Evolving Concepts of Median Nerve Decompression in the Carpal Tunnel

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Compression neuropathies, formerly thought to be rare, have been recognized with increasing frequency over the past 25 years by clinicians who deal with problems of the hand. This apparent increase in incidence in fact reflects a profound increase in awareness and accuracy of diagnosis—both factors which are based on a clearer understanding of the anatomy, pathophysiology, and subsequent clinical appearance of each of these fascinating clinical entities. Of all the compression neuropathies, median nerve compression at the wrist (carpal tunnel syndrome) is the most frequently encountered and best understood. As our understanding has evolved, there has also grown an appreciation for the subtleties and nuances that accompany this entity, and this has been mirrored in a number of modifications in treatment, both surgical and otherwise.

We wish to review the salient features and current understanding of compression neuropathies of the median nerve at the wrist, with emphasis on points that have proved particularly valuable in our own clinical practices. We will also stress those points of technique that reflect a shift from early practices. Such a personal review is necessarily biased, and we will attempt to add balance by including a selected (rather than encyclopedic) listing of both complementary and opposing viewpoints from the published literature on the subject.

ANATOMY

The carpal tunnel or canal is a closed space whose dorsal, radial, and ulnar boundaries are the bones of the carpus and whose volar limit is the transverse carpal ligament. The transverse carpal ligament attaches to the pisiform and hamate on its ulnar aspect and to the scaphoid tubercle and trapezium on its radial edge. Thus, all the boundaries of the canal are either bony or ligamentous and are nonyielding in character. The nine extrinsic flexor tendons of the fingers and thumb pass through the carpal tunnel surrounded by the synovial membranes of the ulnar and radial bursae and accompanied by the median nerve.

PATHOPHYSIOLOGY

The common denominator of the various specific etiologies of carpal tunnel syndrome is a relative volumetric increase in the contents contained within the carpal tunnel. This can be accomplished either by an actual

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increase in volume or decrease in dimensions. Since all the boundaries of the carpal tunnel are nonyielding, an increase in content results in increased pressure within the canal and subsequently upon all of the structures contained therein. Increased pressures within the carpal tunnel, long assumed to be present in symptomatic patients, have been clearly demonstrated by Gelberman and Lundborg using direct measurements.

A large and ever-growing list of etiologic factors that can elevate pressures within the carpal tunnel and cause symptoms of median nerve compression has been compiled (Table 1). These factors can be divided into acute and chronic conditions, ranging from carpal fracture-dislocation with direct impingement of bony segments into the carpal tunnel to nonspecific synovial swelling of uncertain cause. The symptoms produced, regardless of which inciting agent is responsible, are fairly consistent and reflect our current understanding of the effects of either acute or chronic pressure on peripheral nerves.

A detailed and technical review of this current understanding is beyond the scope of this discussion, and the interested reader may refer to more extensive treatises. Briefly summarized, however, it appears that increased pressure on a peripheral nerve interferes with local nutrient blood flow to the nerve. If this local low flow state is transient and mild, then the pathologic state produced may be transient and full recovery of function expected (eg, the neurapraxia of Seddon, grade I lesion of Sunderland). Infarction of the involved nerve represents the opposite end of a pathophysiologic spectrum containing almost endless variations and degrees of severity. In addition to interfering with local hemodynamics, pressure applied to a peripheral nerve has been shown to be capable of producing characteristic and definitive microstructural changes within the neural and supporting structures. It is uncertain at this time which of these two factors predominates in the clinical setting, although it is probable that the more transient neuropathies with rapid recovery are primarily caused by local hemodynamic alterations, and the states in which actual microarchitectural changes are present correspond to more recalcitrant, long-lasting compression neuropathies (eg, axonotmesis of Seddon, grade IV lesion of Sunderland).

### Table 1. Some Causes of Carpal Tunnel Syndrome*

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<tr>
<th>Category</th>
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<tr>
<td>Inflammatory</td>
<td>Rheumatoid flexor tenosynovitis</td>
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<td>Non-specific flexor tenosynovitis</td>
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<td>Thrombosed median artery</td>
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<td>Vascular tumors</td>
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<td>A-V fistulas (hemodialysis)</td>
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<td>Anatomic Anomalies</td>
<td>Enlarged or abnormally located flexor muscles</td>
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<td>Ganglion</td>
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<td>Lipoma</td>
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<td>Trauma</td>
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**CLINICAL APPEARANCE**

Historically, carpal tunnel syndrome has occurred most frequently in women between 30 and 50 years of age. The female to male ratio most quoted in the literature is 3:1 and parallels our own experience, although the age range of our patients seems to be somewhat younger.

The most common complaint is intermittent numbness in the sensory distribution of the median nerve, with the long and index fingers affected more often and more severely than the thumb. Tingling and pain are frequently concomitant complaints. Initially, symptoms are intermittent but become constant in more advanced, longstanding cases. Many patients first note symptoms at night, when they are awakened from sleep by tingling discomfort in the median distribution, often described as "pins and needles." This predilection for night-time symptoms has been ascribed to sleeping in the fetal position with wrists...
Evolving Concepts of Median Nerve Decompression

725

flexed, to direct pressure on the wrists by the head, and to changes in total body fluid distribution resulting from lying horizontally. For unclear reasons, the discomfort is often relieved by shaking the hand or hanging it in a dependent position.

In contrast to night symptoms, many patients will note the onset of symptoms at work or while performing repetitive tasks. Furthermore, activities such as repeated fist clenching or prolonged gripping, as in grasping a steering wheel, will initiate symptoms, while cessation of such activities brings relief. The pain of carpal tunnel syndrome may occasionally radiate proximally, causing confusion with other pain complexes of the forearm, elbow, and shoulder.

Careful questioning and examination will usually pinpoint the site of symptomatic origin at the wrist, even when the patient has not noted this as the source. Carpal tunnel syndrome should thus be considered in the differential diagnosis of almost any obscure symptom complex involving pain in the upper extremity.

Progression of the intermittent episodic nature of the symptoms in carpal tunnel syndrome to either more prolonged or even constant discomfort or numbness usually signals more severe disease, although the degree of actual nerve damage found at surgery, as well as the potential for recovery of function, seems to be more related to the severity of compression than to its longevity. Put another way, marked compression for a relatively short period of time has the potential to produce more neural damage than minimal compression over a longer period of time.

Weakness and atrophy of the median-innervated thenar muscles tend to occur later in the course of the disease, usually after sensory loss has been present for a prolonged period. The patient's perception of this motor palsy is often that of weakness of grasp, particularly pinch, and a clumsiness and loss of dexterity in manipulating small objects. In our own clinical practice, motor weakness in carpal tunnel syndrome patients has been seen with less and less frequency. This is probably owing to a more widespread recognition of the classical symptom complex within the general medical community. As a result, patients are referred for treatment much earlier in the course of this disease than was previously the case.

Not all patients' symptoms involve the entire median nerve sensory distribution in the hand. Indeed, our own patients most commonly complain of symptoms in only a portion of the sensory area subserved by the median nerve—usually the long and index fingers or the long and ring fingers (ie, those areas served by one of the common digital nerves).

EXAMINATION

Sensory Disturbances

Sensory disturbances, as detected by alterations in appreciation of touch as well as abnormal static and moving two-point discrimination, should always be sought and any abnormality in distribution of normal sweating noted. The 30 and 256 CPS tuning forks are also useful. Objective evidence of sensory loss, however, is commonly not present as the modern-day patient will present for evaluation before such severe neural damage has occurred.

Tinel's Sign

Tinel's sign can be elicited in the majority of patients. Tapping with the finger or a reflex hammer over the course of the median nerve at the wrist, just proximal to the flexor retinaculum, will transiently reproduce the numbness and tingling of which the patient complains. Excessively firm percussion of the nerve may produce paresthesias even in normals. Simultaneous evaluation of the opposite wrist (if it is normal) may help identify such situations. The presence of Tinel's sign in patients with symptoms of carpal tunnel syndrome has been quite reliable confirmatory evidence in our experience and has been present in approximately 80 per cent of our patients. The absence of Tinel's sign does not, however,
rule out the presence of carpal tunnel syndrome.

**Phalen’s Wrist Flexion Test**

Phalen’s wrist flexion test is another helpful confirmatory sign. The wrist is placed in unforced but acute flexion for 60 seconds. This posture causes an increase in measured pressure within the carpal canal, particularly marked in carpal tunnel syndrome patients. The test is positive when maintenance of the flexed posture reproduces or exacerbates the patient’s symptoms of numbness or paresthesias. We have found this test to be positive in over 70 per cent of our carpal tunnel patients. Like the Tinel’s sign, absence of a positive Phalen’s test does not completely rule out carpal tunnel syndrome, but its presence is strong confirmatory evidence.

Motor involvement, as evidenced by muscle weakness or even obvious thenar atrophy, is not commonly noted in modern hand surgery practices. When present, however, it is best detected by viewing the thenar eminence in profile and comparing it with the asymptomatic hand. A flattening of the thenar eminence can sometimes be seen relatively early (the so-called shelf sign) and is due to selective atrophy of the abductor pollicis brevis muscle.

The abductor pollicis brevis is the thenar muscle that is most easily tested in terms of pure median motor function (cross-innervation of this particular unit by branches from another nerve is exceptionally rare). The patient places his or her hand on a flat surface, palm up, in the “military salute” position (adducted against the second metacarpal) and is asked to palmarly abduct the thumb against the resistance of the examiner’s finger. Contraction of the abductor pollicis brevis can be easily visualized, and strength can be compared with the opposite hand. Atrophy of the opponens pollicis and flexor pollicis brevis occurs relatively late in patients with median nerve compression.

Occasionally, muscle fasciculation can be seen in the thenar muscles and is a valuable sign because it is an early sign of motor involvement. Pulp-to-pulp pinch strength is also useful and should be measured and recorded. Direct evaluation of opposition is performed by the popular technique of having the patient place the tips of the thumb and either the ring or small finger together and resist attempts to break this pinch circle. This examination has seemed to us to be less reliable and less specific than the abductor pollicis brevis evaluation because the patient may use the adductor pollicis and ulnar-innervated portion of the flexor pollicis brevis and thus still be able to demonstrate considerable pinch power.

**ELECTRODIAGNOSIS**

Sophisticated electrodiagnostic techniques are available to assist in diagnosis of peripheral nerve compression syndromes, and their applications are most widely developed and best understood in relation to compression neuropathies of the median nerve at the wrist.

Slowing of sensory conduction across the wrist has proven to be the most sensitive of such tests, with prolongation of the distal sensory latency above 3.5 msec being present in 85 to 95 per cent of surgically confirmed cases. The motor analogue of slowed sensory conduction, a delayed distal motor latency, may also be measured but is a somewhat less sensitive index. Lastly, electromyographic evaluation of the thenar muscles may show signs of denervation, but only in more advanced cases where actual axonotmesis has already occurred.

We do not routinely use electrodiagnostic studies when evaluating patients for median nerve compression. Our experience has paralleled that of other authors in this regard. In patients with fairly typical symptoms and localizing wrist signs (positive Phalen’s test or Tinel’s sign), electrodiagnosis has not contributed additional information of clinical value. Patients with typical symptoms and confirmatory physical findings have uniformly gained symptomatic relief from surgical therapy even if electrodiagnosis has been interpreted as normal or minimally abnormal. Conversely, false-negative clinical...
diagnoses (patients with absent clinical findings but abnormal electrodagnosis) have not been encountered. Further, there has been no good correlation noted between the degree of electrodagnostic abnormalities and either the severity of symptoms or the amount of nerve abnormality noted at surgery.\(^{17,45}\) For these reasons, we do not routinely evaluate electrophysiologic parameters in patients with clinical presentation of carpal tunnel syndrome. Rather, we reserve such additional (and expensive) tests for patients with questionable diagnoses, those in whom the site of compression cannot be clinically localized with reasonable certainty or who are suspected of having multiple compression loci, and those whose symptoms may reflect systemic disease or generalized neural degenerative disease rather than a localized mechanical process. The latter situations can be readily suspected from a careful general history and physical examination and diagnosed by further appropriate blood tests. In the final analysis, the patient's own cerebral "computer" remains the most sensitive detector of early signs of nerve compression.

**TREATMENT**

Treatment of median nerve compression may be either surgical or nonsurgical. As experience accumulates, the indication for each modality becomes increasingly clear.

**Nonsurgical Treatment**

Nonsurgical treatment of carpal tunnel syndrome is most appropriate when symptoms are mild or intermittent, relatively short in duration, or when the inciting agent is expected to be transient in nature. The most common such situation is carpal tunnel syndrome of pregnancy, where symptoms are related to fluid retention and edema. Resolution of symptoms is almost universal following the end of the pregnancy, and all attempts should be made to alleviate the patient's discomfort with nonoperative means. These include splinting, local steroid injection, diuretics, and other means of edema control.

Patients whose symptoms appear to have been precipitated by a recent change in work habit can also often benefit by cessation or alteration of the provoking activity and nonsurgical therapy. Nonsurgical treatment modalities used include: (1) oral anti-inflammatory agents, (2) splinting and elevation, and (3) local steroid injection. Systemic administration of diuretics and steroid preparations has been described, but, in most cases, we have not thought that the use of such systematically potent agents was appropriate or advisable.

When consulted by a patient with mild, intermittent symptoms of median nerve compression at the wrist and confirmatory clinical findings, our general therapeutic approach is a graded one. We first try to determine if any particular occupational or recreational activity appears to be the inciting cause and, if so, recommend its temporary cessation. Simultaneously, the patient is fitted for a removable volar splint that extends from just proximal to the MP flexion crease to the proximal forearm and holds the wrist in neutral or slight dorsi-flexion. The ideal position of immobilization seems to be one in which the thumb metacarpal is slightly abducted and flexed. This position drops the nerve away from the transverse carpal ligament and makes the ligament less tense. The splint is worn at night (if the patient's symptoms occur only at night) and during the day, if indicated. Simultaneous trial of oral anti-inflammatory agents, such as salicylates or ibuprofen, may also be helpful. We formerly used stronger anti-inflammatory agents such as indomethacin, but now prefer to avoid them because of their potential for harmful side effects.

If nonoperative treatment is to be successful at all, clear-cut improvement or even relief of all symptoms will usually occur within 1 week. Patients who note no significant relief with splinting at 1 week will usually not benefit from persistence in such efforts. In patients who do not respond to this regimen, we next proceed to local steroid injection.

Steroid instillation into the carpal canal is
indicated in patients with intermittent symptoms and in whom objective findings such as loss of two-point discrimination or thenar atrophy are not present. It can also be used as a provocative test to confirm the diagnosis. Other relative indications for steroid injections include those patients in whom surgical therapy is contraindicated by either age or poor general medical condition. These latter situations should be quite rare, since the magnitude of the surgical treatment for carpal tunnel syndrome is modest in terms of systemic stress and can easily be carried out using local anesthesia if necessary.

Technique of Steroid Injection

A mixture of 0.5 mL of soluble steroid and 0.5 mL of Xylocaine is used. The injection is carried out at the level of the distal wrist crease with a short 25-gauge needle angled distally to enter the carpal tunnel. This site of injection is less painful than direct injection through the transverse carpal ligament. Our preferred skin puncture site is just ulnar to the palmaris longus tendon as it crosses the distal wrist flexion crease. Injection here places the needle ulnar to the median nerve but still locates the tip within the carpal tunnel. Injection of steroid preparations (or anything else) directly into the median nerve can be deleterious and should be avoided. If paresthias are elicited when the needle enters the canal, it should be withdrawn slightly and redirected prior to injection to avoid such complications. The injection itself may temporarily aggravate symptoms since a temporary increase in total volume content of the carpal canal results. As long as the needle itself elicits no paresthesias, one may be confident of the safety of proceeding with injection. Splinting is continued following steroid instillation.

We have noted relief of symptoms following carpal tunnel injection in approximately 40 per cent of our patients so treated, a figure somewhat higher than that usually quoted. This discrepancy may reflect the nature of our referral population: the majority of our patients are seen relatively early in the course of this disease. Of those who respond favorably, however, only about half (20 per cent) gain permanent relief. Patients whose symptoms are not relieved or who have recurrence of symptoms after steroid injection should be considered candidates for surgical intervention. We do not favor repeated steroid instillations because of the risk of tendon rupture or inadvertent nerve injury, and also because the great majority of patients who have recurrent symptoms postinjection will eventually require surgery anyway.

Surgical Treatment

Surgical intervention in carpal tunnel syndrome is indicated in patients in whom nonoperative treatment has failed, in patients who show evidence of thenar muscle weakness or atrophy, in patients with reproducible, objective abnormalities on sensory examination, and in almost all patients whose subjective symptoms are constant rather than intermittent. Some surgeons recommend a trial of conservative treatment for patients in these last two categories, but our own observations suggest that the great majority of these, too, will eventually require surgical management.

Acute Carpal Tunnel Syndrome. Symptoms of acute compression of the median nerve at the wrist have been reported subsequent to a number of traumatic situations, including wrist and forearm fractures, dislocation of the wrist, crush injuries, burns, high-pressure injection injuries, and in reperfusion following transient profound ischemia (as with revascularization). The symptoms and clinical findings are similar to those described for cases of carpal tunnel syndrome in general except that those findings of chronicity, particularly thenar muscle atrophy, are not present. Urgent decompression of the carpal canal (along with fasciotomies of any other involved compartment of the extremity) is indicated whenever post-traumatic acute carpal tunnel syndrome symptoms are more than transient and mild. When in doubt, it
is better to proceed with nerve decompression since the risk of permanent nerve damage seems greater in such patients.

SURGICAL TECHNIQUE FOR MEDIAN NERVE DECOMPRESSION

It can be said that the ideal operation for a given pathologic or pathophysiologic condition might be one that maximizes potential therapeutic benefit to the patient while minimizing potential complications and other adverse effects. The ideal operation for carpal tunnel syndrome should have as its goals: (1) the complete decompression of the median nerve and its branches, and (2) the avoidance of surgical complications including injury to the median nerve, its motor branch, its major and minor sensory branches, and its palmar cutaneous branch. Injury to the vascular structures in the area, namely the superficial palmar arch and its branches, must also be avoided.

The skin incision used in the ideal carpal tunnel operation should allow complete exposure of the transverse carpal ligament and carpal canal, should be anatomically placed to avoid major cutaneous nerve injury, should avoid trespassing on flexion creases, and should heal kindly with a good cosmetic result. The surgical approach carried out through this incision should be adaptable to concomitant procedures or unexpected findings, should permit complete visualization of the structures at risk, and should be technically simple. When necessary, it should permit proximal or distal extension. In our hands, the operative approach to be described comes closer than any other to fulfilling these ideal criteria.*

Anesthesia

General anesthesia, regional block (either axillary or intravenous), and local infiltration anesthesia are all equally effective. Most of our patients undergo surgery as outpatients and we have used regional or local anesthesia almost exclusively, depending upon patient preference. A proximal hemostatic tourniquet must always be used to permit the precise dissection and identification of anatomic structures, which is the key to complication-free surgery.

Preoperative Markings

Taleisnik has demonstrated the course of the palmar cutaneous branch of the median nerve and the proper positioning of palmar incisions to avoid damaging it. Neuromas of the palmar cutaneous branch can be markedly symptomatic and disabling; the key to their avoidance is proper incision placement. A mark is drawn on the palmar skin directly in line with the fourth metacarpal ray and is extended proximally to the distal wrist crease (Fig. 1). The trunk of the palmar cutaneous nerve, in its course between the thenar and hypothenar eminences, usually lies well radially to this line. Incisions in this area should, therefore, preferentially be kept ulnar to Taleisnik's line.

We prefer an incision parallel to the thenar crease which, as it crosses the proximal palm, is kept ulnar to the line of the fourth metacarpal ray, and which then curves toward the ulna around the hook of the hamate, terminating at the distal wrist crease. In the routine case, it is unnecessary to incise proximal to this crease. Such an incision permits complete exposure of the transverse carpal ligament, allows direct visualization of all structures at risk during division of the transverse carpal ligament, avoids major cutaneous nerve trunks, and follows normal anatomic creases, thereby permitting favorable healing and minimal scarring. It is important that the incision curve around the hook of the hamate rather than passing directly over it (such a scar location tends to cause prolonged local postoperative tenderness, probably because of its positioning directly over bone at a frequently traumatized site). It is fair to note

*This particular operative technique was first demonstrated to one of the authors by Robert Horner, M.D., in 1975. With minor changes, it has been used continuously by both authors since 1975.
Frederick R. Heckler and Michael E. Jabaley

Figure 1. Incision design for carpal tunnel decompression. A line is drawn along the axis of the fourth metacarpal ray. An incision parallel to the thenar crease, which passes ulnar to the line of the fourth metacarpal ray, is outlined in the interthenar region, curving around the hook of the hamate and terminating at the distal wrist crease. Note that both the ulnar nerve in Guyon's canal and the median nerve in the carpal tunnel are easily accessible through this incision. Extension of the incision more proximally onto the forearm is unnecessary in most cases as the entire length of the transverse carpal ligament underlies the incision, which terminates at the distal wrist crease. The palmar cutaneous branch lies radial to the main trunk of the median nerve and is avoided by this particular incision placement.

that even this incision will divide some of the more distal filaments of both palmar and ulnar cutaneous nerves, but these seem not to cause clinically evident neuromas or pain problems as long as the more proximal trunk of the palmar cutaneous nerve has been avoided.

Extension of the incision proximal to the distal wrist crease should be avoided, as this area regularly heals with a thicker, more prominent scar than incisions confined to palmar skin. We extend the incision proximally only as required by local pathologic states encountered, and never for the usual, standard carpal tunnel release. When proximal extension of the incision is necessary, it is best done in zigzag fashion.

Operative Technique

The incision is designed as noted previously. The arm is exsanguinated with an Esmarch bandage and the hemostatic tourniquet inflated to 75 to 100 mm above the systolic blood pressure (Fig. 2). The skin is incised and the palmar fascia visualized without raising medial and lateral skin flaps, as this would endanger palmar cutaneous branches that are located in the extrafascial subcutaneous layer at this level. The palmar fascia is sharply incised from distal to proximal, stopping when the thicker distal edge of the transverse carpal ligament is reached. This incision is facilitated by tenting the palmar fascia up and away from the underlying structures using hooks in the skin edges. The edge of the palmar fascia can be protected for later repair (see description of closure for details).

Dissection is initiated distally in the palm at the level of the superficial volar arch and continued proximally toward and through the carpal canal. We prefer this approach for two reasons: (1) dissection and identification of the various structures at risk can be done prior to entering the tight confines of the carpal tunnel, and (2) this preliminary dissection can be performed by simple scissors spreading and retracting in the soft areolar and fatty palmar tissues. Since sharp dissection is unnecessary, this approach affords protection against inadvertent injury to poorly visualized or anatomically aberrant structures.

The major concern, of course, is for iatrogenic injury to normal or anatomically variant structures, including the superficial palmar vascular arch, cross-over connections between ulnar and median nerve branches, and most importantly, the motor branch of the median nerve. Many variations of this latter structure have been described, both in terms of its site of origin.
Evolving Concepts of Median Nerve Decompression

...and its course. For this reason, it is essential to visualize the motor branch during this dissection, and the easiest and most consistent place to do this is in the palm at the site where the nerve approaches and enters the thenar muscles. This site, just distal to the distal edge of the transverse carpal ligament, is quite consistent, and most motor branches, regardless of site of origin or variations in course, can be located here. Even if one of the rare situations is encountered where the motor branch enters the thenar muscles at another point, the surgeon will at least be alerted to this situation prior to beginning dissection in the tight carpal canal where visualization is more difficult.

After incision of the palmar fascia distal to the ligament, the first structure to be located is the superficial vascular arch. Gentle, blunt spreading with scissors in the palmar fat allows easy visualization of the arch and its common digital branch to the ring-long finger interspace. This latter structure in turn serves as a handy guide to the common digital branch of the median nerve to these same fingers lying just adjacent to it. Most commonly, this is the ulnarnest median nerve, and dissection can easily be continued proximally, again by spreading the palmar fat. Since no sharp dissection is required, risk to local structures is minimal. Regardless, proximal dissection should be done under direct vision since branches from the ulnar nerve to the median digital nerves are sometimes present and must not be injured.

As dissection proceeds proximally, the distal edge of the transverse carpal ligament is encountered. Simultaneously, it can be noted that the main median trunk has been reached.

Division of the transverse carpal ligament is not initiated yet. Rather, dissection is carried in a radial direction, again using only blunt separation of fatty perineural tissues, and the motor median branch is specifically identified. Identification of the motor branch is facilitated by placing traction on the thenar edge of the skin incision at right angles to the palmar surface, a maneuver that makes the motor branch stand out in its course to the thenar muscles. Once identified at its site of muscular entry, the origin of the motor branch from the main trunk, normal or variant, is easily noted.

At this relatively early point in the operation, the surgeon has identified and can easily protect all important structures at risk and has done so prior to initiating sharp division of the transverse carpal ligament. Division of the ligament can now proceed with relative assurance. We prefer to insert a hemostat between the ligament and the median nerve, visualize the exposed segment of nerve, and then incise that portion of the median nerve until complete division of the ligament is accomplished. In addition, the antebraehial fascia is incised for a short distance proximal to the wrist crease. This latter maneuver can be accomplished by elevating rather than cutting the overlying skin and fat, thereby avoiding injury to cutaneous nerve branches contained therein.

This particular incision easily allows simultaneous evaluation and release of Guyon's canal, if indicated. This passageway is also most easily located in retrograde fashion by tracing the vessels of the superficial palmar arch back toward the ulnar artery into Guyon's canal. Here, the ulnar nerve and artery can be directly visualized and decompressed, if necessary.

Closure of the incision is in one layer using nonabsorbable sutures in the skin. The divided transverse carpal ligament is not sutured or reapproximated, but there seems to be a good reason for suturing the overlying palmar fascia. The transverse carpal ligament holds the pillars of the hand together to form the proximal transverse arch, and an effort should be made to preserve this arch.

Intraneural Neurolysis

This procedure has been advocated as an adjunct to carpal tunnel decompression. It involves epineurotomy with dissection and lysis of fibrosis between fascicles and fascicular groups. Loupe or microscopic magnification is required and the latter is preferred. Such extensive dissection carries its...
Figure 2. Operative technique.

A. The operative incision has been designed as noted in Figure 1. The anticipated positions of the transverse carpal ligament, the median nerve, and the palmar cutaneous branch are also outlined. Most compressions of the median nerve occur beneath the distal portion of the ligament (that area which lies in the center of this incision). B. The skin incision is made and carried down through the subcutaneous fat to the palmar fascia. This fascia is tented up with hooks and then incised. C. The common digital nerve leading to the long–ring finger interspace is easily located by a blunt spread technique in the loose palmar fat. Dissection will be continued proximally along the ulnar side of this common digital branch until the thickened distal edge of the transverse carpal ligament is reached. Note that no sharp dissection is required in this area, thereby minimizing risk to vital structures during the dissection. D. Before beginning dissection into the tight confines of the carpal tunnel, blunt dissection is next immediately carried to the radial side of the main trunk of the median nerve. Traction upward on the thenar edge of the skin incision will allow easy localization of the motor branch of the median nerve (here demonstrated with the curved hemostat). Having visualized this structure as well as the main trunk of the median nerve, the common digital nerve branches, and the superficial palmar vascular arch and its branches, all vital structures at risk have now been seen and can easily be protected. E. The surgeon next proceeds with relative assurance into the tight confines of the carpal tunnel, incising the transverse carpal ligament from distal to proximal. This is done by placing a hemostat between the median nerve and the transverse carpal ligament and incising the ligament by proceeding proximally along the ulnar border of the median nerve. F. A few centimeters of antebrachial fascia are incised subcutaneously proximal to the distal wrist crease by elevating the overlying skin and fat.
Figure 2. G, The completed dissection, showing the wide exposure of the median nerve and all other regional structures including the common digital branches, the thenar motor branch, and the median main trunk. Note that this incision gives wide access to all structures of concern without extension proximal to the wrist crease. H, Guyon's canal is easily explored through the same incision by tracing the superficial palmar vascular arch vessels proximally toward the ulnar artery. The tip of the hemostat is entering Guyon's canal. I, A different patient, viewed from the thenar side (fingers to the right, forearm to the left), in whom carpal tunnel release as well as release of Guyon's canal has been carried out. Note the ease of total exposure of both structures. The probe is in Guyon's canal, which is being decompressed. J, A typical postoperative result at 3 months. As long as the incision is confined to the palm and follows natural crease lines, an excellent cosmetic result is expected.
own risks of both direct mechanical nerve injury and additional postoperative scarring owing to the dissection itself, and one must always be sure that the potential benefit outweighs the risk before undertaking internal neurolysis.

Indications for internal neurolysis are limited to those conditions in which there is scarring or fibrosis of the external and/or internal epineurium. These conditions produce constriction within the nerve, which is unlikely to be relieved by ligament division alone. Indications include: (1) muscle atrophy, (2) fixed sensory loss, (3) severe causalgia, (4) previous unsuccessful operation, or (5) a history, such as crush fracture, suggesting that neurolysis will be necessary. It can be seen from the foregoing that internal neurolysis is indicated only in the more advanced cases. When it is indicated, we do not carry out complete internal neurolysis, but rather approach only the involved fascicles. An example would be a patient who has a fixed sensory loss with abnormal two-point discrimination in the long finger, but only intermittent symptoms in the remainder of the median nerve distribution. In such a situation, the involved nerve branch is located distally and then its fascicles traced proximally through the main trunk of the nerve in the carpal canal. The remainder of the nerve is not disturbed, thereby limiting the risk of postdissection fibrosis or injury to intraneural blood vessels.

POSTOPERATIVE CARE

At the completion of wound closure, a volar splint is applied that extends from just proximal to the MP flexion crease to the proximal forearm and that holds the wrist in slight extension and the thumb in opposition. This posture is maintained for 2 weeks and for a third week at night only. A flexed posture of the wrist must be avoided during the initial healing phase to prevent prolapse of the contents of the carpal canal through the open transverse carpal ligament. Active finger flexion and thumb motion are initiated within 24 hours of surgery and encouraged throughout the recuperative course. Sutures are removed in 6 to 9 days. Patients are advised to avoid strenuous activity for 6 weeks. There seems to be no problem in resuming activities that involve fingertip opposition, but the gripping of objects, such as hammer handles or pliers, may be painful for several weeks.

It is important to recognize that recovery from median nerve compression is based on the pathologic condition of the nerve and does not always occur at the same rate of speed. If the changes in the nerve are biochemical and not structural (neurapraxia), then recovery will be prompt and may be noticeable in a matter of days or weeks. Conversely, if axonotmesis is present and a segmental demyelination has occurred, the patient should be told that recovery may require 6 months or longer. In either case, patients should be aware that gradual improvement may occur for 2 to 3 years and subtle improvement should be anticipated for at least that long. It should be borne in mind that not all fibers or fascicles are equally injured and not all recover at the same rate or to the same degree.

The rehabilitation process can be helped by early initiation of range-of-motion exercises, edema control, and scar massage. Strengthening exercises should be added when pain and early inflammation have subsided.

SPECIAL SITUATIONS

The motor branch of the median nerve may occasionally traverse the transverse carpal ligament independently in a separate fibrous canal, and the palmar cutaneous nerve commonly does this. For this reason, it is possible to have an isolated compression neuropathy of either of these nerves, either as part of or completely independent of the carpal tunnel syndrome. Specific exploration and, if necessary, decompression of each respective nerve should be carried out when isolated thenar weakness is the presenting symptom, when thenar weakness is present in any carpal tunnel patient, and
CONCLUSIONS

Carpal tunnel decompression has become one of the most commonly performed hand operations and one of the most successful. When diagnosis is made relatively early, improvement or complete relief of symptoms is the rule. Complications are infrequent, and few reports in the literature offer analyses of postoperative problems. Such complications, when they do occur, can be catastrophic, for they involve important sensory or motor branches of the median nerve. The absence of large reported series of complications does not necessarily mean that they do not occur. Several patients have been referred to us over a 10-year period with incorrectly placed incisions and postoperative palmar cutaneous nerve neuromas. Two patients have been seen with surgical injuries to the median motor branch, and we are aware of three patients with major hematomas requiring reoperation following laceration of the palmar vessels during carpal tunnel release. Finally, we have reoperated on a number of patients with incompletely divided ligaments, a problem that may be the result of blind ligament division through a transverse wrist incision.

We strongly believe that the key to avoidance of such problems is to approach surgery of the carpal tunnel as a nerve dissection and decompression rather than as a ligament release. Such a change in mind set will engender the modest adjustments in technique requisite to complication-free nerve surgery. The requisites to such surgery are complete visualization and subsequent protection of all structures at risk. Only some forms of longitudinal incision will allow such visualization, and the previously popular transverse wrist incision is mentioned only to be condemned.

The surgical approach described has proven to be easy to use, easy to teach, and reliable. No originality is claimed for either the incision or the surgical approach described, but we do think that their merits have not received adequate emphasis.

REFERENCES


