

## The Cerebellum and Central Dysdiadokokinesia

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One of the more interesting things I have learned during my practice and application of chiropractic neurology is the neurophysiological basis for spinal stability. The intrinsic spinal muscles, unlike other muscles associated with the striated voluntary muscle groups, are involuntary in nature. It is simple to demonstrate this to your patients. Tell them to move their pinkies up and down and side to side. *Then* ask them to laterally flex and rotate the L5-S1 vertebral complex to the right. Of course, they will just laugh.

The reality in terms of functional movement is that these joint complexes do move in these directions (assuming they are not subluxated and thus moving improperly to begin with). The movements are based on reflexogenic responses generated in the brain - specifically in the [cerebellum](#). Proper treatment to promote spinal stability, therefore, must be cerebellar in nature for the patient who happens to be in chronic, unremitting pain secondary to instability.

The cerebellum; where do I start? The cerebellum may very well be one of the most misunderstood areas of a very misunderstood brain. It is at once one of the oldest areas of the brain (fish have a vestibulo-cerebellar cortex that allows them to swim in a balanced manner), and one of the more complex and "new" areas of the human brain. Some researchers feel, in fact, that it is the development of the cerebellum that is responsible for the growth spurt of the rest of the brain; in essence, that it was the cerebellum which made us human.

The cerebellum is made up of a whole host of different neuronal cells: purkinje cells, granular cells, molecular cells, etc. It also has many major nerve centers within it that control a good many functions, such as balance and motor control. It is a very neuron-dense area, such that it is estimated there are more synaptic connections in the cerebellum than there are stars in the universe! Just like any electrical output generator, it can be understood that the cerebellum can be a great generator of "electricity," and electricity is what makes all our engines run.

Because of its concentrated neuronal density, however, the cerebellum is also very blood-dependent, meaning that any loss of oxygenation will affect the cerebellum profoundly, and any toxins taken into the bloodstream will have a high probability of producing a cerebellar consequence. The reasons for these changes into the cerebellum can be numerous.

One way to make sense of its sensitivity is this: When one has had a few too many drinks at the bar and is dumb enough to drive (a dumbed-down cerebellum means a dumbed-down frontal cortex, which will most likely lead to dumb decisions), [the state trooper will put you through a series of sobriety tests. These tests are actually called cerebellar tests.](#) Finger to nose, walking in a straight line, standing on one leg; these are all things controlled by the cerebellum. When alcohol has shut down the cerebellum, one cannot perform these functions properly. Take it a step further, and you may very well see those same symptoms in someone with vertigo, muscle weakness or a visual disorder. And thus, of course, it would make sense that the cerebellum is involved in each of these (and more).

A correlate to this thought process, then, is that stress, sickness and toxins in the body can all have

central manifestations, such that we may present with cerebellar dysfunction and thus, increased likelihood of low back pain. Dr. John E. Sarnow's book *Healing Back Pain* is all about meditating your back pain away, and he claims an 85 percent success rate with his technique of mental therapy. Based on the neurophysiology of the cerebellum and the spine, I do not doubt his numbers.

The cerebellum has two lobes, with the left cerebellum stimulating the right cortex and the right cerebellum stimulating the left cortex. The cerebellum has nuclei within it that control a number of functions related to balance, motor function and visual controls. Hence, a dysfunctional cerebellum is linked to many chronic pain syndromes and disorders such as vertigo, [Parkinson's disease/dystonia \(reflexogenic muscle contractions\)](#), and major muscle weaknesses. Most recently, the cerebellum has been linked to sensory functions, such that we now realize it is the cerebellum that senses where we are in our environment on a constant basis and guides our control of movements through feedback mechanisms.

Regardless, the nuclei in the cerebellum fire into the area acoustica in the pons. As the name might imply, this is also the area that receives direct stimulation from the ear pathways that are so integrally involved in our balance state. The vestibular nuclei fire into the ascending and descending medial longitudinal fasciculus; the ascending portion fires into the motor eye centers, which is why our eyes are so integral to balance, and the descending portion fires into the ventral spinal cord, where we see a reflexogenic spinal muscle response to whatever movements we have made. In essence, the cerebellum senses movement through vestibular and muscle-related reflexogenic afferents, and then sends an efferent response to our eyes and muscles to make sure we maintain balance and do not fall over. This is a much-oversimplified explanation for the moment, but it is all we really need to get where we want to go with this discussion.

*Dysdiadokokinesia*, as you may or may not remember from your board exams, is the ratchety movement associated with a misfiring motor system, specifically a misfiring cerebellum. The traditional board question involved running the heel of the foot from the knee to the shin, and we needed to observe a breakdown in movement that would signify some central cerebellar disorder. This is, in fact, a valuable test that I often use to differentiate areas of cerebellar involvement.

Central dysdiadokokinesia occurs when we see this same breakdown in movement in the intrinsic spinal muscles. Think of it: The same motor systems that show instability with a misfiring cerebellum will likely produce spinal dysfunction. This being the case, you can see how spinal treatment for low back pain that does not enhance spinal stabilizing mechanisms is more likely to fail. Evaluation of the spinal stabilizers need not require a neurology diplomate degree, either.

Here is what I do in the office: Have the patient stand in front of you and place your hands over their iliac crest. Ask them to raise each leg (as if you were checking for motion palpation movement of the PSIS) and then to slowly lower each leg. If, upon lowering the leg, you feel (and often see) a breakdown of muscle movement right at the lumbar spine (you will feel an actual ratchety vibration of the low back musculature as the spinal system struggles for stability), this is indicative of central cerebellar breakdown and spinal instability.

This finding, as you would expect, can have major implications. If the patient lacks spinal stability as evidenced by this positive finding, the likelihood of their holding an adjustment is significantly decreased due to the simple fact that they have no reflexogenic muscle control. This patient may very well re-subluxate by just walking around the office. These patients often become (or are) chronic, and they tend to feel relief for a day or two after a good adjustment, and then are right back where they were again.

The ability to increase spinal stability and help these patients hold their adjustment must be a "central-based" treatment, or a cerebellar specific approach to their care. The interesting thing here is that the cerebellum receives many, many afferents from the spinal joints. Thus, a good adjustment will increase the frequency of firing of the cerebellar integrative state and increase the likelihood that the patient will do well. (A bad adjustment, on the other hand, is likely to deafferentate the patient and decrease firing rates, making the patient worse).

Remember, the eyes and ears are also involved in this area for stability of spinal muscles. What I do many times in the office is integrate eye movement with the preferential side of needed adjustment, assuming that the patient's neurological state can support the treatment. For example, if their left cerebellum is weak, I may have them look to the right at the same time as I adjust their left cervical spine in a coupled movement, thus increasing the integration of the descending medial longitudinal fasciculus and increasing the likelihood that the adjustment will have greater motor stabilizing effects. I may have them listen to music or a metronome at the time of the adjustment to maximize central effects. I then immediately re-check for the signs related to the instability.

If there is an immediate stability change seen, then I know we are on the right track and it becomes an individual approach as to how long it will take to bring the patient to full stability and function. Some will respond in one adjustment, and some chronic patients may take up to six weeks to allow for muscle firing and strengthening to occur. The sky is the limit here; we are limited only by our imaginations as to what we can do to increase spinal firing rates secondary to cerebellar integration.

Keep in mind that this advice is contingent on the central state of the patient's neuraxis, and that they would be able to support the treatment as recommended. Not every neuraxis can tolerate flashing lights on their left side while having a metronome going and adjusting their necks in a coupled fashion.

In summary, look for this central breakdown of muscle stability in your patients and look to central cerebellar mechanisms as a possible culprit for those chronic pain patients who just do not seem to respond to and hold your adjustments. Adding a little more afferentation may be just the ticket to getting those patients out of pain and helping the patient stay well.

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