

# What's Normal: The Cervical Spine

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# What's Normal? The Cervical Spine

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Left to Right: James B. Talmage, MD; Robert B. Snyder, MD; J. Wills Oglesby, MD

#### INTRODUCTION

In prior issues of the AdMIRable Review, we have examined "What's Normal?" in the lumbar spine (Winter 2024), knee (Summer 2024), and shoulder (Summer 2023). In this issue we will review "What's Normal?" in the cervical spine.

The most basic imaging study of the cervical spine is plain, old-fashioned, radiographs. These are euphemistically frequently called "x-rays" although x-rays are invisible electromagnetic waves (like photons of light or the microwaves in your kitchen appliance). Plain radiographs can detect significant fractures, although CT scan is better at detecting less obvious fractures. Key findings to correlate with symptoms like disc herniation and foraminal stenosis are basically not recognizable on radiographs.

A common misperception among attorneys, physicians, and even radiologists is that loss of the normal sagittal plane curve (lordosis) in the cervical spine is evidence of muscle spasm in the posterior cervical paraspinal muscles and thus proof of acute injury. The "normal" cervical spine posture when viewed from the side (or on a sagittal image) is a curve with the concavity facing posteriorly – or "apex anterior."

This is a misconception (false statement). Brooks (2015) in the AMA Guides Newsletter reviewed the poor science behind this false concept, its origin in the 1930s and 1940s, and its erroneous adoption by textbooks. The Systematic Review and Meta-Analysis by Guo (2018) combined studies of over 15,000 asymptomatic normal adults. 64% had cervical lordosis on imaging, meaning just over one-third of normal asymptomatic adults had "loss of lordosis" on cervical imaging. In addition, the prevalence of this findings was the same combining studies of asymptomatic adults and comparing that to the prevalence in patients presenting with neck related symptoms. A study of 488 Japanese patients (Matsumoto 1998) with acute "whiplash" (neck related symptoms after motor vehicle crash) compared to 495 normal asymptomatic volunteers found the same prevalence of "loss of lordosis" (kyphosis) on imaging in both groups at various ages.

Matsumoto recruited 497 initially asymptomatic volunteers in a study we will look at in detail later is this article. He and Okada (2009) reported that in 10-year prospective follow up some of the originally asymptomatic volunteers had developed neck, shoulder, or upper limb symptoms, but whether they had, or did not have, lordosis on the initial imaging had no correlation with whether they developed symptoms over the next 10 years.

The Brooks (2015) article used the interesting analogy of a bow used to shoot arrows. The bow has a curve ("lordosis") and a string. If the string is shortened (as in "spasm" of the posterior spinal muscles) the bow curvature increases (lordosis would increase). Thus, the frequent finding in medical records of "spasm" by palpation of the posterior cervical muscles alleged to decrease the length of the string and yet **decrease or eliminate** the lordosis is false.

This frequent finding in a medical record of "loss of lordosis" as an indication of palpable "spasm," acute injury, or symptoms is not accurate and should be ignored by the reader of medical records.

#### RELIABILITY OF IMAGING

Cervical spine imaging is used for supporting the diagnoses of cervical radiculopathy (causing upper-limb pain, numbness, or weakness) and/or cervical myelopathy (spinal cord compression causing upper-limb, lower-limb, or bowel/bladder symptoms). However, these diagnoses and the imaging must be accompanied by appropriate symptoms and physical examination findings.

Radiculopathy can be caused by disc herniation, foraminal stenosis, lateral recess stenosis, or lateral spinal cord compression. Myelopathy is typically caused by central or lateral spinal cord compression. These clinical syndromes are most commonly investigated by MRI (Magnetic Resonance Imaging).

The lateral recess is rarely mentioned in cervical MRI reports, so stenosis in the foramina (bony canals) through which nerve roots exit and cervical spinal cord compression in the central canal are the two finding on reports which are used to correlate with symptoms and clinical signs of radiculopathy or myelopathy. Reviewing the published literature on reliability (can two radiologists agree on the presence or absence of disc herniation or nerve root compression in the cervical spine) or the analysis of the reports of radiologists on actual cases is hindered by the fact that there are multiple published systems (Hutchins 2022) to evaluate potential cord and/or nerve root compression. Unfortunately, radiology reports on cervical MRIs and attending surgeon's review of the MRI images rarely state which system is being used to determine the presence of and the severity of herniation, stenosis, or neural element compression.

In the knee, shoulder, and lumbar spine reliability studies, the MRIs of symptomatic patients and asymptomatic people are frequently mixed together for interpretation. In the cervical spine most studies on MRI reliability use a smaller number of patients, all of whom have the same or similar pathology. This high prevalence makes the agreement based on chance very high, and thus the kappa values very low, compared to the studies we examined in other body parts. Thus, kappa values are not instructive for most of the cervical spine MRI reliability literature. This literature (Braga-Baiak 2008, Lee 2016, Lee 2020, Seo 2023) can be summarized as inter-observer agreement on the presence or absence of central canal stenosis or foraminal stenosis is 80-90%. This may

explain some of the not infrequent disagreements between the interpretation of radiologists and surgeons viewing the same imaging.

One of the few studies (Okada 2009) with two radiologists looking at both symptomatic and asymptomatic individuals found an interobserver kappa of 0.63 for spinal cord and/or dura compression, and a kappa of 0.88 for foraminal stenosis.

Matsumoto's 1998 study mixed 0.5 Tesla and 1.5 Tesla MRI (done at multiple institutions). The higher the Tesla, the stronger the magnet used, and this is roughly equivalent to having more pixels per inch in your TV or computer monitor. The higher the MRI unit's magnet strength in Tesla units, the more detailed the images. If a 1.5 Tesla MRI unit ages out of service in the U.S. today, it will usually be replaced by a new 3 Tesla unit. A study of 102 asymptomatic volunteers with a mean age of 46 using 3 Telsa MRI (Lee TH 2013) found a kappa of 0.97 for disc herniation.

At times the BWC Medical Directors review cases on appeal with very different interpretations of the same MRI images by the attending surgeon and the radiologist. We, at times, state we cannot make a clear determination in such cases. It is not surgically possible to "unpinch" (decompress) a nerve root or spinal cord that is NOT "pinched." We often point out that the discrepancy in interpretation cannot be resolved in our review and suggest the surgeon and the radiologist meet, review the imaging, and issue a joint addendum. Sadly, we are still waiting for this to happen, even though both doctors would likely learn from the interaction.

The "Lee" system of determining stenosis (Seo 2023) of the central canal and neural foramina appears to have the best reliability. A review of that system is published as "open access" so it can be downloaded without cost by anyone with internet access.



The Lee grading system for cervical central canal stenosis. **A**. Schematic drawing of normal cervical central canal and the corresponding T2-weighted sagittal magnetic resonance image show cervical spine without central canal compromise. There is no obliteration of cerebrospinal fluid space around the spinal cord. Deformity or signal alteration of the spinal cord is not observed. **B**. Schematic drawing of mild cervical central canal stenosis and the corresponding T2-weighted sagittal magnetic resonance image show mild central canal stenosis with over 50% obliteration of cerebrospinal fluid space at the C5/6 disc level, without deformity or signal alteration of the spinal cord. **C**. Schematic drawing of moderate cervical central canal stenosis and the corresponding T2-weighted sagittal magnetic resonance image show moderate central canal stenosis. Ventral aspect of the spinal cord is compressed and deformed. There is no signal change of the spinal cord. **D**. Schematic drawing of severe cervical central canal stenosis and the corresponding T2-weighted

sagittal magnetic resonance image show severe central canal stenosis and the corresponding 12 weighted narrow at the C5/6 disc level with severe deformity of the spinal cord. There is focal medullary hyperintensity at the compressed segment.



The Lee grading system for cervical neural foraminal stenosis. **A**. Schematic drawing of normal cervical neural foramen and the corresponding T2-weighted axial magnetic resonance image show normal bilateral C5/6 neural foraminal without nerve root compromise. **B**. Schematic drawing of non-severe neural foraminal stenosis and the corresponding T2-weighted axial magnetic resonance image show non-severe neural foraminal stenosis of the left C5/6 neural foramen. The narrowest width of the left side neural foramen is less than (but more than 50% of) the extraforaminal nerve root width. **C**. Schematic drawing of severe neural foraminal stenosis of the right C5/6 neural foramen. The narrowest width of the neural foramen is less than 50% of the extraforaminal nerve root width.

#### WHAT'S NORMAL?

The best and most frequently quoted study on what's normal in asymptomatic adults is the prospective study started in 1993 (Matsumoto 1998). 497 Asymptomatic adult volunteers had an MRI of the cervical spine. Manual laborers were a small percentage of the participants, and this study may underestimate the prevalence of findings in manual laborers. The people studied had no current neck, shoulder, or upper limb symptoms, and no history of disease or trauma to the neck or brain. Compression of the dura and/or spinal cord from a disc protrusion was present in 50% of volunteers, while foraminal stenosis was uncommon at only 0.4%.

Ten years later (Okada 2009) 223 of the 497 volunteers were available for restudy by MRI. Aging (degenerative) changes progressed in 81% of the cases. Compression of the spinal cord and/or dura increased from 50% to 82 % of the individuals, while foraminal stenosis increased from 0.4% to 8%. While these individuals were initially asymptomatic, neck pain developed in 10%, shoulder stiffness in 30%, and upper limb numbness in 4%. No individual had neurologic exam findings related to the cervical spine, and no individual had neck surgery. None of the progression of the five categories of MRI aging (degenerative) changes correlated with sex, smoking, alcohol, sports, or BMI. The older individuals were at the beginning of the study, the more progression of aging changes occurred over the next 10 years. The individuals who developed symptoms had more progression of aging changes in general, and older individuals were more likely to have developed symptoms. Age appears to have been the dominant factor in both MRI worsening (progression) and symptom onset.

Twenty years after the original MRI study (Daimon 2018), 193 of the 497 persons were re-examined and re-imaged. This follow up rate of "only" 39% is actually good for studies on 20-year outcomes. The mean age for these individuals was now 57. Aging or degenerative changes had progressed for 95% of these people. The progression (worsening) of findings was 83% for posterior disc protrusion, 86% for spinal cord/dura compression, and 19% for foraminal stenosis. Foraminal stenosis was the only MRI finding on the recent MRI that correlated with symptoms, and only with arm pain (perhaps surprisingly not arm numbness). None of the other four categories of aging changes (including posterior disc protrusion) correlated with stiff shoulders, neck pain, headache, tinnitus, or upper limb numbness. A posterior disc protrusion (herniation) was present in over 50% of those now in their 30s, over 80% of those now in the 40-59 age group, and in over 90% of those now older than 60 years old.

A study of 102 asymptomatic volunteers with a mean age of 46 using 3 Telsa MRI (Lee TH 2013) found a higher prevalence of all degenerative findings than in other published studies, with 81% having some degree of a disc "pathology" (bulging in 67%, protrusion in 29%, extrusion in 23%). As in all studies, the C5-6 disc space had the highest prevalence of findings.

Cervical spine MRI does not predict future outcomes. A systematic review of 12 published prospective cohort studies was hindered by lack of standardized definitions of MRI findings, dissimilar populations studied, and dissimilar outcomes assessed (Hill 2018) but concluded that there was no consistent association between MRI findings and future clinical outcomes.

A study of 18-22 year-old Italians applying for admission to the Italian Air Force Academy (Romeo 2018) studied each applicant by 3 Tesla MRI due to the very high forces experienced by the neck of fighter pilots. Out of 350 asymptomatic applicants 23% had a "normal" MRI. Disc bulging was present in 49%, disc protrusions (a type of herniation) was present in 18%, and disc extrusion (a higher-grade herniation) in 8%. The authors concluded that degenerative/aging changes occur much earlier in life than generally appreciated.

The prevalence of MRI findings that might explain symptoms highlights the problem of "base rate neglect" (Janssen 2021) in which the treating physician has a patient with symptoms, gets an MRI, and then assumes the findings on MRI must explain the patient's symptoms, never considering that the MRI finding might be asymptomatic.

Thus, the correlation between the patient's history and physical exam is critical. This takes time with the patient in the examination room-which the current U.S. model of health care does not appreciate. Neuropathic pain from nerve root compression produces pain and frequently sensory loss in the exact same location at different times (Finnerup 2016). Most medical records do not describe the location of, and boundaries of, pain and/or sensory loss well enough to permit a reviewer to verify the symptoms or sensory loss are constant in location, and thus potentially correlate with an MRI finding. If physicians used a human figure "pain drawing" and the physician drew in and labelled the pain and sensory loss locations, reviewers would have a much easier time interpreting medical records.

Complicating such assessments, shoulder conditions (Kouri 2018, Katsuura 2020, Brusalis 2024) and peripheral nerve entrapments (e.g. carpal tunnel syndrome, ulnar nerve neuropathy at the elbow) must be differentiated from cervical radiculopathy or upper-limb myelopathy.

Further complicating such assessments, the dermatomes of sensory change, and the myotomes of muscle weakness are frequently not as diagramed in medical textbooks. The "nerve roots" that are frequently compressed leave the spinal cord as multiple "rootlets," that further distally fuse to form a nerve root. A more central compression of "the nerve root" may compress some of, but not all of, the rootlets.



Source: https://www.orthobullets.com/spine/2004/spinal-cord-anatomy

This may explain why patients with a particular nerve root compression syndrome often present with a variety of symptoms and neurologic findings.

In addition, textbook descriptions of the dermatomes of sensation served by a single cervical nerve root and the myotomes of muscle function served by a single cervical nerve root assume everyone has the same anatomy in the cervical spine and brachial plexus. Anatomic dissections of cadavers have confirmed the "textbook" or "Netter diagram" of the brachial plexus is present in only 37-77% of individuals (McAnany 2019). Thus, many cervical radiculopathy patients present with symptoms and neurologic findings that do not fit with (not identical to) the textbook descriptions. Some present with just neck and trapezius pain (posterior shoulder) while only some have the expected symptoms in the arm. Intradural connections between the rootlets of cervical nerve roots and "pre-fixed" and "post-fixed" brachial plexus congenital anomalies help to explain this clinical problem.

The textbook brachial plexus is composed of C5 through T1 nerve roots. In the "prefixed" plexus, there is a substantial contribution of C4 and minimal or no contribution of T1 to the plexus. In a "post-fixed" plexus there is minimal or no contribution of C5 and a substantial contribution of T2 to the plexus. Thus, the location of symptoms and neurologic findings are generally constant for a particular person but vary considerably from person to person with the same apparent nerve root compression syndrome by imaging (Ross 2016, Rainville 2017, McAnany 2019, Riew 2019).

The conclusion of these publications is the consistency of symptom location and physical exam neurologic findings on different dates can help confirm that the imaging finding of spinal cord or nerve root compression is the likely cause of the symptoms and neurologic findings, and they help confirm that the imaging finding is not an asymptomatic normal aging change. However, this consistency cannot definitively localize the exact site of neurologic compression. Operative intervention as a treatment option can be determined by consistency, but the exact site to be addressed surgically is determined by imaging.

The famous bank robber Willie Sutton, when asked "why" he robs banks, is reputed to have replied, "That's where the money is." The symptoms and exam and their consistency over time help determine WHO to offer surgery to, but the imaging determines WHERE to operate. Correlation between the consistent pattern of symptoms and signs and the imaging should be discussed in detail in the medical records.

One last thought to explore is the urgency of surgical treatment for spinal cord compression. The finding of "myelomalacia" (increased T2 signal within the compressed spinal cord) is frequently stated as an indication for surgery. It may represent spinal cord scarring, but myelomalacia on MRI does not predict physical exam finding of myelopathy (Nemani 2014) or predict the outcome of surgical decompression of the spinal cord (Shakil 2025).

While serious spinal cord injury after minor trauma is known to occur with pre-existing ossification of the posterior spinal ligament, ankylosing spondylitis, and instability at the Occiput-C2 level, there are very few reports of serious spinal cord injury after minor trauma in those with known pre-existing spinal cord compression from common degenerative changes (Bednarik 2011, Rhee 2013, Chang 2015). This is despite the surprising frequency of spinal cord compression in asymptomatic adults in the studies noted above. High violence activity is usually prohibited for these individuals – contact sports, subduing suspects in law enforcement, etc.

#### CONCLUSION

Hopefully this review of "What's Normal?" will help the reader understand medical records that include imaging of the cervical spine, and the challenges of analyzing medical records.

Jump to *references* for this article.



# The Pain Conundrum in Injured Workers\*

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It is the nature of workers' compensation injuries that they almost always involve pain, and many workers endure pain for the remainder of their lives. Successful treatment of that pain is complicated by several factors: some medical providers who look for easy or quick answers and sometimes the patients themselves complicate their treatment. The following scenarios provide examples of how the treatment of pain can go off track and how it can be different.

#### SCENARIO #1

Ms. Smith is a sixty-year-old woman who has suffered with chronic pain after a simple lifting injury 20 years ago. The initial MRI revealed no acute structural pathology (only degenerative bulging discs and annular tears) and indicated she had no more than a soft tissue injury. She never had surgery after her injury but was sent to pain management. For two decades, she received multiple interventional procedures (trigger point injections, epidural injections, sacroiliac joint injections, and nerve ablations), and several rounds of physical therapy. She was prescribed chronic opioids early on (despite all these interventional procedures). She is now on Fentanyl patches and Hydrocodone, Neurontin, Flexeril, and Ambien through a pain clinic, and Xanax and Cymbalta from her primary physician. She is overweight, has chronic depression and anxiety, is diabetic, and has untreated sleep apnea. Despite all this medication, she reports her pain level is eight out of ten, and she spends most of her days in her recliner. When her pain clinic closed, she was "miserable" because she felt she could not manage life without her pain pills. Now she has gone to a new pain management specialist.

What can the new doctor do to manage this type of complicated case? Does the doctor just continue the "status quo" medications Ms. Smith wants even though he knows she is not doing well on them? Does he continue the drugs she is on when all the medication treatment "guidelines" question continued opioid treatment because the risks far outweigh benefits? Or does the doctor do what is best for the patient in the long run by following the updated science and guidelines and provide more proven treatment, which has a better chance to improve Ms. Smith's quality of life and function, as well as provide her safer treatment?

#### SCENARIO #2

About 10 years ago, a heart-tugging article appeared in a paper from the wife of a chronic pain patient who had committed suicide. Her husband had been on opioids for years when suddenly his pain clinic reduced his prescription dosages from a high level of opioids to much lower dosages. His pain increased significantly as his ability to function decreased. His wife said that her husband felt "there was no remaining hope" and that life was not worth living so he ended it. His wife blamed his death on "all the new state laws" that caused his doctors to stop prescribing opioids for chronic pain. She felt the law change was inhumane.

How could this man's treatment been different to avoid this tragic outcome?

There were definite risks to the high dose opioids he had been taking. Pain management is much more than just writing opioids. His initial treating physicians could have initiated him on a safer level of opioids. They could have carefully explained the reasons behind national and state guidelines on opioid dosages and the dangers of using opioids for treating chronic pain. This man could have been encouraged to consider "alternative treatments" that were available and provided potential solutions that could improve his chronic pain, ability to function, and quality of life. In other cases, such alternatives have resulted in the reduction and even elimination of opioid usage.

Why were these patients in these scenarios and scores of others like them not given the pain management treatment that could afford them the best chance for a life worth living?

Some possibilities are:

#### PATIENTS' UNREALISTIC ASSUMPTIONS

When patients come to a new doctor they often "just assume" that the new doctor will continue the same regimen of drugs they had been on for a long time. When the doctor does not give them the drugs or dosages they were previously given, they ask "Why then did they send me to a pain specialist if you're not going to write my pain pills?"

Or a patient has a surgery followed by a prolonged period of taking opioids (often high dosages), and then after 3-6 months, the surgeon tells them "I can't write the prescriptions anymore because of the new laws, but the pain specialist will."

Now, the new pain specialist must *convince the patient* of the risks of opioids, especially high dosages which include:

- respiratory depression, which can especially occur when taken in conjunction with other centrally acting agents that are sedating (muscle relaxers, anxiety and depression medications, sleeping pills, and nerve pain medications).
- potential addiction and physical dependency as tolerance increases for the prescribed drugs.
- opioid hyperalgesia (a paradoxical condition when the patient's pain taking opioids over time can increase rather than decrease).
- depression caused by these medications (they can be "downers" emotionally).
- sexual dysfunction.
- cognitive dysfunction.
- and many others.

The treating provider MUST gain the TRUST of the patient to try the recommended treatments. Then the pain specialist must attempt a *slow* weaning of the opioids while installing **alternative treatments**. There are several types of non-opioid treatment in the literature to successfully manage chronic pain, which are supported by "science." Some examples are:

- Non-opioid medications there are anti-depressants that can work on both musculoskeletal pain as well as nerve pain, AND they work on the depression and anxiety that is so often experienced by chronic pain patients.
- Cognitive Behavioral Therapy typically a psychologist works with patients to
  reteach them to accept the pain, not give in to it, and to learn how to "turn the
  volume down on the pain" so they can become more functional. The psychologist
  works on "fear-avoidance" and "catastrophizing" tendencies that so many longterm opioid using patients have.
- Acupuncture has been shown to be beneficial in many types of pain and is much safer than taking so many pills.
- Yoga / "Mindfulness" treatments are combination of exercise, stretching, and meditation and are safe treatments.
- Progressive exercises and nutritional management with weight loss so many patients do not understand that movement will not harm them, but NOT moving will. Anti-inflammatory diets have also been shown to have a beneficial effect on chronic pain.
- Electrical modalities TENS units/H-wave units are simple modalities that many times can provide excellent pain control and avoid the use of harmful medications.

Such alternative treatments have a much better chance of long-term success in treating these centralized chronic pain syndromes.



THE PATIENT'S LACK OF UNDERSTANDING THAT FALSE-POSITIVE FINDINGS ON IMAGING STUDIES CAN LEAD TO MISINTERPRETING THE "PAIN GENERATOR"

This misunderstanding about what exactly causes the pain can consequently lead to a patient's demand for inappropriate treatment of their pain.

In the first scenario above, the findings on the imaging studies were probably not caused by her injury, which means, more importantly, they were probably not causing her symptoms. In cases like this, the pain specialist should explain that the findings are sometimes "normal for your age" and are not caused by the injury, much less causing the pain the patient is having. Patients often state, "I have read my report and it states I have a torn disc (annular fissure/tear) or a bulging disc." Patients may assume the findings were caused by the work injury. If they had never had MRI's or x-rays of their back, they would have been unaware of existing degenerative back issues. They become confused and often distrustful because the doctors "all say different things" in terms of explaining what is causing their pain. The doctor should explain to the patient that the literature clearly documents the incidence of false positives/age related changes, such as disc bulges and even herniations that do not correlate with patients work injuries. Doctors may not feel it is worth the conflict they will experience when they try to discuss these issues with their patients. Sometimes they are not aware of the literature indicating this information about "false positive imaging study findings."

In Ms. Smith's scenario, she had a minimal mechanism of injury, no findings on imaging studies of "acute injury" structurally, yet her life morphed into years of treatments. In the end, Ms. Smith was totally miserable and non-functional. The pain generator had become "centralized" and could no longer be identified as a structural musculoskeletal or nerve injury. Consequently, opioids and injections and even potential surgery would not be successful. The pain was driven by the opioids themselves (opioid hyperalgesia), which can happen in cases of long-term use like Ms. Smith's.

### AN UNWILLINGNESS OF THE TREATING PROVIDERS TO LEARN FROM EVIDENCE-BASED LITERATURE AND INCORPORATE THAT LEARNING INTO THEIR PRACTICE

This can lead to inappropriate treatment of their patients' pain.

Medical providers must be educated and convinced that opioids usually do not have long-term success but do have significant risks. It is much easier and less stressful for providers (and their patients) to just continue the opioids and spend minimal time with their patients. There are even documented cases of doctors writing extremely high dosages of opioids without considering other alternatives to pain management and being resistant to any change – even when presented with "the science" indicating the danger of opioids. Patients can be very insistent about getting high dosage opioids, which also makes it difficult for providers to refuse their demands.

Also, there is the financial disincentive for spending the extensive time required to explain the problems with opioids to their patient and to discuss other options. The resistance of the medical provider to "change course" can be attributed not only to ignorance of the updated literature, but also arrogance. Some doctors do not like being told they are wrong. Overcoming this "mindset" of some medical providers can be difficult. Compounding the problem, traditional teaching in medical schools has not placed an emphasis on alternative treatments. Sadly, this mindset can have deadly consequences for patients.

#### IN SUMMARY

The appropriate management of chronic pain patients, in workers' compensation especially, is usually very "complicated." However, if providers spend the appropriate time in educating and listening to patients, try to successfully determine the true pain generators, study the evidence-based medicine literature, and consider alternative treatments to opioids, successful outcomes can often be achieved for patients with far less chance of harm in the long run. Success for patients in such cases makes dealing with all the complications worth it. There is no need for patients to believe they will be condemned to lives of misery without opioids. And yes, the sad outcome of the gentleman in the second scenario did not have to happen. He did not have to "lose hope." With appropriate care, he could have gone on to have a good quality of life and not suffer in such pain forever when his opioids were decreased. Our task is to prevent his tragedy from happening to others with workers' compensation injuries.

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# A primer on the legal approach to aggravations of preexisting conditions

By Jane Salem, staff attorney, Nashville



Jane Salem

Last summer, the Tennessee Workers' Compensation Appeals Board wrote, "This appeal highlights the often daunting challenge of quantifying causation in circumstances where an employee is alleging a work-related aggravation of a preexisting condition."

Daunting indeed and an understatement. For over a century, courts have been wrestling with these cases, and it remains a hot topic to this day. The good news is, additional guidance is coming soon from the full Tennessee Supreme Court.

#### LOOKING BACK

The Supreme Court first considered the issue in *Tennessee Eastman Corporation v. Russell* in 1924, just five years after the Workers' Compensation Law took effect.

In *Russell*, the employee worked the night shift in a distillery and was found dead one morning, appearing to have fallen from a platform. The employer argued he suffered from "epileptic fits," but the trial court found he wasn't epileptic. Instead, the judge concluded the employee had been "previously injuriously affected physically by the fumes and odors arising from the vats or stills," and that this likely happened again, causing the deadly fall.

The high Court didn't cite case law from other states but relied on treatises—books by learned scholars—to find the injury compensable: "[A] pre-existing weakness or disease will not prevent the injury from being the result of an accident, if the accident is the immediate cause of the injury," they wrote. Further, "an acceleration or aggravation of a pre-existing ailment may be a personal injury, in the sense of the compensation laws."

Fast-forward to 1948. In *Swift & Co. v. Howard*, the Supreme Court held, "When an employer employs a workman *he takes him as he is* and assumes the risk of having a weakened condition aggravated by some injury which might not hurt or bother a perfectly normal, healthy person." (Emphasis added). The principle of taking the employee "as is" remains good law. The Appeals Board cited it in summer 2023 in a case that was appealed to a Supreme Court Special Workers' Compensation Panel. The employee unsuccessfully challenged the constitutionality of the Reform Act of 2013 before a Supreme Court Panel in *Worrell v. Obion County School District*.

More than three decades after *Swift*, the Supreme Court narrowed the compensability of work aggravations in *Boling v. Raytheon Co.* In this 1969 opinion, the high Court made a distinction for cases where the aggravation resulted solely in an increase in pain. They wrote, "what we have here is an employee with a disabling injury or disease not related to employment, but the employment does aggravate the disabling injury or disease by

making the pain worse. This situation does not constitute an 'accident' as this word is used in our workmen's compensation statutes."

The high Court placed another limitation in 1987 in <u>Smith v. Smith's Transfer Corp.</u> The justices concluded that the employee's "work for defendant aggravated her pre-existing condition by making the pain worse but it did not otherwise injure or advance the severity of her [preexisting condition] or result in any other disabling condition. Thus, we find plaintiff did not sustain an injury by accident within the meaning of the Worker's Compensation Act[.]"

More than two decades passed until the full Supreme Court released <u>Trosper v.</u> <u>Armstrong Wood Products</u> in 2008. In Trosper, the justices settled the question of what an employee must show for an aggravation to be compensable. They noted inconsistent results from earlier appellate cases and gave the following rule:

"[T]he employee does not suffer a compensable injury where the work activity aggravates the pre-existing condition merely by increasing the pain however if the work injury advances the severity of the preexisting condition or if as a result of the preexisting condition the employee suffers a new distinct injury other than increased pain then the work injury is compensable."

#### LOOKING AHEAD: COMPENSABILITY UNDER THE 'NEW LAW'

Shortly after passage of the Reform Act in 2013, the newly-created Appeals Board questioned the viability of *Trosper*, in a case where compensability was challenged at the interlocutory (nonfinal) stage of a case. The Board pointed out that the *Trosper* Court applied the remedial construction to find the injury compensable. The Reform Act did away with that and requires courts to construe the workers' compensation law fairly and impartially. The Act also defined "injury" to exclude "the aggravation of a preexisting disease, condition, or ailment unless it can be shown to a reasonable degree of medical certainty that the aggravation arose primarily out of employment."

The Appeals Board reversed the trial court's ruling after an expedited hearing that the employee was likely to show he would prevail at trial that his aggravation of a preexisting condition arose primarily out of employment. The case is <u>Miller v. Lowe's</u> <u>Homes Centers, Inc.</u>, released in 2015.

Judges on the Court of Workers' Compensation Claims haven't cited *Trosper* ever since. But notably, a Supreme Court Panel did just a couple years ago in <u>Edwards-Bradford v.</u> <u>Kellogg Company</u>, a case where the date of injury was after the new law took effect.

Last summer in The AdMIRable Review, we informed you that a complex case involving the aggravation of an employee's preexisting knee osteoarthritis was heading to a

Tennessee Supreme Court Workers' Compensation Panel.

In the case, *Edwards v. Peoplease*, the Appeals Board split. The majority cited several post-Reform Act cases and gave guidance for trial judges: "[A] court can consider several probative factors in determining whether a work-related accident contributed more than fifty percent in causing the need for medical treatment, including but by no means limited to: (1) evidence that employee was asymptomatic prior to the work accident but became symptomatic after the work accident; (2) evidence that the employee had no functional limitations to the injured body part prior to the work accident but had functional limitations after the work accident; and (3) evidence, or a lack of evidence, of an 'anatomic change' to the body part or condition in question."

The majority concluded the trial court erred by finding that the primary cause of the employee's need for bilateral total knee replacements was a work accident, not the employee's underlying severe osteoarthritis.

The dissent, however, concluded that sufficient evidence supported the finding that the work accident caused new or increased symptoms that led to functional limitations and that the need for the knee replacements was "hastened" by the work accident.

Notably, the Appeals Board in Edwards didn't cite Trosper or Edwards-Bradford.

The big news now is that instead of a Supreme Court Panel, the full Tennessee Supreme Court will decide the case.

The full Court rarely hears workers' compensation cases anymore. It also hasn't tackled a case involving an aggravation of a preexisting condition in nine years and not since passage of the Reform Act. In that time, however, plenty of aggravation cases have been before the Appeals Board and Panels—which shows how truly "daunting" these cases can be. They're often medical-intensive. Also noteworthy is the fact that the high Court asked the parties to brief the issue of how medical testimony showed be considered by an appellate court. Is the trial court's decision considered "de novo" (of new) or for an abuse of discretion? The outcome of that question is important because it might affect a party's decision on whether to appeal an order.

Perhaps the Supreme Court will tell us in *Edwards* if/when *Trosper* applies. Regardless, *Edwards* is likely to be the next seminal case on the topic, for application for decades to come. We'll let you know the outcome in an upcoming issue of The AdMIRable Review.



# CPP Registry Reaches over One Hundred Physicians

In late February, the Certified Physician Program (CPP) Registry reached an important milestone by appointing its one hundredth physician. Over the fall and winter, the number of applications for appointment to the CPP and MIR Registries increased significantly. The lion's share of these applications came from mostly larger groups and a few independent clinics who see the benefits to increased reimbursements.

Physicians who seek appointment to the CPP Registry must complete a free online course entitled *Best Practices for Treating and Evaluating Injured Workers*. They must also be certified in the AMA Guides, 6th Edition, through an <u>approved vendor</u>. Because of the cost and time commitment, AMA Guides, 6th Edition, certification is often the greatest hurdle that physicians face when seeking appointments to the CPP and MIR Registry.

The recent surge of physician applications is due primarily to the Bureau providing free on-site training to groups of 10 or more. If requested, the Bureau may provide this free training to one or more neurologists, psychiatrists, pulmonologists, ophthalmologists, urologists, dermatologists, and otolaryngologists that are interested in treating injured workers. To be eligible for the free AMA Guides training, physicians must first (1) register and take the Bureau's *Best Practices for Treating and Evaluating Injured Workers* course, and (2) submit an <u>application</u> for appointment to the CPP.

The length of a training session depends on the specialty and includes a test. For more information regarding these training sessions, please contact <u>Jay.Blaisdell@tn.gov</u>.





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