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Cervical Whiplash: Considerations in the Rehabilitation of Cervical Myofascial Injury

SUMMARY

Cervical whiplash, usually the result of impact injuries at high speed, is a typically 20th-century problem. This article describes the biomechanical stresses that result in whiplash and outlines the treatment programs that can be expected to help the patient through the four stages of recovery to achieve functional rehabilitation. (*Can Fam Physician* 1986; 32:1871–1876.)

SOMMAIRE

Le coup de fouet cervical antéro-postérieur, habituellement le résultat de blessures d'impact à haute vitesse, est un problème typique de XX^e siècle. Cet article décrit les tensions biomécaniques que provoque ce traumatisme crânio-cervical ainsi que les programmes thérapeutiques susceptibles d'aider le patient à franchir les quatre phases de rétablissement dont l'objectif est la réadaptation fonctionnelle.

Key words: cervical whiplash, biomechanics, rehabilitation

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WHIPLASH INJURY to the neck is a significant 20th-century problem without a natural parallel. Before the present century it was probably rare. Historically, perhaps, a parallel can be cited in the ramming of triremes or the collision of chariots, which must certainly have taken a toll on the unwitting crews. Sadly, the victims have not passed on to us their experiences, possibly because the circumstances were so often terminal.

Whiplash injury occurs when a pulse of impact energy is generated by vehicular impact at considerable speed; the consequent body-reaction movements exceed the normal voluntary motor-loop speed. By the time a passenger makes a response, the body part is at an angle and a displacement different from those to which the muscles are responding. It may be the less surprising, therefore, that the protective and adaptive biologic mechanisms against movement injury seem often to fail in these circumstances. It would seem that this type of event poses a biologic challenge as yet unmet by evolution.

The physician confronting the problems of diagnosis, management and report of motor-vehicle injury is often reminded of how little is known of the nature of such injury and its recovery pattern, how limited are the tools of diagnosis and, by contrast, how much is expected of physicians by insurance companies and members of the legal profession. Adding to the difficulty is the problem of the patient who suffers chronic pain apparently refractory to earnestly prescribed treatment.

The author of this article offers some explanations and suggestions, gleaned from the literature, useful to the management of this vexing problem.

Biomechanics

For the person involved in an auto collision, acceleration of head, neck, torso, limbs and spine occurs at different times following impact; the precise times are determined by the proximity of the body part to the car itself. The thighs provide a friction base that keeps the legs and pelvis almost immobile relative to the auto, and this immobility is further facilitated by the lap belt. If the forces of decelleration are considerable, as they are in a head-on collision, there may be lap bruising and knee-cap injury from dash impact.

The low back or the thoraco-lumbar junction may be hurt by being suddenly arched or flexed as the torso moves in an arc while the pelvis is fixed from below. A soft seat back and poor or absent chest belting will facilitate this occurrence.

The head's contact with the car is only through the neck. The head is, in effect, at the end of a long lever which begins at the base of the spine; if the low back and torso move even a few degrees, the movement will be magnified in overall angular displacement by the time the forces reach the head.

Initially, in a rear collision, the rider's body, through inertia, seems to go backwards relative to the accelerating car. Potential energy is developed and stored in the cushion, springs and metal frame of the seat back. After initial impact the car stops accelerating and begins to slow. As acceleration

lessens, the potential energy is converted into kinetic energy, and the seat back thrusts the rider's torso forward. The head, still arcing backwards from the car acceleration, is now dragged forward. The neck is rapidly deformed as the extension arch is converted into a flexion arch. Segment by segment the neck must switch conformations from extension to flexion. The ultimate acceleration of the head is greater than the peak acceleration of the car, since the time interval for it to catch up to the body is shorter. Severy et al.,[1] disregarding the damping effects of muscle, have found experimental forces of 11 G in dummies.

There are vectors of upward thrust and horizontal forces. These cause the neck to undergo stretch (traction) and translation (forward-and-back horizontal sliding of elements). If the head hits the wheel, dash or windshield, a compressive injury will occur.

Protective Mechanisms and Vulnerability

The primary defense is the elasticity of myofascial tissues, ligaments and joint capsules. The chest and shoulders will stop the head and neck in front and side motion; extension is opposed only by the back. Angulation of 122 degrees is achieved, while the physiologic maximum is 75 degrees; this differential explains, in part, the relatively greater severity of extension injury. Most of the initial movement is completed before the nervous system can react purposefuly. Reflex loops require 30–40 msec, but conscious reaction loops require more than 100 msec. Most of the initial movement is over by then. Sensory input generating a brief conscious decision leads too slowly to motor responses. As Figure 1 shows, with this latency the motor response might well be completely opposite to the needs of the situation, now dramatically altered.

Head rests are reported to reduce the incidence of neck-extension injury by 24%. The shoulder belt works most effectively in collisions from front or back, preventing the rider from windshield exit. If the impact forces are high enough and protective clothing skimpy, the shoulder or chest will be bruised. The shoulder belt does not always prevent facial or dental injury from impact with dash, steering wheel or windshield. Belts do not offer much protection in side collision; the rider's head or shoulder may strike the door or an adjacent passenger. In rear collision more neck sprain occurs among those wearing seat belts according to Deans[2] perhaps because the restrained torso cannot damp the rate of head acceleration.

For the elderly, neck injury can be very serious. The degenerative spine—over age 50, 50% of patients will have some radiologic changes, and over age 65, two-thirds will show such changes—is biomechanically "stiffer", behaving more like a single long bone than like a set of articulating structures. Deforming forces are less evenly and effectively dissipated, and more damage is done. Vestibular contusion may cause vestibular dysfunction, symptomatic as dizziness. Proprioceptive signals from the facet joints of the neck cannot compensate if the neck is injured. Poor vision, strong lenses or cataracts may prevent visual compensation. The brain no longer knows the head's location in space, leading to vertigo.

Predisposing factors of other illnesses must be considered. Rheumatoid arthritics may fracture or dislocate the odontoid, since the bursa between the dens and the cruciate ligament is a site of inflammation. The rheumatoid bursitis may cause an osteoporotic change in the dens and predispose to a pathologic fracture, or weaken the ligament and predispose to a pathologic rupture. Systemic Cortico-steroid may induce osteoporosis and traumatic symptom masking. Ankylosing spondylitis, DISH or degenerative disc disease will predispose by stiffening the spine.

Provided that accidents are separated by six to 12 weeks, the tensile tissue strength should preclude excessive effects of a second accident. However, the risk of degenerative change may be increased, since repetitive microtrauma is postulated as a cause of degenerative disease. The victim's anxiety and frustration can be expected to increase. Multiple experiences of trauma can produce a neurotic state of fearfulness.

Injury from Vehicular Collision

Each accident must be analysed in its own right. Auto speed and damage are not reliable parameters, although devastating injury seems to be far less likely if collision speed is below 20 MPH. Age of patient, angle of head at impact, and impact force and direction are relevant.

Common sense must prevail. The driver of a dump truck hit by a VW Beetle is not likely to suffer much

Figure 1
Flexion Whiplash: The First 360 Milliseconds

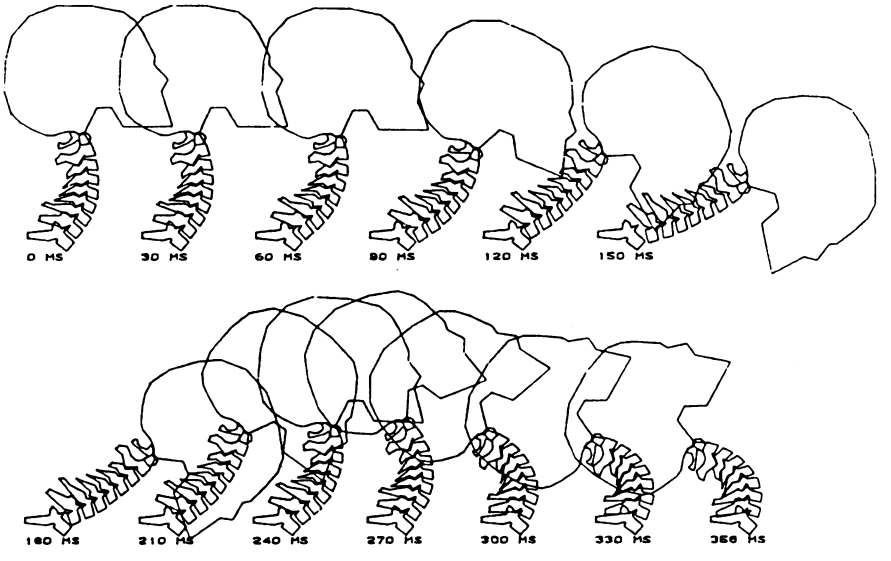

Figure reproduced courtesy of the *Journal of Biomechanics*.

Source: Merrill T, Goldsmith W, Deng YC. Three-dimensional response of a lumped parameter head-neck model due to impact and impulsive loading. *J Biomech* 1984; 17:81–95.

trauma, no matter how vociferous the complaints. Many examples of this type of inappropriateness are seen each day, and perhaps should be called the "*Northumberland* syndrome". (While crossing the Atlantic in the Second World War, the escort cruiser *Northumberland* crossed the path of the more massive *Queen Elizabeth* liner, which split the cruiser in two. The captain of the *Queen Elizabeth* was said to have remarked that he had felt a bump at the time.)

Beware of the late-onset symptom. Most authorities accept a "window" of symptom onset of up to 48 hours, occasionally extended to three days post trauma. Sports-minded readers will recall that stiffness may occur the morning after the season's first game. It is hard to accept a much later onset as relevant. How often does one strike a thumb with a hammer, only to awaken a week later and say "Ouch!"? (Neurologic symptoms such as thoracic outlet syndrome may appear months later, of which more will be said below.)

The severity of injury does seem to correlate with time of onset. Immediate onset pain, with a crack sensation and gradual soreness of the throat or dysphagia, signifies a considerable tissue injury with prevertebral retropharyngeal swelling, the latter sometimes visible on the lateral view of the cervical X-ray. By contrast, back ache occurring for the first time several weeks post trauma probably reflects the effect of spasm and the chronic alteration of posture, anxiety, increased body weight and reduced fitness brought on by the indolence of a prolonged illness.

It is clinically useful to classify soft tissue strain of the neck in categories, such as 'mild', 'moderate' and 'severe'. Mild injuries may develop immediately or more slowly after injury, will heal rapidly, with minimal work-time loss and a symptom-free status about six months post injury. 'Mild' may inadvertently connote the trivial; perhaps one should term this level of injury a 'first-degree spinal myofascial strain, uncomplicated', reserving the use of 'complicated' for deep lacerations, nerve contusions and other injuries which may prolong recovery or adversely affect outcome.

Whiplash victims with moderate injury (second-degree spinal myofascial strain) may develop symptoms over 24 hours, though 75% will appear within 12 hours, as noted by Deans.[2] These persons will experience serious problems with substantial work loss of weeks or months, but will recover a normal lifestyle within six months to two years. The physician must regard with scepticism the patient who remains symptom free for 24–48 hours, and who has neither much spasm nor substantial spinal restriction on initial examination the day after the trauma, but who then progressively deteriorates to a level of prolonged disability and ultimately takes one or more years to recover. Of course, consideration must be given to pre-disposing physical factors or psychogenic overlay. Valid symptoms often include severe headache, parasthesiae and significant early spinal range restriction. Within one year, about 50% of patients in the 'moderate' category will have recovered to the level of 'functional recovery': a full range of activities of daily living will be restored, but often with intermittent symptoms of rheumatism in damp or cold, and intolerance of a prolonged neck position or of extreme turning or extension. After 18–24 months, almost all patients will have reached functional recovery, although some report recovery up to five years later.

Severe injuries (third degree) are disabling in the very long term or even permanently. There may be a fracture or dislocation, or a concussion with more than momentary loss of consciousness and some post-traumatic amnesia. Certainly, symptom onset delayed by two to three days would be incompatible with this category. About 10%–15% of motor vehicular cervical injuries fail to achieve a functional recovery even after the passage of two to three years. This failure may be the result of physical impairment, minimal brain damage from head injury, or chronic pain syndrome.

A functional recovery may be considered achieved when a patient can carry out a full spectrum of normal activities, with a frequency, and to a level of intensity and duration, reasonable for his/her age, skill and needs. A painter who cannot maintain neck extension for 50 minutes in each hour is incapacitated; not necessarily so a lab technician.

Overall, a patient with a neck injury has an 85%–90% chance of achieving a functional recovery. Anywhere from 40%–70% of such patients retain some degree of intermittent, unpleasant, unnatural (but not serious) symptoms in the injured tissues, according to authors such as MacNab[3] and Gotten. The symptoms may be periodically distracting, but create no serious problem in relation to the ability of the injured person to perform daily activities. When seen against the background of normal human spinal complaint incidence and prevalence, it is obvious that most of us, as we get older, will experience some degree of unsustained discomfort in the neck or back after sport over-exertion, gardening or garbage detail, or a poor night's sleep, as well as from rheumatism or arthritis.

The chances of achieving a functional recovery decrease with the duration of symptoms post injury. In my experience, at least 50% of the more seriously injured will have recovered by the end of the first year; a further 25% may recover during the next six months. Only a 15% chance of recovery remains after that point. Although this percentage is small, it still represents a respectable number, and implies that a conclusion that the patient has failed to recover, prior, at least, to the eighteenth month post injury may be premature.

According to Hohl[5] 39% of patients suffering neck injuries may develop premature radiologic changes by the second or third year. Ten per cent will develop clinical symptoms of arthritis.

Complicating Factors

The chronic pain syndrome seems to develop in patients predisposed through immature or maladaptive pain- and stress-coping mechanisms. Pain-avoidance behaviour so dominates the lifestyle of these patients that activity is minimal. The syndrome seems to occur inversely in proportion to the intensity of physical injury.[6] It rarely serves a biologic function or is supported by objective findings. Immature pain-reaction patterns give rise to self-defeating behaviour, then to 'learned inadequacy'. This latter symptom is considered a behavioural disorder, and behaviour-modification techniques may assist recovery, especially if they are instituted early on, before the behavioural patterns become fixed as habits. As described by Addison[7] and Florence,[8] the patient

can be taught to "work to quota" rather than "to tolerance".

Other real, if misdiagnosed, conditions include the post-traumatic anxiety neurosis, which is characterized by symptoms of anxiety such as palpitation, diaphoresis and panic attacks. Post-traumatic stress syndrome involves repetitive reliving of the event, fear of being in cars, and consequent abandonment of even pleasure trips, as well as symptom preoccupation. It is a Pavlovian response and is gradually modified by repetitive positive experiences. This type of problem must be viewed sympathetically as a learned response, an undesirable habit to be overcome. It is closely related to the rape experience and other "assaults" of a deeply personal nature. Its prevalence is variously cited as ranging from 19%–35%.

Role reversal by the family is the most 'deadly' of the possible complications to recovery. When the dominant member is struck down by pain and impaired in daily function, some degree of love and sympathy are necessary to recovery. When family members overwhelm the patient by pampering and protection, the challenges of daily activity are buffered, and recovery slows or even stops. We need some challenge each day if we are to maintain contact with reality and some semblance of normal perspective and objectivity.

Rest is not benign; it may be pathologic. Soft tissues shorten if not regularly stretched. Muscular capillary profusion and energy stores will 'atrophy' in the absence of exercise of at least 60% maximum contraction effort at least once daily. Cardio-respiratory fitness deteriorates 1% per day with immobility. After a few weeks of rest, patients may complain of spinal or peripheral joint tightness, general fatigue, loss of energy and of general strength, and inability to cope with pain, personal problems or the challenge of work. For the elderly patient, the problems may also include orthostatic hypotension and sensory deprivation symptoms.

Family role reversal may occur out of genuine concern for the patient, or manipulation by the patient, or because the new dynamics of the family relationship develop maturity and independence in its members. Such overcompensation must be stopped, or a pathologic and potentially irreversible state of dependency will be induced in the patient.

Assessment

Useful diagnostic tests for signs of whiplash injury include assessment of active and passive range of motion, identifying locations of tender tissues, and neurologic investigation of sensation, strength and reflex. More provocative tests of compression, traction and neck-muscle strength can help to delineate structural pain (on compression) from myofascial pain (traction and muscle tests); and radicular parasthesiae (with impingement) from brachial plexus irritation (inclined traction).

Every physician should be prepared to make a few tests for non-physiologic responses such as straight-leg-raise,[10] pelvic pseudo-rotation, and vibration propagation across the midline. Florence[8] reported an incidence of 20% malingering in chronic pain-clinic patients, some of whom wanted to end their self-destructive behaviour.

Treatment Programs

Convalescence is a concept which should be replaced by the more active notion of rehabilitation. It is possible to "rest to excess". Passive management of whiplash injury may ultimately prove not to have been benign.

Recognizing that there is a soft tissue strain injury with relatively prolonged, and sometimes substantial, impairment (inducing a potentially significant amount of stress and emotional change) should prompt the physician to initiate development of a rational, progressive, multi-phase program of treatment. One may view this as a program. At each level of this program consideration must be given to:

- range of active motion of the injured spine;
- strength of the related musculature;
- postural control and balance of opposing soft tissues;
- general muscular power;
- general aerobic fitness;
- emotional state;
- level of activities of daily living (ADL).

In the First Stage (acute post trauma) the patient presents with pain, spasm and restriction of movement. The most immediate need is for pain control, sleep preservation and anxiolysis. So much anecdotal information is provided by well-meaning laymen that attitudes and expectations may become fixed in a negative way. It is productive to have a short typed handout for the whiplash patient which stresses the fundamental fact that healing can be expected, and that the symptoms will inevitably ease.

Typically, NSAID and anti-spasmodic medication, as well as strong analgesics are prescribed. The NSAID drugs are less effective for whiplash than for acute arthritis. Perhaps this is because of the shear mass of injured tissue concentrated within the neck area of the whiplash patient, or because suppression of inflammation is more likely to facilitate a block of the inflammatory process in an otherwise progressive synovial tissue reaction where much of the substrate tissue remains essentially unharmed and can revert rapidly to normal. Trauma, by its very nature, establishes almost immediate and more widespread tissue damage before the first pill is swallowed. Much of the NSAID benefit may be the result of its analgesic property. Pharmacokinetically, a nocturnal dose is metabolized more slowly, giving a longer dose half-life but lower peak of effect. It is thus a more rational prescription for sustained analgesia and sleep.

Use of enteric-coated NSAID medication is desirable, since the already distressed patient is never grateful for iatrogenic gastritis (10%–15% incidence).

So-called 'anti-spasmodic medication' may be primarily sedative in action. To obtain true muscle-contraction inhibition, one would require a drug such as dantrolene sodium. The patient would report profound general fatigue from the uncoupling of calcium from muscle membranes. Diazepam works at central levels on anxiolysis, but also seems to have a role in the GABA system of neural transmission at the cord level. This double action makes diazepam a useful drug, provided the physician recognizes its pharmacokinetics. The avid fat-cell absorption makes the blood level difficult to regulate; within a few days the dosage can be reduced with the same net serum level. Since diazepam can be highly sedative and may make the perceived tasks of the day "more

mountain than molehill", the physician must be cognizant that it may cause depression. Short half-life analogs such as oxazepam may be more desirable. The other prevalent antispasmodics are probably primarily anxiolytic and sedative. Grosshandler et al.[11] observed that there is no convincing evidence of true spasm in muscle, as compared to cramping; on EMG, spasm is silent. Travell and Simons[12] emphasized that the palpable band is an area of physiologic contracture within the muscle which may impair metalolism and cause hypersensitivity (trigger points), but may respond to local injection.

Initially, the patient may require a collar for analgesia. Since soft collars do not truly restrict movement, the physician should not consider the use of a collar a therapeutic necessity. After the first few weeks, and certainly by the end of the fourth week, the use of the collar should be discouraged other than when shopping or driving, and neck movement should be encouraged. At this stage, a critical prophylactic achievement is making the patient understand that 'hurt' does not equal 'harm', and that movement should not be avoided lest some dire consequence follow.

Early physiotherapy is generally used to promote analgesia. Deep or superficial heat, ice and massage help to control the patient's overall pain level. Not surprisingly, the relief does not last much beyond two hours after treatment. Nonetheless, physiotherapy restores to the hands of the patient the overall feeling of control of the symptoms. At this stage a limited range of gentle motion is undertaken with the patient's co-operation.

Weinberger[13] has stated the position held by many physiatrists who oppose early traction. Although light traction may ease spasm, more distractive forces do reproduce the traction vector of injury and may increase pain and even damage torn tissues.

Most physicians oppose chiropractic or other paramedical manipulation at this stage. Sudden, distractive or rotational movements are potentially harmful to tissues which have not yet recovered full tensile strength. The effect on crack fractures, subluxable facet joints, end-plate fractures or undisplaced compressive fractures can only be speculated.

Unfortunately, X-ray and bone scan imaging do not provide enough information on early whiplash injury, and there is rarely significant benefit from early cervical X-rays.

Stage Two (post acute) is heralded by the easing of spasm, allowing more movement. This improvement should begin within the first four weeks following injury. The rigid spine, intolerant of any movement two to three months after injury is most likely the result of vigorous overprotective guarding rather than of pathology. There is no reason to assume that the patient with soft tissue injury—likely a more widespread but less intense quantity of injury—will be worse off in the absence of X-ray change than will a fracture or surgery case.

As time passes the rehabilitative program should become progressively more active. Physiotherapy should take the leading role in coaxing the patient along. Physicians can help greatly by dealing with the fears of the patient during office visits and by being directive and specific in allowing resumption of activity. Excessive rest and precaution at this stage is counter-productive and almost inevitably assures a passive attitude to recovery in the patient in the longer term.

Drug use becomes increasingly less efficacious with the passing weeks. Extra-Strength Tylenol supplemented by occasional NSAID or diazepam will likely be sufficient to control pain. Again, in analogy to known quantities of pathology as in cervical fracture or surgery, the need for prolonged high-dose codeine or Percodan, in my view, is doubtful; it is at this stage that overtreatment for symptom control can lead to dependency on diazepam or narcotics. Sleep deprivation has usually ended, although the toss-and-turn complaint of interrupted sleep may remain. Beware of the trap of inducing artificial sleep. Use of tricyclic antidepressants such as amitriptyline 25 mg−75 mg QHS is helpful. This medication affects non-REM sleep, influencing fibrositic type myofascial pain, as well as affecting the central pathways mediating pain.

Trigger-point injection with procaine prn has been advocated, as has the technique of the freezing spray. Even dry needling or saline may be effective. Cortisone is not always necessary for therapeutic effect (see Table 1).

There is little justification for a formal physiotherapy program extending beyond the fourth month following injury. Exceptions exist, in the patient with shoulder pain and restriction or other complicating phenomena of injury. Once spinal range and girdle strength are re-established, attendance is mostly for pain control; the effectiveness of the modalities does not extend more than a few hours post treatment. The risk of overdependency increases markedly. The subjective perception of treatment failure is enhanced. Patients invariably expect treatment to result in pain-free status. Instead, it should be stressed that recovery of *function* is the primary goal. Pain control, particularly headache or limb parasthesiae, will come only with the fullness of time and resumption of normal activity.

Chiropractic management and advice to patients varies with the practitioner's style of practice. Logically, as with any of the medical treatment paradigms, the program should taper in response to the achievement of therapeutic goals. Unfortunately, from a physiatric point of view, those goals often fail to include restoration of specific and general fitness, a failing which may foster passivity and dependency in patients. Fortunately, more progressive chiropracters emphasize recovery of activity, diet and exercise, while tapering the attendance in keeping with the improving clinical picture. There is no acceptable published data that supports manipulative treatment over other forms of musculoskeletal treatment.

There is no basis in medical care paradigms for fixed-time schedules of

Table 1[11]
Common Trigger Points: Areas of Referred Pain

Trigger Point	Pattern of Reference
Trapezius	Shoulder, neck, side of head
Levator Scapulae	Shoulder, neck and arm, medial border of scapula
Scaleni	Chest, upper extremity, medial border of scapula
Infraspinatus	Front of shoulder, deep in joint, medial border of scapula
Rhomboids	Along vertebral border & paraspinal muscles

treatment, or for treatment responses not being apparent until months have passed. There can be no justification for a fixed-duration treatment schedule within the paramedical community.

Stage Three is marked by the patient's recovery of almost full range of motion and strength, paralleled by the resumption of many normal activities, albeit with upper limits to the intensity and duration, as well as constraints on the spectrum of activity. Complaints of headache, rheumatism, and intolerance of prolonged positioning or extreme turning are typical. Vigorous return to sport or work is painful or even impossible. The patient's motivation and pain tolerance are critical to the pace of recovery after this point.

Patients with first- or second-degree spinal myofascial strain should have reached the third level of recovery sometime between the eighth and sixteenth week post trauma. Recovery is rarely later, unless it is complicated because of true radicular damage, reflex sympathetic dystrophy, concussion, or post-traumatic emotional disorder.

The most treatable problems at the third stage are those of tissue tightness, and posture. Postural problems derive from chronic positioning adaptions producing such results as imbalance between pectoralis and the rhomboid muscles' rest length, secondary parasthesiae and deconditioning of muscles and cardium.

From a survivalist perspective tissue tightness is a rational biologic adaptation to injury. By adapting to become less elastic, the myofascia may become more resilient to further injury. The body learns by such experiences when the injured person is a professional racing car driver or a diver at Acapulco. Delaying the adaptation until the second experience has poor strategic benefit. The next time may be fatal. Much of the contracturing, however, is reversible by slow sustained stretch and isometric exercise aided by simultaneous heating (by diathermy, not superficial means) to soften the target tissues.

The most common parasthesia is a pins-and-needles sensation felt diffusely in the arm, usually ipsilateral to the side of the neck with the more severe symptoms and spasm. At origin it is probably the result of irritation of the brachial plexus by the adjacent thickened fibrous bands and spastic muscles or by compression by contractured muscles similar to the thoracic outlet syndrome (TOS). There is usually no vascular compromise, and it is often the upper elements from C5, C6 or C7 rather than those from C8 as in TOS. The objective neurologic tests aided by NCT, F-wave studies and EMG are usually negative. The condition may be termed a cervico-brachial neuralgia (neuritis if there are objective findings).

The altered posture of a painful neck and trapezius may result in the shoulder being held forward of the normal position for comfort, resulting in pectoralis contracturing; the first rib may rise as the scalene muscles shorten. The altered posture puts a chronic pull on the scapular muscles. These changes may produce a pectoralis minor syndrome, with the nerves intermittently pressed in near the origin of the pectoralis minor at the coracoid process or a costo-clavicular syndrome with compresssion between the clavicle and first rib. Treatment of this condition requires an assessment of posture and anatomical relationships such as the distance from the midline to the apex of the humeral head. Stretch of the pectoralis and scalenius and strengthening of the trapezius, levator and rhomboideux muscles is necessary. Trigger-point injections are helpful.

For most patients the goals of reducing spinal tightness and postural imbalances, and of achieving weight control and general muscular and aerobic conditioning can be accomplished with home programs, biking, skating, cross-country skiing, pool and fitness-club attendance. These patients need rely only minimally on physiotherapy, chiropractic or massage.

The last stage (symptom resolution) in the recovery of most patients is also the longest. At this stage, functional recovery is complete. Physical examination fails to elicit any sign of abnormality except for local tenderness or discomfort at extreme positions of range. Patients' complaints include tightness and rheumatism, intermittent but unresolved. Generally, any parasthesiae will have resolved.

This is the stage of awaiting the final healing of injury leading to the final plateau in recovery. There is no longer a need for formal treatment, although pursuit of fitness can be helpful, at least in enhancing pain tolerance and altering pain and complaint threshold. For second-degree spinal myofascial injuries, the plateau will be reached between 12 and 36 months post injury. Only 15% of patients fail to achieve a full functional recovery, and some 40%–70% find some mild symptom persistance.

To date there is no objective evidence for stating that any specific treatment is essential to recovery or capable of influencing final biologic outcome. However, the quality of life during recovery, and perhaps the duration period of restricted range and inhibited strength, as well as the timing of return to work and body condition are, in all probability, positively influenced by therapeutic intervention. Of the possible treatment options the most effective seem to be those that incorporate reassurance without creating dependency, provide perspective and insight, and maintain steady pressure on the patient to maximize the recovery by actively and aggressively responding to and managing the pain, the impairments, and the secondary inactivity-related weight gain and deconditioning. ◉

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