Carpal tunnel syndrome: a comprehensive review

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Carpal tunnel syndrome has become the most common clinical entity seen by hand surgeons. It has reached epidemic proportions in the United States and costs more than $2 billion annually. Its pathophysiology is multifactorial and its work-related issues are controversial. The method of detection, surgical treatment, complications, and methods of surgical revision of carpal tunnel syndrome are outlined. Appropriate treatment can be rendered in carpal tunnel syndrome only by using a cautious approach.

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Abbreviations

CTR	carpal tunnel release
CTS	carpal tunnel syndrome
TCL	transverse carpal ligament

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Carpal tunnel syndrome (CTS), which is associated with compression of the median nerve at the wrist, is the most common form of compressive neuropathy in the hand. It is classified as a subtype of "cumulative trauma disorder" or overuse syndrome. Untreated, CTS can lead to severe or irreversible disability. With treatment, however, an excellent prognosis can be expected. Over the past decade there has been a virtual epidemic of this condition, which raises the following questions: What are the main factors influencing this meteoric rise in CTS? How can CTS be prevented? What are the optimal non-surgical methods for dealing with CTS so that operative morbidity, along with its attendant absence from work may be avoided altogether? If surgery is unavoidable, what are the best techniques to relieve pressure inside the carpal tunnel so that the earliest return to full function with minimal residual disability can be achieved? This review addresses these issues and formulates a comprehensive diagnostic treatment approach to CTS based on a review of the current clinical and experimental literature.

Epidemiology

The annual incidence of carpal tunnel surgery in the United States is now estimated at 400,000 to 500,000 cases, at a cost in excess of $2 billion per year [1]. The costs incurred in workman's compensation cases are three times those of noncompensation cases and three times those of nonworkers, with total costs reaching $10,000 per case. Direct costs include physician reimbursement, electrophysiologic screening, and physical therapy. Indirect costs include loss of productivity, litigation, vocational rehabilitation, and permanent impairment awards [2]. In a study of prevalence and work-relatedness of CTS among 127 million US workers [3], 1.47% (1.87 million) reported CTS. Industries with the highest incidence of CTS were mail service, health care, construction, garment, electronics assembly, and food and meat processing. The risk factors seen most often included exposure to repetitive bending and twisting of the hands, white race, female gender, and the use of vibrating hand tools. The peak incidence is between 30 and 50 years of age [4]. The whole concept of work-relatedness, which formerly was accepted without question, is now coming under intense scrutiny.

In order to determine work-relatedness, a detailed work and social history must be obtained. Patients often have avocational activities that may be repetitive, such as gardening, home improvement, crocheting, or quilting. Ac-

cording to the literature [5], there is also a much higher incidence of CTS in obese workers with poor fitness profiles and sedentary lifestyles. Many older workers need to be shifted to less-demanding tasks. There is no guarantee that a given worker can continue a manual job until 65 years of age.

Many patients are highly motivated to use workman's compensation as their "insurance" because their regular health insurance will not pay for lost wages. These patients will try to paint a picture of work-relatedness, thus overestimating the incidence of occupational CTS. Among such patients, it is critical *not* to consider surgery unless there is compelling objective evidence. It is also necessary to differentiate between CTS *caused* by work and CTS *aggravated* by work. Patients should never be encouraged to file for workman's compensation simply because work aggravates their symptoms. These patients do poorly following surgery. The emphasis must be directed toward worksite evaluation, ergonomic intervention, surveillance, general fitness programs, and nerve mobilization exercises.

All this considered, it is clear from well-constructed epidemiologic studies that occupational CTS is more prevalent in workers involved in tasks that require extremes of wrist flexion and extension. In this setting, ergonomic modification has had significant impact. Using tool redesign, stressful postures can be minimized [6]. Other risk factors involve forceful use of the hand, repetition, and vibration. Clearly, the etiology is multifactorial, and I have seen patients with CTS in the nondominant hand when the dominant hand does most of the work.

Historical background

In 1854, Sir James Paget first described the symptoms of carpal tunnel compression caused by wrist trauma. In 1913, Marie and Foix described compression of the median nerve in an autopsy specimen. It was not until 1938 that the symptoms of CTS were correlated with pathoanatomy. Until that time, nerve compression by a cervical rib was thought to be the cause. In that year, Learmonth performed the first carpal tunnel release (CTR) in a symptomatic patient. Beginning in the 1950s, Phalan [7] began a series of studies encompassing over 1200 hands that brought this syndrome into the mainstream of modern medicine [7].

Anatomy

The carpal tunnel is a narrow channel that forms the volar entrance to the hand for nine digital flexors and the median nerve. It is virtually inelastic and bound by carpal bones dorsally, radially, and ulnarly, and, by the transverse carpal ligament (TCL), volarly. The TCL is attached on the radial side to the tuberosity of the scaphoid and on the ulnar side to the pisiform and the hamate. The ligament varies in thickness from 2.5 to 3.5 mm normally and is 3 to 4 cm wide. This ligament coalesces with muscular origins of the thenar and hypothenar muscles. The TCL is about 3 mm thick and 4 cm wide. Proximally, the ligaments are attached to the deep fascia of the forearm. The distal palmar crease is the skin landmark for the proximal border of the ligament. Nine flexor tendons, their tenosynovium, and the median nerve traverse the tunnel on their way to the hand and must be allowed to move freely through it.

The median nerve lies superficial to the flexor digitorum superficialis tendons and lies immediately under the ligament. Four centimeters proximal to the ligament, the median nerve gives off a palmar cutaneous branch that innovates the skin over the thenar eminence. It enters the hand through its own tunnel adjacent to the flexar carpi radialis muscle and superficial to the TCL. This branch can easily be injured during surgery and be a cause of postoperative pain. The median nerve has its terminal branches at the distal edge of the TCL. There, the nerve gives off two main trunks: The lateral trunk branches into the motor branch, the proper digital nerves of the thumb, and the radial side of the index finger; the medial trunk becomes the common digital nerves to the ulnar aspect of the index finger, the middle finger, and the radial side of the ring finger. The first branch to exit is the motor branch to the thenar eminence. It follows one of three courses. The usual position of the motor branch is the extraligamentous recurrent course, where the motor branch leaves the main nerve trunk at the distal edge of the TCL, turns back, and innervates the thenar muscles. The next most common variation is the subligamentous course, where the motor branch leaves while under the TCL. The least common variation is the transligamentous course, where the motor branch takes off under the ligament and pierces the ligament itself. Endoscopic CTR puts the motor branch at risk with this variation [8]. Superficial and ulnar to the TCL lies the volar carpal ligament, which forms the roof of Guyon's canal, (which holds the ulnar artery and nerve.) A recent study showed Guyon's canal to overlap the carpal tunnel by as much as 28%; with wrist extension, it slides even more radially [9•]. This places the ulnar nerve at special risk during endoscopic CTR. Volar to both ligaments lies the superficial palmar fascia, which extends distally to the digits.

Studies by Wilgis and Murphy [10] describe the normal movement of the median nerve through the carpal tunnel. With digital flexion, the median nerve slides proximally into the forearm and, when the fingers extend, it slides distally toward the hand. Extension of the fingers and wrist is the position in which the median nerve is displaced farthest under the TCL in to the hand. Its maximal excursion is 15 mm relative to a fixed point on the

TCL. Longitudinal sliding prevents local stretching and traction injury to the median nerve that would otherwise occur during wrist and finger movement [11•]. The flexor tendons move through the carpal tunnel with a 5 to 7-cm excursion. Szabo *et al.* [12] described a linear relationship between motion of the nerve and tendons so that for any fixed motion of a digit, for example, there will be consistent motion of the tendon and overlying nerve.

The carpal tunnel itself is a relatively inelastic tube that is unable to accommodate changes in the volume or pressure of the carpal canal. Recent dynamic magnetic resonance imaging studies have revealed significant changes in the cross-sectional area of the tunnel with extremes of flexion and extension [13]. This has been corroborated by pressure studies in the carpal canal that reveal a 10-fold increase in pressure when the wrist is flexed or extended as opposed to being in the neutral position [14]. There may also be gender differences in the "volume" of the carpal tunnel, which may explain in part the increased prevalence of CTS in women [15].

Pathophysiology

There is normally ample room in the carpal tunnel for all the structures to move through comfortably. Although the carpal tunnel is open at both ends, it functions as a confined space. The flexor tendons may move across this area 20,000 times in the course of a workday (*eg*, in a clerk typist keyboarding at 60 words a minute). There is minimal relative friction between the tendons and the nerve because of the investing tenosynovium. Some shear forces are generated, however. With chronic, repetitive use of the fingers, it is believed that shearing may result in localized hyperplasia and fibrosis of the investing tenosynovium and mesoneurium about the median nerve [16]. This tissue reaction fills the carpal tunnel and causes compression about the nerve. Studies have shown that the resting pressure across the carpal tunnel is about 2.5 mm Hg at rest and about 32 mm Hg with wrist flexion and extension. This is well below the filling arterial pressure. In CTS, the resting pressure is 32 mm Hg and will increase to 100 mm Hg with flexion and extension [17]. Arteriolar and venular smooth muscle hypertrophy have also been noted in the vessels going to and from the nerve in patients with CTS. Lundborg *et al.* [18] also have described a miniature compartment syndrome in the nerve inside the perineural sheaths. This syndrome is believed to be caused by increased pressure in the intraneural vasculature secondary to venous outflow obstruction. This causes intraneural ischemia.

In addition, compression retards the normal gliding function of the nerves. Nakamichi *et al.* [19] demonstrated that there is significantly diminished sliding in patients with CTS as compared wtih those with normal wrists. If the nerves are unable to glide with normal limb movement, a traction injury occurs with subsequent diminution in electrical conduction. During carpal surgery, an hourglass deformity is commonly seen in the nerve. This is believed to be caused by displacement of axoplasm and telescoping and shearing away of myelin from the center of compression. The above postulates are used to explain the mechanism of CTS in "overuse" syndromes.

In reality, however, anything that will increase the volume of the contents in this relatively inelastic canal will increase the pressure on the nerve. Neurofibromas, lipomas, myelomas, ganglion cysts, hypertrophic "inflammatory" synovitis (such as that seen in rheumatoid arthritis), Reynaud's disease, infection or gout, aberrant muscle bellies (*ie*, proximal origin of the lumbricals) persistent median arteries, hemophiliac hematomas, Colles' fractures, and malunions and carpal instabilities form a partial list. Peripheral neuropathy such as that seen in diabetics and alcoholics may have a superimposed compressive neuropathy. Here, the median nerve is already compromised by the systemic disease, so it is more susceptible to the effect of localized compression. Additionally, alterations in fluid balance can increase the pressure inside the canal. Conditions such as pregnancy, eclampsia, myxedema, renal failure, amyloid deposition, obesity, and even fluid shifts seen during recumbence at night can create symptoms. Szabo and Chidgey [17] have shown that after repeatedly flexing and extending the wrist, recovery to resting pressures took longer in patients with CTS than in normal control. Prolonged fine finger movements with the wrist hyperflexed or hyperextended, such as those seen in musicians or computer operators, or use of the hands for weightbearing functions such as that seen in paraplegics and long-distance cyclists, will elicit the same symptoms. Another clinical entity is double-crush syndrome, which is essentially CTS with a proximal compressive lesion along the course of the median nerve. There may be a compression at the level of the cervical roots (C-5, C-6), the thoracic outlet, or the pronator inlet. It has also been observed that there may be a cumulative effect of compression at multiple sites, meaning that the compression at one site may not be severe enough to register on an electrodiagnostic test, but in total, the cumulative compression may be pronounced.

Persistent low-grade compression can cause endoneural edema, which interferes with endoneural blood flow and ionic axonal conduction. In addition, compression has been shown to slow both orthograde and retrograde axonal transport. Local ischemia stimulates fibroblast activity and if compression is left untreated, local demyelinization and intraneural fibrosis occurs. These changes may be irreversible even after definitive treatment [20].

Clinical presentation

Patients with CTS will commonly complain of numbness, tingling, or pain in the thumb, index, and middle

fingers—which classically corresponds to the anatomic sensory distribution of the median nerve in the hand. This presentation can vary, however, from one finger (*eg*, the middle finger) to the whole hand. Symptoms frequently are aggravated by repetitive exertion and often will awaken patients at night. Relief is sought by shaking, massaging, immersing the hand in warm water, hanging the hand over the side of the bed, or exercising the hand until the symptoms subside. Motor symptoms follow, including diminished dexterity of fine movements such as sewing, writing, or playing a musical instrument. The hand begins to feel "swollen," and patients have difficulty holding objects. Activities that call for raising the hand, such as holding a telephone or newspaper or manipulating a steering wheel, can aggravate symptoms. The dominant hand is affected often and the pain and paresthesias can radiate up the arm to the shoulder. In some reported series, 56% of patients had bilateral symptoms that more commonly affect women. Finally, the patient notes thenar atrophy. It is critical to determine the extent of proximal symptoms so that the possibility of a coexistent cervical disk or neurogenic thoracic outlet obstruction can be determined. There may also be a double-crush syndrome.

Physical examination

Physical examination should consist of sensibility evaluation and provocative testing. Tests of sensibility include sharp/dull, tactile discrimination; two-point discrimination; threshold sensibility; vibration testing; and quantitative sensorimotor testing.

Sharp/dull, tactile discrimination—measures of pain and temperature—can remain normal until late in the disease. Two-point discrimination is the ability to distinguish between one and two points at the fingertips. This is a reflection of the enervation density of slowly reacting fibers in the static test and rapidly reacting fibers in the moving two-point discrimination test, which is the ability to distinguish between one and two points at 6 mm apart. More than 6 mm is considered delayed, and more than 10 mm represents loss of protective sensation. The sensitivity of this test in CTS is low, and two-point discrimination may remain apparently normal until late in the disease.

Threshold sensibility tests the patient's ability to detect fine tactile stimulation with pressure on the fingertips made by fine monofilaments, testing the threshold of slowly adapting sensory fibers. This is an array of filaments of progressive thickness and skin pressure, known as the von Frey or Simms-Weinstein monofilament, that is sensitive in early impairment (83% positivity).

The threshold ability to detect vibration of fixed frequency (128 to 256 Hz) and variable amplitude is helpful in early disease. Vibration testing assesses the threshold of rapidly adapting fibers. Its use as a preemployment screening measure is controversial. Werner *et al.* [21] performed serial vibrograms on a cohort of manual laborers and found that threshold values changed over the course of the work week. (In 1995, Gerr *et al.* writing in the *Journal of Occupational and Environmental Medicine* found that the sensitivity of vibrometry was twice as high after 10 minutes of wrist flexion than before for the detection of CTS.) Werner *et al.* [21], however, maintained that vibrometry threshold perception does not correlate with median sensory latency delay and is not a good early warning screen for CTS.

Quantitative sensorimotor testing is a computer database–driven screening array that determines impulse and sustained pinch and grip strength, as well as the precise psi threshold perception for one- and two-point discrimination for a given dermatome using fine transducers. Early results show that this test may be quite sensitive in early disease [22].

There are four provocative tests for CTS: (1) Tinel's sign can be elicited by lightly percussing the skin along the course of the median nerve. Distally radiating pain or paresthesias will result when the site of compression is tapped (70% sensitivity). (2) Phalen's test is executed by the patient actively flexing the wrist maximally. This effect can be augmented by the examiner pressing a finger against the patient's proximal palm at the same time. Shooting paresthesias into the median nerve distribution within 60 seconds is a positive test (91% specificity). (3) Muscle weakness is tested subjectively by resisted palmar abduction of the thumb against the examiner's finger, feeling for the abductor pollicis brevis tension or atrophy. (4) Injection of a small amount of 1% lidocaine into the carpal tunnel that relieves the pain in the palm or volar forearm can confirm the diagnosis and predict a positive outcome of possible surgical release.

Electrodiagnostic studies

Electrodiagnostic studies, including electromyography and nerve conduction studies, form the benchmark for the objective diagnosis of CTS, despite reports of false-negative results. While there are reports in the literature of excellent results with CTR in symptomatic patients with negative tests, for the most part these studies are the gold standard for diagnosis.

Electromyography

Electromyography tests the electrical activity of the abductor pollicis brevis muscle after motor nerve stimulation. Reduced recruitment of motor units is seen most frequently (35% of cases). Fibrillation potentials and positive waves, which signal median nerve denervation from axon loss, may only be seen in 20% of cases. The

likelihood of a positive electromyographic study is much higher in patients with longstanding motor symptoms. The main utility of electromyography is its ability to detect the more severe cases of CTS and to distinguish CTS from more proximal lesions along the median nerve, ulnar nerve compression, and cervical root compression. Thus, intrinsic muscles that are enervated by the ulnar nerve and forearm musculature need to be evaluated at the same time.

Nerve conduction studies

Nerve conduction studies can be either motor or sensory. Motor conduction involves stimulation of the median nerve at the wrist and antecubital fossa while recording from electrodes overlying the abductor pollicis brevis muscle. The nerve is stimulated, causing an action potential to travel orthodromically down the nerve and chemically across the neuromuscular junction, resulting in a compound motor action potential recorded from skin electrodes overlying the muscle. The time (in milliseconds) it takes the impulse to travel from the stimulation point to the recording point is called the *motor latency*. Subtracting the distal motor latency from the proximal motor latency and dividing the result into the distance between the two stimulation points is the motor conduction velocity in meters/second. This measures conduction across the largest and fastest conducting fibers and is sensitive to focal demyelinization at the compression site. Other nerves need to be checked at the same time to exclude polyneuropathy such as in diabetes, to compare the velocities between the motion of ulnar nerves in the absence of an absolute standard. It can document the level of severity of motor fiber involvement across the carpal tunnel in 70% of cases. Using the "inching technique," in which the stimulator electrode is moved in 1-cm segments across the wrist, is more sensitive but has been far less specific (70% false-positive results) in some series.

Sensory conduction or sensory nerve action potential can be determined by orthodromic (distal to proximal) or antidromic (proximal to distal) stimulation. It records from the largest (15%) myelinated axons in the sensory nerve bundles. These are summations of hundreds of axons firing at the same time, and the amplitude of sensory conduction depends on the number of axons firing and their synchronicity. These nerves need to be measured over short distances and in nerve segments near the skin; otherwise, the amplitude drops off significantly. This is otherwise a very sensitive indicator of focal nerve compression. It is noteworthy that the percentage of true-positive results (60% to 100%) may vary with the finger stimulated and may represent the relative position of a given fiber bundle to the TCL. Truly, compression at any given cross-sectional point in the nerve is uneven. It may be wise to record from the most symptomatic digit. Palmar sensory latencies are measured by stimulating the median nerve in the midpalm at a point 8 cm distal to the recording electrodes at the wrist. This should be less than 2.2 ms and should not differ from the ulnar sensory nerve action potential by 0.2 ms. This test is sensitive in 87% of CTS cases.

There are pitfalls in the overreliance on electrodiagnostic tests. Many physicians who see CTS regularly have seen patients with a classic presentation but normal tests. If only a small number of medium or small fibers is blocked, there may be no delay seen in conduction, as these tests measure readings in the most rapidly conducting fibers. In addition, ischemia and mechanical nerve irritation seen in early CTS may not exhibit a conduction block. Prolonged latencies may not revert to normal even 1 year after surgery, although patients may be clinically improved. The results are operator dependent, and even the sequence of testing can alter the results. Underlying double-crush syndrome or peripheral neuropathy can obscure the results, and there has been poor correlation between the severity of the disease and the magnitude of the numbers. I believe that these tests serve as a useful adjunct to a careful history and physical examination and should be viewed as such [23].

Conservative management

Unless there is obvious thenar atrophy, all patients presenting with CTS should be treated nonsurgically. Nonsurgical treatment generally consists of splinting, nonsteroidal anti-inflammatory drugs, cortisone injections into the carpel tunnel, and vitamin B_6.

Splinting

A forearm-based wrist splint is widely used as the primary initial treatment. There is controversy, however, about whether the brace should be worn all the time or at night only. I prefer nighttime splinting. While the underlying concept is that splinting will prevent extremes of flexion and extension, which have been shown to increase carpal tunnel pressure, most people rarely place their wrists into these positions while awake. If made aware of the risks of keeping their hands in these positions, most people can avoid doing so voluntarily, even in the workplace. With computer operators, ergonomic workplace adjustment should guide the wrist into a neutral position naturally. In addition, there is evidence that trying to use the splint during daily activities will shift stresses higher up on the arm, causing overuse-type pain farther up.

It has been stated that splints serve to reduce inflammation in the region of the carpal tunnel. I know of no study that documents this claim objectively, and all studies that have examined the histology of the tenosynovium in the carpal tunnel in chronic cases show it to be devoid of

inflammation. I believe that full-time splinting immobilizes the median nerve and interferes with its normal gliding pattern. Hence, the same segment of compressed median nerve sits under the TCL at the point of maximal compression all the time. One must be careful with the use of splints because many come from the factory cocked up into extension as much as 30°. I routinely bend splints back to neutral before giving them to a patient. Weiss *et al.* [24] reported that the neutral position of flexion will cause the least intratunnel pressure.

Several studies have examined the symptom remission rate after splinting. Those with the shortest latency had the best results from splinting. However, all studies showed a significant relapse rate. The study with the best results showed that only 40% of patients were symptom-free after 2 years [25].

Nonsteroidal anti-inflammatory agents

Nonsteroidal anti-inflammatory drugs are used as a first-line agent in the treatment of CTS. With the possible exception of CTS caused by inflammatory arthritis (such as rheumatoid disease), the environment of the carpal tunnel is not inflammatory. So it is not clear what effect, if any, these agents have locally. Empirically, I have not seen any effect from the use of these agents. For pure analgesia, there are other drugs such as acetominophen that will relieve pain with less risk.

Cortisone injections

Clinicians who treat CTS nonsurgically generally recommend cortisone injection into the carpal tunnel. This is accomplished by mixing a 1-mL solution of betamethasone, triamcinolone, or methylprednisolone with 1mL of 1% lidocaine and 1mL of 0.25% bupivacaine. A 27-gauge needle is angled 45° distal relative to the palm and introduced at the distal wrist crease in line with the ring finger. As the needle punctures the TCL, a "pop" is felt and the patient is asked to move his or her fingers. If the flexor tendons are pierced, this movement will be painful. The needle is then withdrawn until movement is possible. Injecting the steroid into the tendons exposes the patient to the risk of tendon rupture. If sudden paresthesia is felt by the patient, the needle must immediately be withdrawn because this signals possible puncture of the median nerve. While puncture alone will not injure the nerve, steroid injection into nerve fascicles is highly toxic and must be avoided. With proper placement of the solution, numbness of the hand in the median distribution should result. Care must also be taken not to inject Guyon's canal because it may overlap the carpal tunnel by as much as 30% and be superficial to it.

All series examining the effect of steroid injection have demonstrated significant pain relief in as many as 80% of patients. After 10 months, however, 80% to 90% of these patients report symptom recurrence [25]. It is therefore recommended that injections be reserved for those patients who for some reason cannot undergo surgery over the short term but are otherwise quite symptomatic or for pregnant women whose symptoms should resolve shortly after childbirth [26].

Vitamin B_6

Use of vitamin B_6 (pyridoxine) has been widely publicized in the lay press. This stems from studies dating back to the mid-1970s that described the coexistence of vitamin B_6 deficiency and CTS and the resolution of symptoms with B_6 administration [27]. Most of these studies were descriptive and anecdotal, however, and all were retrospective in nature with small patient populations. It is unclear whether these patients had CTS or B_6 deficiency with peripheral neuropathy that improved with "treatment." More recent studies have shown that in large series of patients with CTS, no vitamin B_6 deficiency was found; and those patients who received B_6 as part of their treatment showed no added benefit over those who did not, including the placebo group [28]. This included both clinical measures and electrodiagnostic markers. At this time, vitamin B_6 administration cannot be recommended for the treatment of CTS.

Diuretics

There is a subset of patients whose CTS is caused by localized edema (*ie*, pregnancy), and there are reports of the use of diuretics as an adjunct in these patients. However, there are no studies to date comparing the use of diuretics alone with other treatment modalities [26].

Exercise

Although most classic articles dealing with the management of CTS do not discuss exercise as a viable option, there has been a recent upsurge of interest in this modality. This is due to the large percentage of patients who fail to improve with standard therapy, to the high cost (both direct and indirect) of surgical care (*ie*, lost work days and productivity) and to the explosive rise in the incidence of CTS over the past 10 years.

The underlying goal of exercise therapy is restoration of the normal gliding function of the flexor tendons and the median nerve that has been disturbed by the compression and adhesions that have developed in the carpal canal as a result of tenosynovial hyperplasia. Seradge *et al.* [29] have shown that intermittent exercise can actually diminish the pressure inside the canal and that this effect persists for some time. Recently, a large retrospective study comparing conservative treatment programs with and without an exercise program showed a significant diminution of symptoms in exercising patients over non-exercisers. In addition to night splinting, patients move their hands through a series of positions designed

to maximize excursion of the nerves and tendons (Fig. 1 and 2). Each position is held for 7 seconds and is repeated five times daily. The hands are soaked in hot water for 4 minutes prior to the exercises and in cold water for 1 minute afterward. This may be tried for 4 to 6 weeks prior to the consideration of surgery [30].

Miscellaneous treatments

In 1995, Monge *et al.* [31] reported a series of 45 patients with type 2 diabetes treated for 12 months with tolrestat, an aldose reductase inhibitor, who showed improvement in pain and paresthesias as well as improvement in sensory nerve conduction velocity between the forefinger and the wrist.

Surgical technique

It is estimated that 50% of patients with CTS will be treated surgically. After induction of intravenous regional or local anesthesia, a tourniquet is inflated to approximately 250 mm Hg. A longitudinal midpalmar incision is created in the line of the ring finger from Kaplan's cardinal line to the wrist flexion crease. Rarely does the wrist crease need to be crossed. The subcutaneous fat at the proximal end of the incision is preserved, as this layer has been shown to contain the crossing terminal fibers of the palmar cutaneous branch of the median nerve. Cutting these nerves will predispose patients to postoperative scar tenderness. Instead, this layer is undermined. The fibers of the palmar fascia are spread, and the distal transverse fibers of the TCL are seen. These fibers are then undermined with blunt dissection with a hemostat and Freer elevator. The distal end is then incised, with the blade staying closer to the ulnar side of the ligament and working distal to proximal. Under the fat layer, scissors will complete division of the ligament. The median nerve is thus exposed and will frequently exhibit an hourglass deformity (Fig. 3). Care is taken to inspect the median nerve for any anomalies, notably the transligamentous motor branch, for it may become injured during this stage. The wound is checked distally to find the superficial palmar arterial arch, and any distal fibers of the TCL are incised. The floor of the carpal tunnel may be inspected at this time in search of ganglia or other masses. Tenosynovectomy is not necessary in routine cases but may be helpful in patients with gout, rheumatoid arthritis, or infections such as tuberculosis. Neither

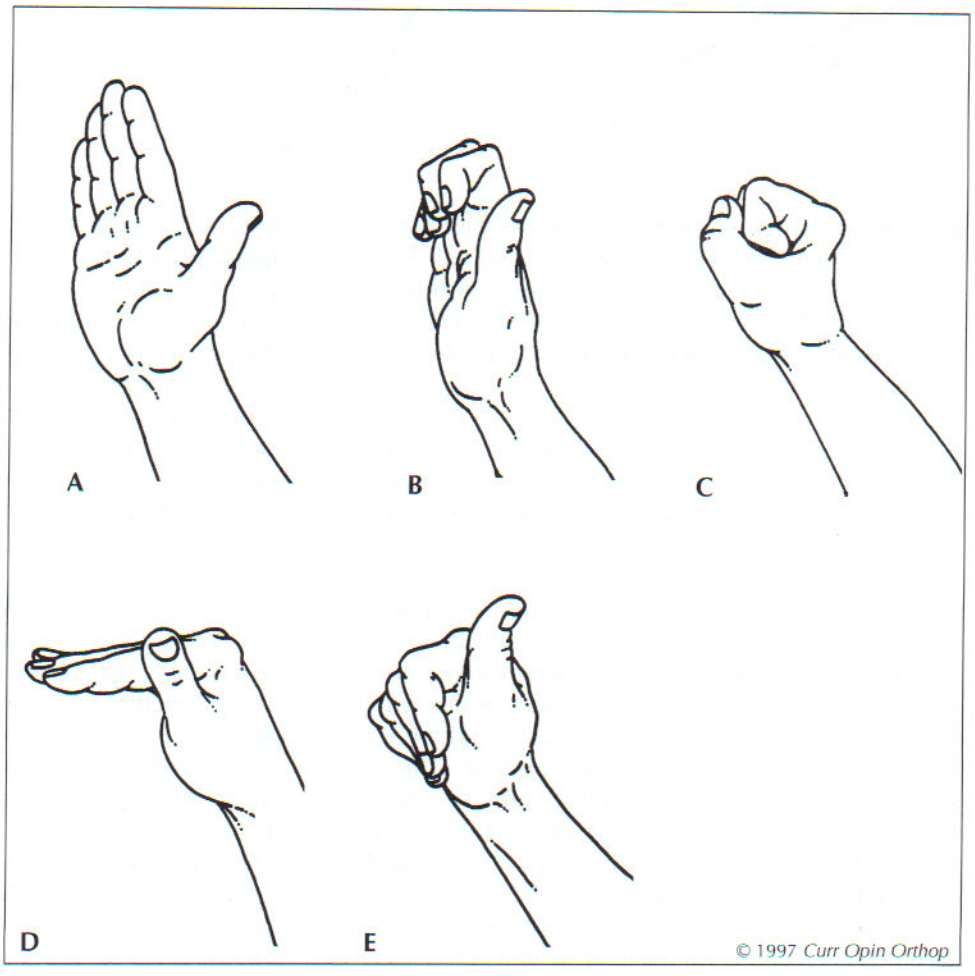

Fig. 1. Five exercises for tendon gliding (*A* through *E*), in which each position is held for 7 seconds and is repeated five times per day.

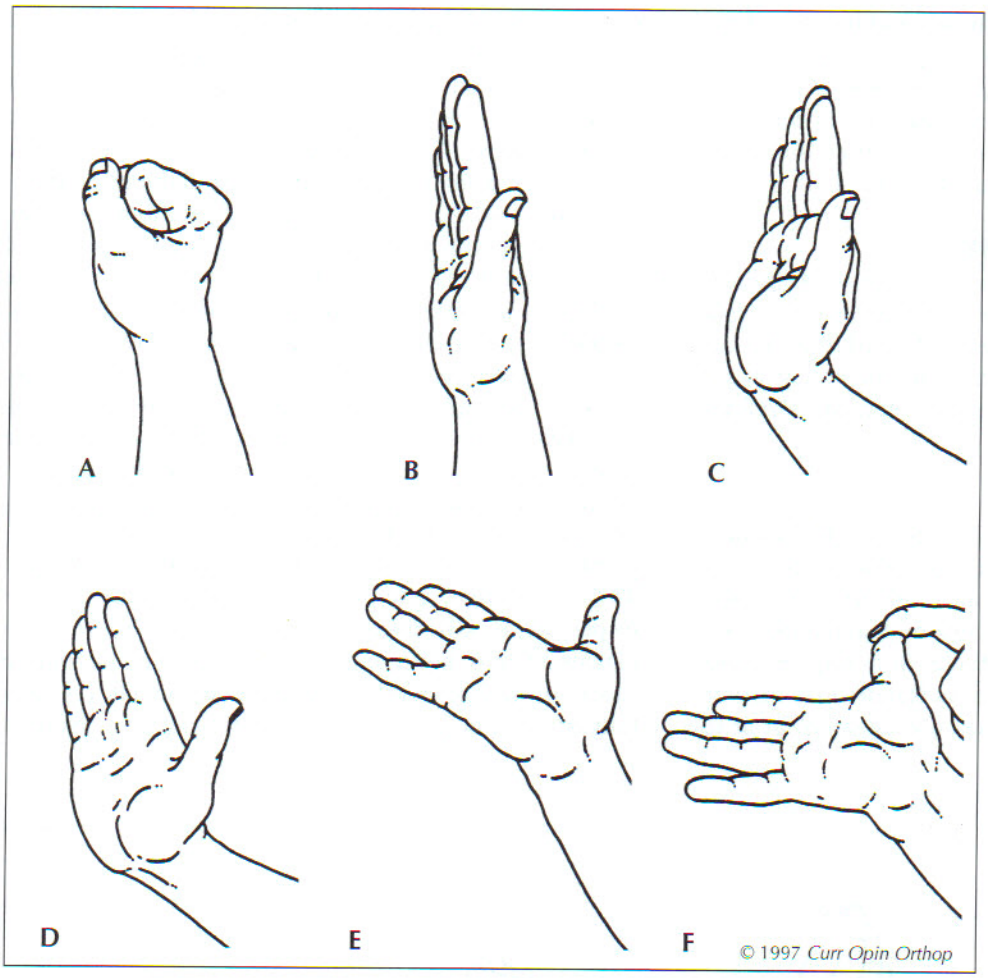

Fig. 2. Six exercises for nerve gliding (*A* through *F*), in which each position is held for 7 seconds and is repeated five times per day. Care must be taken to maintain wrist hyperextension during the exercises in *D*, *E*, and *F*.

internal neurolysis nor epineurotomy is indicated, because they have not been shown to yield any benefits and may actually be harmful and promote postoperative fibrosis [32]. Simultaneous release of Guyon's canal is not routinely recommended because the pressure inside appears to diminish after CTR [33].

Z-plasty of the TCL has been proposed in young patients, but further study is necessary to validate this method and to justify a much more extensive dissection [34].

Magnetic resonance imaging studies have compared the morphology of the carpal tunnel before and after surgery. These studies reveal consistent alterations after open release. There is a change in shape from oval to circular, with a mean increase in volume of 24% [33]. Osterman [35] has shown symptom resolution in 96% of patients, with 84% returning to their original employment. However, postoperative pinch and grip strengths may take up to 6 months to return to normal, and some never do. Katz *et al.* [36] showed that Tinel's and Phalen's tests can be positive even at 2 years. Nancollas *et al.* [37] showed that up to 30% of patients complain of weakness and scar tenderness postoperatively. This is due in part to loss of attachment of the thenar and hypothenar musculature [38]. The dysesthesias and pain experienced preoperatively usually disappear quickly, although many patients will complain of persistent tingling. While the early ischemic changes are reversed rapidly by the release, secondary and more chronic changes take much longer to resolve (if at all) in severe, chronic cases. Higgs *et al.* [39] compared workman's compensation with non–workman's compensation patients and showed significantly poorer results in the compensation group.

Postoperatively, a soft dressing is placed on the surgical site for 10 days, and early motion is begun. Some surgeons cast the patient following surgery to prevent "bowstringing" of the flexor tendons at the carpal tunnel. I believe this consideration to be theoretical only and less of a concern than postoperative adhesion of the median nerve and flexor tendons. In 1995, Bury *et al.* [40] found no benefit from postoperative splinting over a simple bulky dressing. Cook *et al.* [41] demonstrated that splinting postoperatively can be detrimental, with a delay in return to activities of daily living, increased pain, and diminished grip and pinch strength compared with

Fig. 3. The typical intraoperative appearance of the median nerve showing the hour-glass deformity in carpal tunnel syndrome.

patients who mobilized their hands and wrists early. I therefore do not cast but encourage early wrist and finger movement. Generally, patients can perform the same nerve and tendon exercises that they did preoperatively. However, I discourage purposeful use of the hand for 4 weeks. After 10 days, the sutures are removed and scar massage with vitamin E is begun.

Alternatives to the standard open approach

In 1989, Chow [42] described a two-portal endoscopic release and has since described several refinements to this technique. The proponents of endoscopic CTR claim that this method produces less tissue trauma and hence requires a much shorter recovery time and quicker return to work. In 1992, Agee *et al.* [43] compared open and endoscopic release in a prospective multicenter study. These authors showed greater pinch and grip strength and less wound scarring and pillar tenderness early in the recovery period following endoscopic release. There was also an early return to work for this group. In workman's compensation patients, however, there was a marked delay in return to work in both groups [43].

There are also many concerns with the technique of endoscopic release. Equipment is costly, the learning curve is steep, and complications occur even with experienced surgeons [44]. The complication rate has been reported as high as 11% in patients in whom an intrabursal approach was used and 2.2% in patients in whom a subligamentous extrabursal method was used [45•]. Complications include transection of the median nerve or its branches, the superficial palmar arch, or the ulnar nerve; incomplete release of the TCL; and ulnar neuropraxia [46]. Because of these complications, various "mini-open" techniques using specialized instruments have been described. These techniques have not been without complication, however [47]. Recent studies have suggested that the differences between the open and endoscopic techniques have focused on the wide-open classic approach, yet when the mini-open techniques were compared with the endoscopic approach, the postoperative courses were nearly identical [48]. It is clear that endoscopic CTR should be practiced by experienced surgeons with a thorough knowledge of local anatomy. It should not be regarded as a trivial procedure simply because the skin incision is smaller. Intensive training and practice on cadavers is required. Clear-sighted postoperative follow-up is necessary to respond early to potential complications.

Complications of surgical treatment

When performed for appropriate indications, CTR is generally a reliable operation with high success and low complication rates. Complications do occur, however, and a national survey conducted by the American Society for Surgery of the Hand showed that 16% of respondents had performed more than six or more reoperations for failed CTR [49].

There are many reported reasons for failed CTR. The most common cause of recurrent or persistent symptoms is incomplete release of the TCL. Patients with recurrence may have a symptom-free period. However, patients with incomplete TCL release usually have no relief postoperatively. Other reasons for persistent symptoms include an incorrect diagnosis, which can include a proximal compressive lesion such as pronator or double-crush syndrome; nerve adhesion with traction neuropathy; regrowth of the TCL; median nerve subluxation or bowstringing; infection; or technical errors during surgery, such as injury to the palmar cutaneous branch of the median or ulnar nerve or the main trunk of the median nerve. Nerve injury will frequently result in reflex sympathetic dystrophy that can be extremely re-

sistant to treatment [50••]. If the sensory nerve is injured, an obvious neuroma results at the proximal wrist crease. It is better to cut the nerve end distally and flip it back proximally and bury it under the superficialis muscle [50••]. Inadvertent entry into Guyon's canal is not harmful, but needs to be recongnized immediately or the ulnar nerve will be released instead of the median nerve.

Hypertrophic scarring and pillar pain may be unavoidable (the incidence may reach 20%) [50••]. I have seen this subcutaneously even during endoscopic CTR. Key elements are avoidance of an incision that crosses the wrist crease, good intraoperative hemostasis, and disallowance of any resistive activity with the hand during the first month. In the past, proximal transverse incisions were employed and the ligament was sectioned blindly, but this method was associated with a high incidence of accidental injury to the superficial arterial palmar arch. It is safer to open distally and release proximally, undermining the TCL first and staying in the line of the ring finger. Most importantly, if patients return after surgery with persistent complaints, they must be taken seriously and not be thought of as malingering or suffering from "compensationitis."

Techniques for reoperation

A whole genre of surgical procedures has arisen to "reconstruct" failed CTR. The incidence of these reconstructions is reported to be 3% of all CTRs [51•]. These include procedures to "cover" or "wrap" the median nerve [52], free up the nerve from the surrounding scar, and reconstruct the TCL [53]. Procedures that are designed to cover the nerve include turning down muscle flaps using the palmaris brevis, pronator quadratus [52], and abductor digiti minimi muscles [54]. Fat grafts such as the hypothenar fat pad turndown [52] or free dermal fat graft [55] have been used with success. Hunter [53] described extensive neurolysis of the median nerve with actual reconstruction of the TCL for what he termed "traction neuropathy." This procedure is designed to restore nerve gliding and has met with considerable success [53].

Conclusions

It has become clear that CTS is one of the easiest medical conditions to detect and treat and at the same time extremely difficult to evaluate properly and treat comprehensively. Treatment plans need to be individualized, and knee-jerk reactions followed by cookie-cutter treatments must be avoided. Such approaches have proved to be expensive failures and will only continue to be. Every step in the treatment algorithm must be reasoned out and carefully followed if we are to fulfill our patient's expectations. First, do no harm.

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