If you're relying on motion palpation, you will be fooled. If anything, after a cervical MRI rules .

Only Superman with his x-ray vision would be capable of looking at a patient and determining if there was any spinal instability within. And our fingers just aren't that good. The only way to determine if spinal instability is present is to do the diagnostic tests which will reveal ligamentous subfailure. Ligament injuries result from forceful dynamic events and it requires a dynamic diagnostic study to reveal them. Two years after a traumatic event, an MRI can reveal evidence of disc injury, but in a personal injury scenario, the defense can always try to make the case that the herniation occurred sometime after the original event, thereby exonerating their client of blame. But the unusual gapping between bones demonstrated on a DMX study is a lesion characteristic of one thing: the great forces generated in a motor vehicle collision. Because of this, in this instance, DMX is superior to cervical MRI in identifying the organic cause of a somatic pain. Calling whiplash associated pain "nonorganic" in any chronic whiplash patient without first ruling out pathology with a DMX examination has no basis in fact.

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Mythology of Personal Injury: Pre-existing Conditions

Bill Gallagher, DC, CMVI

One of the most frustrating dilemmas I run into with my consulting work with attorneys is when a case finally settles for a lower price than it should have and the attorney tells me that was all they could get because it was a *pre-existing condition*. Pre-existing conditions do create inherent weaknesses that make one more susceptible to further injury. They also require less force to cause injury because the structural integrity has already been compromised. The odds are high that your client has one or more pre-existing conditions. Those odds are even higher than the probability that they have a prior motor vehicle collision in their history.

What is important is for the doctor to document any changes in the affected area and how they may have gotten worse with the collision. It is equally important to document the lack of patient complaints or treatment for any pre-existing conditions prior to the collision. When a doctor documents his lack of prior problems and/or the problems beginning with the crash the insurance company will have a more challenging time using pre-existing conditions as an excuse to settle low. To further refute the adjuster's contention that your client's injuries are all pre-existing conditions we will consider both the legal and the medical precedence.

Legal Precedence:
I offer two citations: Mauer v. US and Gilbert Law Summaries. In Mauer v. United States the 2nd District Court ruled,

> "It is settled principle of tort law that when a defendant's wrongful act causes injury, he is fully liable for the resulting damage even though the injured plaintiff had a preexisting condition that made the consequences of the wrongful act more severe."
> "A plaintiff's recovery for damages caused by a defendant's wrongful act may not be proportionately reduced because of a preexisting weakness or susceptibility to injury such as an osteoarthritic condition or a weakness caused by a previous injury."

By the same token, Gilbert Law Summaries book on remedies states, "As long as the chain of causation is proved, any sort of hospital, medical or other therapeutic care is recoverable." However, in the "but" notes that follows this section, it goes on to explain the following: "If the plaintiff has a pre-existing physical condition that would inevitably have worsened, the plaintiff damages must be reduced to take this fact into account."
The challenge for the defense here is to prove inevitability. I have raised this question many times in a room full of doctors and asked what conditions will inevitably worsened. The best answers offered have been cancer, diabetes, or MS. Diabetes we all agree can be controlled by diet and medication. MS we agree also often goes into spontaneous remission. Neither condition must inevitably worsened. Unless your client is dealing with bone cancer there is no inevitable worsening of their spinal pain, without the given trauma. Even with bone cancer there is the likelihood that the crash made the symptoms worse.

Medical Precedence:
The AMA Guides to the Evaluation of Permanent Impairment is the greatest gift ever presented to doctors and attorneys in the personal injury arena. This book establishes the extent of an impairment caused by an injury. To add strength to its value the standards established in The Guides are based on the most current available research.
Probably the most common excuse for denial is arthritic changes noted by radiologists when they reviewing x-rays or MRIs.
This is directly addressed in the 6th Edition, which is the most current print edition and the one used in Arizona. It states, "Common conditions related to degenerative changes in the spine, including abnormalities identified on imaging studies such as annular tears, facet arthropathy, and disk degeneration, do not correlate well with symptoms, clinical findings, or causation analysis and are not ratable according to the *Guides*." It goes on to say, "Developmental anomalies, including spondylolysis, some forms of spondylolisthesis, kyphosis and excessive lordosis or scoliosis are also not ratable."

A radiographic study that shows degenerative changes at one level does not preclude the probability of injury at another level. This is commonly seen with degenerative changes from a previous crash 20 or 30 years ago that the patient has never complained about. This is an asymptomatic condition that created an inherent weakness and the probability of greater injury with less force and next time around. What is also commonly seen is injuries above or below that pre-existing condition that are themselves new injuries.

Scottsdale chiropractor, Bill Gallagher has taught personal injury seminars across the US and in Europe through the American Academy of Motor Vehicle Injuries. He also offers support to attorneys with Phoenix Medical Legal Services. He can be reached at drbillgallagher@yahoo.com or 480-664-6644.

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What's Your Waist to Hip Ratio? Your Health Depends on It

Ronald Grisanti D.C., D.A.B.C.O., DACBN, MS, CFMP

The waist-to-hip ratio (WHR) is a quick measure of fat distribution that may help indicate a person's overall health.

What does a person's waist-to-hip ratio say about their health?

According to the World Health Organization (WHO), having a WHR of **over 1.0** may increase the risk of developing conditions that relate to being **overweight, including heart disease and type 2 diabetes.**

This may be the case even if other measures of being overweight, such as body mass index (BMI) are in normal range.

The following chart shows how the WHO classify the risk of being affected by weight related health conditions according to WHR:.

Health risk	Men	Women
Low	0.95 or lower	0.80 or lower
Moderate	0.96-1.0	0.81-0.85
High	1.0 or higher	0.86 or higher

Impact on Health

Research shows people who are “apple-shaped” are at a greater risk of certain health conditions than those who are “pear-shaped” (when the hips are wider than the upper body).

These health conditions include:

Cardiovascular disease: One study found that abdominal obesity increased the risk of cardiovascular disease and cancer. Another study found the WHR predicted cardiovascular disease more effectively than BMI or waist circumference. A third study found that WHR is a better indicator of risk of mortality from cardiovascular disease than waist circumference alone.

Type 2 diabetes: A 2016 study found that an increased waist circumference was linked to an increased risk of type 2 diabetes.

Fertility: A 2002 study found that women with a WHR of over 0.80 have a lower pregnancy rate than those with a lower WHR, regardless of their BMI.

A 2006 study with almost 15,000 older people (75 years of age or older), it was concluded that **"waist to hip ratio" is more important than how much you weigh**. The researchers looked at the relationship between waist to hip ratio and Body Mass Index (BMI - how we measure weight for height) and how many people died over the next 6 years.

As it turned out, WHR is even more accurate than BMI for predicting the risks of cardiovascular disease and premature death. In other words, it was "**location, location, location" of fat that was most important - not how much you weigh.**

More studies include related to WHR and Health Issues include:

A 2021 study found that WHR is an accurate tool for predicting hypertension.

A 2015 study showed that increased WHR is a better indicator than BMI for predicting complications in trauma patients.

A 2018 study shared that a high WHR was a significant predictor of death in women with heart failure.

A 2016 study found that a high WHR was associated with hypertension and diabetes.

The Health Benefits of Decreasing WHR

A 2020 study found that decreasing WHR by **5 percent significantly** lowered risks of developing chronic kidney disease in people with nonalcoholic fatty liver disease.

So how do you measure your waist to hip ratio? You need a tape measure and a calculator. Here's how you do it:

1: Measure your waist at the smallest point - usually at the naval or just above it
2: Measure you hips at the widest, largest part
3: Divide your waist measurement by your hip measurement (Waist Measurement ÷ Hip Measurement)

The idea here is that your waist should be smaller than your hips. If your waist is bigger than your hips, than you may have too much fat concentrated around the middle part of your body - something known as "**intra-abdominal obesity".**

The concept is quite simple here in that **intra-abdominal fat is bad for your health.**

Conclusion
Measuring a person's WHR is a quick way to get an indication of:

1. Overall health
2. Obesity levels

Risk of weight-related health conditions
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Hypothalamic Involvement in Cluster Headaches

Gregory Katsaros, DC, DAAPM

ABSTRACT

Cluster headaches are a primary headache disorder within the group of trigeminal autonomic cephalgias. While other primary headache types demonstrate issues such as unilateral distribution, photophobia and cranial autonomic symptoms, unique to cluster headaches is its circadian and circannual rhythmicity. Advanced imaging studies have demonstrated regional hypothalamic involvement during active cluster headache attacks which are not demonstrated during interictal cluster headache periods. These imaging findings along with known hypothalamic involvement of the trigeminal autonomic reflex, nociceptive processing, and circadian cycles have helped further the understanding of the important role the hypothalamus plays in cluster headaches.

CLUSTER HEADACHES

Cluster headaches are a primary cephalgia and are one of the five headache types classified within the category of trigeminal autonomic cephalgias (TAC's). Trigeminal autonomic cephalgias are categorized based upon the commonality of pain in the trigeminal distribution and ipsilateral cranial autonomic symptoms **(1,2).** TAC headaches include cluster, paroxysmal hemicrania, hemicrania continua, SUNCT, and SUNA headache types, with cluster headaches being the most common. The typical pain quality of a cluster headache is sharp stabbing pain of severe intensity. The distribution is unilateral and demonstrates about the ipsilateral orbital region. Ipsilateral cranial autonomic symptoms present during cluster headaches may include conjunctival injection, lacrimation, nasal congestion, eyelid edema, forehead/facial sweating, miosis and ptosis. Attack frequencies occur from 1-8 per day with a duration of approximately 30 to 180 minutes each. The pathophysiology involves activation of the trigeminovascular complex and the trigeminal-autonomic reflex which accounts for the unilateral severe headache and prominent ipsilateral cranial autonomic features. In addition to the autonomic features, there is a circadian rhythmicity unique to this condition which is thought to involve the hypothalamus (**3,4**). This circadian rhythmicity often involves patients reporting the attacks occurring at the same time each day. Most patients with cluster headache also have one or two cluster periods annually with each lasting between 1–3 months. Often, this circannual rhythmicity is seen with the headache bouts occurring in the same month of the year. Although the duration of the cluster and remission periods varies among individuals, these periods remain relatively consistent within the same individual (**5**). The pain producing pathway of cluster headaches projects through branches of the trigeminal and upper cervical nerves to the trigeminocervical complex where nociceptive pathways project to higher centers, while a reflex activation of the cranial parasympathetic outflow provides the efferent loop **(6,7,8,9)**. In addition, there are also direct hypothalamic-trigeminal connections, and the hypothalamus is known to have a modulatory role in trigeminovascular nociceptive and autonomic pathways (**10**). The posterior hypothalamus is functionally connected to the ipsilateral trigeminal system and has an inhibitory role. Dysfunction of these projections may induce a permissive state facilitating the development of a cluster headache attack and influencing its duration (**11).** Similar to migraine, this pain producing pathway contributes to elevated calcitonin gene related peptide (CGRP) in cluster headaches. CGRP is a neurotransmitter which is expressed in and released from a variety of areas within the central and peripheral nervous system, including a subset of polymodal primary sensory neurons of the trigeminal ganglion and regions within the hypothalamus. Functional imaging studies utilizing $H_2{}^{15}O$ positron emission tomography (PET) have demonstrated ipsilateral posterior hypothalamic activation during active periods of cluster headaches **(12,13).** While there are some similarities to migraine including the general distribution, trigeminal release of CGRP, and aspects of the pain producing pathway, hypothalamic activation has not been demonstrated during active migraine headaches **(12)**. However, imaging studies have demonstrated hypothalamic activity on the ipsilateral side prior to the onset of a migraine headache, suggesting that while the hypothalamus may not be contributory during an active migraine headache attack, it may be involved in migraine headache generation (**12,13).** In addition to CGRP, pituitary adenylate cyclase activating peptide (PACAP) has been implicated as a possible contributor to migraine and cluster headache symptomatology. PACAP is a neuropeptide which is currently known to exist in two biologically active forms. It shares close anatomical similarities with CGRP in central nervous system regions associated with migraine pathophysiology suggesting that PACAP may play a similar role to that of CGRP in its contribution to migraines. (**14,15**). With its role as a parasympathetic and hypothalamic signaling molecule, PACAP may also potentially be involved in cluster headaches, although more research is necessary.

HYPOTHALAMUS

The hypothalamus can be described as a collection of nuclei which can be divided into three general regions; the periventricular zone nuclei regulating the endocrine system, and the medial and lateral zone nuclei regulating autonomic and somatic behavior. The hypothalamus plays a major role in controlling most of the endocrine and vegetative functions of the body as well as being an essential motor output pathway of the limbic system. With its important roles in autonomic nervous system control, the trigeminal autonomic reflex, nociceptive processing, and circadian rhythmicity, and these aspects being prominently diaplayed during active cluster headache attacks, the hypothalamus appears to actively participate in cluster headaches.

Trigeminal-Autonomic Reflex and Cranial Autonomic Symptoms

The trigeminal autonomic reflex plays a role in primary headache disorders, however it is unclear whether the autonomic symptoms in trigeminal autonomic cephalalgias and migraine are resultant of heightened direct trigeminal discharge, central generators, peripheral stimulation, or any combination of these. The autonomic nervous system is regulated by nuclei of the hypothalamus and limbic system via the nucleus tractus solitarius (NTS). The associated brain stem nuclei and spinal autonomic ganglia receive afferent input which then eventually projects to the hypothalamus and limbic system. The integration of this autonomic system and the trigeminal nerve gives rise to the trigeminal-autonomic reflex. This is thought to be the basis for the cranial autonomic features demonstrated in cluster headaches and other trigeminal autonomic cephalgias **(16, 17, 18).** The pain in cluster headaches may be of either peripheral or central origin. In peripheral causes, the headache attack is thought to originate from activation of the peripheral afferents activating the trigeminal-autonomic reflex, whereas in central causes, the attack is thought to be resultant of, at least in part, direct activation of the posterior hypothalamus. In both causes, there is stimulation of the superior salivatory nucleus from which parasympathetic outflow activates certain ipsilateral cranial autonomic features of cluster headaches including conjunctival injection, tearing, nasal congestion and rhinorrhea. Symptoms such as miosis and ptosis are thought to result from parasympathetic-induced vasodilation of the internal carotid artery and functional impairment of the oculosympathetic fibers. The pain stimuli are carried through projections to the trigeminal-cervical complex, then onto the thalamus, and finally to the cortical sensory pain processing areas (**11).**

Nociceptive Processing

The hypothalamus has direct ascending and descending connections with the dorsal horn and contains several areas associated with the descending modulation of pain and nociceptive processing. The medial preoptic nucleus (MPO) plays an important role in the autonomic response to pain and has projections to the periaqueductal grey (PAG), nucleus raphe magnus (NRM), and the rostroventromedial medulla (RVM), all of which are areas involved in nociceptive processing **(19, 20**). There are areas within the medial and lateral hypothalamus which inhibit the responses of spinal cord neurons to noxious peripheral stimuli (**21, 22**). The paraventricular nucleus (PVN) has antinociceptive effects (**23**), and the anterior hypothalamus is known to suppress the response of wide dynamic range neurons in the dorsal horn (**21, 24**).

Circadian Rhythms

The hypothalamus is involved in the circadian rhythmicity of essential physiologic functions including the sleep-wake cycle **(25**). The suprachiasmatic nucleus (SCN) is recognized as the central control center of biological rhythmicity and has influence over other regions of the brain. It also has direct projections to the lateral hypothalamic, PVN, preoptic, and supraoptic nuclei **(26,27)**.

DISCUSSION

Cluster headaches are a debilitating primary headache disorder and the most common of the trigeminal autonomic cephalalgias. There are many commonalities between migraine and cluster headaches, and an understanding of these differences is essential for proper diagnosis and treatment. The cranial autonomic symptoms and circadian and circannual rhythmicity seen in cluster headaches are not aspects of migraine headaches. In addition, cluster headaches are not known to be triggered. While there are a variety of exogenous triggers which can initiate a migraine headache attack, no such triggers are known to exist for cluster headaches. Cluster headaches are unilaterally distributed, demonstrate with cranial autonomic symptoms, and display a circadian rhythmicity. Functional imaging studies have demonstrated ipsilateral posterior hypothalamic activation during active cluster headache attacks which are not evident during interictal cluster headache periods. The hypothalamus has both direct and indirect involvement with the trigeminal-autonomic reflex, cranial autonomic activities, and circadian rhythmicity. During a cluster headache attack, there is activation of the trigeminal-autonomic reflex with the release of CGRP. CGRP and CGRP receptors are widely distributed throughout the central and peripheral nervous system, including the hypothalamus. Release of CGRP from perivascular nerve endings causes neurogenic vasodilatation, and increased levels of CGRP have demonstrated to be increased in the cranial circulation during cluster headache attacks. Whether through direct activation in central origination or indirect activation through peripheral afferent stimulation, the hypothalamus appears to play an important role in cluster headaches.

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About the Author

Gregory Katsaros, DC, DAAPM is a Diplomate in Pain Management and a member of the American College of Nuclear Medicine and the International Headache Society. He is the owner of Integrative Pain Management in Tempe, AZ (www.azheadaches.com) and co-owner of Aristotle Continuing Education (www.AristotleCE.com). The focus of his practice is on headaches and nonsurgical musculoskeletal pain.

Your Board of Chiropractic Examiners

Bill Gallagher, DC

The Arizona Board of Chiropractic Examiners is a state governmental agency. As a governmental agency, the purpose of professional state boards is to protect the public from the professionals. That would be better stated as to protect the public from the members of the profession who are acting unprofessionally.

Those of us who are not new to practicing in Arizona may recall how stringent our board was in the past. In fact, the Arizona Board of Chiropractic Examiners earned the reputation of being the most stringent professional board in the country. That has changed.

Probation and fines along with additional continuing education hours were to be expected if you were called before the board. For the times I have attended these meetings and for the problems that I have seen addressed I still see probations, fines, additional CE hours, and even suspensions and revocations of licenses.

In each case I agreed with those consequences and so did the doctor who was on the carpet. I expect that each of them has learned by their mistakes.

Here in Arizona one of the requirements for a new licensee is to attend a state board meeting. With COVID, the option of attending virtually seems to be the first choice for most new doctors. Having attended several of these meetings in the past year I can tell you this is an excellent rule to have in place and I would advise anyone to find the time to attend one of these meetings in person.

Considering the amount of time that they devote to address all the complaints, our board members get paid less than minimum wage. Clearly, they don't do it for the money. It is obvious that they do it because of their dedication to our profession.

I have seen such a variety of cases in the last year ranging from drug abuse to sexual abuse and more than you could imagine in between. I have also seen that when the doctor is on the carpet and must answer for their behavior there is one common question, did you put that in the file? Some of the variations might be did you write that down or did you include a copy of that letter in the patient's file?

Bottom line every complaint that I have seen before our state board has come down to documentation. Obviously, this is one of the reasons we have a biannual requirement of two hours of continuing education on documentation and record keeping.

What I have seen there has caused me to make some changes in my continuing ed classes on documentation. I have also learned so much about what kind of mistakes we make in our practices.

Record keeping it is a major concern and as I mentioned comes up with every case. This includes when a patient complains of substandard care which seems justified when the board gets a copy of the file and finds that there was substandard documentation.

Drug abuse and sexual abuse cases are regularly addressed by our board. For the largest drug free profession there is a certain level of irony when I hear a case of drug abuse before the board. Sexual abuse in the past was usually about a male doctor overstepping the bounds with a female patient. Today when Harry met Sally may also involve when Sally met Harry or Sally met another Sally.

For those cases that have gone before the board every single doctor was asked, “did you put that in the patient’s file?”

With our chiropractic physicians license comes certain privileges and certain responsibilities. Simply by being able to call ourselves professionals we have raised the bar and must hold ourselves and each other to I higher standard.

New techniques in PT disjoint income tax laws as we know it

Craig Wolman, CPA

How can a new tax save you money? If you're thinking this is some kind of joke, we're not here to crack you up as taxes are no laughing matter. In fact, the mere thought that a tax can actually save people money, is *sacrum*legious!

In joining the ranks of approximately 30 other States, for tax years beginning January 1, 2022, Arizona has enacted the Pass-Through Entity Tax (PTET) which applies only to taxpayers whose business is treated as a partnership or S-corporation for federal income tax purposes. PTET is not mandatory as it is considered an elective procedure.

So why would anyone voluntarily choose to pay this tax?
Here's a little backstory:

The 2017 Tax Cuts and Job Act (TCJA) limited the amount of allowable itemized deductions of State and Local taxes (SALT) to $10,000 For high-income taxpayers, this amount is easily eclipsed when factoring in state income taxes, property taxes (especially with multiple homes) and personal property taxes (i.e. annual car registrations).

The IRS in 2020 announced that partnerships and S-corporations may deduct SALT payments of its owners at the entity level when calculating taxable income or loss, thus lowering the federal income tax assessed on the owners of PTEs (i.e. individuals, shareholders, and / or LLCs)

By allowing PTE owners to deduct more than the Federal limit of $10,000, the government encourages owners of pass-through entities to increase their annual SALT intake!

States seem to care more about the heart-health of its taxpayers. Although PTET **reduces** Federal taxable income, the amount of PTET deducted is added back so that State income tax is calculated on the entities' actual operating income. However, the amount of PTET paid is taken as a **credit** on the PTE owners' tax return to **lower** their State income tax liability.

PTET, for Arizona is calculated at a rate of 2.98%, equaling the maximum flat tax rate (also new for 2022).

In a nutshell, PTE owners receive an increased Federal deduction for paying the same amount of State taxes as they are subjected to pay; just by manipulating the source of the tax payment from individual bank accounts to the business bank account.

The TCJA expires in 2025. After that, it's anybody's guess what the laws will be like but in the meantime, we at MillerWolman CPAs can work with you on PTET and other efficient tax strategies to help you avoid the #1 reason chiropractors get in trouble with the IRS……. back taxes! .

Had a hunch you've heard that before but if not, we stand corrected.

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Radiology Case—Brief Report

Sacral-Coccygeal Chordoma Mimicking as Low Back Pain

Gregory Katsaros, DC, DAAPM

T2 MRI demonstrating large chordoma

CT Lumbar spine reformatted sagittal image demonstrating chordoma and bony erosion

A 54 year old patient was seen for a primary complaint of low back pain. The patient had chronic low back pain which had increased over the past 6 to 8 months. About three months prior, the patient was seen at their primary care physician's office and was prescribed analgesics, lumbar spine x-rays, and physical therapy. The patient chose not to have the x-rays performed. After the course of physical therapy and without much improvement the patient sought chiropractic care. The patient was evaluated and referred for an MRI of the lumbar spine which demonstrated a large sacral-coccygeal chordoma. A subsequent CT of the lumbar spine was then performed.

A chordoma is a rare cancerous tumor occurring along the spine. It is derived from remnants of the notochord and is a type of sarcoma. Approximately 50% of chordomas are found within the sacrococcygeal region and 35% within the spheno-occipital region, with the remainder seen throughout the spine. A chordoma typically erodes the bone from which it arises. In this case, the sacrococcygeal chordoma formed a well defined mass that bulged from the anterior surface of sacrum and eroded the patient's S3, S4, and S5 segments and also the coccyx. The chordoma also compressed the bladder.

Most chordomas grow slowly. Pain is usually the first symptoms in patients with a sacrococcygeal chordoma. Occasionally extension of the tumor into the pelvis can lead to symptoms such bowel and bladder dysfunction or other symptoms secondary to nerve root compression. Spheno-occipital chordomas usually demonstrate with headaches and cranial nerve dysfunction. Thoracic and lumbar region chordomas typically present with non specific musculoskeletal pain.

Treatment is by surgical resection followed by high dose radiation therapy. Due to the high rate of recurrence after treatment, MRI follow up with and without contrast should be performed every 6 months for the first 5 years followed by yearly imaging.

In Memoriam

We can see further and dream bigger because of the wisdom of those who came before us

LELIA IRENE ESCH SCHLABACH, DC

Lelia Irene Esch Schlabach DC, 96, went to be with the Lord on Wednesday, October 26, 2022, in Phoenix, Arizona. Born February 11, 1926, in Pigeon, Michigan, she was the daughter of Henry and Barbara Esch. She married Abe J. Schlabach in 1947 after graduating from Palmer College of Chiropractic and moving to Arizona. Lelia and Abe joined her father's practice at North Phoenix Chiropractic in 1967. She was still adjusting and caring for patients until age 91.

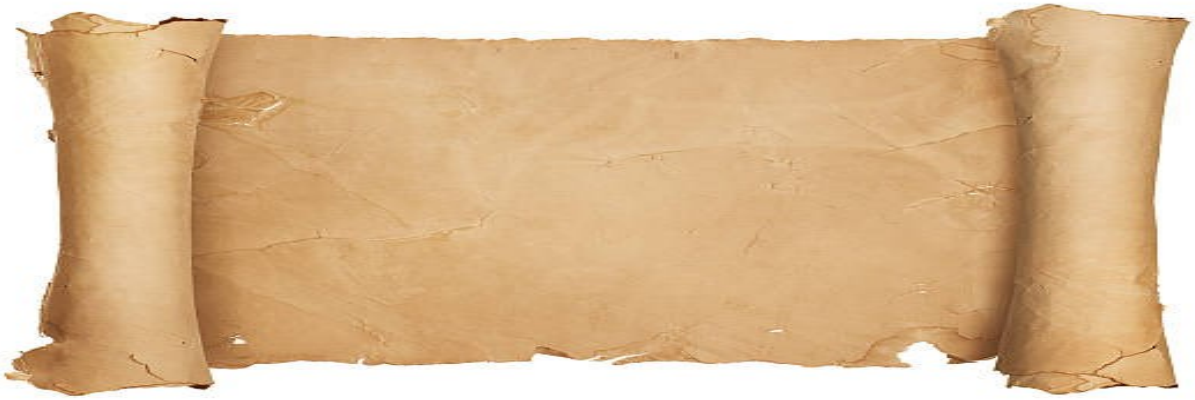
She was a member of Sunnyslope Mennonite Church from its inception in 1947, serving as teacher, mentor and elder in the church. Her philosophy on life, was to dedicate everything in life to our Lord Jesus Christ. She prayed for family, friends, patients, and strangers. She would openly share the love of the Lord with anyone, as attested to by her patients.

To say she was very involved in the advancement of her profession is an understatement. She served all chairs of the Arizona State Chiropractic Association, ending as president. She was appointed chairman of the Arizona Board of Examiners and served 3 years. She was the first women on Board of Trustees of Palmer College of Chiropractic and a founding member of the Board of Trustees for Palmer West (San Jose) and served 9 years. She served on multiple committees for both Palmer colleges and was a member of the Sigma Phi Chi Sorority.

She often said her 4th job was serving as a board member. She served on the board of directors of Friendship Retirement Corporation (Glencroft) from 1980 to 1995. Then later, on the board of directors of the Friendship Foundation. She received the 2004 Trustee of the Year award from the AZ Association of Housing the Aging for her work with Glencroft. Her last board of directors' position was with the Sunnyslope Historical Society, where she enjoyed being involved in the preservation of the local history.

She is survived by daughter, Gretchen Krehbiel (Kenneth Krehbiel) and granddaughters, Jessica Gardner (Mason Gardner) and Alyssa Krehbiel. She was preceded in death by son, John Schlabach and husband, Abe J. Schlabach D.C.

Colossians 3:23 Whatever you do, work at it with all your heart, as working for the Lord..



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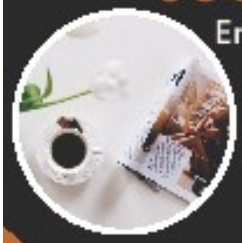

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