CASE REPORT: MYOFASCIAL PAIN SYNDROME: A DOUBLE CRUSH-LIKE APPEARANCE

R. KEVIN PRINGLE, DC,^a DANIEL L. RICHARDSON, PHD,^b AND
ROBERT S. SHIEL, PHD^c

^aPrivate Practice of Chiropractic, Houston, TX. ^bProfessor, Department of Chiropractic Practice and Department of Biochemistry, Nutrition, National University of Health Sciences, Lombard, IL. ^cAssociate Professor, Department of Diagnosis, National University of Health Sciences, Lombard, IL. Submit requests for reprints to: Dr. R. Kevin Pringle, 4107 Pinebridge, Spring, TX 77388. Paper submitted September 6, 2002.

ABSTRACT

Objective: To discuss the case of a patient with myofascial pain syndrome that appeared similar to double crush syndrome.

Clinical Features: This is a case report of a 45-year old female who presents to the private practice clinic with the same signs and symptoms of carpal tunnel syndrome, 1 year post-carpal-tunnel surgery. The history is consistent with injuries that result in double crush syndrome and thoracic outlet syndrome. The patient history includes a motor vehicle accident (MVA) in 1963 that resulted in a prolonged hospital stay and fracture of a cervical vertebral (C4) body. She fractured the olecranon process of the right elbow during a fall in 1970's. She has smoked approximately 1 pack a day for 20 years. Her job involves long periods of sitting at a keyboard and in a car. All of these portions of the patient history are possible factors in the clinical signs and symptoms of double crush syndrome.

Intervention and Outcome: Treatment involved chiropractic manipulation and physical therapy modalities for the elimination of pain and returning the patient's full range of motion in the wrist, elbow, shoulder and cervical spine. After range of motion was restored, a home exercise program was initiated. The home exercise program included a return to past sporting activity and active, resistive (Theraciser band) activities for the elbow and cervical spine. Also a reduction in smoking was initiated as part of the patient's treatment plan.

Conclusion: It is our contention that patients who have electromyographic (EMG) demonstrable carpal tunnel syndrome need a cervical spine workup and a period of conservative care to include skilled manipulation, physical therapy and home exercises before surgery is recommended. (*J Chiropr Med* 2003; 2:66-74)

KEY WORDS: Myofascial Pain Syndrome; Double Crush Syndrome; Manipulation; Botanical Therapies; Somatoform Disorders; Doctor-Patient Relations

INTRODUCTION

Myofascial pain syndrome is characterized by the presence of active trigger points that refer pain to remote locations (1). There are also latent trigger points but they do not produce pain and may cause restricted movement (1). The classic finding for active trigger points is the presence of hyperirritable, taut band of muscle (1-3). As a result of the active trigger points many signs and symptoms of classic peripheral nerve disorders can be, and are, mimicked by the trigger points. These trigger points should be a member of the differential list for most, if not all, peripheral nerve disorders. There are many names by which trigger points have been called and described in the literature; examples include muscular rheumatism, myalgia, myogelose and interstitial myofibrositis (2,3).

The diagnosis of the myofascial pain syndrome in active trigger points is based on history and physical examination that includes the active search for the active trigger points. The taut, palpable band of hyper-irritable muscle is the classic description of the changes in muscle associated with myofascial trigger points. The symptoms of myofascial pain syndrome are referred pain in specific patterns, range of motion is limited, muscle may be weaker when tested, "jump sign", local twitch response and constant pressure by examiner causes pain in referred zone to intensify. Historically, acute overload of stress can produce the changes and a long-term overload can produce gradual onset of myofascial trigger points (1-3). Also, a precise pain drawing could be useful in the diagnosis of the specific trigger points (2,3).

Treatment of myofascial trigger points are stretch-and-spray procedure, ischemic compression, dry needling, anesthetics and ultrasound (1-3). Each treatment regime has demonstrated effectiveness. Lewit has also stated that inactivation of the trigger point should occur in conjunction with joint mobilization (2). One in the absence of the other may predispose the patient to recurrence of the active trigger points (2,3).

As stated previously the active trigger points in myofascial pain syndrome that can mimick the signs and symp-

toms of many peripheral nerve disorders. Some that are mimicked are carpal tunnel syndrome, ulnar tunnel syndrome, thoracic outlet syndrome and double-crush syndrome (4-7).

Double crush syndrome is a disorder of which pain, numbness and weakness of an extremity due to 2 or more compression or compression-like abnormalities of peripheral nerves. Double crush syndrome may be a commonly misdiagnosed disorder of the upper extremity and cervical spine (8) but the actual diagnosis remains controversial (9-11), controversial enough that some authors suggest abandoning it as a diagnosis (10). This may be the result of conflicting data reported as to the significance of carpal tunnel with some cervical radiculopathy (12) and cervical arthritis (12,13). As a result of the conflicting data there are failed carpal tunnel surgeries that have and may respond to conservative management with manual therapy. There has also been the suggestion that an overlap of thoracic outlet syndrome, carpal tunnel and double crush syndrome may occur (8,14). The findings of 1 syndrome may be misinterpreted and/or not diagnostically linked to the appropriate syndrome or diagnosis, leading to failure of recommended treatment plans whether conservative or surgical management. Further confusing the diagnosis is the possibility for double crush syndrome to occur along any peripheral nerve at different places (15). Clinical data reported has centered on the large nerves of the upper extremity (16).

In the upper extremity and cervical spine there are multiple structures along the path of the median nerve and its more proximal parts and the ulnar nerve and its more proximal parts which may cause irritation (11,15). Some of the structures which can irritate the median nerve are a cervical rib, enlarged transverse process of the C5-C7 vertebral units, scalene muscle hypertrophy, drooping shoulders, intervertebral disc protrusion, superficial and deep heads of the pronator teres and the carpal tunnel's transverse ligament (17-26). There also are metabolic conditions which predispose patients to symptoms both distally and proximally; these include pregnancy (18,25), diabetes (21), excessive weight gains (18), thyroid disorders (18,22,27), and more. Lastly, traumatic child deliveries and/or childhood injuries may produce traction on the nerve roots and/or peripheral nerves.

Upton and McComas proposed a hypothesis based on the phenomenon of axoplasmic flow (28). The Upton and McComas hypothesis was preceded by the description of the link between cervical spine and shoulder and carpal tunnel syndrome symptoms (14). Yet this theory

of altered axoplasmic flow remains controversial and unproven (28). The normal physiology and function of the nerves is that messenger substances, trophic materials, are synthesized within the cell body and transported along the nerve fiber. Damage of a nerve fiber and failure of delivery of trophic material beyond a point of constriction results in nerve damage. But this hinderance of the delivery of trophic material may occur at more than 1 site along its pathway, thus the diagnosis of double crush and not simply thoracic outlet or carpal tunnel syndrome of and by themselves (28). One author suggests that the double crush syndrome lesions are not rare in the presence of thoracic outlet syndrome findings (19).

The effects of peripheral nerve entrapment by both mechanical and metabolic mechanisms appears to result in the same pathophysiological mechanism (30). The alteration in the axoplasmic flow (12,28) and denervation phenomenon will produce the signs and symptoms of clinical syndromes (8). It is sometimes difficult to distinguish between which mechanism is the culprit. Diabetic neuropathy (25,31), vitamin deficiencies (23), alcoholism (25), cervical acceleration-deceleration injuries (32), cervical ribs (29) and cervical spondylosis (10) are a small list of causes which can simulate double crush syndrome and other similar peripheral neuropathies, i.e. carpal tunnel syndrome and thoracic outlet syndrome (19).

Carpal tunnel was first recognized in 1854 by Paget. Carpal tunnel syndrome is a relatively common clinical syndrome (17,18,24,26) that affects women 5 times more than men (18,24,25) and 1% of the population (17,25). The signs and symptoms of carpal tunnel affect the hand, wrist and distal arm, they are: (1) loss of sensation of the thumb, 2nd digit and median half of the 3rd digit (18,20,24-26,29,30), and (2) muscle wasting of the thenar eminence (23) is evident in prolonged cases, associated with compression of the median nerve in the carpal tunnel (17,18,20,22,24-26,29,30). Historically carpal tunnel syndrome is usually insidious (17,24-26,29) but acute traumatic carpal tunnel syndrome does occur.

This diagnosis can be confirmed by EMG examination of median nerve (18,20,22,24-26,33) and is considered the gold standard (20,33). But the clinical diagnosis can be made based upon subjective presentation of the patient (34,35). In some patients there is evidence of multiple nerves being affected; this may include the ulnar nerve (21,36). There is some evidence to suggest the myofascial pain referral patterns from the shoulder, upper arm and forearm muscles will appear like thoracic

outlet syndrome or carpal tunnel syndrome. This is sometimes referred to the wrist only and not the myofascial referred pain patterns (37). In an unpublished study by Upton and McComas which is described in Simpson and Fern's paper (38), they found that 81% of patients with EMG-demonstrable peripheral neuropathy also had evidence of a cervical root lesion. The impaired flow of the trophic material beyond the area where damage may have occurred can predispose the patient to carpal tunnel-like symptoms and/or carpal tunnel syndrome, carpal tunnel-like symptoms if the damage occurs proximally to the wrist and carpal tunnel syndrome if the damage occurs in the carpal tunnel (24).

Thoracic outlet syndrome is a disorder of the compression of a portion of the brachial plexus and vascular structures associated with the brachial plexus in the thoracic outlet (29,39). Thoracic outlet syndrome symptoms are vague (39) and can mimic carpal tunnel syndrome. More of the possible symptoms are (1) weakness of all the intrinsic muscles with sensory loss over the ulnar side of the hand (30), (2) discoloration of the hand (29,30,39), (3) Raynaud's phenomenon (29,39), and (4) decrease in the pulse (30). The patient may not be able to accurately describe the areas of paraesthesia, making the diagnosis difficult (40). This is typical of the vagueness of the symptoms associated with thoracic outlet syndrome. The orthopedic tests for thoracic outlet syndrome are not highly sensitive or specific for thoracic outlet syndrome. Thus, thoracic outlet syndrome from the scalenes (29,41), cervical rib (14,29,41), clavicle (29,41), morphological changes of the 1st rib (41,42) or pectoralis minor impingement, may be vague and difficult to differentiate or diagnosis. The high level of false positive for these tests are misinterpreted and treatment of the area is often initiated without further distal examination which reproduce the patient's symptoms (39). The symptoms of thoracic outlet syndrome can be provoked by raising the arms above the shoulder, like Wright's test (29,39) or lifting a weight. The symptoms are often nocturnal, awaken the patient several times during the night and may follow ulnar or median nerve distributions. Lastly, in a series of papers (43,44) there is a description of a variant of thoracic outlet syndrome from myofascial trigger points. These papers describe the treatment as manipulation, some passive physical therapy modalities, postural awareness, an at-home-stretching program and myofascial release technique were performed (43).

The structures described above in the cervical spine area can cause and are linked to the symptoms of carpal tunnel syndrome which can be also associated to the

possible double crush syndrome. The vascular component of thoracic outlet syndrome further makes TOS different from carpal tunnel and can be impinged by the same structures in the neck and upper extremity. But since double crush may have its origins in the cervical spine or thoracic outlet it may be similar to thoracic outlet syndrome symptomatically and etiologically but has additional distal changes of the neuronal axon.

In Rydevik's study on compression of nerves and the effects, he found that venous blood flow was stopped at 5–10 mm Hg in some venules (45). The venular congestion suggested to be an important pathophysiological mechanism in nerve compression syndromes (46). They also found that 10 mm Hg was sufficient to induce a reduction of the transport of methyl-glucose to nerve roots (45). These are caused by trauma, disc herniation and spinal stenosis (45). Thus, when the impairment of nutrients to the nerve roots occurs, possibly mechanical and/or chemical, the patient is predisposed to symptoms distally along the nerve pathway. It is possible that smoking can cause retrograde venous stasis and increase the pressure a nerve is under, thus hindering the flow along a given nerve.

Another consideration is T4 syndrome, upper extremity paraesthesia with or without dull headache that arises from the thoracic spine (47), and was described by Maitland (48), McGuckin (49), and Grieve (50). The T4 segment appeared to most frequently implicated but T2-T7 can be involved (47). It appears to be a related to an upper cross syndrome clinical presentation with anterior head-carriage, rolled forward shoulders, internal rotation of the arms and protracted scapula. Signs and symptoms include dull headache, glove-like paraesthesia and is more commonly found in women than men. Treatment recommendations include postural exercises, stretching exercises and manipulation (47).

CASE REPORT

A 47-year old white female suffered from bilateral wrist and hand numbness and tingling, right elbow pain and cervical spine stiffness. The numbness and tingling was described as covering the 1st and 2nd digits on both hands. Her wrist and hand problems had been present on and off for the previous 3 to 4 years.

She did not recall any traumatic event recently that may have precipitated the onset of the numbness and tingling in her hands. She was involved in a motor vehicle accident in 1963 where she suffered a C4 vertebral body fracture and the right olecranon when she fell and

hit the elbow on the pavement. She was hospitalized for 10 days following the motor vehicle accident and released with a cervical collar and prescription for pain killers. She had also been in 10 other "less severe" motor vehicle accidents. She stated that she was working as an insurance salesperson and had previously spent time writing a book. When asked, both of these endeavors required extensive time working on the computer and completing forms and that working on the computer precipitated the hand and wrist numbness and tingling. She could not recall any 1 activity that made the wrist better. But she did report temporary relief following the carpal tunnel surgery on both wrists. But the symptoms returned in both wrists prior to her seeking care in our office. At this point she states that she is frustrated with her current medical care and the physician was ambivalent toward her concerns. Lastly, she is a 1.5–2 pack a day smoker for the past 30 years.

She complained of headaches and a stiff neck that seem to be worsening due to stress. She had not been working because her surgeries, and her family was going through upheaval that she felt burdened her extensively. Her headaches and neck stiffness was bilateral and occasionally unilateral, around the eyes and temple regions. Relaxing and resting seem to decrease the frequency and strength of the headaches.

Vital signs were normal. The heart and abdomen examination were deferred during the initial examination but the lung examination was performed due to her long-term smoking and revealed slight crackles at the base of both lungs, but tactile and vocal fremitus and voice sounds were normal.

Orthopedic testing revealed a decrease in range of motion of the cervical spine, visually, in extension, right and left lateral bending, and right rotation. Overpressure at the end of active extension range of motion was painful in the suboccipital area and there was pain at the paravertebral musculature with lateral bending, which she described as "muscle pulling." Tests for nerve root irritation were negative for radicular symptoms but did cause muscle pain in the cervical and upper thoracic spine from being stretched. Trigger points were palpated in the suboccipital triangle bilaterally, medial head of triceps on the right, pronator teres bilaterally, anterior scalenes bilaterally and brachioradialis on the right. The pronator teres point on both arms referred into the hands similar to the chief complaint. The brachioradialis and medial head of the triceps trigger points on the right both referred pain into the right hand similar to the chief complaint. The anterior scalenes and the suboc-

cipital muscles referred pain along the temporal and frontal areas of the head similar to her headaches.

Baseline neurological testing, dermatomes, myotomes and monosynaptic reflexes were performed and provided no helpful diagnostic information. The myotomes were +4 or +5 and the decrease was associated with pain from muscle stretch in the cervical spine. Dermatomes were equal bilaterally and circumferential sensory testing was unable to determine any nerve root involvement. Lastly, reflexes were normal.

Radiographs were not performed of any area because the surgeon had recently performed plain films of the cervical spine and wrist. The films were requested and provided by the surgeon for evaluation. The 3 view cervical spine films revealed the following findings:

1. Multiple anomalies of the atlanto-axial articulation including spina bifida occulta and deformity of the atlanto-axial joints.
2. Kypho-scoliosis of the cervical spine.
3. Degenerative discopathy C4–5 with mild posterior marginal hypertrophic spondylosis.
4. Anterior notch deformity at the anterior superior corner of C4 probably from old flexion injury.
5. Moderate arthrosis of the posterior joints throughout the cervical spine.
6. Due to anatomical variance, it was difficult to assess the significance of C7 transverse process being larger than T1 transverse process.

At this point she was diagnosed as suffering from:

1. Double crush-like syndrome secondary to myofascial trigger points.
2. Cervicogenic headaches secondary to myofascial pain syndrome.
3. Degenerative disk disease of the C5–C6, C6–C7 intervertebral disk spaces.
4. Possible de-innervation-supersensitivity-like pain syndrome from the motor vehicle accident in 1963 and the other previous motor vehicle accidents.

A treatment plan was instituted to attempt to increase range of motion of the cervical spine, decrease the frequency of headaches and decrease the numbness and tingling effects of the myofascial trigger points in both hands. Initially, the treatment plan was 4 visits per week for the first 2 weeks before re-evaluation. Manipulation of the cervical spine, shoulders, elbows and wrists was performed; on some days, manipulation was not tolerable in the right elbow and right wrist.

Manipulation was a high-velocity, short-lever thrust which is typical in the clinical practice. The cervical spine manipulation was performed with her lying supine. It seemed that the manipulation was easier to perform when the soft-tissue massage was done before the manipulation.

The shoulder, elbow and wrist were manipulated with the patient seated. The shoulder was adjusted with the arm completely flexed at the elbow. The doctor reaches around the patient with both hands and grasps the elbow. Moving the arm through passive movements with overpressure applied, "restrictions" were attempted to be identified. To add more support to the patient's shoulder, the patient leaned back against the doctor's chest (sternum) and abdomen. Then, when a "restriction" was found a high-velocity thrust was applied along the plane of limited movement.

The elbow was also performed with the patient seated. The adjustments performed were (1) A radial head rotatory adjustment and (2) Medial-to-lateral glide movement of the olecranon. First, the radial head will be discussed. The arm was flexed to 90 degrees with some pronation and internal rotation of the arm. A thenar pad contact on the radial head was taken with the elbow in 90–95% of extension. A quick high-velocity thrust around the positive "x-axis" and along a positive "z-axis" was given.

Lastly, the wrist was also adjusted with the patient seated. The arm was flexed to 90 degrees. A bilateral thumb contact was taken on the ulna and radius. The wrist was flexed to 90 degrees and a longitudinal tractional force was applied to both the radius and ulna. Then, a "whip-like" movement is made by moving the wrist from flexion to neutral wrist positioning while an impulse is applied to the radius and ulna by the bilateral thumb contact. It appears in this case that a combination of these adjustments with the other modalities was useful in the restoring cervical spine range of motion, eliminating double crush symptoms, alleviation of headaches and the alleviation of the neck pain.

Cross-friction massage to the trigger points was done following 15-minutes of cryotherapy massage. The cryotherapy was used to decrease the pain associated with compression of the trigger points during cross friction massage.

Ice packs were used over the suboccipital muscles, pronator teres and right medial triceps post massage to alleviate some the post-massage pain from the compression of the trigger points and expected hyperemia (51).

She was asked to begin a 20–30 minute walking activity daily to begin aerobic conditioning. This treatment plan appears to have been effective during the first 2 weeks. She was 90% pain free in her left wrist and hand, and about 50% pain free in the right wrist and hand after 2 weeks. With the improvements using the above treatment plan, the same treatment plan was continued for the next 6 weeks with a change in the frequency of visits. Weeks 3–5, she was seen 3 days a week, and week 6 she was seen 2 days a week; weeks 7 and 8, only 1 visit a week. At the end of 2 months, the patient was pain-free in her left wrist and had not suffered any numbness and tingling of the left wrist. The right wrist and hand still had occasional tingling 10% of the time.

An intriguing bit of information was discovered during the subjective questioning during the assessment of the patient after a weekend. She was unable to smoke the entire weekend and reported that she did not have any numbness and tingling, but 1 week later she had begun smoking again and her right wrist and hand was again occasionally tingling. Lastly, she was able to return to work full-time and was also able to return to playing tennis 2 days a week; she had been unable to do for parts of the past 4 years due to the elbow and wrist pain following playing.

DISCUSSION

In this case manipulation, a passive soft tissue therapy and an at-home exercise plan seem to be effective in treating a double-crush-like syndrome secondary to myofascial trigger points. There are some other conservative management strategies that may have helped in treating this patient more effectively. A more vigorous smoking cessation program, acupuncture or hypnotherapy could have been instituted earlier. Smoking seems to be a confounding factor responsible for a portion of the numbness and tingling.

The complication rates from surgical management of the carpal tunnel syndrome associated with the recurrence of the symptoms following surgery can be the consequence of the surgery itself (52) or an underlying predisposing condition (52) that was not recognized as the primary cause of the carpal tunnel syndrome. Further making the diagnosis more difficult is the possibility of double crush syndrome and myofascial trigger points which can mimic carpal tunnel syndrome.

The patient was frustrated by what she perceived as ambivalence of the surgeon when he told her "the pain is in your head" after the failure of the surgeries. Indi-

viduals who experience chronic pain may find their complaints of subjective distress discounted by medical professionals who in the absence of an identifiable biological explanation define the problem as somatoform or the pain as imaginary. For double crush, the absences of observable evidence of pathology together with the ambiguous status of the illness in professional health care may preclude the possibility of a diagnosis for many sufferers.

This failure of the system to identify the source of the patient's problem leads to the delegitimization of the patient's experience of the symptoms. There are 2 types of delegitimizing encounters between doctor and patient. The first is based on the insignificance of the symptoms and the second, and more damaging, on the definition of the illness as somatoform or even worse, as "all in the patient's head." The reality constructed for sufferers of double crush syndrome through delegitimizing experiences is that their illness is not 'real' at all but rather a fabrication based either on the needless exaggeration of everyday complaints, in which case they are malingerers or on the perception of imaginary symptoms in which case they are psychologically ill. In either case, the self-doubt, the threat of stigma, the social isolation that results, the psychological paralysis induced by the ambiguities of the illness, and the shame about being wrong about really being sick all contribute to the suffering of the patient (53). Failure of health care to name the patient's condition leaves patients feeling not only betrayed but also with no means of coping. Without diagnosis there is no treatment, no way of combating the illness. Not knowing makes life decisions and planning for the future difficult or impossible (54).

Using lack of physical findings as the sole determining factor in concluding a patient has a psychological disorder can lead to misdiagnosis. For example, Riley & Roy (55) found that 25% of patients diagnosed as having pseudo-neurologic symptoms were found to have bona fide neurological disorders over the course of 5-year follow-up. The diagnosis of somatoform and other psychological disorders consequently should not be made on the basis of failure to find adequate physical cause alone. The different psychological disorders have other criteria and demographic features that allow for a more positive diagnosis and not a diagnosis of exclusion.

Conversion (pseudo-neurologic) symptoms typically have onset between ages of 10–35. When symptoms occur at later ages the likelihood of a general medical condition is high. In addition, conversion symptoms are typically of short duration.

Pain disorder has chronic pain but the diagnosis requires that psychological factors are judged to have an important role in the onset, severity, exacerbation or maintenance of the pain. These psychological factors may be either the presence of other psychological disorders or identifiable psychosocial stressors. The physician should look for a temporal relationship between the symptoms and these factors.

Somatization disorder presents with pain symptoms in combination with others. The criteria require a minimum of 4 pain symptoms, 2 GI symptoms, 1 pseudo neurologic and 1 sexual. This pattern of unexplained symptoms with onset before age 30 is unlikely in patients without the disorder (56).

In addition, physical complaints are often the identifying features of psychological disorders other than the somatoform disorders. Patients with depressive or anxiety disorders will often present with musculoskeletal pain, GI distress and headaches and not recognize or minimize the emotional or psychological symptoms. Thorough evaluation of the patient's psychological state should always be integrated into the on-going health assessment of the patient and not left as a last resort.

There are also common fracture of the upper extremity that could have been considered a predisposing factor in the failure of the carpal tunnel release. The fractures are one of those etiologies that make double crush syndrome a reality and needed consideration with carpal tunnel if the patient history indicates such. Olecranon fractures have been associated with median and ulnar neuropathies (32). The effects of old cervical spine fracture in this case may have some of the effects of the double crush syndrome and headaches because of the possibility of "presondylosis"-denervation supersensitivity changes (57).

Tradition medical management of carpal tunnel syndrome (CTS), thoracic outlet syndrome (TOS), and double-crush syndrome is divided into conservative and non-conservative surgical approach. The medical conservative approach involves prescription medication, splinting and steroid injection (14,58). The medications and steroid injections work to mask the signs and symptoms of inflammation but has detrimental effects on the soft-tissues in and about the injection site. The types of medications typically used are non-steroidal anti-inflammatories drugs (NSAIDs) (17–19,22,25,26,29), corticosteroids (17,18,22,25,26,29), and diuretics (22,35,40,58). NSAIDs include ibuprofen and naproxen among others. NSAIDs have adverse effects of the gastrointestinal tract, central nervous system and metabolic

system (59). The adverse effects to the gastrointestinal tract are due to the intolerance of the gastric tract and result in gastritis; this can be diminished by using a buffering agent. This side-effect can be the result of irritation of gastric mucosa from an undissolved tablet, non-ionized salicylate absorption by the stomach, or inhibition of prostaglandin synthesis which provides a protective layer to the gastric mucosa (59). In animal studies it is the past, prostaglandin inhibition, that seems to make the gastric mucosa vulnerable to ulceration (59).

There are a number of natural anti-inflammatory medications that may be as useful and without the side effects of gastritis leading to gastric ulcers. Alternatives to the use of NSAIDs include bromelain, tumeric, and evening primrose oil. They act to decrease inflammation by affecting the arachidonic acid and leukotriene cascades (60) (See Figure 1). The effects of these 3 natural anti-inflammatory botanicals are high in gamma-linolenic acid (GLA) and act to inhibit arachidonic acid synthesis.

Another treatment component associated with dietary counseling and its effects on the inflammatory process

includes the elimination of pro-inflammatory food products such as meat, dairy fat, wheat, corn and shellfish; these increase the amount of arachidonic acid. If the patient is using NSAIDs and is unwilling to use the alternatives, then demulcents and mucogenics should be supplied. These include botanicals such as slippery elm and marshmallow, which can be found in several products.

Corticosteroids are used for the treatment of inflammatory conditions. Corticosteroids are produced in the adrenal cortex and include mineralocorticoids and glucocorticoids. The major glucocorticoid is cortisol and is the synthetic glucocorticoid analogue. The corticosteroids block all the known pathways of eicosanoid metabolism. This is done by inhibiting the phospholipase A-2, which acts to liberate arachidonic acid. In turn arachidonic acid is acted upon by leukotrienes and lipoxygenase. The reason corticosteroids are used in place of NSAIDs is that NSAIDs only inhibit the cyclooxygenase pathway and not lipoxygenase (60). The side-effect of corticosteroids are avascular necrosis (especially of the hip), fatty embolism, tendon rupture (42), micro-trauma, alteration of biomechanical properties of the ligamentous tissue (42), and osteoporosis. In Kapelanos'

Figure 1: Prostaglandin and leukotriene production

paper it was found that the ligamentous changes associated with corticosteroid use are decrease in tendon weight, decrease in load to failure rate and decrease in energy to failure rates but no significant difference in healing time when compared to saline injections (42). These are not all of the side-effects but provide a list that demonstrate the need for an alternative to the corticosteroids. Some of the alternatives to the corticosteroids include bromelaine, curcumin and chamomile tea. The bromelaine acts by increasing prostaglandin synthesis at the PG3 level. Curcumin acts to increase alpha linoleic acid which is a precursor to the eicosapentaic acids. Lastly, the chamomile tea contains apigenin which will act to increase linoleic acid in the PG1 series. All three without the detrimental effects as the synthetic glucocorticoid (60). An additional point is that 1 paper found that 15 weeks of injections were necessary for the changes to occur (61).

Diuretics are used to treat the symptoms of CTS, TOS, and double-crush syndrome. Diuretics have been used to treat fluid volume and electrolyte abnormalities. These drugs act to block the transport functions of the renal tubules (59). Figure 1 shows the renal tubule transport system and the action sites of the diuretics (59). A major concern with the use of diuretics is the number of contra-indications that exist with their usage. Some of the alternatives that may be use in place of diuretics include hawthorn and dandelion. These act by increasing the glomerular filtration rate as a result of increased contraction force of the heart as well as a direct action on the distal tubule, which is similar to Thiazides.

Lastly, vitamins and minerals may be consider important in treating patients with CTS, TOS, and double-crush syndrome. The B-6 and B-12 vitamins appear to be essential for treating nervous tissue dysfunction (18,22,24,26) but like much of this topic is also a controversial topic (22,23). Magnesium and calcium supplementation is also important for maintaining muscle strength and function, because possible disuse leads to atrophy and cramping (61,62).

The amelioration of the last 10% of pain and paraesthesia in the right hand and the cessation of smoking can be pure coincidence but there exists some evidence to the contrary. Nicotine has several effects on the cardiovascular system, including a direct effect on vasculature, and an indirect effect on the adrenal gland. The release of adrenaline causes vasoconstriction in the periphery and an increase in heart rate and blood pressure. The direct effect is also evident as it blocks and stimulates the cholinergic system. High doses of nicotine in heavy

smokers causes a blockage of musculoskeletal contraction. Another action of nicotine is to increase platelet adhesiveness, leading to an increased tendency for clotting in the periphery. We do not suggest that this patient had a clot of any sort. But the first two actions of nicotine can have an affect that may possibly increase the effects CTS, TOS, and double-crush syndrome in an already vulnerable area. The discontinuation of smoking and the enhanced oxygen carrying capacity of the blood may have enhanced the resolution of symptoms. Lastly, enhanced oxygen carrying capacity is associated with the decrease in carbon monoxide, which can be found as high as 6% in tobacco products. This can lead to an increase of carboxyhemoglobin of up to 10% in smokers, thereby decreasing the oxygen concentration in the blood (63).

REFERENCES

1. Travell JG, Simons DS. Myofascial pain and dysfunction: The trigger point manual. Baltimore, MD: Williams & Wilkins; 1992.
2. Simons DG, Travell JG. Myofascial pain syndromes. Textbook of Pain. Chapter 25.
3. Simons DG. Myofascial pain syndrome due to trigger points. International Rehabil Med Assoc; 1987.
4. Hong CZ, Simons DG. Pathophysiologic and electrophysiologic mechanism of myofascial pain trigger points. Arch Phys Med Rehabil 1998;79:863-70.
5. Hong CZ. Myofascial trigger point injection. Crit Rev Phys Rehabil Med 1993;5:203-17.
6. Hong CZ. Consideration and recommendation of myofascial trigger pint injection. J Musculokel Pain 1994. p. 229-59.
7. Rosen NB. The myofascial pain syndrome. Phys Med Rehabil Clin North Am 1993;4:159-72.
8. Osterman AL. The double crush syndrome. Orthop Clin North Am 1998;19: 147-55.
9. Richardson JK, Forman GM, Riley B. An electrophysiological exploaration of the double crush hypothesis. Muscle Nerve 1999;22:71-7.
10. Johnson EW. Double crush syndrome: a definition in search of a cause. Am J Physical Med Rehab 1999;76:439.
11. Wilborn AJ, Gilliatt RW. Double-crush syndrome: A critical analysis. Neurology 1997;49:21-9.
12. Mariano KA, McDougle MA, Tanksley GW. Double crush syndrome: chiropractic care of an entrapment neuropathy. J Manipulative Physiol Ther 1991;14:262-5.
13. Upton A, McComas A. The double crush in nerve entrapment syndromes. Lancet 1973;2:359-62.
14. Naska AO. The role of thoracic outlet syndrome in the double crush syndrome. Ann Hand Surg 1990;9:331-40.
15. Flatt DW. Resolution of a double crush syndrome. J Manipulative Physiol Ther 1994;17:395-7.
16. Osterman AL. The double crush syndrome. Ortho Clin North Am 1988;19: 147-55.
17. Demopulos GA, Urbaniak JR. Carpal tunnel release: comparing the options. J Musculoskeletal Med 1996;13:51-8.
18. Katz RT. Carpal tunnel syndrome: a practical review. Am Family Physician 1994;49:1371-9.
19. Davis PT, Hulbert JR. Carpal tunnel syndrome; conservative and nonconservative treatment. A chiropractic physician's perspective. J Manipulative Physiol Ther 1998;21:356-62.
20. Hennessey WJ, Kuhlman KA. The anatomy, symptoms, and signs of carpal tunnel syndrome. Physical Med Rehabil Clin North Am 1997;8:439-57.
21. Vaughn NM, Pease WS. Postoperative complications of carpal tunnel surgery. Physical Med Rehabil Clin North Am 1997;8:541-53.
22. Kraft GH. Carpal tunnel syndrome in patients with peripheral neuropathy: it can be evaluated and treated. Physical Med Rehabil Clin North Am 1997;8:577-95.

23. Franzblau A, Rock CL, Werner RA, Albers JW, Kelly MP, Johnsto EC. The relationship of vitamin B6 status to median nerve function an carpal tunnel syndrome among active industrial workers. *J Occupational Environmental Med* 1996;38:485-91.
24. Greenly LW. Carpal tunnel syndrome. *Chiropr Technique* 1998;10:190.
25. Steyers, Schelkun PH. Practical management of carpal tunnel syndrome. *Physician Sportsmed* 1995;23:83-8.
26. Greensspan J. Carpal tunnel syndrome: A common but treatable cause of wrist pain. *Postgrad Med* 1988;84:34-43.
27. Spindler BA, DeBon AL. Nerve conduction studies and testing of carpal tunnel syndrome. *J Hand Surg* 1982;7:260-3.
28. Morgan G, Wilbourn AJ. Cervical radiculopathy and co-existing distal entrapment neuropathies: Double-crush syndrome? *Neurology* 1998;50:78-83.
29. Atasoy E. Thoracic outlet compression syndrome. *Orthop Clin North Am* 1996;27:265-303.
30. Dawson DM. Entrapment neuropathies of the upper extremities. *New England J Med* 1993;329:2013-8.
31. Brown MJ, Asbury AK. Diabetic neuropathy. *Am Neurol* 1984;15:2.
32. Kumar A, Lennard TA, Margherita A. Extremity trauma in motor vehicle accidents. *Physical Med Rehabil: State of Art Reviews* 1998;12:99-109.
33. Vender MI, Truppa KL, Ruder JR, Pomerance J. Upper extremity compressive neuropathies. *Physical Med Rehabil: State of Art Review* 1998;12:243-62.
34. Huffman JD. Electrodiagnostic techniques and conservative treatment of thoracic outlet syndrome. *Clin Orthop* 1986;207:21-3.
35. Karas SI. Thoracic outlet syndrome. *Clin Sports Med* 1990;9:297-310.
36. Twogood DA. Carpal tunnel syndrome: A new way of thinking. *Am Chiropr* 1998;20:42-3, 47.
37. Golovchinsky V. Double crush syndrome in lower extremities. *Electromyogr Neurophysiol* 1998;38:115-20.
38. Simpson RL, Fern SA. Multiple compression neuropathies and the double crush syndrome. *Orthop Clin North Am* 1996;27:381-8.
39. Karas SE. Thoracic outlet syndrome. *Clin Sports Med* 1990;9:297-310.
40. The medical advisor. The complete guide to alternative and conventional treatments. Time-Life Books. 1996. p. 234-7.
41. Nichols HM. Anatomic structures of the thoracic outlet. *Clin Orthop* 1986;207:13-20.
42. Kapetanos G. The effect of the local corticosteroids on the healing and biomechanical properties of the partial injured tendon. *Clin Orthop* 1982;163:170-9.
43. Sucher BM. Thoracic outlet syndrome-a myofascial variant: Part 2. Treatment. *J Am Osteopath Assoc* 1990;90:810-45.
44. Sucher BM, Heath DM. Thoracic outlet syndrome-a myofascial variant. Part 3. Structural and postural considerations. *J Am Osteop Assoc* 1993;93:334, 340-5.
45. Rydevik BL. The effects of compression of the physiology of nerve roots. *J Manipulative Physiol Ther* 1992;15:62-6.
46. Sunderland S. Brachial plexus injuries. *Clin Neurol Neurosurg* 1993;95(Suppl):S1-2.
47. DeFranca GG, Levine LJ. The T4 syndrome. *J Manipulative Physiol Ther* 1995;18:34-7.
48. Maitland GD. Vertebral manipulation. 5th Ed. Boston, MA: Butterworth; 1986. p. 344.
49. McGuckin N. The T4 Syndrome. In: Grieve GP, editor. *Modern manual therapy of the vertebral column*. New York, NY: Churchill Livingstone; 1986. p. 238.
50. Grieve CP. Common vertebral joint problems. New York, NY: Churchill Livingstone; 1983. p. 238.
51. Chamberlain GJ. Cyriax's friction massage: a review. *J Orthop Sports Physical Ther.* 1982;4:16-22.
52. Idler RS. Persistence of symptoms after surgical release of compressive neuropathies and subsequent management. *Orthop Clin North Am* 1996;27:409-16.
53. Kleinman A. The illness narratives: Suffering, healing and the human condition. New York, NY: Basic Books; 1988.
54. Ware NC. Suffering and the social construction of illness: The delegitimation of illness experience in chronic fatigue syndrome. *Med Anthropology Quat* 1992;6:347-61.
55. Riley TL, Roy A. Pseudoseizures. Baltimore, MD: Williams & Wilkins; 1982.
56. DSM-IV. American Psychiatric Association. Washington, DC; 1994.
57. Gunn CC. Prespondylosis and some pain syndromes following denervation supersensitivity. *Spine* 1980;5:185-91.
58. Chang MH, Chiang HT, Lee SSJ, Ger LP, Lo YK. Oral drug choice in carpal tunnel syndrome. *Neurol* 1998;51:153-6.
59. Katzung B. Basic and clinical pharmacology. 7th Ed. Connecticut: Appleton; 1998.
60. Seaman DR. Clinical nutrition for pain, inflammation and tissue healing. Hendersonville, NC: NutrAnalysis; 1998.
61. Werbach MR. Healing through nutrition. New York, NY: Harper-Collins; 1993.
62. Werbach MR. Nutritional influence on illness: A sourcebook of clinical research. 2nd Ed. Tarzana, CA: Third Line Press; 1987.
63. Ray O. Drugs, society and human behavior. St. Louis, MO: Mosby Company; 1983.