Cervical Trauma and Chronic Pain
Fibromyalgia caused by Cervical Injury
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Fibromyalgia is a chronic pain condition that is associated with neuroendocrine dysfunction and a complex of multi-system symptoms. In order to be diagnosed as having Fibromyalgia a patient must have eleven of eighteen tender points tender to less than four pounds per square inch pressure, and a chronic non-restorative sleep disturbance lasting more than three months. Fibromyalgia patients have reduced levels of the branch chain amino acids, reduced serum levels of serotonin, epinephrine, norepinephrine and growth hormone and increased levels of substance P in the spinal fluid. [12] Fibromyalgia is 13.3 times more common in patients following cervical injuries than it is in patients who have had lower extremity injuries. A recent study from Israel documented a 22% prevalence of Fibromyalgia 1 year after auto accidents causing whiplash injuries in comparison with a 1% prevalence after accidents involving leg fractures. [6]

Patients with Fibromyalgia have augmented central processing of nociceptive stimuli in comparison with pain free controls. When measured by EEG fibromyalgia patients have changes of greater amplitude on both sides of the brain in response to painful stimuli. Pain-free controls responded on one side of the brain with smaller evoked potentials. Gibson noted an increase in late nociceptive evoked somatosensory response in 10 FMS patients as compared to 10 controls. SPECT scanning of the brain of a patient with Fibromyalgia shows reduced blood flow in the caudate nucleus of the thalamus, which is a site of central pain processing. Fibromyalgia patients have the characteristic pain of allodynia in which non-noxious stimuli such as light touch are processed as painful.[13, 2]

There is evidence that pain perception in Fibromylagia patients is different in quality. In normal controls the pain threshold increases gradually between 80 and 160 pressure units. It spikes dramatically at 160 units of pressure. Fibromyalgia patients show a linear increase in pain intensity with pressure intensity at every level from 80 to 200 units of pressure. Animal studies show this same pattern due to central sensitization and lowered firing threshold in the dorsal horn cells.[3]

In our Fibromyalgia clinic we have identified five distinct etiologies of Fibromyalgia. One is associated with prolonged emotional or physical stress and the subsequent adrenal depletion, leaky gut and food allergies. One is associated with exposure to organic chemicals, heavy metals or pesticides. One appears to be genetic and seems to be associated with food allergies or with increased need for enzyme substrate in the liver detoxification pathways or serotonin pathways. One type occurs after immunizations or viral illness. And there is one type of fibromyalgia that occurs after whiplash injuries, cervical trauma or after surgery. The post-surgical cases are thought to occur when the neck is hyper-extended during intubation and constitutes a cervical injury.

My discussion is going to focus on the fibromyalgia or chronic pain associated with cervical injuries. We have “successfully” treated 23 patients with this type of Fibromyalgia in our clinic. Successfully is placed in quotes because we are able to reduce or eliminate their pain and provide palliative care quite easily. Returning the patient to full health is challenging but has been done in enough cases to provide hope and therapeutic direction and to suggest a model for the pathophysiology of this type of fibromyalgia.

Patient Presentation and History
The pain diagram and symptom complaint describes burning, aching midscapular pain, shoulder, neck, arm and hand pain that begins soon after the cervical trauma and persists to varying degrees from the time of the injury to the date of presentation. After some period of time, usually one to six months post injury, the pain generalizes to the whole body, particularly down the thoracic and lumbar paraspinals, the gluteals and down one or both legs often into the feet. The pain is usually described as burning, aching, and tingling and occasionally as stabbing. The patients generally rate the pain as varying between a 4 and a 9/10 on a visual analogue scale. Most describe their hands and feet as feeling cold since the accident or injury. The patients have an affective response to the pain that is characteristic. The pain is particularly bothersome. Most have chronic headaches that may be quite severe. Many have been on narcotics or Neurontin for some time.

Imaging
Among the 25 cases we have seen that fit this historical profile many did not have MRI studies. When MRI studies were done or available from prior physicians they all showed a disc bulge or a contained herniation usually at C5-6 or C6-7. In most of the cases the films had been read as normal by the reading radiologist. They were subsequently read as having a central bulge or contained herniation by our consulting radiologist. Plain film x-rays showed anterolisthesis or retrolisthesis above or below the disc bulge in three cases. Flexion extension films were not done in all cases but when they were done they showed slight segmental hypermobility, or increased translation, at the level of disc injury or just above it. Degenerative changes were common when the injury was more than five years old. One case showed no sign of a disc injury and it was suggested by her neurologist that she had a spinal cord traction injury.

Physical Examination
The physical examination invariably showed hyperesthesia at one or more upper extremity dermatomes, usually C4 or C5, and normal upper extremity reflexes. Dermatomal sensation was normal in the lower extremities in all cases. In most cases the patellar reflex was hyperactive and the achilles was normal. Two patients had a very hyperactive abdominal reflex. Muscle strength in all of the cases tested was +5/5 in the upper extremity. Cervical compression produced neck pain and an increase in shoulder pain in most cases. Measurement of the eighteen tender points by algometer usually showed the required eleven out of eighteen necessary for a fibromyalgia diagnosis unless the patient was on narcotic medication.

Hypothesis of Pain Facilitation
In order to link these cases of Fibromyalgia with the cervical trauma we have to make a case for the association between cervical injury and central pain processing abnormalities. The connection lies between the cervical discs and the anterolateral pain pathways of the spinal cord.

Neuroanatomy
“Noxious stimuli” or pain impulses travel toward the spine on two types of fibers. There are very fast myelinated A delta fibers that conduct at about 5-30 meters/second and carry heat and mechanical stimuli. They are associated with well-localized sensations of sharp pricking pain. The other set of pain fibers are small slow unmyelinated C fibers that conduct at .5-2 meters/second and carry a wide variety of high intensity mechanical, chemical and thermal stimuli. They are associated with diffuse long-lasting burning pain. [13]

These pain fibers enter the cord in the lateral division of the dorsal horn, synapse with second order interneurons in the cord, cross anteriorly and ascend towards the brain in the anterolateral

pathways, the neospinothalamic and the paleospinothalamic tracts. The faster A delta fibers cross the cord in lamina 1 and form the neospinothalamic tract carrying sharp localized pain. The second pathway carries the slower C fibers, A delta and A beta fibers and crosses in lamina V to form the paleospinothalamic and the spinoreticular components of the anterolateral system. This pathway carries deep diffuse chronic pain and displays the phenomenon of prolonged after discharges and facilitation when large fiber input is suppressed and then followed by C fiber stimulation. [13] This becomes important later when we talk about how chronic pain becomes facilitated.

Cervical Trauma

We know that cervical trauma causes cracks in the annulus and fractures in the end plates that expose the nucleus pulposus to the spinal fluid either directly or via the spinal vasculature. In their 1992 paper on disc injuries in cervical trauma, Taylor and Twomey describe the pathological changes in the cervical discs created by trauma. They did a comparative study of the cervical spines from 16 subjects who died of major trauma and 16 subjects who died of natural causes. 15 of the 16 trauma subjects showed clefts in the cartilaginous endplates. The cartilaginous end plates are important because they are vascularized whereas the disc itself is avascular. The vasculature extends from the end plate into the epidural space. [19] Age related changes in the discs are found extending from the uncovertebral joints medially toward the center of the disc and do not involve the end plates.[24] Posterior disc herniations and facet hemarthrosis were also observed in the trauma group and absent in the control group. [24]

"The outer annulus of cervical discs is innervated and cervical discs may be more extensively innervated than lumbar discs. [4, 9, 17]…The rim lesions we have described usually involve the outer part of the annulus. The delayed healing and predisposition to premature degeneration after the experimental production of rim lesions in the intervertebral discs of sheep, with the tendency to vascularization of these lesions, suggest that something similar may be responsible for chronic pain associated with soft-tissue injuries to the cervical spine. This study and the study of Davis et al [10] show that neck extension sprain, with posterior disc herniation, may be associated with injury to the spinal cord such as localized petechial hemorrhage in the anterior columns or vascular damage to the anterior spinal or radicular arteries."[5] In the cases studied by Taylor and Twomey x-rays taken before microscopic examination did not show fractures, dislocations or subluxations. "Clinical studies show that rim lesions and traumatic herniations are demonstrable in survivors of motor vehicle trauma in the absence of vertebral fractures." [24]

Patients with chronic residual cervical pain following trauma have symptoms that persist for years after soft tissue injuries should have healed. The damage to the cartilaginous end plates and discs provides a model for that persistence. Rim lesions produced surgically to a depth of 5mm in the discs of sheep, do not heal when followed for a period of 18 months. Only the outer third of the annulus shows the capacity to heal. These disc and rim lesions extend inward to at least that depth with deformation of the annular lamellae and degeneration of the nucleus pulposus. This phenomenon helps to explain the persistence of the injury.[24]

Nucleus Pulposus as a Neurotoxic Agent

We know that the nucleus pulposus causes an inflammatory response in nerve tissue. [21] Nucleus pulposus material was implanted in the epidural space in pigs near the cauda equina. Implantation of retroperitoneal fat was used as a control. Nerve fiber degeneration, axonal swelling, increased axoplasmic density and marked attenuation and splitting of the myelin sheaths were noted in the animals exposed to nucleus pulposus material. Nerve root conduction velocity was significantly lower in the nucleus pulposus exposed nerve roots than in the control nerve roots. This study was the first time it had been demonstrated that the nucleus pulposus

could produce reduction in nerve conduction velocity and nerve fiber degeneration without a mechanical compression of the nerve root. [20]

Nerve conduction velocity	Day 1	Day 3	Day 7
Control	84+/-2	83+/-4	76+/-11
Nucleus pulposus	63+/-9	45+/-16	45+/-19

We know that the nucleus pulposus elicits an inflammatory response as indicated by leukotaxis and an increase in vascular permeability. The exact mechanism of the inflammatory response has not been verified. It wasn't clear whether the inflammatory reaction was induced by the nucleus itself or from substances being liberated from other tissues as a response to the interaction with the components of the nucleus. Glycoproteins, immunoglobulin G, phospholipase A_2 and hydrogen ions have been proposed as possible mediators of this inflammatory damage. [20]

Marshall proposed a "chemical radiculitis" in which the annulus fibrosis is weakened by degeneration and finally ruptures under the stress of a traumatic episode. The nuclear fluid, which may be highly irritating to nerve tissue is then ejected into the peridiscal tissue.[15] The inflammatory properties of the nucleus pulposus have been demonstrated in hogs[21], dogs[16] and rabbits.[7] Macrophages predominate in an area of tissue injury within a few days and secrete the by-products of phagocytosis, including hydrogen peroxide, lactic acid and PLA_2. PLA_2 is an important lipolytic enzyme in the arachidonic acid cascade. This process intensifies and prolongs the inflammatory responses. Proinflammatory substances such as interleukin1 and other cytokines may also activate PLA_2 and other proteolytic enzymes that are found in disc tissue. Phospholipase A_2 is present in high concentrations in herniated and painful discs. [23]

"It is concluded that immediate neural response is a direct effect of PLA_2, based on its chemical composition…. In a long term PLA_2 study 3 days after the application (of PLA_2) breakdown of myelin sheaths, unclear axonal margins and vacuolar degeneration were observed...The PLA_2 found in the herniated human disc may be neurotoxic around the immediate exposed tissues…The evidence for neurotoxicity included loss of spontaneous nerve discharge after PLA_2 application and absence of response to mechanical stimulation in previously responsive units." [23]

"Central processing of pain may arise from the neurotoxicity or recruitment of silent units. Long-term consequences of this neurotoxicity could include neurodegeneration, neural regeneration, neuroma formation and ectopic nerve impulses all of which can be sources of pain. [1, 8] Peripheral nerve lesions can produce spinal cord changes that may contribute to deafferentation pain. These changes include sprouting of myelinated fibers into lamina2 of the spinal cord and increased discharge of dorsal horn neurons."[15, 23]

Fibromyalgia and Central Pain

Robert Bennett has described the central sensitization of pain perception in Fibromyalgia patients in wonderful detail. Pro-inflammatory cytokines (interleukin-1 and 6 and tumor necrosis factor)…sensitize second order dorsal horn neurons (Lamina V slow C multimodal pain neurons) through an NMDA-substance-P-nitric oxide cascade.[2] Mountz used SPECT scanning to demonstrate reduced blood flow to the thalamus and caudate nucleus where pain stimuli are processed in Fibromyalgia patients as compared to normal controls. [18] In acute pain, blood flow is increased in these areas. Bennett also points out that lesions of the lateral thalamus often result in a pain syndrome characterized by affective distress, and aching, burning and tingling

pain that is exacerbated by normally innocuous stimuli such as light touch, known as allodynia.[1,2]

Chronic Pain and Cervical Trauma

All of these pieces come together in the spinal cords of patients who have had cervical trauma. We hypothesize that the exposure of the nucleus pulposus material to the spinal fluid via the cracks in the annulus created by cervical trauma, causes an inflammatory response in the spinal fluid. This inflammatory response may be mediated by phospholipase A_2 and its associated cytokines.

Nerve destruction such as that shown in dogs, pigs and rabbits would be created by these neurotoxic inflammatory chemicals in the spinal cord in response to exposure to the nucleus pulposus. Phospholipase A_2 has been shown to be so neuro-toxic that it is capable of damaging the pathways in the anterolateral system. This inflammatory response is dose related. [22,23] We hypothesize that this inflammatory substance creates a chemical lesion in the paleospinothalamic tracts. These tracts carry pain information up the spine to the thalamus, caudate nucleus and the cortex and ascend the cord in the anterolateral portion of the lateral column. The paleospinothalamic tract is the outermost of the two tracts and carries diffuse deep chronic pain sensation.

The anatomic proximity of the cracks in the discs and endplates could expose this system to high levels of phospholipase A_2. This tract is immediately adjacent to the site of the disc herniations demonstrated by Taylor and Twomey in their study of cervical trauma cases.[13, 24]
The inflammatory damage to the anterolateral columns could operate in one of two ways. If the damage was minor and simply reduced the firing threshold of the axons pain traffic up the cord would be facilitated and enhanced creating the profuse allodynia seen in Fibromyalgia patients.

If the inflammatory damage progressed to damage of the paleospinothalamic nerves it would effectively create a chemical deafferentation. We have seen how inflammatory chemicals reduce the firing of the nerves and slow nerve conduction. The trauma induced physical damage to the anterolateral pathways and the cord seen by Taylor and Twomey add another possible etiology for the deafferentation phenomenon. Deafferentation, whether caused by chemical disruption or physical trauma and damage in the ascending pain pathways would produce what is essentially a thalamic pain pattern. I find this mechanism the more likely of the two I have proposed.

“Central pain can arise not only from pathologic lesions in the thalamus but also from neurosurgical lesions placed anywhere along the nociceptive pathway from the spinal cord and brain stem to the thalamus and cortex. …The sensations are unpleasant and abnormal, often unlike anything the patients had ever felt before: spontaneous aching and shooting pain, numbness, cold, heaviness, burning and other unsettling sensations that even the most articulate patients find difficult to describe. Central pain is particularly distressing emotionally.” [13]

Fibromyalgia and Central Pain

Fibromyalgia patients with a cervical trauma etiology have been describing this type of pain to me for four years. The description of the pain sensations associated with central or thalamic pain is precisely word for word what I have heard in patient histories in more than forty patients. Deafferentation in the anterolateral pathways is capable of creating the tract lesions that produce the thalamic pain symptoms we see in fibromyalgia patients. Patients with fibromyalgia not caused by cervical trauma do not have the same quality of pain that cervical trauma patients describe. Their pain is diffuse and achy but it lacks the disturbing affective neuropathic intensity

seen in the cervical-trauma mediated fibromyalgia patients. This affective intensity is characteristic of thalamic or central pain. The similarities between centrally mediated pain and the pain described by this group of fibromyalgia patients and the differences between treatments effective in this group of fibromyalgia patients and other types of fibromyalgia patients led to the development of this hypothesis.

The clinical picture suggests that the chronic central nerve pain facilitates the sympathetic nervous system causing a chronic fight or flight response, especially when the disc is damaged at the C5-6 level causing stimulation/facilitation of the C5 sympathetic ganglion. The sympathetic response is characteristic. The body's repair systems are put on hold, circulation to the digestive system is reduced, myofascial circulation is altered, immune system function is compromised, the adrenals produce elevated levels of endogenous cortisol and are constantly taxed to keep up and the system gradually experiences more and more dysfunction. When the gut is compromised in this fashion for a year or more it is more prone to dysfunction including "leaky gut" and the resultant food and systemic allergy reactions. Elevated endogenous cortisol levels cause thinning of the gut wall and may impair transport of the branch chain amino acids. The branch chain amino acids are necessary precursors of neurotransmitters including serotonin, epinephrine, norepinephrine, oxytocin and dopamine. They are essential co-factors in phase one and phase two liver detoxification pathway function. Branch chain amino acid levels are reduced in Fibromyalgia patients.[11]

By the time the patient has been in this condition for one to two years the symptoms have generalized into the classic neuroendocrine chaos we call "fibromyalgia".

Treating Cervical Trauma Induced Fibromyalgia

This would all be interesting as an academic exercise but it becomes compelling when one is able to treat and reverse these effects. In February of 1999 we happened upon a way of treating patients with this symptom profile in our clinic. Out of desperation and trial and error we happened upon a frequency combination and current polarization technique that eliminated chronic full body pain in one patient. By the end of 1999 we had treated 25 of these patients, by the end of 2002 we have treated 150.

These patients were unresponsive to previous medical treatments, to functional and natural medicine approaches and to microcurrent treatment of myofascial tissue. Indeed, in some cases their pain was worsened by microcurrent myofascial treatment. One patient had been treated for over a year in our clinic and had had cervical disc surgery, lumbar disc surgery and a shoulder repair following an auto accident. None of these therapies changed her pain. Her case is one that illustrates and supports the deafferentation hypothesis. Repairing the cervical disc did not change her pain. She moved away from Portland in 1998 and returned in October 1999 after we had developed this treatment protocol. She left the treatment room pain-free for the first time in two years. She still has some arthritis pain but the debilitating neuropathic pain is gone. We hypothesize that the damage to the anterolateral pathways created by the disc injury perpetuated her pain. We must also hypothesize that the microcurrent and the frequencies somehow change the conductivity and function of the injured cord areas and restore proper function. Her case is typical of this group.

Treatment Method

A standard two-channel microcurrent instrument with three digit frequency settings and a two-place multiplier provides microamperage current modulated by frequencies between .1 hertz to 999 hertz with three digit specificity. There are several frequency combinations that have been effective. We use either 40hz or 284 Hz on channel A and 10hz on channel B or 321hz on

channel A and 10hz on channel B. The current must be used polarized positive at the upper cervical spine and negative at the sacrum or feet depending on the extent of the patient's pain in order to achieve the desired effect. The cervical contact has to wrap around the neck at least to the exiting nerve roots. In our clinic we use graphite conducting gloves that are plugged into the microcurrent instrument to provide this current distribution.

When treatment in the clinic was effective in reducing the patient's pain, the patient was sent home with a home microcurrent unit made by Rehabilicare. (800 676-6489) It is the only unit on the market that has the capacity to provide polarized current and a range of different frequencies, including those we find useful, on two different channels. When the patients use a home microcurrent unit they must use 2x3 inch pads touching over the spinous processes and wrapping the neck laterally to the exiting nerve roots. Smaller pads do not have the desired effect. The patients are instructed to use the home unit often enough to keep their pain below a 3/10 VAS.

Some patients found that after an initial period of wearing the home unit 12 to 24 hours a day for about a month they could reduce their wearing time to four hours every day or every other day to maintain the pain relief.

Clinical Controls

The patients were always treated while laying face down on the table. Sham treatment with the machine turned off was done in about one quarter of the cases, at first by accident and later as an intentional control. The sham treatment is easy to do because the current is subsensory. Sham treatment never produced pain reduction or tissue softening. The patients were always blinded to the frequency used. Four frequency combinations and both biphasic and polarized current were applied in every case. The patient was always blinded to the current polarization. Portions of the treatment are done with the gloves wrapped around the neck without a hand in the glove to remove the effect of the operator's field.

Results

Patients with disc injuries and the characteristic pain pattern were helped only by 40Hz and 10Hz applied polarized positive at the cervicals and negative distally. Patients with pain associated with a central injury above the cervical level were helped by 320Hz on channel A and 10 Hz on channel B. There have been no patients with this history and symptom combination in whom this treatment was ineffective. 23 of the 25 seen have responded well. Use of microcurrent in this fashion also seems to reduce or eliminate the chronic headaches experienced in this group. One patient with a cord traction injury had pain relief during treatment but suffered severe headaches when the microcurrent was withdrawn. She was not able to use the microcurrent constantly so treatment was abandoned. One patient with childhood onset of pain following cessation of childhood seizures experienced good relief after the first visit but no relief in subsequent treatments and treatment was abandoned.

All patients with "simple" posttraumatic cervical trauma centrally-mediated pain, regardless of chronicity, experienced relief with one of the two frequency combinations. No other frequency combinations were effective. Only one of the frequency combinations is effective on any given patient, except for one patient in whom 320/10 was useful on the first visit and 40/10 useful thereafter. This treatment protocol is not effective in any other type of pain. One patient required treatment of myofascial pain first and then cord treatment was effective. Sham treatment did not produce any change in any patient.

One patient had been diagnosed by an infectious disease specialist as having a herpes type infection in the spinal cord. This patient did not respond to the 40/10 or the 320/10 protocols. There is one frequency thought to be useful for shingles, 230hz on channel A and 430hz on channel B. After four unsuccessful attempts to provide relief using other protocols, this frequency combination was used polarized positive at the neck and negative at the feet for one hour. The patient's leg pain was gone within ten minutes and she was pain free for six weeks after seventeen symptomatic years. Repeated treatment has consistently reduced her symptoms for weeks at a time. No other frequency combination or treatment protocol has been effective in reducing her generalized pain.

Side Effects
In all but one patient, there were no side effects except for skin irritation from the continuous use of microcurrent adhesive pads. Some of these patients have been using the current in this fashion daily for ten months and have had no ill effects. One patient had headaches following treatment and treatment was abandoned.

Precautions
When a disc is herniated causing frank cord compression or when there is bony cord stenosis application of microcurrent in this fashion causes an almost immediate increase in pain. In the two cases in which this response was noted, treatment was discontinued immediately and the pain receded over the next few hours back to pre-treatment levels. In one case this response to treatment resulted in an order for an MRI and successfully predicted the presence of cord compression. The patient was referred for surgery.

Follow-up Treatment
After the first visit the patients varied in their response. All of the patients were fitted with a home microcurrent unit capable of generating the frequencies listed and polarizing the current in the desired manner except for the herpes patient. Most patients followed up with care to relieve the residual myofascial hypertonicity. All reduced narcotic medication; most eliminated it except for occasional exacerbation episodes. After years of moderate pain most were adrenal depleted. We followed them with nutritional supplements and microcurrent treatment aimed at rehabilitating the adrenals, liver, kidneys and gut. When possible or appropriate patients were referred for physical therapy with Ola Grimsby Institute trained physical therapists to stabilize the intrinsic muscles and support the injured areas of the cervical spine. As the patient reconditioned biomechanical difficulties were treated as they arose either with chiropractic treatment, posture and ergonomic recommendations, physical therapy, facet injections, or epidural injections. Treating the cord-mediated pain was fairly simple. Helping the patient to achieve full recovery has definitely been a multidisciplinary project.

With the exception of the last four patients listed, all of the above patients are doing well. AK has had Fibromyalgia for 18 years. After eight weeks of treatment she was off all pain medication. At her first visit she had 14/18 tender points. After eleven treatments eight weeks later she had four of eighteen tender points and was sleeping through the night without medication. She had OGI physical therapy for reconditioning and is still doing very well. Her response is optimal for this group. The least successful patient responses have been due to inability to tolerate the adhesive pads on the skin and inability to keep the pain below a 3-4/10 because the home unit could not be used.

Discussion
Microcurrent provides electron flow to the tissue using physiologic amperage. It increases ATP production and restores normal membrane conductance in injured tissue. It seems reasonable

that microcurrent would allow return of normal membrane stability and would restore function in nervous system tissue including the dorsal horn neurons and apparently to the spinal cord. The use of specific frequencies to treat specific conditions dates back to electro-therapies used in the early 1900's. These frequencies seem to be effective when applied with microcurrent therapy. The clinical response certainly suggests that the effects are very frequency specific. The DC current must be polarized in order to achieve the desired effect but the mechanism by which polarization creates the effect isn't clear.

Microcurrent therapy has been effective in a group of patients whose pain has been otherwise intractable and untreatable. The uniform success even in this relatively small group of patients suggests several things.

1. It suggests that this type of fibromyalgia is based on a true physiologic / neurologic response and is not, as some have suggested, a somatiform disorder.
2. It suggests that further study into the mechanisms of spinal cord inflammation, deafferentation and cord-mediated central pain is worthwhile.
3. It suggests that microcurrent is a low risk, low-cost treatment for this condition that deserves further study.

Fibromyalgia - Neuropathic Pain – Spinal Cord Mediated Pain
Case Results 2/99 through 3/2000

Patient	Pain at first visit	General pain range	Years Chronicity	Cause	Pain at end of visit	Frequency used **
FE	7/10 (N)	4-8/10	26yrs	Motorcycle accident age 19	0-1/10	320/10 first visit 40/10 after
AH	8/10	5-8/10	3yrs	MVA	2-3/10	320/10
CMK	5/10 (N)	4-8/10	18yrs	MVA	1-2/10	320/10
MV	8/10	6-8/10	1 yr.	MVA	0/10	320/10
LC	5-6/10	4-8/10	3yrs	Fall	0/10	40/10
HB	5-6/10	4-8/10	50yrs	MVA	0-1/10	40/10
ST	3-4/10	3-6/10	3yrs	Lifting overhead	1/10	40/10
AK	5-6/10 (N)	3-8/10	18yrs	MVA	0-1/10	40/10
MH	5/10	4-8/10	5yrs	Using a pick in hard soil	0-1/10	40/10
CC	7-8/10	5-8/10	3yrs	MVA	1/10	40/10
AS	5-6/10	5-8/10	6yrs	Supine childbirth-pulling on straps	0-1/10	40/10
DW	5/10	5-7/10	10yrs	Following surgery	0-1/10	40/10
CW	4/10	4-7/10	7yrs	Fall – thoracic disc bulge	0-1/10	40/10
AC	7/10	5-8/10	18yrs	MVA	2/10	40/10
NM	7/10	5-8/10	2yrs	MVA	0-1/10	40/10
LN	8/10	5-9/10	2yrs	MVA	0-1/10	40/10
BH	5/10 (N)	4-9/10	25yrs	MVA	3-4/10	40/10 effective only after FSM muscle work
DC	9/10	7-9/10	2 days	MVA	2/10	40/10
TH	7/10	3-8/10	3yrs	MVA	0/10	40/10
SW	8-9/10	6-9/10	6mo	MVA	2-3/10	40/10
BGM	7-8/10 (N)	4-8/10	18yrs	Incline Village Herpes	1-2/10	230/430
SW	8/10 (N)	5-9/10	8yrs	MVA	3/10	40/10
EH	8/10 (N)	4-9/10	4yrs	Disc Compression	2-4/10	40/10
KS TX ineffective Dis-continued	5/10	4-8/10	1yr	MVA cord traction injury	2/10	40/10 severe headache after use
EV Effective first visit not effective thereafter	5/10	3-7/10	25yrs	Pain started when childhood seizures stopped	0-2/10	320/10

**Frequencies were polarized positive at the neck negative distally.
(N) Indicates pain level while on narcotics.

Blood Data

Blood samples were taken from a patient with this history and symptom profile who had a seven-year chronicity. The samples were collected as blood spots on chromatographic paper and analysed by a chromatographic immuno-chchemist at NIH. The first sample was taken before treatment and the subsequent samples were taken as the pain dropped and/or frequencies were changed to address different tissues. On each treatment date 90 minutes elapsed between the first and final sample.

Sample	DATE	IL-1	IL-6	IL-8	TNF-α	IFNγ	SP	CGRP	VIP	NY	β Endorph	Cortisol	Serotonin
MK1	5/11/00	392.8	204.3	59.9	299.1	97.2	132.6	100.8	8.5	18.1	5.2	15.5	285.6
MK2	5/11/00	288.5	200.8	47.6	265.7	99.8	127.5	97.6	10.2	13.7	7.1	12.6	309.2
MK3	5/11/00	103.2	121.7	21.3	96.5	73.7	82.4	61.3	32.9	7.2	21.4	33.7	202.1
MK4	5/11/00	52.6	33.9	11.4	43.4	32.6	38.2	22.4	48.4	5.1	69.1	78.3	169.5
MK5	5/11/00	21.4	15.6	4.8	20.6	11.4	10.5	8.6	69.9	6.6	88.3	169.9	289.6
MK1	5/14/00	218.7	165.9	45.7	205.6	75.9	99.6	81.4	4.9	10.7	9.4	12.9	250.0
MK2	5/14/00	113.2	87.3	21.6	151.8	44.7	102.5	80.9	26.3	6.5	36.7	61.8	203.7
MK3	5/14/00	45.6	40.7	5.8	33.3	26.5	41.7	37.9	39.1	14.5	89.5	149.3	366.2
MK1	5/17/00	145.9	100.5	42.6	114.2	80.6	144.6	113.3	12.5	11.8	11.6	11.8	322.4
MK2	5/17/00	61.5	47.2	10.4	71.9	39.3	55.7	37	28.4	7.2	88.6	78.9	259.3
MK3	5/17/00	10.6	11.6	5.1	22.1	5.9	9.4	11.6	71.8	6.0	115.9	182.6	410.6
Normal Range		0-25 pg/ml	0-25 pg/ml	0-25 pg/ml	0-25 pg/ml	0-25 pg/ml	0-30 pg/ml	0-20 pg/ml	0-20 pg/ml	0-20 pg/ml	0-35 pg/ml	5-25 ug/ml	100-300 ng/ml

KEY:
IL-1: Interleukin 1
IL-6: Interleukin 6
IL-8: Interleukin 8
TNFα: Tumor necrosis factor alpha
INFγ: Interferon gamma
SP= Substance P
CGRP: Calcitonin gene related peptide
VIP: Vasoactive intestinal peptide
NY: Neuropeptide Y
β Endorphin: beta endorphin

Bibliography

1. Bennett GJ: *Neuropathic Pain*: Textbook of Pain, Melzak & Wall editors, 3rd edition, London: Churchill-Livingstone, 1994: pp. 201-24

2. Bennett RM: Emerging concepts in the neurobiology of chronic pain: evidence of abnormal sensory processing in Fibromyalgia. *Mayo Clinic Proceedings* 74: 385-398, 1999

3. Bendtsen L: Evidence of qualitatively altered nociception in patients with fibromyalgia. *Arthritis and Rheumatism* 1998; 41: 1966-71

4. Bogduk N: The innervation of the cervical intervertebral discs. *SPINE* 13: 2-8, 1988

5. Bohlman H: Acute fractures and dislocations of the cervical spine. *Journal of Bone and Joint Surgery* 61A: 119-1142, 1979

6. Buskllа D, Neuman L, Valsberg G, Alkalay D, Wolfe F.: Increased rates of fibromyalgia following cervical spine injury: a controlled study of 161 cases of traumatic injury. *Arthritis and Rheumatism;* 40: 446-452, 1997

7. Cavanaugh JM, Ozaktay AC, Yamashita T, Avramov A, Getchell TV, King AI: Mechanisms of low back pain. *Clinical Orthopedics*; 335: 166-80, 1997

8. Chen C, Cavanaugh JM, Ozaktay AC, Kallakuri S, King AI: Effects of phospholipase A_2 on lumbar nerve root structure and function. *SPINE*; 22: 1057-64, 1997

9. Cloward RB: Cervical discography: Mechanisms of neck, shoulder and arm pain. *Annals of Surgery* 150: 1052 – 1064, 1959

10. Davis SJ, Teresi LM, Bradley WG, Ziemba MA, Bloze AE: Cervical spine hyperextension injuries: MR findings. *Radiology* 180: 245-251, 1991

11. Guyton AC: Textbook of Medical Physiology, Seventh Edition, WB Saunders Co., 1986

12. Juhl J, Fibromyalgia and the serotonin Pathway, *Alternative Medicine Review,* Vol. 3, Number 5, 1998

13. Kandel E, Schwartz J: Principles of Neural Science, second edition. Elsevier Science Publishing Co., Inc., New York, 1985: pp. 331-336

14. Lombard MC, Larabi Y: Electrophysiological study of cervical dorsal horn cells in partially deafferented rats. In: Advances in Pain research and Therapy. New York: Raven Press, 1983: 147-54

15. Marshall LL, Trethewie ER, Curtain CC: Chemical radiculitis. A clinical, physiological and immunological study. *Clinical Orthopedics*; 129: 61-67 1977

16. McCarron RF, Wimpee MW, Hudkins PG, Laros GS: The inflammatory effect of nucleus pulposus: A possible element in the pathogenesis of low back pain. *SPINE*; 12, 760- 764, 1987

17. Mendel T, Wink CS: Neural elements in cervical intervertebral discs. *Anatomic Record* 223: 78A, 1989

18. Mountz JM, et al.: Fibromyalgia in women: abnormalities of regional cerebral blood flow in the thalamus and the caudate nucleus are associated with low pain thresholds levels. *Arthritis Rheum;* 38: 926-938, 1995

19. Netter F: Atlas of Human Anatomy, Ciba-Geigy, 1991, plate 159

20. Olmarker K, Rydevik B, Nordberg C. Autologous nucleus pulposus induces neurophysiologic and histologic changes in porcine cauda equina nerve roots. *SPINE*; 18: 1425-32, 1993

21. Olmarker K, Blomquist J, Stromberg J, Nannmark U, Thomsen P, Rydevik B: Inflammatogenic properties of nucleus pulposus. *SPINE*; 20: 665-669, 1995

22. Ozaktay AC, Cavanaugh JM, Blagoev DC: Phospholipase A_2 – induced electrophysiologic and histologic changes in rabbit dorsal lumbar spine tissues. *SPINE*; 20: 2659-68, 1995

23. Ozaktay AC, Kallakuri S, Cavanaugh JM: Phospholipase A_2 sensitivity of the dorsal root and dorsal root ganglion. *SPINE*, Vol. 23, Number 12, pp1297-1306, 1998

24. Taylor, JR and Twomey, LT: Acute injuries to cervical joints. An autopsy study of neck pain. *SPINE,* Vol. 18, Number 9, pp1115-1122, 1993