LITERATURE REVIEW

The Effects of Mild Compression on Spinal Nerve Roots with Implications for Models of Vertebral Subluxation and the Clinical Effects of Chiropractic Adjustment

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Abstract — This review attempts to analyze the clinical relevance of nerve compression as a component of the vertebral subluxation and if the chiropractic adjustment can lead to the correction of the nerve pressure. Literature searches were conducted on the World Wide Web at the Pub Med website. There is evidence of nerve compression at the level of the intervertebral foramen (IVF) occurring anywhere from 15.4% to 78% of levels inspected. Most of the spines inspected were already prescreened to eliminate those that were definitely known to have nerve compression problems. Pressures as little as 10 mm Hg can alter the nerve root and dorsal root ganglion's abilities to function normally. In the normal range of motion the pressures generated in the IVF may exceed 30 mm Hg. When considering the concept of a joint fixated in a diminished sphere of its normal range of motion in conjunction with the mild pressure increases, it becomes apparent that nerve function can be significantly altered. The chiropractic adjustment can effect a restoration of normal H-reflex in compressed nerve roots without altering the H-reflex at uninvolved levels. The major variables of compression are the rate of onset, the amount of pressure generated, and the time maintained. Another major variable in the recovery is age. The younger the nerve tissue the better chance for a full recovery. The concept that a vertebral subluxation can induce pressure increases at the level of the IVF is supported by the literature. This increase, though seemingly mild, is enough to alter nerve function. The garden hose theory or hard bone - soft nerve explanation of vertebral subluxation is considered by some to be archaic but appears to be a valid entity at least in the lower cervical spine. More research is needed to decipher the susceptibility to mild pressure increases throughout the spine.

Key Words: Spinal nerve root, compression, dorsal root ganglia, chiropractic, adjustment, vertebral subluxation, manipulation

Introduction

Spinal nerve root compression has generally been considered a result of long-standing degeneration, disc involvement or other severe pathology. It is not considered a relevant factor in the pathogenesis of vertebral subluxation by some. Nor is it considered that relevant in a non-elderly population. This paper will first review the pertinent literature associated with the neurophysiologic effects of nerve compression and then it will examine the literature regarding the applicable biomechanics. Any gaps in knowledge will attempt to be identified. Other models of vertebral subluxation are not under question in this review. This paper is meant only to explore the possible relevance of the hard bone-soft nerve model and related colloquial terminology of vertebral subluxation.

Leach[1] discusses previous research to assess the possible clinical significance of such phenomena. His conclusions on each theory have a similar theme: that each may occur, that hard neurologic signs would be present, but that neither occurs frequently as part of the subluxation concept and therefore are not relevantly important as part of chiropractic theory. Leach talks of compression in relation to muscle motor function and

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sensory changes but not to health status. He makes a few exceptions as regards cord compression and its relation to certain rheumatoid conditions and Sudden Infant Death Syndrome (SIDS). Haldeman[2] echoes Leach's views of the unlikeliness of compression and its relief from an adjustment. In reviewing the Mercy Conference guidelines[3] there is no mention of neural involvement as the cornerstone of the vertebral subluxation. Banks et al[4] and Gottlieb[5] explore the implications of birth trauma with possible cord and brain stem damage and future health status including SIDS, integrating the concept of chiropractic care as a plausible intervention. Gutmann[6] expresses that upper cervical spine exams should be mandatory on all difficult deliveries as well as with any developmental health problems because of the relationship between the atlanto-occipital joints, atlantal nerve compression, and the brainstem. In a recent review of models of vertebral subluxation, Kent[7] states that "...the nerve root compression hypothesis is far from obsolete." Konno[8] has shown that a pressure of 10-mm Hg, just slightly more than the initial threshold of touch, applied intermittently, is enough to decrease muscle action potential (MAP) approximately 20%. The interesting part of Konno's work is that the MAP decrease remained constant and the pressure applied was continuous, not intermittent.

A definite dichotomy of thought exists on the subject of nerve compression and its part in the vertebral subluxation theory. This paper will focus particular attention on the pathological changes that take place in the spinal nerve when exposed to varying degrees of compression. Once a general picture of the pathophysiological mechanisms has been established, a review of the literature of the current understanding of biomechanics and the effects at the level of the intervertebral foramen will be undertaken. Once the evidence has been presented, an integration of the findings will be done with textbook knowledge. In this way a better understanding of the clinical significance of spinal nerve compression may be achieved.

Methods

Literature searches were conducted on the World Wide Web using the Pub Med Medline[48] search engine. During the time period of searches, the format of Pub Med changed. All of the searches were done utilizing the old Pub Med in the advanced format. The main search term used was "root, spinal nerve" which generated 16,122 hits under the MeSH search category. Additional search terms under the MeSH category were "compression" which generated 2052 hits, "edema" which generated 23 hits, and "immune" which generated 120 hits. The final search used was "axoplasmic flow" which generated 5,739 hits and then adding "compression" which yielded a reduction to 30.

Two articles, deemed particularly relevant, Alleyne[41] and Farmer[43] were then entered into the search engine and the [see related articles] option was utilized. For the first, 116 hits were generated. The latter generated 182 hits.

There were no limitations set on the years searched. Articles were selected that dated from the late 1980s or sooner. Older articles would only be selected for inclusion if relevant recent research could not be obtained. In reviewing the hits generated the title was the first exclusionary item. If the title did not convey a relevance to the review, no further investigation was attempted. When the title conveyed some relevancy to the present study the abstract was read. The main inclusionary criteria for the abstracts were evidence of compression alterations of neural function under low-pressure conditions.

Review of Literature

As a starting point it is necessary to review the recent findings about the effects that compression has on the nerve root, including the dorsal root ganglia, dorsal and ventral ramus, and the dorsal and ventral roots. As a general rule any experiments done on animals other than humans automatically entails a certain leap of faith that the findings are applicable to the human species. Certain minute details of anatomic and physiologic relationships could easily be missed that change the impact that compression has at the vertebral level in man.

Another critical point is that most nerve compression studies deal with the response of motor nerves of muscle and peripheral sensory nerves. The response of the internal organs and the fibers associated with them may have different qualities that change the sequelae of compression.

In human studies done on cadavers the sample population's mean age is typically skewed in the direction of the elderly. Correlations drawn to the population as a whole are then suspect. There is no way to know for sure when certain conditions that are found actually began. There is also no way to know if they are preventable. Also with cadaveric studies, when the investigators test for statistical significance they are testing for significance within the population of cadavers studied. It is not believed that the investigators test for significance as it relates to the whole population i.e. are findings in 10 cadavers relevant enough to draw conclusions to the whole population?

A final general critique of compression studies is that there are different methods of quantifying compression. A number of quantification methods exist such as size estimates of the nerve root; comparing the linear change in diameter from initial (100%) to final (25%, 50%, etc.), pressure estimates; comparing no pressure (0 mm Hg) to greater pressures (10, 50, 100 mm Hg, etc.), and weighted estimates; where a particular pressure is represented as a function of mass (i.e. 10g). These are valid quantifications, but ways of converting them for comparison are unknown.

Direct Compression

Olmarker et al.[9] examined the anatomic differences of the pig cauda equina. They found that in the smaller animals the spinal nerve had a subarachnoid space up to the point of exit from the spinal canal. This is different from a human model in that these nerves are freely floating in the cerebrospinal fluid (CSF). The arterioles and venules of the pig at this level are located outside the endoneurium but enclosed by the root sheath. Inside the endoneurium only capillaries were present.

In an earlier work, Olmarker et al.[10] studied compressive effects on the pig cauda equina. This experiment did not compress the dorsal root ganglia (DRG) and was actually caudal to the DRG. The compression took place at the level of C_{x1} (1st coccygeal nerve root). They used a balloon apparatus for the compression model.

The findings in this study identify the average minimum pressure required to stop the vascular flow. The arterioles required an

average 127-mm Hg (SD 18 mm Hg), the capillaries 40 mm Hg (SD 6 mm Hg) and the venules 30 mm Hg (SD 10 mm Hg). A decrease in the size of the vessels was noted at 10-50 mm Hg below the level for cessation for each. Arterioles began to pulsate at roughly 30-40 mm Hg below the occlusion pressure, this did not happen to the arterioles or venules.

Upon re-circulation hyperemia was noted and was more pronounced in the venules. It was present in the first minute of re-circulation and gradually diminished over a 5-10 minute period. Blood vessels were identified afterward that were not previously visualized microscopically. Edema developed in the first 10 minutes, but only in the vessels compressed for 2 hours at 50 or 200 mm HG.

Continuing with the same model of compression, Olmarker et al.[11] studied the effects of methyl glucose transport as altered by compression. They define R as the radioactivity of the specimen (the nerve root) and R_{co} as the radioactivity of the normalized blood. A figure is derived, R/R_{co}, which represents the percent departure from normal concentrations. This figure offers a concept of decreased nutritional transport. The methyl glucose molecule has a particular size that may or may not relate to other forms of nutrition that effect the function of nerve roots, assuming of course that the sizes of the particulate matter are relevantly effected by compression. One aspect that is not addressed is the DRG and effects on its nutritional support system. This importance will become more relevant with other research findings about distinct differences of the effects of compression on the spinal nerve and the DRG.

The nerves were exposed to rapid (<1.0 seconds) and slow (20 seconds) onset compression at 10, 50, or 200 mm Hg. Just the application of the compressive device brought about a significant reduction of the R/R_{co}. The control R/R_{co} was measured at 79% and the differing levels of compression were compared to this figure giving a percent reduction from control. The sham procedure elicited an R/R_{co} value of 69%; this is a 14% reduction. In the slow onset mode of compression, R/R_{co} values were 65%, 40%, and 27% for the 10, 50, and 200 mm Hg respectively. This represents a reduction from control of 18% for 10 mm Hg, 49% for 50 mm Hg, and 66% for 200 mm Hg. In the rapid onset compression group the R/R_{co} values were 56%, 27%, and 4% for the 10, 50, and 200 mm Hg respectively. This represents a reduction from control of 29% for 10 mm Hg, 66% for 50 mm Hg, and 95% for 200 mm Hg.

Olmarker et al.[12] studied the edema formation related to nerve root compression. Using pigs as mentioned before, pressures of 50 and 200 mm Hg were applied for 2 minutes, 10 minutes or 2 hours. Their results show edema formation to be more pronounced in rapid onset compression than in slow onset compression. Edema is quicker to form with a higher pressure. Most importantly the edema formation appears to be dependent on the duration of compression. Unfortunately they only kept the compression for a maximum of 2 hours and the minimum pressure studied was 50 mm Hg. Too much speculation would be involved to draw conclusions for lesser compressions and edema over longer periods of time, say 10-20 mm Hg for days or months. In the discussion the authors talk of a compartment syndrome occurring in the IVF caused by edema from the compression injured nerve root. The danger with this type of pressure chamber is that the DRG is in the IVF and houses all the sensory cell bodies. In this study with the pressure being applied at the cauda equina the edema is likely to be cleared away better by the surrounding cerebrospinal fluid.

Turning their attention to impulse propagation, Olmarker et al.[13], using their same mode of testing on pigs, found that the amplitude of muscle action potentials (MAPs) responded similarly with greater reductions at greater pressures and greater reductions with quicker onsets. Their lowest pressure level studied, 50 mm Hg, did not exhibit statistical significance for reduction in amplitude of MAPs at either rapid or slow onset. Here again the study is limited because they only held the compression for 2 hours and the lowest pressure level used was 50 mm Hg. Although the trend in their data would suggest that perhaps lower pressures for longer time frames may induce a reduction in amplitude of MAPs, that conclusion cannot be drawn from this study.

Cornefjord et al.[14] applied ameroid constrictors just cranial to the DRG of the first sacral nerve root in pigs mimicking a gradual onset of compression over 20 days. They used 3.5 or 2.5 mm constrictors that compressed the nerve root with the slow absorption of surrounding fluids. The authors go to great lengths to explain the constrictors and analyze the time frame it takes for them to reach maximum constriction. They do not however explore this relationship with the size of the nerve root. There is no way to arrive at a quantified compression as it relates to a pressure reading.

Both size constrictors were found to reduce nerve conduction velocity (NCV) significantly within 1 week with a slightly greater reduction seen at 4 weeks. Most animals underwent severe changes. Earlier on, at 1 week, endoneurial bleeding and signs of inflammation were common. At 4 weeks, epidural proliferation of fibroblasts and capillaries were more common. With 3.5 mm and 2.5 mm of compression after 1 week, there was severe splitting of the myelin sheaths, dense axoplasm, a severe reduction of axonal volume and myelin sheaths with complex wrinkling proximally and distally to the compression. The authors feel that although they could not rule out nerve fiber loss, the observed nerve tissue shrinkage was mainly due to atrophy.

Konno et al.[8] working with a model similar to Olmarker,[9-13] placed one balloon at C_{x1}-C_{x2} and the other at C_{x2}-C_{x3} and compressed the pig's cauda equina. Two modes of compression were utilized. The first was termed intermittent where both balloons were inflated and deflated simultaneously. The second mode termed continuous/intermittent where the cranial balloon was left inflated and the caudal balloon was inflated and deflated. The testing was done for 8 cycles over 2 hours. Pressure onset applications over 15-20 seconds of either 10 or 50 mm Hg were used. The cycles were 10 minutes inflated and 5 minutes deflated. MAPs were recorded every 5 minutes.

During intermittent compression, decreases of roughly 30±21% and 31±22% in amplitude of MAPs for the 10 and 50-mm Hg groups respectively were seen. The difference from control was significant but the difference between the two was not. The increase in pressure to 50 mm Hg did not change the reduction in the intermittent group. They elicited close to a 100% reduction of MAP amplitude in the 50-mm Hg continu-

ous compression mode. The difference in the 50-mm Hg group between the intermittent and the continuous groups was considered to be statistically significant.

Mao et al.[15] published experimental data on double level compression. They used dogs with two balloons for compression, one at L_7 and the other at S_1. They used 10 mm Hg of pressure and recorded NCVs at 1 week and 1 month.

The NCV was significantly lower after 1 week and 1 month in the double compression. The NCV at 1 month was greater than at 1 week indicating a recovery had occurred that was statistically significant. The measurements taken at the cranial compression zone exhibited a similar pattern of recovery. In the caudal zone, the reduction of NCV was significant at 1 week and 1 month. There was no statistically significant difference between 1 week and 1 month. This implies that at the caudal zone the recovery was either not occurring or not occurring as fast. They attribute this to an adaptation to the applied pressure by the nerve tissue and vascularization.

Kikuchi et al.[16] in a similar double level compression of the dog cauda equina demonstrated a tolerance to additional compression. They applied compression at 10 mm Hg for 1 week then applied additional compression with a second balloon at 50 and 100 mm Hg. Their results show that the nerve tissue was less susceptible to the additional compression.

Kim et al.[17] studied the dog cauda equina and exposure to chronic compression. They compressed the 6th and 7th lumbar segments by 25%, 50%, or 75% of their original size. As previously mentioned, no correlation to a pressure can be made.

The sensory evoked potentials (SEPs) in the 25% group showed a minor delay in latency postoperatively that recovered by 2 weeks and remained so through 3 months. SEPs in the 50% group showed marked increases in latency and decreases in amplitude postop there was a limited recovery in both at 2 weeks but no more so at 3 months. In the 75% group, SEPs completely disappeared postop, returned at 2 weeks, but still were markedly below normal and remained so through 3 months.

The motor evoked potentials (MEPs) in the 25% group showed a 22% increase in latency postop that recovered by 2 weeks. The 50% group had a complete absence of MEPs postop. The MEPs returned at 2 weeks but were still decreased in latency even at 3 months. MEPs in the 75% group were lost at 2 weeks 3 of 6 dogs showed a return at 1month, 4 of 6 by 2 months and 5 of 6 by 3 months. There was paralysis of the tail and urinary incontinence in all 6 dogs of the 75% group, 2 had fecal incontinence.

The authors make note that in the presence of spinal stenosis as evidenced by radiograph, CT scan, or MR image, the symptoms reported by patients do not necessarily correlate with the location of compression.

Echoing the thoughts of that statement Hidalgo-Ovejero et al.[18] produced a report of a patient with L_5 and S_1 related symptomatology. The findings at those levels were minimal to justify the chief complaint of the patient. MRI, CT myelography, and CT scanning revealed stenosis affecting the L_1-L_2 and L_2-L_3 levels. It is hypothesized by the authors that the stenosis cranially actually affected the L_5 and S_1 nerve roots. This caused the patient to perceive symptoms of a lower level lesion.

Takahashi et al.[19] examined 34 human patients with disc herniations. They measured the pressure applied to the nerve roots by using a transducer. They admittedly acknowledge the fact that the laminectomies performed to get access to make the measurements may very well have inadvertently decreased the pressure readings. The pressure reading varied from 7-256 mm Hg. There was no relation between magnitude of pressure and gender, age, duration of symptoms, limitation of straight leg raise, and crossed leg pain. Trunk list did have a significant higher-pressure correlation as did neurologic deficits. Paralysis, foot drop, and bladder dysfunction had very high-pressure correlations. An incidental note of their findings was that in the prone position disc herniation created higher pressures than canal stenosis.

Hanai et al.[20] took recordings of the wide dynamic range (WDR) neurons in lamina 4-6 at L_7 of cats. They used approximately 40g of pressure applied by a clip for 3 minutes to the dorsal root or DRG. With compression to the dorsal roots there was an initial burst of activity followed by a rapid reduction of firing in the WDR neurons. In the DRG, compression maintained repetitive firing throughout the compression period and for about 20 seconds after the release of pressure there remained a mild increase of firing. The DRG, in response to compression, therefore evokes a prolonged level of high frequency responses in WDRs that is not seen in dorsal root compression.

In response to brushing with compression to the dorsal root and the DRG, the receptive field expanded and the animals' responses were enhanced. In response to noxious pinch in the dorsal root and DRG compression, the receptive field size and the animal's responses remained unchanged. The number of spikes evoked by both noxious pinch and brushing were the same for the dorsal root and DRG compression.

Ramer et al.[21] performed loose ligation or complete ligation of the sciatic nerve in rats unilaterally. In the complete ligation group they also transected the sciatic nerve distally to prevent retrograde axoplasmic flow. They were looking at the time course of sympathetic nerve sprouting and changes in threshold for withdrawal to mechanical and thermal stimuli. The rats were tested postop at 4, 7, 14, 21, and 28 days. Four days after the chronic constriction injury there was sympathetic sprouting in the DRG. In the transected nerves however, the sprouting was not detectable until 14 days. These results were much more pronounced on the ipsilateral DRG but were seen to occur contralaterally. The authors feel that the difference may be due to the availability of products of Wallerian degeneration. These products would theoretically be available by retrograde axoplasmic flow which couldn't occur in a severed nerve.

In the other area investigating changes in threshold the authors found that from postop to day 28 there were increases in sensitivities to thermal and mechanical stimuli. A significant change for both occurred by 14 days postop with mechanodynia preceding thermal sensitivity.

In another study using rats, Hu et al.[22] studied the effects of acute and chronic nerve compression along with the effects of just inflammation. At the L_5 level they placed a small stainless steel rod into the intervertebral foramen (IVF). The rod was removed after 30 seconds for the acute group and was left for a month in the chronic constriction group. In the third group they injected carrageenan (1%) into the IVF to cause inflammation.

No spontaneous removal or casting off of body parts (autoto-

my), changes in gait, or changes in posture occurred in any group for the month following surgery. Cutaneous hyperalgesia was present in the chronic compression group, measured as a latency of withdrawal to noxious heat, from day 5 to day 42 postop. The inflammation group showed a significant change of increased hyperalgesia from day 5 to day 21 postop. The acute compression group showed no significant change in hyperalgesia. The authors do admit that the measured changes were significant most of the time, indicating that at certain unmentioned time periods the changes were not of significance.

The authors found spontaneous activity to be present in 21.5% of fibers in the DRG of the chronic compression group. All these fibers were deemed to be myelinated on the grounds that they displayed conduction velocities of 4.4-38.5 m/s. In contrast only 1.98% of A-type fibers exhibited spontaneous activity in the control group. The patterns of activity appeared to fit into three categories. The first was a regular interval of repeated interspikes 33.3% of fibers. The second was a pattern of irregular interspike intervals 24.7% of fibers. The third was a pattern of bursting activity with periods of silence, which could either be regular or irregular 42% of fibers. When a 5 g weighted mechanical force was applied to the normal DRG group and the chronic compression DRG group, the number of units responding were not significantly different. The response of the fibers from the DRG compression group were significantly greater in magnitude and duration.

In a similar model of chronic compression in the rat, Zhang et al.[23] inserted stainless steel metal rods into the L_4 and L_5 IVFs to cause a chronic compression injury to the DRG. Spontaneous activity was recorded in large size cells of the chronically compressed DRG. This was significantly higher than in controls. The spontaneous firing continued for more than 3 minutes. There was evidence that some of the spontaneous large size cells were of cutaneous origin. For the large and medium cells the mean threshold current was 50% lower and was 30% lower in the small sized cells. In the medium cells, but not the large or small, the duration of the after hyperpolarization and the peak amplitude of the action potential were significantly greater. Testing the peripheral axons of the compressed neurons revealed no loss of axonal conduction. There was also no difference in mean conduction velocities between DRG and peripheral nerves.

There was mechanical hyperalgesia present in all rats at day 1. This lasted through all 14 days of testing. There was no evidence of autotomy or deformation of the paw. There was evidence of crossover but this was not statistically significant.

Histological analysis revealed that the rod and DRG were often covered with a connective tissue enriched with blood vessels which proved difficult to remove. On the surface of the DRG at the area of injury there was an increase in vascularization. There was also an increase in the amount of "noneuronal" cells around each neuronal cell body.

Song et al.[24] repeated the same protocol as Hu[22]. They did not have an inflammation group. None of these rats exhibited autotomy or abnormal nail growth. They exhibited an ability to escape a prod with gait of a normal fashion suggesting there were no permanent motor deficits. Both of the groups showed abnormalities of gait and posture at day 1 postop. While this lasted in the acute group for only a few days, in the chronic group it typically lasted for 2 weeks. For some of the rats in the chronic compression group the abnormalities lasted up to 5 weeks, the end of the observation period.

In the chronic compression group the withdrawal threshold response to pressure and heat decreased significantly below baseline at day 1 ipsilaterally. It remained there for rest of the testing period (35 days) except at day 35 for the heat withdrawal. Tactile allodynia (withdrawal to a cotton wisp) rose significantly at day 1 and then gradually declined back to baseline by day 20. There was a significantly greater withdrawal to a cold plate but no mention of the time frame. The ipsilateral and contralateral skin temperatures were not significantly different.

Nerve fiber analysis revealed spontaneous activity that was significantly greater in the A and C type fibers. This was more evident during the first week than it was during the remaining period of up to 35 days. After sciatic nerve transection the spontaneous activity remained in the A and C type fibers. After dorsal root transection proximal to the DRG they became silent. The authors suggest that this activity therefore arises from the DRG and this contributes to the cutaneous thermal and mechanical hyperalgesia observed.

Tabo et al.[25] compressed the dorsal roots of rats central to the DRG at L_4-L_6 with silk ligatures. They were loosely ligated either once or twice. The mechanical withdrawal threshold showed a significant reduction on the operated side in both groups beginning at day 3. This peaked from the second to fifth week then gradually recovered. On the contralateral side a delayed threshold reduction was also observed. There was a significant increase in hypoalgesia (thermal paw withdrawal) beginning immediately after surgery and recovering after 1 week. No crossover of hypoalgesia was noted. There was an ipsilateral reduction in weight bearing for 5 weeks. For 1-week post surgery there was a reduced force of hind limb withdrawal to noxious heat. Later at weeks 5-9 on the contralateral paw there were greater forces of hind limb withdrawal to noxious heat. Changes in response to the application of cold plates were not present.

During the time the animals were exhibiting thermal hyperalgesia and mechanical sensitivity, neuronal responses were no different than those of the animals where surgery was performed with no silk placements (sham procedure). This was at the 5-week period and mechanical sensitivity had increased bilaterally. At 22 weeks the authors observed increased spontaneous firing and enlarged mechanical receptive fields ipsilaterally in the dorsal root compression group. The mechanical hypersensitivity had resolved contralaterally and was decreasing ipsilaterally at this time. The changes seen in the receptive fields and the spontaneous firing were too slow to account for the mechanical and thermal sensitivities. There were also no contralateral neuronal changes.

Sugawara et al.[26] analyzed the excised dorsal roots and DRG of dogs exposed to mechanical compression or hypoxia. These experiments were done in an oxygenated artificial CSF. The mechanical thresholds for compression to elicit firing were much lower in the DRG than in the dorsal root (36.0g and 73.5g respectively). The DRG firing was also of a longer duration. Under hypoxic conditions the DRG showed firing and decreased thresholds for mechanical stimulation. The dorsal root showed similar firing under hypoxia but no change from

mechanical stimulation. These changes disappeared upon the reintroduction of oxygen.

Yoshizawa et al.[27] compressed the 7th lumbar nerve root of dogs with a 5mm long silastic tube and did histologic and physiologic analysis as well as assessment of the blood-nerve barrier function. The silastic tube had an internal diameter of 3 mm. At 1 month there was thickening of the dura and arachnoid membranes that the authors reported corresponded to the alteration of the blood-nerve barrier. Endoneurial fibrosis and Wallerian degeneration became obvious at 6 months. NCV did not change as long as there were some large MFs. When the large MFs disappeared at 12 months there was a concurrent decrease in the NCV. The amplitude of the compound action potentials decreased along the same time frame as the decrease of the large MFs. Nerve root edema was observed from 1-12 months. There were 2 pathways for intraradicular edema formation: first by paracellular transport by opening of the tight junctions and second by increased transcellular vesicular transport. The authors concluded that compression basically causes a degeneration of nerve fibers. Repeated micro-traumas are responsible for the damage done. Nerve fibers begin regeneration as soon as they are damaged. When comparing recently damaged fibers to those damaged long ago the latter fibers have regenerated to a far greater extent.

Boulu et al.[28] proposed mechanisms of progression for acute and chronic compression based on a compilation of research. The authors feel acute compression begins with venous congestion followed by intraneural edema and reduced axonal transport. Finally complete blood stasis and motor and sensory deficits. Chronic compression starts with the dura and arachnoid showing signs of hyperplasia. Wallerian degeneration begins and there is a reduction in the large myelinated sensory fibers. Ephapsis from large myelinated proprioceptive neurons to nociceptive neurons, fibrotic build up and Wallerian degeneration occur at 6 to 12 months.

In young and aged rabbits Toh et al.[29] studied compression of the cauda equina at S_2-S_3. They utilized 3 grades of compression. Grade 1 where they compressed 1/9 of the anteroposterior (AP) diameter of the cauda equina, grade 2 with 2/9 of the AP diameter compressed, and grade 3 with 3/9 of the AP diameter compressed. Their acute compression was applied fully within the first month. Their graded compression was applied gradually at 1 and 2 months. It should be noted that the application of compression in this study was of a far longer time duration than most other studies. Upon examination by light microscopy acute compression showed a larger increase in the number of small MFs of the younger rabbits compared to the older rabbits. This process of an increase in the number of small MFs is associated with regeneration. Unfortunately the authors do not elaborate on the exact classification of the small MFs. Degenerative findings such as separation of the axon sheath, circumferential cleavage, and/or infolded loops of myelin were observed more frequently in the aged group with graded compression. The increase of the number of small MFs after graded compression in the aged rabbits was less than that after graded compression in the young rabbits. The findings after 3 months of graded compression in the aged rabbits were similar to those of the acute compression in the young rabbits. The findings would indicate that there is a lack of the regenerative process of small MFs associated with aging.

Oishi et al.[30] inspected 50 human cadavers without obvious neurological or neuromuscular diseases. The mean age was 77 years old. A group of the cadavers were assigned the status of radiculopathy. The criterion for the radiculopathy group was a finding of an IVD half the size of surrounding IVDs at C_5-C_6 on x-ray examination. The radiculopathy group showed an increase in the number of small MFs in the ventral and dorsal roots. There were also findings of narrowing of the lumen of the endoneurial vessels and perineurial fibrosis. There were decreases of large axons as well as total transverse fascicular area (TTFA), large MF number, and the ratio of large MF to total MF. Increased were the axon fiber ratio, axon myelin ratio, MF fiber density, and the ratio of small MF to total MF. There were no differences in the axon diameter or total MF number. In the ventral group there were greater numbers of small MFs and small radius axons. The authors attribute the decrease in TTFA of the ventral root to the large axons becoming smaller or atrophic and the myelin sheath becoming thinner relative to axon size. The pathologic changes were similar in both the ventral and dorsal roots. There was an absence of active axonal degeneration as well as ongoing demyelination and loss of MFs. There was a significant decrease in the number of large MFs without decrease in the number of total MFs. On the indented side of the axon the myelin sheath was thinner in relation to axon size as well as fewer large axons.

Examining specimens obtained during surgical decompression for trigeminal neuralgia Rappaport et al.[31] found a significant disruption of tissue ultrastructure. There were few remaining axons in the area of most severe damage. Those that did remain were completely demyelinated. There were more surviving axons adjacent to these areas but these showed severely disrupted myelin sheaths. The authors referred to these areas as zones of demyelination or dysmyelination. These zones occupied a large majority or all of the cross sectional areas of the samples. The zones of demyelination and dysmyelination were interdigitated rather than continuous. Collagen fibrils showed a striking overproduction in the extracellular matrix. There were areas of pure condensed collagen forming one or more massive clumps. In the zones of demyelination there was a reduction of axon population. Due to the denuded axons there were signs of probable cross talk between fibers. Direct exposure to the extracellular medium of the axolemma was also present.

The authors categorized the zones of dysmyelination into four main forms. 1) Thinning of the myelin sheath in relation to the axon size of swollen fibers. 2) "Crenulated myelin invaginations, and swelling of glial cytoplasm associated with the inner mesaxon." 3) "Atrophy or disappearance of the ensheathed axon." 4) Areas interpreted as regenerating sprouts in surviving myelin sheaths usually of very large fibers.

They identify 3 key features of trigeminal neuralgia. The first is neural triggering where there is a persistence of a painful sensation after a momentary stimulus indicating an ongoing self-sustaining neural firing process. The second is neural amplification where the activity triggered is more intense than the triggering event. And the third is the stop mechanism which is when spontaneous cessation of the neural activity occurs. These findings are relevant because in 8 of 12 of the patients in this

study there was clear evidence of arterial compression of the trigeminal nerve. The authors do not offer an explanation as to the cause of the arterial compression.

Chen[32] presents the findings of 8 patients with a cervical disc protrusion and concomitant C_2-C_3 compression as verified by CT myelography or MRI. The signs and symptoms present were numbness of the arms and hands (6/8), neck pain (2/8), lack of fine motor control of the hands (8/8), dysesthesia or hypesthesia to pinprick (7/8), allodynia or hyperesthesia (1/8), proprioceptive loss (1/8), tingling and numbness of the fingers that begins distally and moves proximally (5/8), difficulty walking and loss of balance (3/8), and motor deficits (6/8). Although not tested for statistical significance this list may give a rough outline of the likelihood of encountering these signs and symptoms with cervical nerve compression. Unfortunately the author does not mention symptoms that are not of muscle or sensory origin.

Gallant[33] studied the effects of compression on axoplasmic flow and membrane potential in squid axons. Squid axons are large (0.3 to 1.0 mm in diameter) unmyelinated fibers.The axons were studied *in vitro* with compression from a capillary tube applied for 10 seconds at pressures of 0.5, 5, 20, and 100 g/mm.

The 0.5 g/mm and 1 g/mm pressures compressed the axon by up to 90% which was reversible upon removal of the capillary tube. The addition of a 1 g/mm pressure is not explained by the authors. The axons were completely compressed in the 5, 20, and 100 g/mm groups and recovery was slow. Axoplasmic evaluation with an 80 power binocular microscope revealed a progressive amount of damage. At 0.5 g/mm the axoplasm was intact, only fine threads remained between the 2 halves after 5 g/mm, and by 100 g/mm the axoplasm was often completely severed. The 0.5-g/mm pressure did not effect axonal transport. Progressive worsening of the axoplasmic flow occurred from 5 to 100 g/mm of pressure.

Membrane potential testing revealed that during the compression all action potential activity stopped for all compressions from 0.5 to 100 g/mm pressures. Recovery after compression was complete for the 0.5 g/mm group and 88% for the 5-g/mm. Some of the 20 g/mm recovered the action potentials and none of the 100-g/mm group recovered.

Dahlin et al.[34] studied the effects of compression on axonal transport in the vagus nerve of rabbits. The nerve was subjected to compression from an inflatable compressed air system at approximately 5-mm distal to the nodose ganglion for 8 hours. The placement of the compression apparatus without inflation (sham procedure) brought about a minor transport block that was not statistically significant. The findings for 20 mm Hg of compression show a slight accumulation of both retrograde and anterograde transport. Retrograde transport was found to be significantly different from sham but the anterograde was not. Whether or not the anterograde transport was significantly different from a control is not known because the authors failed to test without the chamber applied. The 30-mm Hg group showed marked inhibition of anterograde and retrograde transport both proximally and distally to the compression zone.

Dahlin et al.[35] produced another study on axonal transport. The vagus nerve of albino rabbits was the subject of either ligation or compression. In the ligation group 2 ligatures were attached 20 and 40 mm from the ganglion. A small air compression chamber was attached roughly 20-30 mm from the ganglion for the compression group. It is not made clear by the authors whether these were applied cranially or caudally. In the compression group a ligature was made 10-15 mm distal to the compression chamber. In the ligature group a marked accumulation of the radiolabeled proteins occurred proximal to the proximal ligature and around the distal ligature. The compression chamber alone (sham) caused no accumulation. The authors back up their claim of "no accumulation" with measured low accumulation results which are not tested for significance against a control. In examining the transport graphs there is a clear difference between the sham compression graph and the normal vagus nerve graph. Why this was not elaborated on by the authors is unknown. It seems entirely possible that the compression apparatus imparted some pressure from its application in and of itself. For slow axonal transport the 20 mm Hg group showed a minor accumulation but this was not statistically significant when compared to sham procedure. Again there is no test against a control. At 30 mm Hg of pressure for 8 hours there was a marked accumulation of the slow axonal transport labeled proteins in the area of compression. For the radiolabeled proteins of fast axonal transport, 20 mm Hg was sufficient to induce a significant block.

Spinal Biomechanics

According to the definition in the Association of Chiropractic Colleges Paradigm Statement,[36] the definition of a vertebral subluxation "...is a complex of functional and/or structural and/or pathological articular changes that compromise neural integrity and may influence organ system function and general health." Having already reviewed the pathophysiologic changes associated with compression, it becomes evident to review the recent literature findings that pertain to "articular changes" in such a way that they might affect neural function at the nerve root level. Only such findings that are complimentary to or in contrast to general anatomical knowledge will be discussed.

Xu et al.[37] dissected 20 cadavers, 10 male and 10 female aged 62-74 years old. Among the findings that are of significance in the cervical spine the dural sac of the nerve roots abuts the medial border of the superior pedicle on its course to the IVF and in the IVF the nerve root lies directly on top of the inferior pedicle. Given the age of the cadavers in this study it seems relevant to include the average disc height but this was not done. It may be possible that the disc heights were significantly shorter than a younger population and if so the nerve roots may have been slightly more elevated in their course.

Tanaka et al.[38] examined 18 cervical spines from cadavers (10 men and 8 women) aged 52 to 98 years old. Their findings were that the IVF was shaped like a funnel, the medial portion being narrowest and widening as it coursed distally from the vertebral canal. The nerve root's shape was just the opposite; it was conical and widest centrally. The ventral root lay inferior to the dorsal root in the IVF and was 2/3 the size of the dorsal root. The nerve root, when removed from the dura, was 25% to 33.3% the size of the foramen in diameter. The fibrous tissue of the posterior longitudinal ligament blended with the fibrous tissue of the nerve root and laterally was continuous with the fibrous tissue of the vertebral artery. The DRG and the segmental nerve

were found to lie posteriorly and medially to the vertebral artery. There were 30 of 144 nerves found to be compressed. Protruded discs and uncovertebral arthroses were responsible for 18 of these anteriorly. The other 12 were compressed posteriorly by the superior articular process and the periradicular fibrous tissue. The compressions took place at the entrance zone of the IVF where the root was largest in comparison to the narrowest region of the IVF. The different compressive agents varied due to variations of anatomical structure for each individual and each level involved.

Ebraheim et al.[39] confirm the finding of the fibrous connective tissue sheath at the cervical IVF. They performed dissections on 28 cadavers (11 male and 17 female) 47-91 years old. A fibrous tissue was found to connect the nerve root, vertebral artery, uncinate process, and the uncovertebral joint together as a unit.

Alleyne et al.,[40] in studying the cervical spine, also demonstrated this fibrous connective tissue and its interrelationship with the vertebral artery, DRG and ventral root. They studied the spine of 10 adult cadavers reported to be "all elderly." The authors do not offer any comment on the condition of the cadavers (i.e. whether they ruled out specimens due to severe degenerative conditions). They found 73% of the DRG/ventral root complexes compressed from C_3-C_6 by the vertebral artery, C_3-80%, C_4-70%, C_5-75%, and C_6-67%. The authors do not comment on the levels below C_6. A build up of collagenous tissue is reported at the sites of compression.

Lu et al.[41] examined the loss of IVD height and its effects on the IVF. Their analysis was done on 16 cadavers (6 male and 10 female) 56-74 years old, and without significant degenerative spondylosis. They studied the C_4-C_5, C_5-C_6, and C_6-C_7 disc spaces in the neutral position. The measurements for the IVF were made through the midpedicle. This probably corresponds closely to the area Tanaka et al.[38] found to be the narrowest in the IVF. The results showed that a 1mm narrowing of the IVD produced a reduction of foraminal cross-sectional area of 20-30%. All the measurements were taken with the removal of all soft tissue structures in the IVF.

Utilizing 5 cadavers Yoo et al.[42] examined the changes in IVF dimensions associated with flexion/extension and flexion/extension and rotation. The cadavers were 35-70 years old without gross defect, previous injury, or surgery. They measured the narrowest dimension of the IVF with "finely graded circular blunt probes." Their reference for levels tested states C_5-C_7. It is not clear whether they implied the superior or inferior IVFs of those levels. They found that with flexion from neutral to 20-30° there was a statistically significant increase in the size of the IVF and with extension, again from neutral to 20-30°, there was a statistically significant decrease in the size of the IVF. The tests for flexion and extension at 10° did not show a statistically significant change in size. At 30°, there was a 13.2% decrease of the IVF in extension and a 10.6% increase in the IVF in flexion. The combination of rotation and flexion/extension did not produce a statistically significant change in foraminal dimension. The trend though was that in extension, ipsilateral rotation increased the IVF and contralateral rotation decreased the IVF, and in flexion ipsilateral rotation decreased the IVF and contralateral rotation increased the IVF.

Farmer et al.[43] studied the pressure generated in the cervical IVFs at C_5-C_7. They used 8 fresh cadaver specimens, one 40 years old, the rest 60-70 years old. They were selected to exclude any signs of spinal disease or knowledge of previous spinal or shoulder surgery. A balloon filled with saline was inserted into the IVF. Once inserted, the pressure recording system was zeroed with the neck in neutral. They measured the pressures generated during 20° and 40° of flexion and extension with and without the arm abducted in the coronal plane. In extension they found statistically significant increases in pressure at all 3 levels. At 20° of extension the pressure increases were 33.4 mm Hg at C_5, 36.9 mm Hg at C_6, and 22.6 mm Hg at C_7. At 40° of extension the pressure readings were +41 mm Hg at C_5, +38.9 mm Hg at C_6, and +39.9 mm Hg at C_7. In the flexion studies only C_5 registered results that were statistically significant. They were +16.3 mm Hg at 20° and +26.3 mm Hg at 40°. The results of flexion on C_6 and C_7, although not found significant, were -2.4 and -0.6 mm Hg at 20° and 40° respectively for C_6 and +13.9 and +10.6 mm Hg at 40° respectively for C_7. The arm abduction series yielded mixed results. Here again the extension series were statistically significant. They report the results as an average for all three levels, -18.8 mm Hg at 20° and -27 mm Hg at 40°. In the neutral position a clear pattern of reduced pressure emerged but the changes were not statistically significant (i.e. reduced less than 10 mm Hg). The flexion series were also not significant. The pattern was mixed. At C_5 a general small increase in pressure, at C_7 a general small decrease, and at C_6 a decrease at 20° and an increase at 40°.

Turning to the lumbar spine, Hasegawa et al.[44] studied the spines of 18 fresh cadavers, 35-86 years old. They found the L_5-S_1 disc height to be significantly thinner at the mid- and posterior section than the rest of the lumbar spine. The cross-sectional areas of the IVFs were not significantly different for any levels of the lumbar spine. The cross sectional areas of the nerve roots were largest at L_5-S_1 and progressively got smaller moving cranially. As would be expected then, the ratio of nerve root/IVF based on cross-sectional areas grew larger as one moved inferiorly from L_1-L_2 to L_5-S_1. Out of 100 foramina studied there were 21 that showed signs of the nerve root being compressed. The agent of compression was bulging of the inferior ligamentum flavum anteriorly in all cases. The exclusion of cadavers with degenerative changes such as osteophytes or disc bulge explains why these causes of compression are not represented in this population. The largest posterior disc height observed with a compressed nerve was between 8 and 9mm. The largest IVF height observed with a compressed nerve was between 21 and 22mm. The authors arbitrarily assign a critical dimension based on the prevalence of compression versus the total number of specimens at a certain height. The critical height for the posterior disc was assigned at 4mm and for the IVF was 15mm.

Inufusa et al.[45] studied the effects of flexion/extension on the lumbar spine. The cadavers ranged from 35-87 years old. There were 37 of them. In this study the cadavers were assigned to a pre-selected group. Each cadaver was frozen in a position and studied as such. With this methodology one cadaver's results in say flexion could not be compared to the same cadaver in extension. The averaged results of this study are very limited. If a particular cadaver is anomalous in its motion segments it is limited to only one segment of the study. They only studied one

motion segment, not the whole lumbar spine. Nerve compression was noted mostly in the extension group at 33.3%. In the neutral and flexion groups 21.0% and 15.4% of the segments had nerve root compression respectively. Facet subluxation with bulging of the ligamentum flavum was the usual cause of compression although posterior disc bulge was also associated with compression. Smaller disc heights and foraminal dimensions were found to be significant in compression. Measurements in the nerve compression group were a posterior disc height of 5.38 +/- 2.25mm and an IVF height of 18.11 +/- 2.75mm. These met a statistical significance of $P < 0.05$. Too many of the figures in this study do not meet statistical significance and therefore are not reported here.

Floman et al.[46] utilized H-reflex testing on patients with unilateral disc herniations at the L_5-S_1 level. The study involved 24 patients 20-50 years old. The disc herniations were confirmed by CT or MRI. The patients displayed symptoms of sciatica in an S_1 distribution, positive straight leg raise, and 16 had a decreased Achilles' reflex. The average duration of symptoms was 2 months. Thirteen people had an abnormal H-reflex of the gastrosoleus muscle ipsilaterally. Significant differences were found in mean amplitude and latency on the involved side. Post-manipulation, the H-reflex amplitude showed a significant increase towards normal on the affected side, H-reflex amplitudes on the unaffected side were unchanged. The latencies on the affected side changed showing a trend towards normal but this was not statistically significant. Eight of the patients did not display an abnormal H-reflex nor did they display a change in H-reflex post-manipulation. Three of the patients were either difficult to manipulate or measure H-reflex, and were therefore excluded.

Discussion

Comparing the dorsal root to the DRG it seems likely to be the DRG that is responsible for the most aberrant activity due to compression. The dorsal root in response to compression exhibits WDR neuron firing during compression[20] and increased firing in response to hypoxia.[26]

The DRG exhibits firing with afterhyperpolarization of the WDR neurons.[20] There is sympathetic sprouting in response to compression[21] and increased firing of large MFs.[22] Chronic compression causes spontaneous activity of large sized cells, decreased mean threshold current, no change in NCV, and in the medium sized cells increased afterhyperpolarization and peak amplitude of action potential.[23] The A and C type fibers show spontaneous activity with an ability to recover somewhat over time.[24] The dorsal root has a higher threshold to mechanical stimulation than the DRG.[26] The firing duration of the DRG is longer.[26] Under hypoxic conditions the DRG exhibits increased firing and a decreased susceptibility to mechanical stimulation.[26] As long as there are large MFs present there is no change in NCV, once these fibers are gone the NCV decreases.[27] The amplitude of the compound action potential shows a gradual decrease as the large MFs decrease.[27]

Pressure gradients show that the axoplasm is progressively destroyed with greater pressures.[33] The nerve alterations that occur as a result of pressure are a reduced nutritional supply starting at 10 mm Hg.[11] At 20 mm Hg there is a significant block of fast axonal transport[35] and a slight accumulation of antero- and retrograde transport.[34] The slow axonal transport is severely effected at 30 mm Hg.[35] Flow within the venules stops also at 30 mm Hg[10] as well as a marked inhibition to antero- and retrograde transport. Flow in the arterioles is stopped at 40 mm Hg.[10] By 100 g/mm pressure the axoplasm is completely severed.[35]

Sensory changes that may occur as a result of compression are an increased sensitivity to mechanical stimulation.[21,24,25] This may be the first to show[21] and the last to leave.[25] Increased thermal sensitivity typically follows this and disappears sooner.[21,24,25] Heat sensitivity lasts longer with greater pressures.[22] Increased sensitivity to soft touch, if present, typically resolves the fastest.[24] There should be no significant bilateral skin temperature changes in the limbs.[24] Ranking the order of likely to occur symptoms, we have from most to least, lack of fine motor control, dysesthesia, hypesthesia, motor deficits, numbness and tingling, allodynia, and hyperesthesia.[32] The longer the pressure, the longer there are changes in gait and posture.[24]

Histological changes and ability to recover are age dependent with regeneration more common in the young.[29] A quicker onset rate of compression induces greater inflammatory changes.[13] The higher the pressure level, the greater the inhibition of normal activity.[10,11,33,34,35] Multiple levels of compression induces greater changes also.[8,15,16] Once compression has taken place the tissue changes contribute to an increased resistance to additional compression.[16] The SEPs and MEPs show this resistance with an initial delay in latency that shows the ability to recover even though the pressure is maintained.[17]

In the beginning stages of compression there is endoneurial bleeding and inflammation,[14] reduced nutritional supply,[10] venous congestion and intraneural edema,[28] and inhibition of axoplasmic flow.[28,34,35]

Once compression has been maintained there is proliferation of fibroblasts and capillaries.[14] There is an increase in connective tissue, vascularization, and noneuronal cells.[23] The axoplasm becomes dense with reduced axonal volume.[14] Hyperplasia of the dura and arachnoid[27,28] occurs in the intermediate stages. In this approximate time, separation of the myelin sheath,[14,29] thinning of the myelin,[31] and atrophy of the axons[31] occurs. Fibrotic buildup of collagenous tissue ensues[27,28,30,31] along with the onset of ephaptic transmission.[27,28,31] Approximately one year after compression has been maintained there are no more large MFs and NCV decreases.[27] This is pressure, time and onset rate dependent; at the low end of the scale the large MFs may be almost unaffected; at the high end they may degenerate very quickly. Wallerian degeneration begins at around 6 months and by 12 months is clearly evident.[27,28]

The chronically compressed roots will show an increase of small MFs, axon/myelin ratio, and MF density.[30] They exhibit a decrease in large axons, large MFs, fascicle size, and lumen of the endoneurial vessels.[30] There is however no change of axon diameter.[30]

In the cervical spine the position of the nerve root puts it in contact with the medial border of the superior pedicle and lies directly on the inferior pedicle.[37] The nerve root is widest as it enters the IVF and thins as it moves distally out the IVF.[38] The IVF in contrast is thinnest at the medial portion and widens moving distally.[38] This creates a situation where the thickest part

of the nerve root is in the thinnest part of the IVF in the cervical region. The root at this level occupies 25-33.3% of the IVF without its dural sheath.[38] It is connected by a fibrous tissue to the posterior longitudinal ligament, the vertebral artery, the uncinate process, and the uncovertebral joint.[38,39]

The IVF size in the cervical spine increases with flexion and decreases with extension.[42] There is a tendency here for an increase in cervical IVF size with extension/ipsilateral rotation and flexion/contralateral rotation.[42] The tendency to decrease the cervical IVF size happens in extension/contralateral rotation and flexion/ipsilateral rotation.[42] The IVF dimensions appear to be similar throughout the lumbar spine.[44] The nerve root increases in size as you descend down the lumbar region.[44] Unfortunately no relevant data was obtained on the thoracic spine.

Compression is reported to occur in 20.8%[38] and 73%[40] of cervical spine segments. In this region the reported agents causing compression are the vertebral artery[40] and discs, uncovertebral arthroses, superior articular process, and periradicular fibrous tissue.[38] In the lumbar region, 21% of the nerve root complexes are reported to be compressed in the neutral position.[44,45] Extension apparently increases the rate of compression here to 33.3% and flexion decreases it to 15.4%.[45] The culprit most often cited as the cause is the ligamentum flavum. The critical dimensions for the lumbar region are a posterior disc height of 4 mm[44] - 5.38 mm[45] and an IVF height of 15 mm[44] - 18.11 mm.[45]

The pressures generated in the IVF do not correlate well however with the dimensions. Extension, as expected, generates higher pressures in the IVF from C_5-C_7.[43] Flexion does not give expected results showing increased pressures at C_5 and a tendency to increase pressures at C_7.[43]

With documented disc herniation and concomitant altered H-reflex parameters spinal manipulative therapy improves the H-reflex amplitude deficit without affecting the uninvolved side.[46] The H-reflex latency showed improvement but only one treatment was tested and longer term possibilities are unknown.[46]

Conclusion

A review of terminology is warranted in lieu of the previous mentioned study. The definition of manipulation is a force applied to a joint in its passive state to restore normal joint motion.[48] The definition of an adjustment is a force applied with specific intent to allow the body to correct nerve interference.[48] Floman et al.[46] report using manipulation. In the "Patients and methods" section there is not a single mention of joint restriction anywhere. In fact they do not mention restriction of motion in the whole article. They do however talk of signs of altered nerve function in the form of an abnormal H-reflex. They also use the "manipulation" as a way to facilitate improvement of the neural disturbance exhibited by the altered H-reflex. Therefore it would seem that the authors were imprecise in their terminology, they actually performed chiropractic adjustments even though they are not listed as being chiropractors.

Stephenson[49] and Strauss[50] speak of the Mental Impulse, that it was a specific message, sent from brain cell to tissue cell to effect a specific task. Although including this concept may invoke controversy, excluding it would be neglectful. As such then, the action potential could be considered a part of the Mental Impulse, much like say Morse code. If you change the dash and dot patterns the message becomes gibberish. Alterations of action potential would therefore constitute an alteration of a part of a message thereby altering its effect. Other aspects of the mental impulse to consider would be the nutritional state of the nerve fiber and the ability to transport larger molecules in a retro- or antero-grade fashion. Pert[51] echoes this concept in her book *Molecules of Emotion: The Science Behind Mind-Body Medicine:*

> "Though a key fitting into a lock is the standard image, a more dynamic description of this process might be two voices-ligand and receptor-striking the same note and producing a vibration that rings a doorbell to open a doorway to the cell. What happens next is quite amazing. The receptor, having received a message, transmits it from the surface of the cell deep into the cell's interior, where the message can change the state of the cell dramatically. A chain reaction of biochemical events is initiated as tiny machines roar into action and, directed by the message of the ligand, begin any number of activities-manufacturing new proteins, making decisions about cell division, opening or closing ion channels, adding or subtracting energetic chemical groups like the phosphates-to name just a few. In short, the life of the cell, what it is up to at any moment, is determined by which receptors are on its surface, and whether those receptors are occupied by ligands or not. On a more global scale, these minute physiological phenomena at the cellular level can translate to large changes in behavior, physical activity, even mood."

This quote takes on particular importance when considering the low-pressure effects on axoplasmic flow and nerve cell maintenance and regeneration.

In examining cadavers for signs of compression[38,40,45,46] the authors only consider indentation of the nerve root. Could it be possible that nerve roots have undergone compression injury without visible deformation, where collagenous build up[27,28,30,31] could render the appearance of the nerve root somewhat normal?

Compression can be viewed as a succession of events in the nerve root. These events can be amplified or minimized based on the presence or absence of confounding factors in the microenvironment of the nerve root and the IVF. The rate of onset, length of time maintained, and degree of pressure might be considered the major confounding variables.

The role of ephaptic transmission, or cross talk between nerve fibers, is generally spoken of in terms of generating pain.[28] It seems just as possible for cross talk to occur to other nerve fibers. Nerve fibers that are not responsible for pain, but the functioning of other tissues.

Utilizing NCV tests to detect the presence of nerve compression does not seem to be relevant. NCV alterations do not occur until the loss of large myelinated fibers occurs.[23,27] This apparently doesn't occur until after 12 months of compression.[27] It is the view of these authors that waiting for such a time frame would constitute negligence in clinical practice.

Schafer et al.[52] introduce the concept of joint

misalignment/fixation. This concept is one where the articulations of the spine and possibly extraspinal joints become fixated in their normal range of motion. Fixations happen in such a way that the complex motion segments of the spine can only freely move through a subset of the 3 axes (x, y, z) delineating its normal range of motion. The authors comment that the joint fixation/restriction is not necessary for a vertebral subluxation to occur. Though they feel that the joint fixation/restriction is a major component most of the time. The authors also categorize these restrictions as primary and secondary. The primary restriction is one that is causing a neural disturbance and therefore is termed a vertebral subluxation. The secondary allows the spine as a whole to function as normally as possible and therefore is termed a compensation.

The size of the IVF would seem to be a good indicator of what pressures would be derived there. Boyle's law[53] states, given that temperature is constant, the pressure of a gas varies inversely with its volume. Applied to the IVF the increase in size leads one would to expect a decrease in pressure and vice versa. With the evidence available at this juncture, it seems that the size of the IVF does not correlate with the pressures generated internally. Pressures can actually increase with an increase of IVF size. The only pressure tests available readily indicate that at C_5-C_7 there is enough pressure in certain neck positions that if maintained in a joint (misalignment/fixation situation) there is a definite alteration of neural function including nerve flow. These alterations would therefore alter the quality and/or quantity of the message sent. At the tissue and cellular level, the message received would not be adequate for the function the body demands. The entire body could then theoretically be affected.

In 1973 Crelin[54] published the hallmark paper that supposedly debunked prevailing chiropractic theory of the time. The author's intent was to take fresh cadaveric spines and subject them to various external forces and see if the nerve root in the IVF would be susceptible to compression. The paper abounds with methodological flaws. The author removed all of the areolar tissue in the IVF dismissing it as "flimsy." It is not stated but heavily inferred by this action that the author felt the areolar tissue incapable of transmitting pressure. He uses many different pressure reading gauges to measure the forces applied to the spines but none that measure the forces generated at the IVF. Apparently for an experiment designed to test the chiropractic theory of increased pressures generated in the IVF during vertebral subluxation this was unimportant. The author's main experimental test for nerve impingement is visual observation; he "eyed" it. According to the author there was always plenty of room for the spinal nerve in the IVF. Again, it would appear the unstated principle here is Boyle's Law of Gases. Farmer's[43] work clearly demonstrates that Boyle's Law does not apply since gases only occupy the IVF when you remove all the supporting tissues. An analytical reading of the paper clearly demonstrates that it is erroneous.

When considering the clinical implications of the vertebral subluxation complex that carries a compression component these research studies offer a unique understanding of the pathogenesis. A minor pressure increase occurs at the level of the IVF. This may or may not produce noticeable sensory and/or motor symptoms. These symptoms if present, would most likely be transient and self-limiting. They also would not involve entire fiber groups therefore one might not see sensory changes in an entire dermatome. This would also hold true for any motor disturbances; they would be mild and self-correcting. The neural tissue is self-protective in low-grade compressions and utilizes additional connective tissue that prevents further injury. If there is a continuous, repetitive injury some fibers regenerate while others are damaged. If the pressure is constant and sufficient the larger fibers show a type of atrophy. Such atrophy would not be readily recognizable but would cause disturbances that are more akin to an insidious onset. Future research could focus on the mechanism of reinjury in a nerve that has undergone repair to see if the same pattern of damage occurs. This could possibly explain why, when a person becomes subluxated again, their particular pattern of symptoms returns.

The "garden hose" theory as postulated by BJ Palmer[55] likened the nerve to a garden hose. Pressure on the hose decreased the outflow of water in much the same way as pressure on a nerve would decrease the outflow of mental impulses. This was the basis for the colloquial phrasing "hard bone-soft nerve." The bone does not obviously always contact the nerve. There are other elements such as the endoneurium, the vasculature, and adipose tissue involved at the level of the IVF.[43] The alteration of motion allows the bone and other structures to create a state of increased pressure in the IVF. The increased pressure alters the functionability of the nerve root and DRG. This would change the message received in either a cranial or caudal direction. The resultant action taken in the brain to orchestrate further body function or at the level of the body's cells to effect certain chemical reactions would be changed.

The chiropractic adjustment, applied to a vertebral subluxation with increased foraminal pressures, would work to reduce and correct the vertebral subluxation. The resultant normalization of pressure would allow the body to heal the injured nerve root and DRG and restore the normal transmission of information. This healing process is age and degree dependent. One must consider the age of the person with a vertebral subluxation as the younger they are, the better the chance for a complete correction. One must also consider the onset of the subluxation as the more "violent" the onset, that is high-pressures over short periods of time, the less likely it is for a significant recovery. Posture becomes important in the corrective phase because bad posture could prolong the problem and therefore hamper the recovery. Posture may also be considered a cause, in that the repetitive training of areas of the spine with bad posture may not be conducive to good neural hygiene.

The findings of this report support the hypothesis of nerve compression as a viable process associated with vertebral subluxation. Compression of a nerve root was found between 15.4% and 73% of levels inspected in this review. Most of the specimens used were pre-screened for degenerative changes and gross pathology. The chiropractic adjustment has been shown to restore the normal function of a compressed nerve. The garden hose theory is far from obsolete as Kent[7] explains and seems quite possible to be a major factor in vertebral subluxation. Future research on pressures generated through normal ranges of motion at all spinal levels will help to delineate the importance compression plays at various IVF levels.

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