Chapter 4 - Treatment Protocols of a Whiplash-Associated Disease

…victims of WAD reveal so few, if any, objective findings via the history and physical and because there is little verification by radiological studies.

Numerous injuries result from vehicular accidents even when the impacts are not very big and there is minimal damage to one or both vehicles.

With so little information available, it is understandable that there is so much rejection by insurance carriers.

Failure to decrease symptoms, reduce impairment, or to restore normal function from the available treatment modalities adds to the dilemma facing the WAD patient.

The efficacy of various protocols advocated in treating WAD is decided by the practitioner and is dependent upon careful review of all medical peer-reviewed journal articles.

Of all the symptoms ascribed to the whiplash syndrome, only pain seems to have been evaluated, while other symptoms such as impaired function, dizziness, cognitive impairment, and visual disturbances are equally debilitating yet receive scant review.

CURRENT TREATMENT PROTOCOLS

Current treatment protocols that are advocated and have been reviewed in the medical literature include enforced rest, education, collars, posture education, physical modalities, cervical traction, manipulation, massage, analgesics, tricyclic medications, psychological interventions, trigger point injections, disectomy, cervical fusions, epidural steroid, myofacial release, craniosacral manipulations, soft tissue techniques, muscle energy techniques, and numerous herbal medications.

With such an array of treatments that largely have proved to be essentially ineffectual, as revealed from personal professional utilization reviews and from careful review of the literature, it is no wonder that the whiplashed patient is a "pain in the neck" to all involved. These failed treatment procedures and protocols have exorbitant costs to the medical delivery system, insurance companies, health maintenance organizations, and the incurred legal interventions, invoking a review of their efficacy.

Any treatment modality must depend upon a stated biophysiological abnormality caused by the acceleration-deceleration injury to the soft tissues of the head and neck that must be altered, modified, and restored.

Therapy should address the specific phase of the event presented to the physician.

There are two phases of the event—acute and chronic—and they are discussed in the following sections.

ACUTE PHASE

In the acute phase, there is evidence of inflammation in one or more tissue sites causing "the" pain. This demands early intervention.
Inflammation indicates an accumulation of nociceptive chemicals at the site of sensory nerve endings (Figure 4.1). The quality and quantity of these nociceptor agents determine, in part, the severity of the pain experienced by the patient. The quantity and quality of these agents also sensitize the neurons on the dorsal root ganglion and dorsal horn cell neurons, which make them primary initiators of nociception.

If not abated by prognosis and understood by the patient, the patient's fear and anxiety will enhance the degree of pain. Fear and anxiety along with possible memory of a previous episode or the story of another person having suffered a whiplash may aggravate the pain behavior (Figure 4.2).

**Melzack and Wall's Gate Theory**

In 1965, psychologist Ronald Melzack and anatomist Patrick Wall proposed the Gate Theory in which a spinal gating mechanism in the dorsal horn was thought to modulate the transmission in the dorsal horn from afferent fibers to the spinal cord T cells. Activity in larger fibers tended to inhibit transmission by "closing the gate." Small fibers opened the gate. A "central control trigger" activated and influenced the descending fibers within the cord. These fibers influenced the gate mechanism.

Treatments routinely used in the acute phase that have been found to be ineffectual are rest, analgesic medications, transcutaneous electrical stimulation (TENS), ultrasound and pulsed electromagnetic therapy, and the use of soft collars.

Traction (static, intermittent, or manual) has been found in the literature to be no more effective than moist heat, education, and exercises.

Active intervention is considered resumption of all activities and active exercises with education.

In the author's opinion, with years of clinical experience substantiated by careful review of the literature, there is a role for some of the refuted modalities mentioned but only when used in conjunction with appropriate exercise. The exercise intervention advocated specifically invokes the short neck flexors and long neck extensor muscles with isometric eccentric contractions.

This isometric muscular contraction, in which no motion occurs, contracts the short deep muscles, which are known to stabilize the spine. These repeated isometric muscular contractions, even in the acute phase, diminish the protective "spasm," remove the factors of ischemia from these contracted muscles, and place some tension on the ligaments and tendons. With this approach, which gets the patient involved early in the care of the WAD, pain is reduced.

Contracting the cervical muscles in all planes of rotation increases the range of motion of all segments of the cervical spine.

The muscles that may have been in deconditioned status before the accident begin gaining strength and endurance, which stabilizes the spine to prevent continuation of symptoms leading to chronicity.
The deep cervical muscles are emphasized because they are the muscles that afford cervical spine stability. The other neck muscles that essentially "move" the spine are ultimately involved, but their contraction during the acute phase may initiate pain with resultant fear and self-immobilization, resulting in loss of range of motion.

At first these eccentric contractions of specific deep muscles of the neck are best administered with a therapist so that the "feel" of the contraction and the specificity of proper muscle(s) contracting is verified. Ultimately, a home exercise program evolves as these exercises must be done several times daily and probably for several months. Instructions and supervision of these active exercises is the goal of the therapist.

The deep cervical flexor muscle exercises are performed as follows:
1. With the patient supine on a firm surface the head remains on the ground.
2. The patient then brings the neck to the table, decreasing the lordosis by contracting the short muscles of the neck (see Figures 4.3, 4.4, 4.5).
3. While in the "flat neck" position, rotate the head to both sides to increase range of motion and free the restricted facet capsular limitation.
4. When the desired position is reached, hold the head and neck in that position for a few moments, then relax, and repeat the exercise.

These exercises are done repeatedly during the day to give added strength and endurance to the short neck flexor muscles, improve the posture, and decrease pain and guarding in the patient.

These exercises also diminish the duration of the symptoms by preventing chronic pain as well as protecting against another whiplash incident.

The trapezius muscles, which attach the shoulder to the neck and head, must gradually be addressed in an exercise program.

Circumduction of the shoulders with and without weights in the hands increases range of motion and strengthens the shoulder girdle muscles.

Economically, proper posture in everyday activities is also instrumental in eliminating the symptoms of WAD and should be addressed early in treatment.

"Other" modalities can be added including ice packs during the acute painful phase, heat packs prior to exercise during the reparative phase, and cervical traction, which affords relief and may be instituted as a home program in conjunction with exercise (see Figures 4.6, 4.7).

Medications are also valuable ancillary modalities because they allow the patient to participate in active exercises.

**CHRONIC PHASE**
For the 25% of patients who suffer a whiplash injury that persists for several months, their injury can be considered chronic.

For this group, the therapeutic approach is more complicated and the prognosis more guarded, which is why adequate care of the early acute phase is so important.
In its review of the literature, the Quebec Task Force found no evidence that any conservative therapy, including manipulation and electric nerve stimulation, had any benefit in treating the chronic pain experienced after whiplash.

The task force concluded that 12 weeks after whiplash injury, a multidisciplinary team, which includes neurosurgeons, orthopedists, physical therapists, and clinical psychologists, needs to be invoked.

The Quebec Task Force advocated no specific physical therapy modalities but stated that the use of a water pillow for sleep could reduce early morning pain. Pain could be carefully modified by nonsteroidal anti-inflammatory drugs (NSAIDs) and antidepressants. Patients considered having pain from injured facet joints, which is frequent in WAD translation injuries, can often benefit from intra-articular injections. This procedure is acceptable if benefit results after the first injection, but repeated injections should not be considered if the first injection did not give significant benefit with a lasting effect.

The Quebec Task Force also found no literature on the benefits of surgical intervention for WAD and stated that surgery should not be instituted merely because of "failed physiotherapy."

Posttraumatic progressive cervical ligamentous instability may result following a whiplash injury in spite of an initial "normal" X-ray and may be a cause of prolonged chronic pain and impairment.

It is well known that ligamentous damage can occur in the absence of bony damage. Generally, this is a self-limited problem, but in some cases may result in progressive instability.

Ligamentous stability is hard to document clinically but if unrecognized may lead to a progressive unstable spine with a potential for spinal cord injury.

Strain characteristics of ligaments that result in tensile failures from a superimposed stress have been documented.

Normal loading of isolated collagen fibers that form ligaments can return to normal from 2% to 5% strain.

Failure occurs at 7% to 8% strain in which the ligament undergoes plastic deformity.

A very high-velocity strain, as occurs in whiplash, may result in irreversible plastic change or even disruption.

Under normal conditions, the ligaments allow very little motion between the cervical vertebrae.

In cadaver studies with all ligaments intact, horizontal movement of adjacent vertebra does not exceed 35 mm and displacement of 11 degrees or less.

Clinically, this instability can be verified by flexion-extension radiographs.

Healing occurs in three phases. Phase 1 lasts approximately 72 hours and is a humoral response as in any acute soft tissue injury.
Phase 2 lasts 48 hours to 6 weeks in which there is deposition and synthesis of collagen, which contracts between 3 and 14 weeks and may extend 6 months.

Phase 3 is remodeling, which lasts from 3 weeks to 1 year before the collagen regains its functional capacity.

Remodeling is dependent upon the stresses placed upon it during this phase.

Healing of damaged ligaments occurs by fibrous repair rather than regeneration of the tissue.

Ligamentous injury to the cervical spine after a whiplash injury makes the spine more susceptible to further injury, albeit minor. Other tissues, specifically muscles, must be used to stabilize the spine while ligaments heal. Muscle con- traction affords the moderated forces to the healing ligament that ensure proper remodeling.

A recent study of the genetic factors identified in lumbar disk disease found that patients who sustained a discogenic injury with sequela had a mutation of collagen IX genes, which showed an Arg 103-Trp (arginine- tryptophane) substitution in the alpha 3 chain (Trp3 allele), whereas patients without this mutant did not suffer as severe an injury.

This implies that persons with this mutant genetic factor in their collagen are more prone to sustain a severe injury from a minor stress because of their "weakened" collagen, whereas those without the mutant factor are less susceptible.

The headache experienced by the WAD patient also requires attention.

Greater superior occipital neuralgia is discovered by eliciting tenderness at the base of the skull; this pressure reproduces the headache and confirms the diagnosis, which must be explained to the patient. This diagnosis can be verified and the patient can benefit by injecting an algesic solution with steroid into the nerve at that point.

Cervicogenic headache often responds to treatment of the cervical spine, eg, traction, local ice, and appropriate exercise. Improvement of a post-whiplash headache also reassures the patient that the headache is directly related to abnormalities of the cervical soft tissue.

The best and most effective treatment of WAD is to inform the patient, in understandable terms, of the significance of symptoms, the benefit of resuming all daily activities, and the value of proper exercise to increase the stability of the cervical spine and the healing of the ligamentous structures.

The patient must understand the uselessness of most modalities when used alone and that these modalities are time-consuming, expensive, and ineffectual.

**PROGNOSIS**

Neck disorders are prevalent in the working population, with 43% in a semi-rural community reporting neck symptoms in the previous 6 months and 18% in the previous year. questionnaires and include factors of gender, age, occupation, psychological distress, and psychosocial factors.

These are neck pain on palpation, muscle pain, headache, and pain and/or numbness radiating to the arms, hands, or shoulder area.
DEPRESSION
Patients with WAD who develop chronic pain often manifest depression.

Depression may merely be a temporary "bad mood" associated with frustration of physical signs and symptoms not abating with therapy and time.

This is usually considered to be a loss of interest and pleasure in everyday life for a period exceeding 2 weeks.

More severe depression is known as "dysthymia," which includes a change in weight, sleep disturbance, fatigue, unrewarding sleep, guilt ruminations, psychomotor agitation or retardation, difficulty in thinking and concentration, and even thoughts of suicide.

The question is, which came first, pain or depression? Fishbain et al found depression followed the onset of pain and termed it "consequence hypothesis." incidence of depression than did controls. They concluded that depression may precede pain and that persistent pain exacerbates depression.

Treating depression in WAD patients with chronic pain improves the prognosis of both.

It doesn't matter whether the chronic pain of WAD and depression are causal, coincidental, or mutually exacerbating; all patients with chronic pain are depressed, making it necessary to address the depression.

Adding the diagnosis of "depression" to the diagnosis of WAD suggests that this "condition" preexisted the diagnosis of WAD and is more important that the factors elicited in the diagnosis of WAD.

Unfortunately, this label presents a dilemma for insurance carriers and may imply to the treating physician that depression was preexisting and that the patient may be seeking "secondary gains" from his "accident"

MEDICAL-LEGAL ASPECTS OF WHIPLASH-ASSOCIATED DISEASE
The patient suffering from WAD may have had vague symptoms and no "objective" findings when the vehicle in the accident was only slightly damaged.

The patient may have been inappropriately evaluated and inadequately treated.

These factors understandably cause insurance carriers to be suspicious in their acceptance of financial responsibility.

*Claims have been implied that the introduction of a lawyer in a whiplash claim increased the extent of alleged symptoms and the duration of the condition.*

The other finding was that "people who initially consulted only a physician closed their claims more rapidly than subjects who consulted a chiropractor or physical therapist. Those that did not consult a health care provider got better quicker."
CHRONIC PAIN
One challenging factor of the post-whiplash syndrome is the presence of chronic pain that persists even after any other manifestations have gone and all treatment modalities exhausted.

A research article published in Pain revealed that chemical abnormalities, which result in cortical synaptic reorganization, were found in the brains of patients with chronic pain.

Persistent focal musculoskeletal pain creates chemical and neural synaptic changes in the brain, thus modification of local pain and alteration of cognitive changes to that pain are of value in changing the neural patterns.