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Potential Contributions of Neck Muscle Dysfunctions to Initiation and Maintenance of Carpal Tunnel Syndrome

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A biomechanical perspective of the carpal tunnel (CT) is reviewed that lends itself to an understanding of carpal tunnel syndrome (CTS) from a broader pathophysiological perspective than focusing narrowly or solely on nerve disturbance in the extremity. A wider integration of physiological systems in the etiology and maintenance of CTS is proposed that links muscular dysfunction in the neck and possibly elsewhere to dysfunction at the CT. A significant subset of individuals who develop CTS have a primary contribution from muscular dysfunctions rather distal to the CT itself. Neurophysiological dysregulation of normal inhibitory feedback at the level of the motoneuron pool specifically involving gamma motoneuron impulses may be a primary contributing mechanism. Empirical demonstration of amelioration of CTS symptoms by means of surface electromyography (sEMG) retraining of dysfunctional neck muscle patterns is reviewed as support for the hypothesized link. The specific retraining techniques are described. Future conceptual and research directions are noted.

KEY WORDS: carpal tunnel syndrome; cumulative trauma disorder; repetitive strain injury; surface electromyography; tonic neck reflex.

INTRODUCTION

Carpal tunnel syndrome (CTS) represents one of the more perplexing conditions in the public health arena, is especially prevalent in occupational health settings, and is challenging for pain management specialists. CTS has been linked in

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particular to occupations that require highly repetitive and/or forceful hand movements (Silverstein, Fine, & Armstrong, 1987). Although conflicting reviews of the epidemiology of CTS have appeared (Hadler, 1997; Mackinnon & Novak, 1997), it has been estimated that up to 15% of workers in the highest risk industries (e.g., aircraft and bearing manufacturing, grocery checking, meat packing, sewing, to name a few) are affected annually (Katz, Larson, Fossel, & Liang, 1991; Masear, Hayes, & Hyde, 1986); this is 100 times more than would be expected in an age- and sex-adjusted population (Stevens, Sun, Beard, O'Fallon, & Kurland, 1988). Costs, including those for surgery, and other related expenses or compensation, may exceed $15,000 per worker (Katz et al., 1991; Masear et al., 1986). Yet available treatments, including surgical procedures, are by no means uniformly effective on an enduring basis (Higgs, Edwards, Martin, & Weeks, 1995).

Cardinal features of CTS include symptoms of median nerve involvement (i.e., pain and associated dysfunction in the thumb, index finger, and medial half of the third finger) and occurs when the median nerve is compressed within the carpal tunnel (CT). Anatomically, the syndrome has been divided into three categories: (a) increase in the volume of the contents of the CT, (b) enlargement of the median nerve, and (c) decrease in the cross-sectional area of the CT (Blecker, 1987). However, the vast majority of CTS (and perhaps all occupationally linked CTS) appears to be the result of increased contents of the CT leading to tendonitis and/or tenosynovitis. Indeed, enlargement of the median nerve appears to be rare and a decrease in the cross-sectional area of the CT in patients without rheumatic disease or wrist fractures is unusual (Scelsi, Zandungo, & Tenti, 1989). A number of investigators have observed thickened and edematous synovial sheaths accompanied by histological inflammatory features, particularly in patients with recent onset of symptoms who present for surgery (Castelli, Evans, & Diaz-Perez, 1980; Neary, Ochoa, & Gilliatt, 1975; Phalen, 1966, 1972; Scelsi et al., 1989). On the other hand, based on their study of histological changes in the CT in cadaver patients, Armstrong et al. (1984) have suggested that minor tenosynovitis in the CT is likely to be seen in almost everyone, but it is exacerbated in patients with CTS. Nerve conduction abnormalities in the expected (decreased) direction are the gold standard for the definition of a case (Dorwart, 1984).

Given these findings, explanations for CTS have often focused narrowly on the pathophysiology as it pertains to the nerve disturbance in the extremity without necessarily viewing a wider integration of physiological systems in the etiology and maintenance of CTS. This article attempts to provide a broader perspective on the role of multiple etiologies in the development of CTS. In particular, it is proposed that a significant subset of individuals who develop CTS have a primary contribution from muscular dysfunctions rather distal to the CT itself. Further, neurophysiological dysregulation of normal inhibitory feedback at the level of the motoneuron pool specifically involving gamma motoneuron impulses may be a primary mechanism in this. It has been discovered that this muscular dysfunction can be remediated with surface electromyography (sEMG) techniques.

BIOMECHANICAL PERSPECTIVE

The tendons of the flexor digitorum profundus and superficialis and of the flexor pollicis longus (i.e., the major force producing muscles involved in hand exertions) pass through the CT (see Figure 1). Wrist flexion and extension cause these tendons to be displaced against or past the walls of the CT, including the transverse carpal ligament below and the carpal bones above. Magnetic resonance imaging studies in normal (asymptomatic) persons have demonstrated CT changes (e.g., size, shape, alignment of contents) and median nerve compression (or migration) during wrist flexion and extension (Skie, Zeiss, Ebraheim, & Jackson, 1990; Zeiss, Skie, Ebraheim, & Jackson, 1989).

A tendon sliding over a curved surface, such as in this case, may be analogous to a belt wrapped around a pulley, as suggested by Armstrong and Chaffin (1979; see Figure 2). The biophysical relations, as explained by them, can be described in terms of the force exerted on the pulley as a function of the belt tension, the radius of the pulley curvature, the coefficient of friction between the pulley and the belt, and the angle of pulley-belt contact. The coefficient of friction is normally minimal and relatively inconsequential for estimating force effects. However, when tenosynovitis occurs, both edema and inflammation occur and result in significant exponential increases in the coefficient of friction, thereby increasing the tendon load per unit length.

The relations are also such that the tendon load per unit length, the most critical element in this analogy, is dependent upon the degree of tendon curvature and load. Further, there are direct relations of the contact force between the tendons and adjacent wrist structures with the tendon tension and (inversely) with the radius of tendon curvature. The radius of curvature can be estimated for different wrist thicknesses and the tendon tension can be estimated for given positions of different hand sizes. The total force transmitted from a belt to a pulley depends on how far the belt is wrapped around the pulley, and in this instance would be largely a function of the angle of wrist deviation and of tendon load. Hence, exertions of the hand with the wrist greatly deviated would result in greater total force on the tendons and adjacent wrist structures than with a nearly straight wrist. Tendon force is, then, understood as a function of hand size, position, and load.

Additionally, one mechanism that may contribute to increased load per unit pressure of the tendons is by means of the power grip, one of the most common human hand postures that involves simultaneous wrist extension and active finger flexor contraction. In the presence of active tenosynovitis, there may be a further exponential increase, resulting in more inflammation and/or edema, thereby causing an increase in the contents of the CT and median nerve entrapment (Skubick, Clasby, Donaldson, & Marshall, 1993).

A series of studies provide clinical support for this biomechanical perspective (Gelberman, Hergenroeder, Hurgens, Lundborg, & Allison, 1981; Okutsu, Ninomiya, Hamanaka, Kuroshima, & Inanami, 1989; Rojviroj et al., 1990; Szabo & Chiedgey, 1989), which may be summarized as follows. When patients with symptomatic CTS were compared with normal subjects under conditions that involved changes in wrist position, pressures within the CT were lowest for both groups in

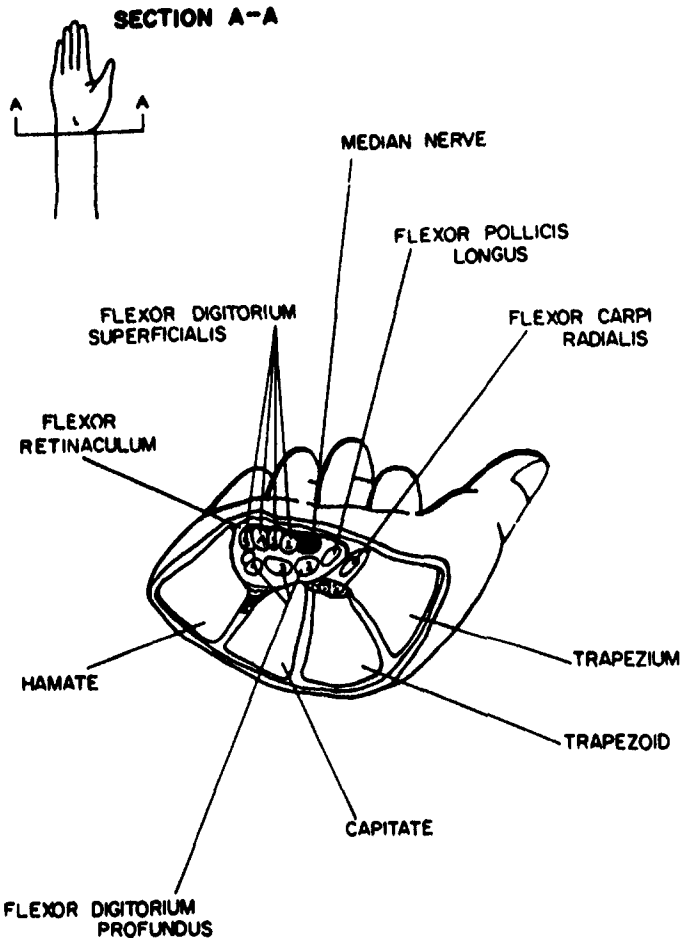

Fig. 1. A cross-sectional view of the carpal tunnel. From "Some Biomechanical Aspects of the Carpal Tunnel," by T. J. Armstrong and D. B. Chaffin, 1979, *Journal of Biomechanics, 12,* p. 567. Copyright 1979 by Elsevier Science. Reprinted with permission from Elsevier Science.

the neutral position. With flexion pressure increased significantly, and even more so with extension. Symptomatic patients exhibited significantly increased pressures for all three positions, compared to normal subjects; this is concordant with the known pathology of tenosynovitis in CTS. Moreover, at least in cases of early and intermediate symptomatology, there was prolonged recovery of pressures to baseline. Apparently, the inflamed synovium increased the volume of the contents of the CT. Given that there is an elevation of pressure in normal subjects when the wrist is deviated implies that these movements (with extension increasing pressure more than flexion) further decrease the volume of the CT. Therefore, it would follow that the effect of the smaller CT volume seen with flexion or extension is superimposed upon the effect of the increasing tenosynovitis, thereby leading to a compromising of the median nerve.

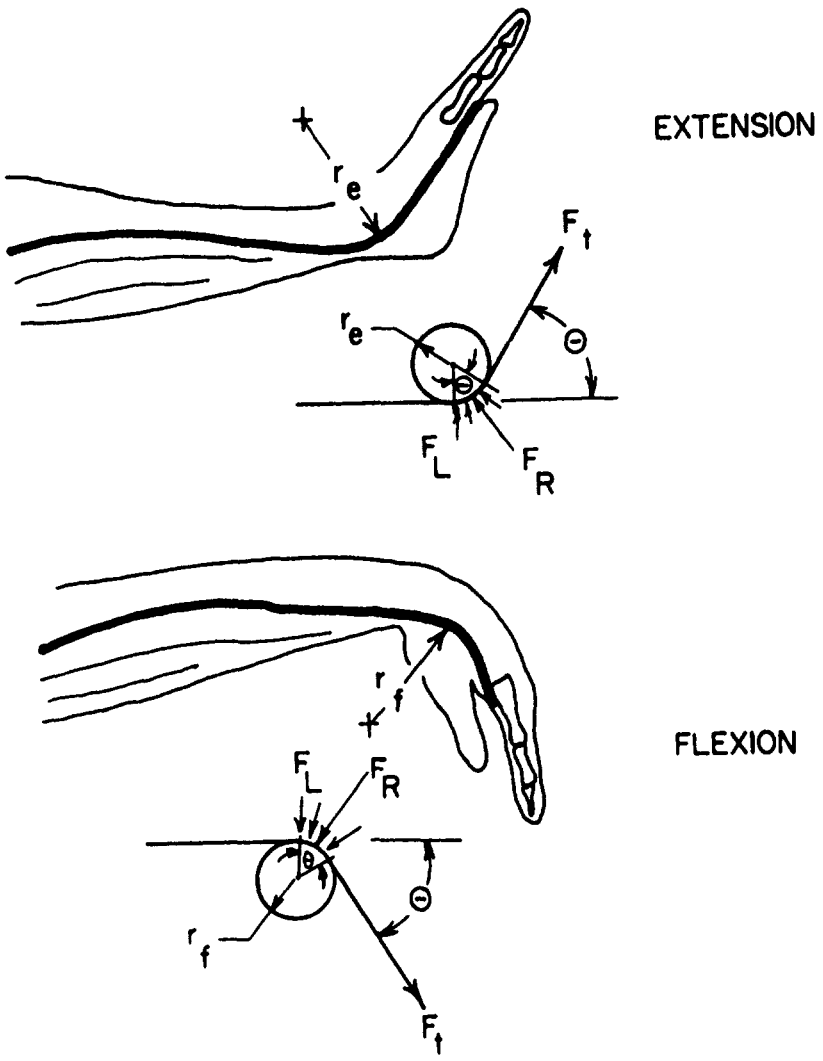

Fig. 2. Illustrations of the belt-and-pulley analogy as applied to wrist extension and flexion, with the flexor tendons being supported by anatomical pulleys with radii r_e and r_f, respectively. From "Some Biomechanical Aspects of the Carpal Tunnel," by T. J. Armstrong and D. B. Chaffin, 1979, *Journal of Biomechanics, 12*, p. 568. Copyright 1979 by Elsevier Science. Reprinted with permission from Elsevier Science. The intrawrist forces, F_L and F_R, and related variables are described in equations and further explicated in Armstrong & Chaffin.

Taken together, these studies support the concept that tenosynovitis in symptomatic patients leads to an increase in the volume of the contents of the CT and this leads to increased pressure on the median nerve. Failure to return to baseline

as described above would be compatible with the notion that repetitive passive flexion and extension lead to an increased tendon load per unit. In line with the belt and pulley biomechanical analogy (Armstrong & Chaffin, 1979), the increased load per unit in an already inflamed synovium would lead to further exponential increases as the coefficient of friction increased. The result would be further increases in the tenosynovitis. As the tenosynovitis increases, the volume of the contents of the CT increases and would lead to progressive median nerve entrapment (Skubick et al., 1993).

THE LINK TO MUSCULAR DYSFUNCTION

Tonic Neck Reflexes

A series of papers in various English and non-English language sources on tonic neck reflexes was summarized by Hellebrandt and colleagues (Hellebrandt, Houtz, Partridge, & Waltos, 1956). They cited the work of Magnus and de Kleijn dating to 1912 which demonstrated that the position of the head modifies the tone of the limb musculature, although this was initially apparent in pathological states and was only eventually observed to be the case in normal subjects. Tonic neck reflexes were subsequently recognized to be a normal feature of human infants. One of the tonic neck reflexes of particular importance to the present discussion is illustrated in Figure 3. In the instance of head rotation to the left, this reflex involves a corresponding extension and abduction of left upper and lower extremities, while the right upper and lower extremities manifest adduction and flexion (i.e., the so-called "fencing" position; Brett & Kaiser, 1997). When the head is rotated to the right, the reverse pattern is seen.

Ultimately all of the tonic neck and labyrinthine reflexes were demonstrated in normal adults. Their universal presence in adult men was confirmed and related to various patterns of association with other factors, such as age (decreased), oxygen deficiency (increased), amount of sleep (increased), and repetitive thumb movements. Corresponding changes in muscle tone were also measured in terms of needle electromyography, subsequently sEMG, and by other objective means.

Hellebrandt et al. (1956) studied the correspondence of postural patterns with electromyography and other technical measurements under conditions of stressful purposeful movements and demonstrated that repetitive fatiguing limb movement exerts reciprocal reflex activity upon the neck muscles. Hence, changing head position affects limb-muscle activity and the converse is likewise true. What results is a positive feedback loop in which head position enhances limb tone, which in turn affects head position. They further demonstrated that muscle fatigue in the neck has a significant effect of increasing limb activity in a predictable manner. The interaction of the wrist extensors and flexors, as well as the sternocleidomastoids (SCMs) and others were among those measured. In experiments involving, for example, various patterns of wrist extension and flexion and head rotation, with stressful unilateral movements, reflex activity was first picked up in the ipsilateral agonist, then the contralateral antagonist, and ultimately the prime mover on the contralat-

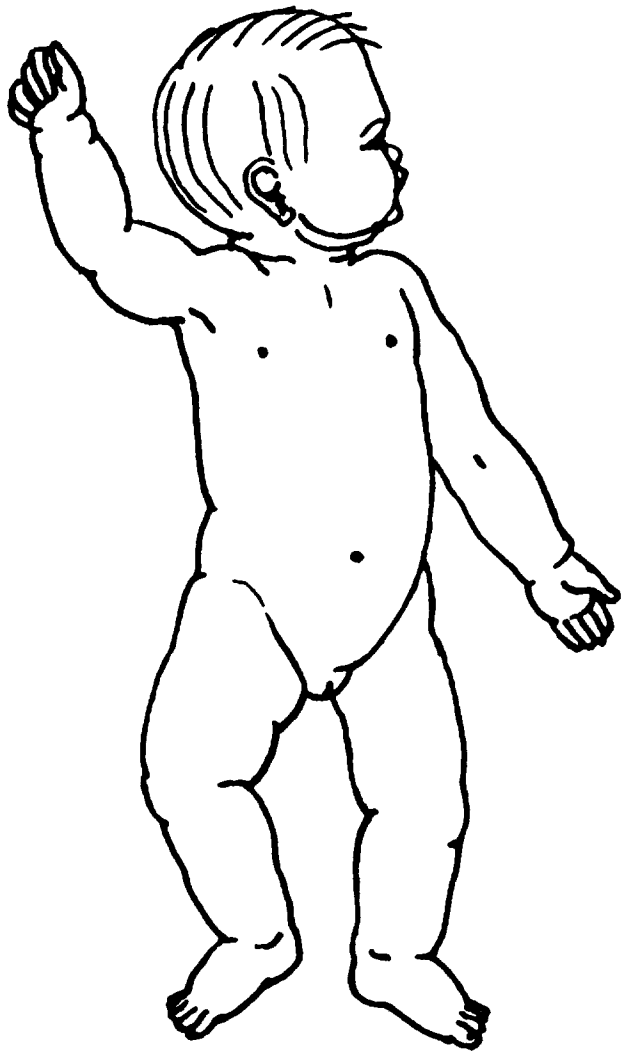

Fig. 3. Illustration of the infant in a tonic neck reflex pose, indicating the relative position of limbs that resembles a fencer (i.e., extension and abduction of upper and lower extremities in the direction of the rotation of the head, with corresponding adduction and flexion of the contralateral upper and lower extremities). From "Neurology of the Newborn" (p. 7), by E. M. Brett and A. M. Kaiser, in E. M. Brett (Ed.), *Paediatric Neurology* (3rd ed.), 1997, New York: Churchill Livingstone. Copyright 1997 by Churchill Livingstone. Reprinted with permission from W. B. Saunders Co.

eral side. More recently, Aielle et al. (1988) found that the degree of head rotation and amount of activity in the flexor carpi radialis were linearly related, with the forearm activity increasing with ipsilateral head rotation.

Another related literature, summarized in Basmajian and DeLuca's (1985) classic work on muscles, demonstrates that the acquisition of much of motor control is achieved through the increasing inhibition of antagonist activity relative to that of the primary movers; and that inappropriate motor activity (common in infants and children), including rudimentary reflexes, may be more likely to reappear under conditions of aging and stress. Taken together, these literatures indicate that the tonic neck reflex as described above is present in normal adults and has an impact upon the muscle activity of the forearm flexors and extensors, and that certain conditions may be more likely to predispose to the reemergence of these more basic reflexes and/or to interaction effects as a function of stress, work load, etc.

Disinhibition Theory of Gamma Motoneuron Dysregulation

Building on Donaldson's (1989; see also Donaldson & Donaldson, 1990; Donaldson, Romney, Donaldson, & Skubick, 1994) pioneering work in the development of certain sEMG applications to the treatment of chronic myofascial pain syndromes, Donaldson and colleagues (see companion article to this paper [Donaldson, Nelson, & Schulz, 1998] for more complete neurophysiological discussion, including diagramming of some of the proposed neurological substrate) have recently explicated a theory that specifies certain conditions under which trigger point activity, related myofascial pain, and/or motor control dysfunctions may be manifested. Essentially, the theory proposes that dysregulation at the level of the gamma motoneuron circuitry occurs under certain circumstances due to changes in afferent generated by muscle stretched upon its length while contracting. The change in the afferent leads to a functionally significant imbalance in excitatory/inhibitory summation effects at the motoneuron pool, thereby producing changes in the interactions of the muscles and leading to disinhibition from appropriate balance of the electrical activity between the two sides of the body. It is proposed that the contralateral muscle develops a trigger point because it is no longer being inhibited correctly. Actually, however, dysregulation of the normal inhibitory feedback mechanism may lead to any one or all three of these phenomena: (a) hyperactivity of the muscle after contraction, (b) excessive electrical activity during movement, and/or (c) inappropriate coactivation with other muscles during movement. Within a broader perspective of motor control, this gamma motoneuron inhibitory feedback system is embedded within a whole interacting hierarchy of multiple levels of motor control (cortical, subcortical, spinal, etc.) that involves recurrent, lateral, and/or reciprocal inhibitory feedback (Windhorst, 1996).

With regard to CTS, while not all of the mechanisms are fully identified, it is believed that excessive electrical activity during movement and inappropriate coactivation with other muscles during movement are the chief correlates of disinhibition that are linked to the reemergence of primitive reflex activity; over time this is hypothesized to be associated with the pathological changes at the CT and development of CTS symptoms. Further, though, from a theoretical standpoint, it is presumed that the dysregulation is correctable by learning and related neuromuscular retraining procedures.

Supportive Findings Pertaining to CTS

Given the above biomechanical conceptualization of the CT, the observed patterns of primitive (tonic neck) reflexes operative under conditions of aging, physical stress, and/or other factors, as well as the potential application of gamma motoneuron disinhibition theory as an explanatory mechanism linking neurophysiological disinhibition to dysregulation of motor activity in the manner suggested by Hellebrandt et al. (1956), Skubick et al. (1993) proposed that CTS may be a consequence of increased forearm flexor and/or extensor activity (i.e., due to increased load per unit) associated with muscular dysfunction. Briefly stated, the argument is: CTS can be viewed biomechanically within the belt and pulley analogy previously described. Increased contents of the CT secondary to tenosynovitis result in median nerve entrapment in the CT. Tenosynovitis is a direct consequence of an increasing load per unit of the tendons. The tendon load per unit increases secondarily to increased muscular activity in the forearms, which is a result of dysfunction of the neck muscles (e.g., SCMs and cervical paraspinals [CPS]). If this were so, it should be possible to demonstrate that varying positions (or movements, e.g., rotation) of the head in patients with CTS should be associated with increased forearm activity. Treatment by changing motor activity of the SCMs and CPS should thereby result in a decrease of corresponding forearm activity.

Accordingly, Skubick et al. (1993) treated 18 symptomatic patients with CTS (average duration of symptoms 10 months) who had their CTS validated with initial nerve conduction studies assessing distal median-motor latency, median palmar-sensory latency, and median terminal-latency indices. Post-treatment nerve conduction studies were also conducted. Fast Fourier analysis of the spectra were conducted to obtain the median frequencies. During treatment no changes in work habits or other activities were encouraged, nor were medications, splints, or other factors altered if they were already in place. Surface EMG techniques measured SCM and cervical paraspinal, as well as forearm flexor and extensor muscle activity during head movement. Both sets of neck muscles were found to be asymmetrical when compared side to side for each homologous muscle (i.e., left versus right SCM, left versus right CPS) during head/neck flexion, consistent with Donaldson et al.'s (1998) disinhibition theory. Specific neuromuscular retraining on a once weekly basis (average length of treatment seven weeks) along the lines recommended by Donaldson (1989; Donaldson et al., 1998) targeting the restoration of balance for the SCMs resulted in the following: reduction of SCM asymmetry that was in turn associated with decreased forearm flexor sEMG activity, and a correspondingly significant decrease in all nerve conduction measures for every subject. Treatment was terminated when subjects either reported symptomatic relief or the nerve conduction studies improved. In no case was treatment continued if the nerve conduction normalized.

Asymmetries of the CPS also came back into balance without specific retraining exercises given for them. Over half of the subjects reported an improvement in CTS symptoms with post hoc analysis suggesting this may be related to SCM median frequency. Indeed, although the median nerves of all subjects showed electrophysiologic improvement, those with SCM frequencies <40 Hz improved the best with the retraining procedures, while those with >40 Hz did not improve in terms of

relief of CTS symptoms. Since retraining was discontinued when the nerve conduction findings were normalized in any given case, this may have resulted in a limitation to the extent of improvement that might otherwise have occurred in some subjects if the retraining had been allowed to go on for a longer period of time. Very clear patterns of time-linked muscle activity patterns were demonstrated and illustrated in figures of sEMG tracings obtained from a representative subject (see Skubick et al. [1993] for these figures and actual summary data results on all subjects).

Alternatively, other muscle dysfunctions may have required correction in order to experience additional symptomatic relief. Further, there may have been irritation of the brachial plexus secondary to scalene entrapment in some subjects. Along these lines, as Travell and Simons (1983) have documented, referred pain patterns for both scalene and infraspinatus muscles directly overlap with the distribution of pain associated with median nerve dysfunction. Indeed, scalene involvement has been suggested by Headley in another report (summarized by Pronsati, 1992). However, these (scalene, infraspinatus) muscles were not specifically examined for myofascial dysfunction but *may* have been involved in some of the subjects' complaints. Other alternative explanations or mechanisms may have been operative as well. Likewise, it is not known if a retraining focus specifically on the CPS would have been equally efficacious; but since the retraining exercise is essentially the same for the SCMs and CPS, this may be a moot point.

NEUROMUSCULAR RETRAINING PROCEDURES DERIVED FROM DISINHIBITION THEORY

The application of this theory to CTS involves sEMG assessment monitoring eight separate channels concurrently of forearm flexor and extensor activity along with the activity of the SCMs and CPS. Both left- and right-sided activity are measured for each muscle. Neck flexion/extension is first performed. The SCMs and CPS are examined for any evidence of asymmetrical activation during what should otherwise be a relatively symmetrical movement (i.e., the maximum amplitudes of the left versus right SCMs are compared as are those from the left versus right CPS). Next, there is a determination of any evidence of cocontraction during head rotation (i.e., cocontraction of forearm flexors/extensors during primary rotational movements of the SCMs and CPS) and for evidence of inappropriate cocontraction of extensors and flexors during wrist flexion/extension.

For example, for a patient presenting right upper extremity CTS symptoms head/neck flexion/extension is likely to demonstrate, via sEMG, an asymmetrical pattern of relative activation, with the left SCM and (sometimes) right CPS yielding higher maximum amplitudes when compared side-to-side for the homologous muscle. Rotation of the head to the left is likely to elicit (abnormal) right forearm flexor activity and left extensor activity. Secondary processes may also be observed; for example, bilateral flexion movements of the wrists will typically yield severe cocontraction of the right extensors (which should be relatively quiet under normal circumstances). Hence, asymmetrical neck activity patterns are observed during the

symmetrical action of neck flexion; cocontraction is observed during the asymmetrical movements of head rotation (i.e., forearm flexors/extensors with neck muscle movements); and cocontraction is observed during wrist flexion involving that of the forearm/wrist extensors with the flexors (essentially having to overcome the excessive extensor activity) on the symptomatic side. The reverse patterns would be expected for patients presenting with CTS in the left upper extremity.

For patients presenting with bilateral CTS complaints, there is often a history of one side developing symptomatically first before the other. For example, if a patient can recall right-sided CTS developing first, then the patterns described above for the patient with right CTS will predominate in the clinical picture and as assessed via sEMG. However, in addition to the cocontraction (during head rotation to the left) of the contralateral (right) forearm extensors but also of the ipsilateral (left) forearm extensors, there is likely to be further cocontraction observed of the left forearm flexors during wrist extension. The rationale for this is that the inappropriately coactivated left extensors result in further inappropriate coactivation of the left flexors, explaining the temporal difference in onset of symptoms.

The treatment strategy is straightforward in the case of unilateral symptoms. For example, the patient with right CTS symptoms would be treated by focusing on the asymmetrical neck (SCM) muscle imbalance. The treatment protocol, following that described by Donaldson et al. (1998) would be to have the patient rotate the head to the right and then go into flexion, hold that position for ten seconds, and then return the head to a neutral position and rest quietly for 50 s. This would be repeated for a total of six times (i.e., 6 min). Three practice sessions in each subsequent 24-h period (for a total of 18 min each day, no more and no less) would be assigned until a restoration of balance is obtained as seen with sEMG maximum amplitudes during neck flexion/extension movements. Once there is a restoration of balance between the two sides of the neck muscles, the inappropriate cocontraction in the forearm extensors/flexors should concurrently subside along with CTS symptoms. In the case of bilateral symptoms, the treatment would begin by targeting the intervention based on the neck muscle asymmetry amplitudes observed during neck flexion/extension. Further work may need to focus on the actual forearm activity to release the overactive flexor activity. Other myotherapy (e.g., strengthening, stretching, and related techniques) may be useful in augmenting the intervention in either the unilateral or bilateral case. However, the relative role of myotherapy appears to be secondary, given that the CTS symptomatology typically resolves with the resolution of the neck muscle imbalance patterns detected and retrained via sEMG exercises.

FURTHER COMMENTS AND FUTURE DIRECTIONS

Many unanswered questions remain regarding this hypothesis and the treatment intervention derived in association with it. We presently have no basis for estimating which proportion of individuals with CTS would demonstrate the proposed sEMG patterns. Certainly, our own clinical experience, which may be affected by referral biases, would suggest that a substantial number of individuals with CTS symptoms

demonstrate such patterns of asymmetrical activation and coactivation. This is an important point bearing further research, however. If there are some people with CTS who are not helped with the sEMG techniques described, then it may be that the techniques, when effective, are more a means of breaking a pain cycle rather than providing support for the conceptualization offered of the neck problem precipitating the CTS. Also, it is possible that the disinhibition manifested in the sEMG findings may be secondary to the CTS symptoms themselves. Alternatively, the physical examiner should remain attuned to the possibility of other "double crush" phenomena being operative in producing apparent regional pain syndromes; that is, any of a variety of other nerve impingements or irritations (or illnesses such as diabetes and its associated nerve damage) may contribute either as a sole source or cumulative etiologic agents for CTS or related syndromes (Hurst, Weissberg, & Carroll, 1985; Slater, Butler, & Shacklock, 1994; Upton & McComas, 1973).

Nonetheless, the sEMG intervention as described in this paper results in reduction of symptoms and restoration of more normal muscle activation patterns. Hence, the inference would be that a primary dysregulation of neck muscle motor control at the level of the motoneuron pool may be a viable explanation. In subjects examined thus far, with restoration of the neck sEMG muscle balance to a more symmetrical representation there is a corresponding decrease observed concurrently in the pain and other CTS symptoms as well as an elimination of the more distant coactivation seen in the other (forearm) muscles. This suggests that the neck muscle imbalance (and the underlying neurological substrate) is a primary contributing mechanism.

In addition, future research should address and document the extent of the associations of forearm extensor and flexor activity with neck (e.g., SCM, cervical paraspinal, scalene) and other muscle (e.g., infraspinatus) activity to identify subgroups of patients with CTS who may have implicated one or more patterns of contributions of neck (or other) musculature dysfunction. The correspondence of these dysfunctions with trigger point or other myofascial indicators is an important additional step (cf. Travell & Simons, 1983; Donaldson et al., 1998). Similarly, an important question to address is the extent to which these findings are unique to CTS or apply to other upper extremity cumulative trauma disorders and/or to overlapping syndromes.

Certainly, also, other factors should be examined for inclusion into a comprehensive formulation of the development and persistence of CTS symptoms. For example, ergonomic and postural as well as affective, stress-related, and environmental aspects may be important in the genesis of the problems and in pointing to other factors that will require correction in order for any improvements resulting from the intervention described here to be maintained. This is particularly salient given the extensive psychosocial context in which CTS and other pain syndromes occur and the controversy that has been generated regarding the validity of cumulative trauma disorders and repetitive strain injuries in general (Hadler, 1997; Mackinnon & Novak, 1997). Nonetheless, a comprehensive multidimensional model of chronic pain may point to the integration of other myotherapy, psychological coping skills training, and related mind-body (e.g., relaxation training, pacing, etc.) interventions as necessary concurrent therapies with sEMG (cf. Caudill, 1995; Novy, Nelson, Francis, & Turk, 1995).

Future studies should be conducted to demonstrate the reproducibility of the beneficial effects of the sEMG techniques described, as well as the most appropriate components in addition to sEMG for correcting the muscular or other dysfunctions observed. Component analyses of multimodality interventions might suggest the most cost-effective ways to reduce the frequency of CTS and perhaps the frequency of surgery or other unhelpful interventions. In addition, there are theoretical implications for understanding the basic mechanisms underlying such dysfunctions, for example, the extent to which gamma motoneuron disinhibition may be implicated in the genesis of such problems in some instances. Overall, a broadened appreciation of the potential role of multiple etiologies in the seemingly common expression of CTS symptomatology may lead to a better appreciation of the diversity of mechanisms underlying or influencing CTS and more appropriate assessment and treatment strategies targeted in the individual case.

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