

# Case Study

## Resolution of Trigeminal Neuralgia Following Subluxation Based Chiropractic Care: A Case Study & Review of Literature

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### Abstract

**Objective:** Since the first century A.D., trigeminal neuralgia (TN) has been regarded as one of the most painful and enigmatic diseases known to man. It has been generally accepted that patients will never be completely free from the condition regardless of the therapy. This case study highlights how chiropractic care has overcome this stigma in a patient who has been relieved of this disease now for over eight years.

**Clinical Features:** Forty year old female presented with right sided TN of six months duration. History included only one major finding of a root canal adjacent to affected area three months prior to onset of painful paroxysms. Patient rated pain 10++ out of 10 on the visual analog scale. Prescriptions included fifteen analgesics including morphine, oxycontin, and percocet. Chiropractic examination revealed upper cervical subluxations. A-P and lateral cervical x-ray analysis determined right C1 laterality and loss of cervical curve.

**Intervention and Outcome:** Diversified adjusting technique and supine rotary breaks to C1 and C2 were utilized. After first adjustment, the pain resolved and patient discontinued use of medications. After eight years of maintenance care, she has not experienced any relapse.

**Conclusion:** This case demonstrates a situation in which chiropractic care has helped a patient successfully manage the debilitating paroxysms of trigeminal neuralgia. Further research is advised to explore whether or not chiropractic should be the first intervention employed in treating TN.

**Key Words:** *Chiropractic; subluxation; diversified technique; [atypical] trigeminal neuralgia; neuropathic pain; antiepileptic drugs; microvascular compression surgery; suicide*

### Introduction

Save a few new drugs hitting the market or a new twist to surgical intervention, treatment for trigeminal neuralgia (TN) has been the same for decades. This is alarming because the treatments do not properly address the cause at the root and, therefore, do not bring permanent resolution. In light of this understanding, the intent of this paper is three-fold:

1. *To offer a thorough, comprehensive overview of the disease with some of the most recent medical and chiropractic research available.*
2. *To shed some light on a disease that has confounded countless physicians for millennia.*
3. *To spark interest for further research to explore whether or not chiropractic should be the first intervention employed in treating TN.*

## Historical Overview

Generally regarded as a condition impossible to completely cure,<sup>1,2</sup> trigeminal neuralgia, also known as *tic douloureux* or *the suicide disease*, has had a rich recorded history dating back to the first century A.D. where it was initially described by Aretaeus of Cappodocia.<sup>3</sup> Since then, many have attempted to illustrate the debilitating, painful paroxysms so common to this enigmatic and elusive disease. English philosopher and physician John Locke penned what is considered to be the very first, comprehensive description in 1677. Below is his explanation regarding one of his patients, the wife of the English ambassador to France, the Countess of Northumberland:

*...such violent and exquisite torment, that it forced her to such cries and shrieks as you would expect from on upon the rack, to which I believe hers was an equal torment, which extended itself all over the right side of her face and mouth. When the fit came there was, to My Lady's own expression of it, as it were a flash of fire all of a sudden shot into all those parts, and at every one of those twitches which made her shriek out, her mouth was constantly drawn to the right side towards the right ear by repeated convulsive motions, which were constantly accompanied by her cries...These violent fits terminated on a sudden and then My Lady seemed to be perfectly well.*<sup>3</sup>

Due to its elusive nature, surgical interventions have varied dramatically over the documented lifespan of TN. By 1756, the French surgeon Nicolas André coined the disease "tic douloureux" describing several patients with TN treated by neurectomy.<sup>4,5</sup> André believed that the paroxysms, or sudden *tics* of pain, were caused by compression of facial sensory peripheral nerves. After reproducing the pain experimentally, he confirmed his hypothesis and removed trigeminal adhesions with a caustic solution of mercury water.<sup>8</sup> His technique of using cauterizing stones ensured that there was minimal blood loss and less likelihood of recompression by an accumulated blood clot.

English physiologist and surgeon, John Hunter, describes the elusiveness of the disease and subsequent treatments in his 1778 treatise:

*This pain is seated in some one part of the Jaws. As simple pain demonstrates nothing, a Tooth is often suspected, and is perhaps drawn out; but still the pain continues, with this difference however, that it now seems to be in the root of the next tooth: it is then supposed with by the patient or the operator, that the wrong Tooth was extracted; wherefore, that in which the pain now seems to be, is drawn, but with as little benefit. I have know cases of this kind, where all the Teeth of the affected side of the Jaw, have been drawn out, and the pain ahs continued in the Jaw; in others, it has had a different effect, the sensation of pain has become more diffused, and has at last, attacked the corresponding side of the tongue. In the first case, I have known it recommended to cut down upon the Jaw, and even to perforate and cauterize it, but all without effect. Hence it should appear, that the pain, in question, does not arise from any disease in the part, but entirely a nervous affection.*<sup>3</sup>

Sadly, stories of useless tooth extractions and needless surgeries are still common today, as we see in the case history of our patient in this study.

As knowledge and science progressed, more accurate theories developed into the epidemiology of TN and surgical intervention followed suit. Sir Walter Dandy, one of the founding fathers of modern neurosurgery, set the bench mark when he outlined his theory of vascular compression as a cause of TN. Subsequently, he pioneered the posterior fossa approach for the treatment of TN. In 1934, Dandy identified the major compressing vessel as the anterior inferior cerebellar artery. Peter Jannetta popularized Dandy's theory and was the first neurosurgeon to apply the operating microscope to the problem. He observed the almost universal occurrence of vascular channels compressing the trigeminal nerve in patients with TN and devised a technique for nondestructive microvascular decompression (MVD) of the nerve.<sup>6</sup> Since the 1960's, MVD has become the standard to which each of the other surgical treatments for TN have been compared.<sup>3</sup>

By the nineteenth century, physicians began to look for non-surgical alternatives to managing TN. Zakrzewska notes that with the successful response of bromides in treating seizures during the 1850's, we see a subsequent influx of antiepileptic drugs (AED) being used to alleviate TN pain by the 1860's.<sup>7</sup> Treatment remained static for about 100 years until carbamazepine was first used in 1962 and shortly became the drug of choice today.<sup>3,7-8</sup> It has evolved into not only becoming the gold standard of TN pharmacotherapy, but it is now a primary diagnostic tool; confirming a patient's diagnosis of TN due to a positive response to the drug.<sup>3,8</sup>

## Epidemiology

The elusiveness of TN becomes evident when considering its epidemiology. Much of the literature today cites research done in the 1990's stating that the U.S. annual occurrence is 4-6% per 100,000 patients.<sup>7,9-13</sup> Consequently, the NIH considers it a "rare disease,"<sup>14</sup> yet that may be a misnomer due to misdiagnoses, mismanagement, and poor research which has confused the data significantly.<sup>7,9</sup> Zakrewska, for example, points out that international studies in the UK and Holland suggest a much greater frequency of 28 per 100,000.<sup>7</sup>

TN generally occurs in the middle-aged and elderly, with women being affected twice as much as men.<sup>11-12</sup> Pediatric cases consistently are reported, albeit much more infrequently.<sup>11-12</sup> Certain anatomical differences are thought to put women more at risk than men, but no substantial research has been done on this. Quoting de Lange's work, Sens and Higer hypothesize that the reason TN occurs more in the elderly is due to elongation of the vertebral artery, which tends to occur with age. Subsequently, it is presumed that the arteries only approximate or touch the nerve in younger people without neuralgia and that compression of the nerve is due to further elongation and age-related dilation of the vessels in the more aged.<sup>16</sup>

## Prognosis

There is noteworthy lack of research concerning the prognosis of TN,<sup>2,7,17</sup> although chiropractic and medical literature

share numerous case studies of significant pain relief and temporary remission being achieved. Zakrzewska indicates that in the initial phases, there are often up to six months or more of pain remission, but these periods tend to shorten in duration as relapses occur more frequently.<sup>17</sup> Sadly, there is little hope for sufferers of TN for complete resolution under the current medical model. Chiropractic and medical research either directly or indirectly states that complete and permanent remission is highly unlikely to impossible.<sup>2, 15, 22</sup>

### Diagnostic Considerations

TN can only be diagnosed on clinical features, so a thorough history is paramount.<sup>5,17</sup> Moreover, since there are no medical devices to establish a conclusive diagnosis, the efficacy of the AED carbamazepine has become popular in identifying the disease. When TN is suspected, an MRI is recommended to rule out secondary causes.<sup>3,8</sup>

TN is a severe, generally unilateral neuropathic pain located within the sensory distribution of cranial nerve five (CN V), the trigeminal nerve.<sup>2, 17-19</sup> Zakrzewska provides the following as gold standards to prevent misdiagnosing patients due to common differential diagnoses attributed to facial pain:

- Each single burst of pain lasts on average under two minutes.
- There is no pain between paroxysms.
- Most patients will initially experience complete remission of pain for weeks or months.
- The pain is described as sharp, shooting, electric shock-like.
- The pain is provoked by light touch activities, but attacks of pain can also be spontaneous.
- The pain is always located in the distribution of the trigeminal nerve and first division trigeminal pain is rare.
- The severity of the pain can vary, especially if medication is used. However, when the disorder is at its peak the pain is suicidal, grossly impairs quality of life and leads to weight loss.
- Sleep disturbance tends to occur if the pain is severe.
- Depression is often noted and there are reports of patients committing suicide or feeling suicidal.
- Extremely rare to have any autonomic symptoms or signs.
- In atypical trigeminal neuralgia patients report prolonged pain of a lowered intensity and quality (i.e. burning, tingling or dull, after the main attack of pain).<sup>17</sup>

Without knowing when the next painful attack will occur, people with TN find themselves terrified to do even the most remedial tasks like brushing their teeth or combing their hair because they're afraid they may trigger a paroxysm. Their lives are spent in a constant state of tension and anxiety fearing this unknown. Thus, depression and suicidal tendencies are common and should be noted to confirm diagnosis when reviewing the patient's history. The literature confirming the suicidal tendencies of someone suffering through TN paroxysms is alarming.<sup>2, 9, 17-20</sup> Burcon notes that approximately half of those people who live with TN for more

than three years commit suicide;<sup>9</sup> thus, TN has been coined "*the suicide disease*" by lay people. The other coined term, *tic douloureux*, refers to the accompanying unilateral grimace precipitated by the pain.<sup>3</sup>

The significance of determining an accurate diagnosis is vital to ensure proper treatment and to prevent malpractice. Significant side effects from unnecessary tooth extraction, improper drug administration, and surgical procedures should be avoided at all costs. Subsequent risk factors are mentioned below. See Table 1 for a list of differential diagnoses.

### Etiology

Due its neurological nature, it is understood that the pain mechanisms in the trigeminal system are altered; the reason why remains a point of contention. Demyelination commonly observed at its root entry zone leads to ephaptic transmission, where action potentials jump from one fiber to another. A lack of inhibitory inputs from large myelinated nerve fibers plays a role and a reentry mechanism causes an amplification of sensory input and excruciating pain when triggered (i.e. hyperexcitability).<sup>11</sup> It has generally been accepted that compression of the trigeminal nerve root by a blood vessel is the cause.<sup>5</sup> The most commonly accepted theory explaining this pathophysiology is Devor et al's ignition hypothesis.<sup>7, 22</sup> They contend that:

*The ignition hypothesis of trigeminal neuralgia is based on recent advances in the understanding of abnormal electrical behavior in injured sensory neurons, and new histopathologic observations of biopsy specimens from patients with trigeminal neuralgia who are undergoing microvascular decompression surgery. According to the hypothesis, trigeminal neuralgia results from specific abnormalities of trigeminal afferent neurons in the trigeminal root or ganglion. Injury renders axons and axotomized somata hyperexcitable. The hyperexcitable afferents, in turn, give rise to pain paroxysms as a result of synchronized afterdischarge activity. The ignition hypothesis accounts for the major positive and negative signs and symptoms of trigeminal neuralgia, for its pathogenesis, and for the efficacy of treatment modalities. Proof, however, awaits the availability of key experimental data that can only be obtained from patients with trigeminal neuralgia.*<sup>21</sup>

Sens and Higer contend that the most common cause of this phenomena are tortuous arteries in the prepontine space; namely that the trigeminal nerve is being compressed by the superior cerebellar or basilar arteries.<sup>16</sup> In a study where they investigated five patients via MRI, they observed tortuous vessels compressing the fifth cranial nerve on the side of TN symptoms in all patients.<sup>16</sup> According to De Lange, this is called "trigeminal looping."<sup>23</sup> De Lange observed trigeminal looping in 43.5% of the 159 angiograms he observed.

Stiles and Evans concede that the integrity of the myelin sheath has historically been the focal point, though the only agreement is that the trigeminal sensory system is dysfunctional and not caused by any one factor.<sup>3</sup> TN has also been linked to multiple sclerosis, brainstem disease, and hypertension.<sup>3</sup> Likewise, an article written by the NIH states that in most cases a cause is not found.<sup>1</sup>

## Treatment

Due to misdiagnosis, many patients go untreated for extended periods of time. Personal health histories generally indicate a long list of visits to various medical and dental specialists in an attempt to get relief from the paroxysms. Patients are usually prescribed analgesics and anti-depressants by their general practitioner and are oftentimes referred to a dentist or oral surgeon. As John Hunter described over two hundred and twenty five years ago, TN pain has (and still is) oftentimes falsely attributed to dental pathology. Consequently, it is quite common for teeth to be extracted, but to no avail.<sup>9</sup>

The gold standard of medical management is the utilization of AED's; carbamazepine and oxcarbazepine being the drugs of choice. As no drugs have been developed specifically to relieve TN related symptoms, these drugs have traditionally been used to help the frequency and intensity of attacks. Originally designed to treat epilepsy, AED's help TN because they slow down the entire nervous system.<sup>7,9</sup> Typically, these drugs are quite effective in alleviating pain temporary, yet their efficacy is lost within time. As Zakrzewska points out, only one study has proven the long-term effectiveness of carbamazepine.<sup>7</sup>

When resistance develops, either the dose increases or one of five common surgeries is administered. When neither of these therapies works, the patient is at high risk to committing suicide. In many cases, increasing the dose or undergoing surgical procedures proves ineffective. In fact, as many trials indicate, increasing the dose oftentimes proves counter-productive in that symptoms are amplified.<sup>7</sup> Some random control trials have been conducted to substantiate the usage of pharmacotherapy, but due to the nature of the disease, many patients are so terrified to deviate from any effective drug regimen they may be under that current research has been delayed because of a lack of willing participants.<sup>7</sup>

Some of the more common side effects to pharmacology are: ataxia; fatigue, drowsiness and lethargy; vomiting; loss of muscle tone; constipation and nausea. Thus, there is a need for individualization of medication therapy based on comorbidities and the use of other medications.<sup>7</sup>

Besides microvascular decompression, all the other surgical therapies utilized (i.e. rhizotomies) to treat TN focus on destroying the sensory fibers of the trigeminal nerve. Below is a brief summary provided by the NIH highlighting the five most common surgical procedures available:

- Balloon compression works by injuring the insulation on nerves that are involved with the sensation of light touch on the face. The procedure is performed in an operating room under general anesthesia. A tube called a cannula is inserted through the cheek and guided to where one branch of the trigeminal nerve passes through the base of the skull. A soft catheter with a balloon tip is threaded through the cannula and the balloon is inflated to squeeze part of the nerve against the hard edge of the brain covering (the dura) and the skull. After 1 minute the balloon is deflated and removed, along with the catheter and cannula. Balloon compression is generally an outpatient procedure, although sometimes the patient may be kept in the hospital overnight.

- Glycerol injection is generally an outpatient procedure in which the patient is sedated intravenously. A thin needle is passed through the cheek, next to the mouth, and guided through the opening in the base of the skull to where all three branches of the trigeminal nerve come together. The glycerol injection bathes the ganglion (the central part of the nerve from which the nerve impulses are transmitted) and damages the insulation of trigeminal nerve fibers.

- Radiofrequency thermal lesioning is usually performed on an outpatient basis. The patient is anesthetized and a hollow needle is passed through the cheek to where the trigeminal nerve exits through a hole at the base of the skull. The patient is awakened and a small electrical current is passed through the needle, causing tingling. When the needle is positioned so that the tingling occurs in the area of TN pain, the patient is then sedated and that part of the nerve is gradually heated with an electrode, injuring the nerve fibers. The electrode and needle are then removed and the patient is awakened.

- Stereotactic radiosurgery uses computer imaging to direct highly focused beams of radiation at the site where the trigeminal nerve exits the brainstem. This causes the slow formation of a lesion on the nerve that disrupts the transmission of pain signals to the brain. Pain relief from this procedure may take several months. Patients usually leave the hospital the same day or the next day following treatment.

- Microvascular decompression is the most invasive of all surgeries for TN, but it also offers the lowest probability that pain will return. This inpatient procedure, which is performed under general anesthesia, requires that a small opening be made behind the ear. While viewing the trigeminal nerve through a microscope, the surgeon moves away the vessels that are compressing the nerve and places a soft cushion between the nerve and the vessels. Unlike rhizotomies, there is usually no numbness in the face after this surgery. Patients generally recuperate for several days in the hospital following the procedure. A neurectomy, which involves cutting part of the nerve, may be performed during microvascular decompression if no vessel is found to be pressing on the trigeminal nerve. Neurectomies may also be performed by cutting branches of the trigeminal nerve in the face. When done during microvascular decompression, a neurectomy will cause permanent numbness in the area of the face that is supplied by the nerve or nerve branch that is cut. However, when the operation is performed in the face, the nerve may grow back and in time sensation may return.<sup>12</sup>

Side effects of the above surgeries include: sensory loss; anesthesia dolorosa; eye and masticatory problems; permanent deafness; and death. Microvascular decompression is reported as having the least occurrence of negative side effects, though none of these modalities can guarantee 100% pain relief after two years.<sup>17</sup> Note that the results of these surgical interventions vary greatly and the literature is not conclusive. Long-term trials are needed to substantiate any claim in supposed effectiveness as there is virtually no literature concerning recurrence. One Chinese study published this year by Xu-Hui et al. claim overall favorable, long-term outcomes from their purposed percutaneous retrogasserian glycerol rhizotomy trial they conducted. Out of the 3370 patients, they report that 99.58% received initial pain relief. Out of the 2750

patients that were still being monitored, 35% had pain recurrence at various intervals from 1 to 23 years; the major complication being facial numbness. Most patients recovered within half a year.<sup>7</sup> Last year Matsuda et al. reported a 51% success rate in their gamma knife surgery trial on 104 patients, though, this study only tracked their patients for 68 months.<sup>8</sup>

### *Classification*

TN is traditionally classified as being primary (idiopathic) or secondary to lesions of the trigeminal (gasserian) ganglion sensory root or root entry in the pons.<sup>10</sup> There are, however, various sub-classifications. With the advent of terms like “atypical” trigeminal neuralgia and the confusion that generally surrounds this pathology, Eller et al. propose a new classification scheme for TN and related facial pain syndromes in an attempt to guide the various differential diagnoses into a standardized framework by using objective and reproducible criteria. The mainstay of this criterion is the patient’s medical history.<sup>5</sup> The system is as follows:

1. Trigeminal neuralgia Type 1 (TN1)
2. Trigeminal neuralgia Type 2 (TN2) Both referring to idiopathic, spontaneous facial pain that is either episodic (TN1) or constant (TN2).
3. Trigeminal neuropathic pain resulting from unintentional injury to CN V via trauma or surgery.
4. Trigeminal deafferentation pain resulting from intentional injury to the nerve by peripheral nerve ablation, gangliolysis, or rhizotomy in an attempt to treat TN or other related facial pain.
5. Symptomatic trigeminal neuralgia resulting from multiple sclerosis.
6. Postherpetic trigeminal neuralgia following a cutaneous herpes zoster outbreak in CN V distribution.
7. Atypical facial pain describing a facial pain secondary to somatoform pain disorders and requiring psychological testing for diagnostic confirmation.<sup>5</sup>

We find this classification useful, but certainly not conclusive. As we will see below, not all trigeminal-related pain is spontaneous. There needs to be a category for what is generally referred to as atypical trigeminal neuralgia. This disease presents with a less intense, constant pain with intermittent, excruciating paroxysms.<sup>17</sup> Eller et al’s intentions are appreciated particularly due to the elusive nature that has confounded health care providers for millennia. They have begun to simplify the TN definition and classification process based on symptoms so that even the most inexperienced health care provider can diagnosis a patient confidently and offer proper advice regarding the various treatment options and prognoses.

### **Case Report**

#### *Patient History*

A forty year old female presented with right sided TN of six months duration. Besides living a highly stressful lifestyle including daily caffeine intake, minimal exercise, and smoking a pack of cigarettes a day, history included one major finding: root canal adjacent to affected area (lower right molar) three months prior to onset of painful paroxysms. Patient rated pain

10++ out of 10 on the visual analog scale; describing the pain as a constant throbbing between jaw and ear, with paroxysmal attacks described as “ice pick or letter opener” being jabbed into her ear canal. Nothing triggered the pain. When patient arrived to office, she had already been to the emergency room 2 times, had seen 5 different specialists (EENT, neurologist, and neurosurgeon), and 3 dental specialists. During that duration, she was prescribed and took a total of 15 different medications including morphine, oxycontin, and percocet. She had one tooth extracted in an attempt to treat the pain, but to no avail. Health history indicated that every aspect of life was affected; including work, sleep, recreation, and daily routines.

The pain occurred more at night which precipitated insomnia and terror to sleep on the right side of body; sometimes getting only 1 hour of sleep. She couldn’t focus at work because of the constant, toothache type of throbbing in her face. Patient held (and still does at the time of this paper) a management position with great responsibility that required her presence so she missed only a minimal amount of work. This added to her anxiety and complicated her condition because she never got the rest she duly required.

The financial burden was increasing exponentially with all the co-pays for doctors’ visits and prescriptions. Due to the extreme torment, the patient couldn’t even listen to music or read, which aggravated her mental state because she was an avid music lover. At the time she presented into the office, she was severely depressed. In fact, she reported that she was suicidal, living on pins and needles terrified of the next, inevitable paroxysm. Her neurologist advised a procedure that would, according to the patient, “drill a hole in [her] head,” presumably microvascular decompression.

No MRI was performed; the diagnosis was purely based on past history and the presenting chief complaint. According to the patient, he told her that he would “kill the nerve,” and could help with the pain, but the surgery had serious potential side effects; namely, fatality. Taken so aback by his recommendation and the possible risk of death, she decided to take her sister-in-law’s advice and give chiropractic a chance as a “last resort.”

#### *Examination, Intervention, and Outcomes*

Thomson-Derefield analysis yielded a left and right cervical syndrome. Palpation results yielded spastic, inflamed, and edematous cervical paraspinal musculature. Balance test positive bilaterally. Coordination test positive; finger-nose-finger. Foramina Compression test positive. Romberg’s test positive. A-P and lateral cervical x-ray analysis determined right C1 laterality and loss of cervical curve.

Thomas analysis and Diversified adjusting technique were utilized. With patient prone, functional leg check was not level in the second flexed position when she rotated her head to the right and also to the left; thus, indicating a right and left cervical syndrome (i.e. subluxation at C1 and C2) which were corrected by a supine rotary break to atlas and axis. The functional leg-length was then re-checked after the adjustment and there was no longer a leg-length discrepancy when she turned her head; thus, correction to the subluxations was assumed to have been made. Patient had the following care

schedule: 3 visits per week for the first 4 weeks; 2 visits per week for the following 8 weeks; 1 visit per week for the next 40 weeks; 2 visits per month for the last 7 years. Within one month there was complete resolution of all the above findings. 100% reduction in spasm, inflammation and edema in paraspinal musculature. 100% resolution of spinal tenderness. 100% restoration of cervical range of motion [see Tables 1 & 2]. 100% resolution of *Balance*, *Coordination*, *Foraminal Compression*, and *Romberg's* tests. Details are discussed below.

#### *Neurological and Orthopedic Tests*

The *Coordination test*, *finger-nose-finger*, was performed by asking the patient to alternately touch her nose and the examiner's finger as quickly as possible. Abnormal movement (ataxia) is generally observed if the patient overshoots or moves in an abnormal wave; oftentimes in a wavy motion or jig-jagged fashion. Normal performance of this motor task depends on the integrated functioning of multiple sensory and motor subsystems: position sense & visual pathways; lower & upper motor neuron; basal ganglia; and cerebellum.<sup>24</sup>

When considering that the trigeminal ganglion serves the same function as the dorsal root ganglion (DRG) in the spinal nerves, we can infer that a lesion in the trigeminal ganglion is possibly attributing to a motor dysfunction in the basal ganglion.<sup>18</sup> CN V is associated with many of the brainstem reflexes (e.g. turning the eye toward a flash of light, sudden noise, touch on skin, autonomic processes in chewing).<sup>18</sup> Moreover, all sympathetic and parasympathetic axons of CN III, VII, and IX follow branches of the trigeminal nerve to their target tissue(s) which explains the patient's inability to properly perform the finger-nose-finger coordination test.

The *Foraminal compression test* is an assessment for cranial nerve root encroachment indicating cervical nerve root pathology. To conduct the test, the patient's was seated and the examiner rotated the patient's neck while exerting strong downward pressure on the head. Pressure is first applied with the head in a neutral position and then with the head rotated to the right side (side of complaint). The test was repeated bilaterally. With the neck rotated and downward pressure applied, the intervertebral foramen closes. Foraminal encroachment is presented by localized pain. Pressure on the nerve root is presented by radicular pain. Reproduction of the complaint is a positive finding and suggests foraminal encroachment, as occurred in her case.<sup>25</sup> Foraminal encroachment is directly linked to various pathological conditions including neurofibroma (most common), herniated meningocele, and tortuous vertebral arteries.

Of significance to our study on TN are the tortuous arteries affecting CN V. As we learned above, tortuous vasculature has been coined the main cause of TN. If, for instance, chiropractic adjustments can relieve this pressure, TN could possibly be corrected. There have been many studies on chiropractic's role in managing hypertension; most recently a study by Bakris *et al.* who set up a double-blind study to substantiate whether an upper cervical adjustment could affect blood pressure.<sup>27</sup> After observing consistent and repeatable results of patients' blood pressure decreasing after a chiropractic adjustment, they concluded that one manipulation

in the upper cervical area has the effect of two hypertension medications."<sup>27</sup>

From this, we can logically deduce that if a cervical chiropractic adjustment can affect blood pressure, then perhaps it affects arterial function. If the vertebral artery is relieved, then it is also very possible that its cranial branches and peripheral vasculature can be effected; namely, the superior cerebellar or basilar arteries. As we have seen above, these arteries have been most commonly attributed to compressing the CN V, which is undoubtedly the reason they have been a point of focus concerning TN.

To conduct *Romberg's test*, the patient was asked to stand with her feet together (touching each other) and then close her eyes. With the examiner remaining as close as possible in case she lost her balance, she began to sway which signified a positive finding. With the eyes open, three sensory systems provide input to the cerebellum. These include vision, proprioception, and vestibular. When patients' close their eyes, as in the case of the patient in this study, visual input is removed and instability is brought out.<sup>25</sup>

Though, not conclusively a TN attributed issue, we suspect some connection due the proprioceptive nature of the test and CN V<sub>3</sub>. The mandibular nerve (CN V<sub>3</sub>), for instance, relays proprioceptive input from the periphery, particularly from stretch receptors of the muscles of mastication, teeth, and gums. Moreover, one of the sensory trigeminal nuclei, the mesencephalic nucleus, is a significant center for proprioceptive impulses in the head and face.<sup>26</sup> Collaterals from mesencephalic sensory axons terminate in the trigeminal motor nucleus, providing the afferent arc for the monosynaptic jaw (masseter reflex). Being proprioceptive in nature, a lesion in this nucleus could possibly disturb central processing in other areas of the brain (i.e. the trigeminal nerve itself and corresponding structures).

#### *Diversified Technique*

The chiropractic specialty used to treat the patient in this case study was the "diversified" technique. The keys to diversified have always been the importance of accurate, specific diagnosis of the active lesion, and the structural environment of that lesion within the patient.<sup>28</sup> Who and when the term "diversified" was coined is unclear, although most attribute it to Joe Janse. He proposed that chiropractic is not an alternative to medical science, but rather a complementary science.<sup>29</sup>

Gitelman and Fligg indicate that this technique attempts to correlate appropriate, adjustive spinal manipulative procedure with joint dysfunction while taking into consideration the additional factors of muscle dysfunction.<sup>45</sup> It maintains that muscle dysfunction is important whether it is the primary factor in either the segmental or overall abnormal movement pattern that has stressed the area to the point of being symptomatic. Otherwise, it is secondary to the joint dysfunction.<sup>45</sup>

Today, over 95% of practitioners are reported as utilizing this modality according to the American Chiropractic Association (ACA).<sup>30</sup> It entails a high-velocity, low-amplitude thrust that

oftentimes results in a cavitation of a joint (a “cracking” or “popping” sound indicating a pressure release in the vertebral joints).

In our study, the patient received specific adjustments that undeniably brought her TN into remission. The examination confirmed a “right and left cervical syndrome.” Clemen explains that fixations at the right or left side of vertebra are responsible for continuous nerve impingement.<sup>31</sup> Thus, the vertebral subluxation can be detected by various diagnostic measures. For instance, restricted range of motion in flexion, extension, and lateral flexion planes in the occiput-atlas motor unit as indicated in the patient in our study.<sup>31</sup>

The patient received supine rotary breaks to C1 and C2. The primary objective of this procedure (when adjusting a subluxated segment) is to introduce a line of drive to that fixated segment that will restore the previously restricted motion.<sup>32</sup> This was performed by utilizing the following common procedure established by Szaraz and described by van Schalkwyk and Parkin-Smith:

1. Patient supine with the cervical spine deviated away from the side of contact.
2. Contact is made with the index finger, the wrist of the contact hand being straight.
3. The line of drive is laterally deviated toward the lateral flexion fixation. The thrust is sudden, short amplitude pectoral thrust.<sup>32</sup>

#### *Actual Diagnosis*

When the patient presented into the office, she came in with a diagnosis of trigeminal neuralgia from her neurologist. After evaluating her case in lieu of her health history and physical examinations, it is clear that she had *atypical* trigeminal neuralgia, a rare form of the disease. As we’ve seen above, the literature is conclusive of the following: TN pain is intermittent, with no pain between attacks; complete remission usually occurs early on; typically there are no positive findings for neurological testing. *Atypical* TN pain, on the other hand, presents with the following: less intense, *constant* pain between excruciating paroxysms; positive findings testing for neurological testing.<sup>5,17</sup>

Medically speaking, proper diagnosis is vital because *atypical* TN is treated much differently than the classic TN due to its resistance to AED’s. It’s even suspected that both have totally different etiologies.<sup>7</sup> For example, we noted above with the ignition hypothesis that there is increasing evidence that changes in the nervous system happens not only peripherally, but centrally. It has been shown that these central changes might be more frequent in *atypical* TN versus classic TN; presenting with an over-activation of the central facilitation of trigeminal nociceptive processing.<sup>7</sup>

#### *Patient’s Experience*

Initially, the patient did not trust her new pain-free state and continued to live the way she had adapted to over the previous 6 months, terrified and paranoid of the “next” paroxysm. When the next attack didn’t come, she gradually became more

confident in attempting to live life normally again. Within a week she decided to listen to music on her way to work and she was able to listen to her favorite music again without agony. She began to sleep better, concentration improved, and she ceased taking any medication. The final step to recovery was when she resumed eating on the right side of her mouth. Up until one month after being pain-free, she still refused to eat on that side because she feared a paroxysm. She explained the experience as follows:

*Dr. Mike told me I should start eating on the right side of my mouth, but I was too afraid. After a couple visits he kept telling me to do it so I decided to give it a try. The next visit, I brought food into the office and with Dr. Mike in the room with me, I took a bite of food on the right side of mouth for the first time in months! I was terrified and tears were streaming down my face. You know what? It didn’t hurt a bit!*

As stated above, during the initial examination, she stated that she considered suicide if things didn’t improve because the symptoms were getting worse. At the time, she was unaware that she was pregnant. Obviously emotional due to all that had transpired the past year, she reported after the birth, “Were it not for chiropractic my daughter would not be here.”

#### **Discussion**

The anatomical connections between the various trigeminal nuclei and TN pain clearly indicate that treating the symptoms (i.e. medically analgesizing and treating peripheral nerves via dental extractions, rhizotomies, or other surgical procedures) will not be affective if the lesion is located in brainstem nuclei. As Guyton and Hall note, pain from TN can usually be blocked by surgically severing the peripheral nerve from the hypersensitive area.<sup>33</sup>

The sensory portion of CN V is often sectioned immediately inside the cranium, where the motor and sensory roots separate from each other. Thus, the motor portions, which are needed for many jaw movements, can be spared while the sensory elements are destroyed. The procedure leaves the side of the face anesthetic, yet it is not infallible. Sometimes the operation is unsuccessful, indicating that the lesion that causes the pain might be in the sensory nucleus in the brainstem and not in the peripheral nerves.<sup>33</sup>

It is purported, however, that chiropractic affects these areas directly. Grostic first described this in his dentate ligament-cord distortion hypothesis.<sup>34</sup> Noting that TN paroxysms could precipitate from irritation of the trigeminal nerve, trigeminal ganglion, or the spinal nucleus, Grostic proposed that,

*By combining anterior rotation of the atlas on the side to which the atlas has laterally deviated with the lateral traction it may be possible to put traction directly on the sensory nucleus of the trigeminal nerve at the level of the first and second cervical vertebra....Direct mechanical-vascular irritation of the spinal nucleus might also explain those cases in which surgical destruction of the ganglion or sectioning of the nerve fails to provide relief.<sup>34</sup>*

He noted that while caring for a patient with TN the rotational misalignment of the atlas appeared to be the most crucial

factor and required the greatest reduction before the painful attacks subsided. Due to the laterality in her atlas (as noted above), this could explain why the patient in our study responded so well to the rotary breaks administered to her atlas and axis. We see 8 similar cases of Grostic's example above in Erickson's text on the upper cervical subluxation complex.<sup>35</sup> This study attempted to validate chiropractic's effectiveness in treating TN due to the cervical-cranial connection, as described by Bogduk.<sup>36</sup>

Bogduk notes that the neuroanatomical basis for cervicogenic headaches, for example, is due to a convergence in the trigeminocervical nucleus between the trigeminal nerve and the first three cervical nerves. He defines the trigeminocervical nucleus as "those cells in the upper three cervical segments that receive both a trigeminal and a cervical peripheral input."<sup>36</sup> Thus, any pain produced by structures innervated by C1 – C3 nerves (e.g. atlanto-occipital and lateral atlantoaxial joint, sternocleidomastoid, trapezius and paravertebral muscles) may be perceived as arising from the trigeminal area of the head. Anatomically speaking, therefore, we see a direct connection as to how and why a cervical chiropractic adjustment, particularly one in the atlas-axis region, can have profound effects on the trigeminal nerve and, thus, on TN.

Another convincing link cervical adjustments may have on TN can be seen in Welch's and Boone's work with sympathetic and parasympathetic responses to spinal manipulations.<sup>37</sup> Well known since the 1970's, the trigeminal nerve is accompanied by both parasympathetic and sympathetic fibers; the former via CN V<sub>3</sub> and the later CN V<sub>1</sub> & 2.<sup>38</sup> As Welch and Boone preliminarily suggest in their 40 patient study, cervical adjustments could manifest a shift in parasympathetic dominance in response to the parasympathetic nervous system.<sup>37</sup> If one could, therefore, stimulate the parasympathetic nervous system (PNS), one would be able to possibly stimulate the trigeminal nerve directly to bring about desirable results. What that dominance would look like exactly is unclear. Further study is needed to confirm and analyze their relationship.

The TN and chiropractic connection was not an isolated instance in the literature concerning chiropractic's efficacy in treating rare diseases. There have been numerous studies, for example, substantiating chiropractic's effectiveness in properly managing and resolving rare and debilitating diseases like cephalgia vertigo, cervicobrachial neuralgia, fibromyalgia, intercostal neuralgia during pregnancy, myofascial pain syndrome, spinal neuralgia, and even increasing the CD4 counts on HIV positive patients.<sup>39-43</sup> Though, certainly not conclusive in the literature, it is evident that different chiropractic adjustments and modalities have been confirmed and documented in managing a number of pathologies.

#### *Subluxation: Chiropractic and Medical Literature*

The term "subluxation" has received much attention in the health care system. See the appendix for a brief historical perspective of the term and concept "vertebral subluxation" based on Terrett's historical account.<sup>45</sup>

In his work that has been paramount to chiropractic, Sharpless demonstrated a number of findings; namely, that spinal nerve and roots are more vulnerable to mechanical stress than peripheral stress; and that vertebral subluxations can cause such stress.<sup>46</sup> The University of Colorado Professor of Psychology devised a compression apparatus for animals to mimic the situation in which bony facets, via tissues, compress nerves in the human body. Referring to Sunderland (1968), Sharpless indicates that it is this kind of situation that most often gives rise to idiopathic peripheral nerve compress syndromes [as in TN].<sup>46</sup>

This hallmark study, clearly indicated that just 10 mmHg (about the weight of a dime) is all that is needed to significantly compress the cord, resulting in neurological interference. "It is only when the joint is fixed in a position yielding a significant increase in pressure," Sharpless said, "that one would expect compression block to develop. This consideration is relevant to the chiropractic definition of a 'subluxation' as a 'fixation of a joint within its normal range of movement, usually at the extremity of that range.'"<sup>46</sup> Thus, validating what Hernricus Hieronymi contended as the vertebral subluxation over 200 years prior, Sharpless' experiment provides scientific proof for many of the claims chiropractors and medical physicians have been making for centuries.

As expected, there have been a myriad of theories defining and attempting to explain the vertebral subluxation. Of specific interest to our study is Kent's work concerning the concept dysafferentation.<sup>47</sup> Dysafferentation refers to imbalance in afferent input presented by increased nociceptor input and reduced mechanoreceptor input. Kent states: "The intervertebral motion segment is richly endowed by nociceptive and mechanoreceptive structures. As a consequence, biomechanical dysfunction may result in an alteration in normal nociception and/or mechanoreception. Aberrated afferent input to the CNS may lead to dysponesis. To use the contemporary jargon of the computer industry, 'garbage in—garbage out.'"<sup>47</sup> This leads to an increased excitability of nociceptive neurons in the central nervous system.

Accepting that a *lesion* is any abnormal tissue found on or in an organism, usually damaged by disease or trauma, the significance this "dysafferentation" theory has to TN can be seen by the following:

1. If the trigeminal nerve or associated nuclei (particularly those extending to the upper cervical regions as discussed above) are injured in any manner, the corresponding lesion can have hyperexcitable sensory affects on the trigeminal nerve.
2. This, in turn, could possibly precipitate TN paroxysms.
3. Moreover, decreased mechanoreceptor activity due to reduced joint movement or pathology in the skin, muscles, or intervertebral discs (IVD), has been linked to ataxia and cerebellar pathology.<sup>44</sup> Considering that the cerebellum is largely responsible for unconscious proprioception, we see another connection with

CN V<sub>3</sub> insult as discussed above.

4. It is, therefore, quite plausible to see how an upper cervical joint and or IVD pathology (i.e. subluxation) affects mechanoreceptors and nociceptors in the upper cervical spinal cord.
5. This, in turn, can affect the trigeminocervico nucleus or the spinal trigeminal tract, which can produce direct, sensory dysafferentation.
6. Thus, CN V hyperexcitability can cause TN.

Kent concludes that: “If afferent input is compromised, efferent response may be qualitatively and quantitatively compromised. Correcting the specific vertebral subluxation cause is paramount to restoring normal afferent input to the CNS, and allowing the body to correctly perceive itself and its environment.”<sup>47</sup> Though, further investigation is needed to substantiate the connection between a cervical vertebral subluxation as a possible cause of TN.

### *Limitations*

There are three notable limitations to this study: using the visual analog scale (VAS) as a diagnostic tool; the lack of research defining the diagnosis “vertebral subluxation;” the classification of this paper as a “case study.” Each will be commented on briefly.

Quite ubiquitous in its application, the VAS has been a simple, concise standard in gaging and monitoring patients’ reported pain concerning their present complaint. As Chen and Lee point out, though, due to pain’s subjective nature and the strong influence of social context, emotion, and other non-physiological variables, trigeminal neuralgia and related facial pain syndromes are difficult to accurately measure.<sup>48</sup> This can often be described as the proverbial “white coat” syndrome, in which patient’s symptoms will be exasperated because of their nervousness being in the same room with a doctor or health care professional.

Moreover, they may even fabricate their ailment(s) for various reasons (e.g. to satisfy a psychological desire for attention, sympathy, etc). The VAS, for instance has been used to determine the efficacy of medical therapies (surgical and pharmacological alike), though it is 1-dimensional. Not adequately addressing the complexity of the measurement of chronic pain, it has not undergone psychometric testing to assess their reliability and validity in TN patients.<sup>48</sup> This can pose problematic particularly in regards to proper diagnoses and appropriate treatment. Not only is it paramount for the patients’ care plan, but proper diagnoses affect the population at large. Approximately 10% of Americans reportedly suffer from chronic pain conditions which estimate a cost to the U.S. upwards to \$80 billion annually in healthcare costs and lost productivity.<sup>48</sup> This usage of funds has significant effect on our national budget, health care expenditure, insurance companies based coverage on such data.

In lieu of these deficiencies, Chen and Lee suggest an 18-item questionnaire that measures pain in patients with TN including key components like quality of life indexes. They see their proposal as a “first step” in the development of a more accurate tool to evaluate the multi-faceted pain in patients with TN. The multi-institutional Initiative on Methods,

Measurement, and Pain Assessment in Clinical Trials (IMMPACT) helped shape their work by defining the 6 core domains to be considered in treatment trials of chronic pain:

1. Pain intensity
2. Physical functioning
3. Emotional Functioning
4. Participant rating of improvement and satisfaction with treatment
5. Symptoms and adverse events
6. Participant adherence to treatment regimens.<sup>49</sup>

Due to our using the VAS, we feel it would be advantageous for further clinical trials to be performed using more appropriate pain scales to confirm our findings.

The second limitation is that the term or diagnosis “subluxation” has detractors alleging that it’s never been defined and ubiquitously accepted for research purposes. As Meeker notes, because we haven’t agreed on what a subluxation is, we have no way to measure it. And without an agreed upon measurable control variable, how then can we honestly “do” research?<sup>50</sup>

Thus, the detractors would argue, we have not previously concluded what exactly a chiropractic adjustment is and what it “does”. Moreover, we don’t even have a universally accepted definition what “health” is. Therefore, according to Meeker, until we define the above terms, we’ll never be able to show that a subluxation is “bad” and that adjustments are “good.”<sup>50</sup> It is evident that much more discussion is needed to resolve this matter and we hope to see a common forum where the issue can be settled.

The final limitation is the fact that this paper is first and foremost based on a retroactive case study. Due to its very nature of being a report on the treatment of individual patients, there are no control groups and are thus generally regarded as having no statistical validity.<sup>51</sup> Thus, it can often be seen as a “descriptive” method, not an “explanatory” one. Furthermore, because only one or a few people are involved, they may not be representative of the general population.

Due to its retrospective nature, problems may also occur because researchers have to rely so heavily on notes and memory. The case study has also been criticized as being subjective and even pseudo-scientific. If done well, though, they can be a good starting point; well-documenting history and exams, and thoroughly reviewing the literature and outcome measures.

A well known “evidence pyramid” places case studies second from the bottom on a 9-tiered scale; with the “gold standard,” Cochrane Systematic Reviews, topping the list<sup>52</sup> [see Table 4].

As may be expected, chiropractic literature concerning TN is limited to case studies and are very few in number. There is a great need for other, more reputable, modalities in the future. Note, though, that this deficiency is not limited to our profession alone. Zakrzewska indicates that the medical field shares the same dilemma.<sup>7</sup>

## Conclusion

It has been over 8 years since the patient in this case study first received chiropractic treatment. In addition to her receiving 100% resolution after one adjustment, it has been remarkable to observe that no attacks have occurred since then. As noted above, this is extremely rare as most people generally enjoy complete remission for only a few months up to 2 years, regardless of the therapy utilized.<sup>17</sup>

After attempting to review the anatomy, chiropractic, and medical literature as thoroughly as possible we see the great need in a multi-disciplinary and integrative approach to treating trigeminal neuralgia. We propose that medical and dental physicians refer TN patients to a well-respected and competent chiropractor who can accurately confirm the disease as the simple diagnosis and pain scale guidelines above indicate. Upon investigation, if the chiropractor detects a vertebral subluxation (especially one at the upper cervical region), then the TN patient should be treated accordingly. If the patient doesn't respond well to a strict and highly monitored care plan, then an appropriate referral should take place to a well-respected and highly qualified health care specialist as determined by their chiropractor.

Our hope is that this paper will empower the chiropractic and medical professional alike to consider TN an approachable disease – perhaps treatable and manageable without the use of pharmacological and surgical interventions

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**Table 1**  
Typical Differential Diagnoses for TN<sup>2, 7, 10</sup>

Atypical Facial Pain
Atypical Odontalgia
Burning Mouth Syndrome (Stomatodynia)
Cluster Tic-Syndrome
Geniculate Neuralgia (Nervous Intermedius Neuralgia of Hunt)
Giant Cell Arteritis
Glossopharyngeal Neuralgia
Idiopathic Stabbing Headache
Intra-Oral Symptoms (e.g. toothache)
Post-Herpetic Neuralgia
Temporal Arteritis
Temporomandibular Disorders
Traumatically Induced Neuralgia
Trigeminal Autonomic Cephalalgias (e.g. cluster headaches      paroxysmal hemicranias, SUNCT, SUNA)
* <u>SUNA</u> : Sort-Lasting Unilateral Neuralgiform Headache Attacks with Cranial Autonomic Symptoms
* <u>SUNCT</u> : Short-Lasting Unilateral Neuralgiform Headache Attacks with Conjunctival Injection and Tearing

Table 2. Initial Examination of Patient Before First  
Chiropractic Adjustment – Cervical ROM

Flexion	24° due to pain
Extension	29° due to pain
Left Lateral Flexion	22° due to pain
Right Lateral Flexion	16° due to pain
Left Rotation	65° due to pain

Table 3. Re-examination One Month Later – Cervical  
ROM

Flexion	50°
Extension	60°
Left Lateral Flexion	45°
Right Lateral Flexion	45°
Left Rotation	80°

## Appendix

The below synopsis of the vertebral subluxation is based off of Terrett's work.<sup>45</sup>

- *1746*: Joannes Hernricus Hieronymi's defined the "vertebral subluxation" as many chiropractors see it today. He wrote, "Subluxation of joints is recognized by lessened motion of the joints, by slight change in the position of the articulating bones and pain...most displacements of vertebrae are subluxations rather than luxations."
- *1820*: Edward Harrison noted that, "...a very slight and partial compression of the cord, or some of the nerves, will disturb the organs to which they run." Thus, he proposed correcting the vertebral subluxation. "In recent cases these subluxations are easily replaced," he said.
- *1821*: Understanding the significant ramifications of the vertebral subluxation, Harrison said, "When any vertebra becomes displaced or too prominent, the patient experiences inconvenience from a local derangement in the nerves of the part. He, in consequence, is tormented with a train of nervous symptoms...the obvious indication for the cure of spinal affections consists in restoring the displaced bones to their natural situation, that the spinal cord and nerves, relieved from injurious pressure and disturbances, may be reinstated in their former abilities."
- *1824*: The first subluxation corrective procedure (i.e. the modern "chiropractic" adjustment) is proposed by Harrison in print. After setting the patient up in a mechanical traction device, Harrison explained what could be the first "activator" type chiropractic adjustment. "...the prominent vertebrae and ribs were then pressed, and driven in the direction of their natural situations, with an instrument held in the right hand....I formerly used my thumbs for pressure; but, [found] the other contrivance much more powerful and easier to be borne."
- *1868*: Little confirms Dr. Harrison's work when he said that it was Harrison who, "thirty years ago, adopted a peculiar means of endeavoring to press the rotate spinous and transverse processes into a more favorable position." Note: D.D. Palmer, probably unaware of Harrison's work, may have prematurely donned himself the originator of this particular vertebral manipulation.
- *1828*: Referring to the significant effects of "spinal irritation," Brown wrote that, "the immediate cause of the pain is spasm of one or other of the muscles arranged along the spine, altering the position of the vertebrae, or otherwise compressing the nerves as they issue from the spinal marrow. When it is allowed to remain for any considerable length of time, it often produces nervous complaints and general bad health."
- *1831*: A memorandum submitted by a group from the medical community to the Massachusetts' Legislature gave an anatomical basis for vertebral subluxations and even suggested its cause in various idiopathic diseases. "Dissection," they stated, "has enabled the anatomist to follow the nerves....This has suggested to the physician the truly philosophical remedy for the painful afflictions of these regions, produced by disordered nerves: viz. to apply remedies to the back [i.e. subluxation] – the less obvious but the true seat of the disease."
- *1839*: John H. Griscom, M.D. discussed that "nerves are frequently irritated at the level of their exit."
- *1843*: J. Evans Riadore M.D., a Fellow of the Royal College of Surgeons in England wrote, "Every organ and muscle in the body is dependant, more or less upon nerves....one or two of the vertebrae may be pressing injuriously upon either the anterior or the posterior root of some nerves....When one vertebra forms exception in the regularity of the spinal line, either by height or distance from its fellows [i.e. subluxation], a serious train of nervous symptoms may supervene."
- *1895*: D.D. Palmer, with incredible insight and skill popularized the systemic science of chiropractic and coined the term, though, as we have seen above, the fundamental hypotheses of chiropractic were in medical literature for over a hundred years by the time he touched the subject.
- *1979*: The New Zealand Royal Commission's statement on chiropractic: "Although the precise nature of the biomechanical dysfunction which chiropractors claim to treat has not yet been demonstrated scientifically, and although the precise reasons why spinal manual therapy provides relief have not yet been scientifically explained, chiropractors have reasonable grounds based on clinical evidence for their belief that symptoms of the kind described above can respond beneficially to spinal manual therapy".