NERVE AND TENDON GLIDING EXERCISES AND THE CONSERVATIVE MANAGEMENT OF CARPAL TUNNEL SYNDROME

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Nerve and Tendon Gliding Exercises and the Conservative Management of Carpal Tunnel Syndrome

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ABSTRACT: While developments continue in the surgical management of carpal tunnel syndrome, little emphasis has been placed on the evaluation of a comprehensive non-surgical treatment. In this study, 197 patients (240 hands) presenting for treatment of carpal tunnel syndrome were divided into two groups. Patients in both groups were treated by standard conservative methods, and those in one group were also treated with a program of nerve and tendon gliding exercises. Of those who did not perform the nerve and tendon gliding exercises, 71.2% underwent surgery compared with only 43.0% of patients who did perform them. Patients in the experimental group who did not undergo surgery were interviewed at an average follow-up time of 23 months (range, 14–38 months). Of these 53 patients, 47 (89%) responded to this detailed interview. Of the 47 who responded, 70.2% reported good or excellent results, 19.2% remained symptomatic, and 10.6% were non-compliant. Thus, a significant number of patients who would otherwise have undergone surgery for failure of traditional conservative treatment were spared the surgical morbidity of a carpal tunnel release ($p = 0.0001$).
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Carpal tunnel syndrome (CTS) has reached epidemic proportions. It is estimated that release of the transverse carpal ligament is among the ten most commonly performed operations in the United States, resulting in a significant contribution to national health care costs.[1] These figures become staggering when the hidden costs are considered, including time off work, lost wages, and diminished workplace productivity.[2] The current non-surgical regimens designed to manage carpal tunnel syndrome—including splinting, non-steroidal anti-inflammatory medication, steroid injections,[3] and ergonomic workstation modification—are frequently ineffective, often leading to recommendation for surgical treatment.[4]

Even following surgery, results may be less than satisfactory. There may be significant postoperative morbidity due to hypersensitive scar, debilitating pain in the thenar and hypothenar eminences, loss of grip strength which may be prolonged, and recurrence of symptoms on return to work.[5] Dividing the transverse carpal ligament alters the mechanism of the thenar and hypothenar muscles and may cause bow-stringing of the flexor tendons.[6] In an effort to minimize these effects, endoscopic carpal tunnel release has been developed and proclaimed to be a major advance.[7] However, reports have described major surgical complications, and further study is required to assess the true role of endoscopy in the management of carpal tunnel syndrome.[8]

In order to improve the efficacy of non-surgical treatment for carpal tunnel syndrome, development of a supplemental approach to standard conservative treatment was needed. To formulate this new approach it was decided to reconsider the "static model" of compression neuropathy that currently guides most non-surgical treatment decisions.

Studies by Wilgis and Murphy[9] and by McLellan and Swash[10] have described the normal longitudinal 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.[9] Extension of the fingers with extension of the wrist is the position in which the median nerve is displaced farthest under the transverse carpal ligament into the hand. It has also been demonstrated by McLellan and Swash[10] that hyperextension of the wrist causes the median nerve to slide distally by 10–15 mm relative to a fixed bony landmark in the carpal tunnel. Displacement of the median nerve during flexion of wrist and fingers is two to

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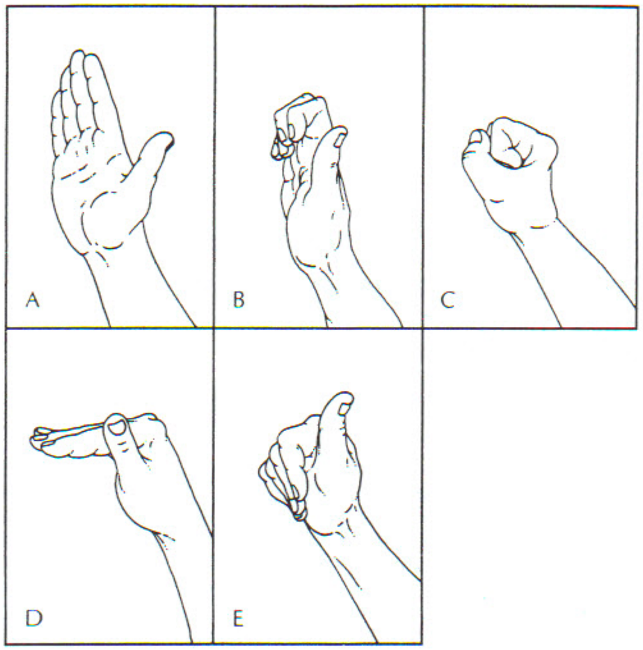

FIGURE 1. The five discrete positions in which fingers are placed in tendon gliding exercises: A, *straight;* B, *hook;* C, *fist;* D, *tabletop; and* E, *straight fist.*

four times greater at the wrist than in the upper arm. Active and passive extension of the wrist and fingers causes the nerve to move approximately 11 mm distally. Flexion of the wrist and fingers moves the nerve 4 mm proximally.[10] Longitudinal sliding prevents local stretching of the median nerve that would otherwise occur during wrist and finger movement.[11]

Szabo et al.[12] showed the differences between median nerve and digital flexor tendon excursion in the carpal tunnel and the linear relationship that exists between median nerve displacement and that of the flexor tendons.[12] This would result in frictional and shear forces between the tendons and the nerve with normal finger and wrist motion. It is conceivable that with chronic repetitive use of the fingers, shearing may result in localized hyperplasia and fibrosis of the investing tenosynovium around the median nerve and flexor tendons in the tunnel. This would retard movement of the median nerve and the flexor tendons in the tunnel, resulting in disturbance of the normal gliding between structures in the carpal tunnel. Ochoa and Marrott[13] felt that the hourglass deformity resulting from displacement of axoplasm and telescoping of myelin away from the center of the compression is due to shear forces.[13] This cannot be explained on the basis of a static model of compression, as shear stresses are evident only in a dynamic model. How is shear generated using a static model?

Armstrong et al.,[14] in a study of normal cadaveric hands, demonstrated that in the central region of the carpal tunnel, presumably at the point of maximal compression, there is an increase in the density of synovium and connective tissue adjacent to the median nerve, associated with thickening of the epineurium. There is also arteriolar and venular smooth-muscle hypertrophy and endothelial thickening. Even "normal hands" seem to experience some degree of reactive change within the carpal tunnel. Exaggeration of these reactive changes could dramatically increase the pressure on the median nerve and its vascular supply, perhaps causing symptoms of compression neuropathy.[14] These histologic changes have been corroborated in clinical observations.[15–18]

Lundborg and Rydevik[20] and Sunderland[21] indicated that stretching of a nerve might damage the perineurium and affect its permeability. With chronic hypoxia, leakage of proteins and inflammatory cells into the fascicles from injured extraneural and epineural blood vessels leads to fibrosis of the nerve. With tension-creating elongation of the nerve of as little as 7%, the cross-sectional area of the nerve fascicle is reduced, causing an increase in intra-fascicular pressure and decreased intra-fascicular blood flow, especially in epineural and perineural venules.[20,21] Lundborg et al.[22] also described a "miniature compartment syndrome" occurring inside the protective perineural sheaths because of venous outflow obstruction within the nerve. They described this phenomenon as being secondary to chronic pressure and perineural fibrosis and resulting in localized neural ischemia.

Sunderland and Bradley[23] indicated that even small increments of stretch on a nerve could seriously impede its normal electrical properties. LaBan et al.[24] described the tethered median nerve stress test for chronic carpal tunnel syndrome, indicating that when the disease is chronic, the median nerve becomes tethered in place in the carpal tunnel and that by hyperextending the wrist as well as the index finger, one can reproduce symptoms of neuropathy. It is thus postulated that the inability of the nerve to glide through its normal path could result in traction injury.

A study by Seradge et al.[25] demonstrated that intermittent active wrist and digital flexion and extension exercise reduces the pressure in the carpal tunnel. We hypothesized that it might be possible to affect the clinical course of CTS in at least some patients by using a specific series of exercises. Postulating that adhesions to the median nerve exist preoperatively, we proposed a regimen that actively forces the median nerve and the flexor tendons to their maximal excursion through the carpal tunnel. Totten and Hunter[26] have described a program for postoperative nerve gliding, which is used to minimize scar adhesions and maximize nerve excursion through the carpal canal, and have reported that the program is effective in the management of postoperative carpal tunnel syndrome, with relief of pain and low recurrence rates. There is, however, only brief reference to the use of this program as part of a comprehensive non-surgical conservative regimen. We postulated that by employing these nerve and tendon gliding exercises, symptom resolution could be affected by 1) stretching the adhesions in the carpal canal, 2) broadening the longitudinal area of contact between the median nerve and the transverse carpal ligament, 3) reducing tenosynovial edema by a "milking action," 4) improving venous return from the nerve bundles, and 5) reducing pressure inside the carpal tunnel. The objective of this study was to ascertain whether symptom resolution could be effected in patients with carpal tunnel syndrome who would otherwise have probably required surgical intervention.

MATERIALS AND METHODS

This investigation of non-surgical management of carpal tunnel syndrome included 197 patients (240 hands) seen between 1988 and 1993. There were 43 patients with bilateral symptoms and 154 patients with unilateral symptoms. All patients presented with symptoms of median nerve compression. Symptoms included volar wrist pain that tended to be more pronounced at night, numbness in either the median nerve distribution or all five digits, dropping of objects, and loss of dexterity. Physical examination of each patient included tests for Tinel's and Phalen's signs as well as two-point discrimination and Semmes-Weinstein threshold tests. Patients who went on to have surgery were treated conservatively for an average of 4 months (range, 1–6 months). There was wide variation in the duration of symptoms and treatment prior to presentation to our clinic (see Tables 1 and 2).

The patients were divided into two study groups: 1) a control group of patients presenting between 1988 and 1991, and 2) an experimental group of patients presenting from 1992 through 1993. Patients who had obvious thenar atrophy, pregnancy-related CTS, or recurrent CTS following a previous carpal tunnel release were excluded from the study. Patients with obvious thenar atrophy were excluded because in most cases surgical release is recommended, and symptoms in patients due to pregnancy may resolve following delivery. Patients who had previously undergone carpal tunnel release were excluded because the goal of this study was to examine the role of the nerve and tendon gliding exercises on patients who had not yet undergone carpal tunnel surgery. It should be noted that patients with other underlying disease (e.g., rheumatoid arthritis or diabetes) were not excluded from the study. In both the experimental and control groups, similar percentages of patients received the traditional treatments of splinting, non-steroidal anti-inflammatory medication, and steroid injections. In addition to these traditional treatments, the experimental group was instructed in the series of nerve and tendon gliding exercises developed by Totten and Hunter.

The exercises were designed to maximize the excursion of the digital flexors and the median nerve through the carpal tunnel. In tendon gliding exercises the fingers are placed in five discrete positions (Figure 1)—straight, hook, fist, tabletop, and straight fist. In addition, the median nerve is mobilized by putting the hand and wrist through six

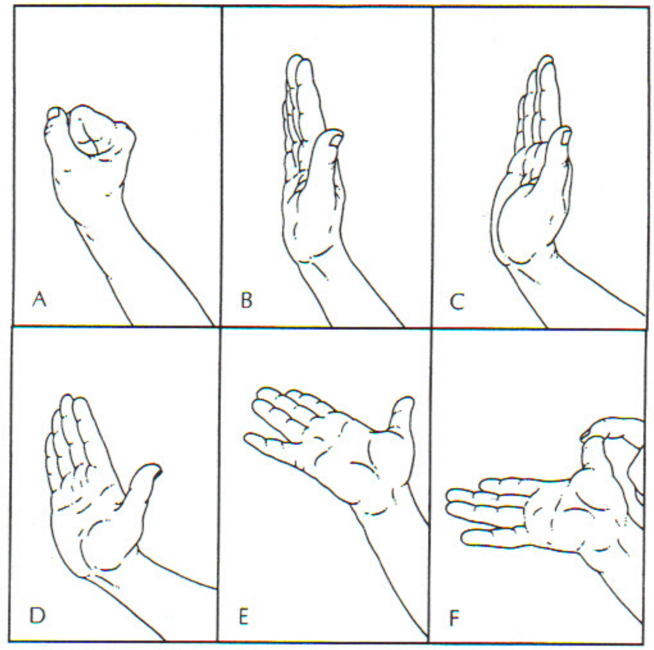

FIGURE 2. *Six positions for mobilizing the median nerve.* A, *The wrist is in neutral, with the finger and thumb in flexion.* B, *The wrist is in neutral, with the fingers and thumb extended.* C, *The wrist and fingers are extended, with the thumb in neutral.* D, *The wrist, fingers, and thumb are extended.* E, *The wrist, fingers, and thumb are extended, with the forearm in supination.* F, *The wrist, fingers and thumb are extended, the forearm is in supination, and the other hand gently stretches the thumb.*

additional positions (Figure 2). Each position was maintained for 7 seconds and repeated five times at each session, with a total of three to five sessions per day. In conjunction with the exercises, patients were also encouraged to perform contrast baths at least twice a day (optimally, three to five times a day). The patient would keep the hand in warm water for 4 minutes and then in cold water for 1 minute. Heat has been shown to increase localized blood flow by vasodilation and also increases connective tissue extensibility, facilitating the gliding of the nerve and tendons. Cold produces analgesia, reducing inflammation and edema.[27]

Both groups of patients, the control (n = 104) and the experimental (n = 93), were matched with respect to gender, age, and duration of symptoms (Table 1). The only difference between the control and experimental groups was occupation, the proportion of clerical and white collar workers being significantly greater in the experimental group and the number of manual laborers significantly greater in the control group ($p = 0.001$). A comparison of all patients who underwent surgery and all patients who did not have surgery was also performed (Table 2). There was no difference in the demographic breakdown (i.e., gender, age, occupation, and duration of symptoms) for these patients.

Both groups were initially followed for an average of 4 months, and those patients in the experimental group who did not undergo surgery were followed for an average of 23 months (range,

TABLE 1. **Demographic Features of Patients in the Control and Experimental Groups (N = 197)**

	Control (n = 104)		Experimental (n = 93)		Significance of Difference
	No.	(%)	No.	(%)	
Gender:					
Male	29	(27.9)	21	(22.6)	
Female	75	(72.1)	72	(77.4)	$p = 0.49$
Age (years):					
<30	5	(4.8)	8	(8.6)	
30–39	38	(36.5)	31	(33.3)	
40–49	33	(31.7)	29	(31.2)	
50+	28	(27.0)	25	(26.9)	$p = 0.75$
Duration of symptoms:					
<6 mo	32	(30.8)	27	(29.0)	
6–12 mo	17	(16.4)	19	(20.4)	
1–2 yr	26	(25.0)	18	(19.4)	
2+ yr	25	(24.0)	26	(28.0)	
Unknown	4	(3.8)	3	(3.2)	$p = 0.82$
Type of work:					
Clerical/white collar	44	(42.3)	65	(69.9)	
Manual	40	(38.5)	15	(16.1)	
Homemaker/retired	16	(15.4)	11	(11.8)	
Unknown	4	(3.8)	2	(2.2)	$p = 0.001$

TABLE 2. **Demographic Features of Patients in the Surgery and No-surgery Groups (N = 197)**

	Surgery (n = 114)		No Surgery (n = 83)		Significance of Difference
	No.	(%)	No.	(%)	
Gender:					
Male	26	(22.8)	24	(28.9)	
Female	88	(77.2)	59	(71.1)	$p = 0.42$
Age (years):					
<30	7	(6.1)	6	(7.2)	
30–39	42	(36.8)	27	(32.6)	
40–49	32	(28.1)	30	(36.1)	
50+	33	(29.0)	20	(24.1)	$p = 0.62$
Duration of symptoms:					
<6 mo	30	(26.3)	29	(34.9)	
6–12 mo	17	(14.9)	19	(22.9)	
1–2 yr	26	(22.8)	18	(21.7)	
2+ yr	36	(31.6)	15	(18.1)	
Unknown	5	(4.4)	2	(2.4)	$p = 0.15$
Type of work:					
Clerical/white collar	57	(50.0)	52	(62.7)	
Manual	32	(28.1)	23	(27.7)	
Homemaker/retired	20	(17.5)	7	(8.4)	
Unknown	5	(4.4)	1	(1.2)	$p = 0.12$

14–38 months). Of the 53 patients in the experimental group who did not undergo surgery, 47 (89%) responded to a detailed telephone interview. The patients in the control group who did not undergo surgery were not followed long-term. Patients were asked about their symptoms, level of function, and whether they would consider surgery if the physician recommended surgical intervention as an alternative. Results were considered "excellent" when a patient was completely asymptomatic and "good" when a patient had occasional symptoms that were relieved by reinstating the exercise program. Results were rated "fair" when a patient had frequent symptoms but still obtained some improvement by reinstating the exercise program. When a patient had continuous symptoms that did not respond to reinstating the exercise program, the results were considered "poor."

In evaluating the results of this study, all data were taken from the patient's records. Because this was a retrospective study, in some instances certain information was not recorded. All signs, symptoms, and treatments that were not specifically noted in the records were considered not present or not performed.

Patient demographics are reported for the total number of patients (N = 197), not the total number of involved hands, whereas clinical signs and treatments are reported for the total number of involved hands (N = 240).

The primary endpoint of this study was surgery. The decision to operate was based on several factors, specifically, 1) patient's perception that conservative treatment had not altered their symptoms to any great extent, and 2) the absence of improvement in clinical signs. All evaluations and surgery were performed by the primary surgeon. The difference in the proportions between the experimental and control groups, on the one hand, and the surgery and non-surgery groups, on the other, were evaluated using a *chi*-square test for significance.[28] In the categories where there were found to be significant differences, *chi*-square analysis was also performed with the unknowns omitted. This was done because of the large number of unknowns in some categories.

RESULTS

In examining the overall effectiveness of nerve and tendon gliding exercises in the conservative treatment of carpal tunnel syndrome, it is worth noting that only 43.0% of patients in the experimental group underwent surgical release compared with 71.2% of patients in the control group (Table 3). This represents a significant decrease in the number of patients who went on to surgery (p = 0.0001).

Tables 1 and 2 show that age and duration of symptoms do not appear to be significant determining factors in whether the patient was more likely to require surgery in either the control or the experimental group. When demographic data were analyzed according to occupation, a significantly higher percentage of manual laborers were found

TABLE 3. **Number of Patients Having Surgery in the Control and Experimental Groups (N = 197)**

	Control		Experimental		Total No. of Patients	Significance of Difference
	No.	(%)	No.	(%)		
Surgery	74	(71.2)	40	(43.0)	114	
No surgery	30	(28.8)	53	(57.0)	83	
Total patients	104	(100.0)	93	(100.0)	197	$p = 0.0001$

in the control group compared with the experimental group. However, when surgery patients were compared with non-surgery patients (Table 2), clerical workers were found to be no more likely to require surgery than were manual workers.

Table 4 shows that there was no significant difference between the control group and experimental group with regard to results of testing for Phalen's sign, Tinel's sign, and two-point discrimination. More than 80% of affected hands exhibited a positive Phalen's sign, approximately 70% had a positive Tinel's sign, and 14% had abnormal two-point discrimination. Table 5 shows that there was no difference in Semmes–Weinstein monofilament test results between the two groups when the unknowns were omitted from the analysis ($p = 0.13$).

TABLE 4. **Physical Findings, Treatment Regimens, and Results of Electrodiagnostic Tests on Hands in the Control and Experimental Groups (N = 240)**

	Control (n = 124)		*Experimental (n = 116)*		*Significance of Difference*
	No.	*(%)*	*No.*	*(%)*	
Physical Findings					
Phalen's sign:					
Positive	105	(84.7)	94	(81.0)	
Negative	14	(11.3)	21	(18.1)	
Not available	5	(4.0)	1	(0.9)	$p = 0.11$
Tinel's sign:					
Positive	81	(65.3)	86	(74.1)	
Negative	37	(29.8)	27	(23.3)	
Not available	6	(4.9)	3	(2.6)	$p = 0.29$
Two-point discrimination test:					
Positive	18	(14.5)	17	(14.7)	
Negative	99	(79.8)	84	(72.4)	
Not available	7	(5.7)	15	(12.9)	$p = 0.14$
Semmes–Weinstein threshold test:					
Positive	26	(21.0)	41	(35.3)	
Negative	49	(39.5)	45	(38.8)	
Not available	49	(39.5)	30	(25.9)	$p = 0.02$*
Treatment Regimens					
Splinting:					
Positive	114	(91.9)	116	(100.0)	
Negative	0		0		
Not available	10	(8.1)	0		$p = 0.01$*
Cortisone injection:					
Positive	63	(50.8)	51	(44.0)	
Negative	61	(49.2)	65	(56.0)	$p = 0.35$
Non-steroidal anti-inflammatory medication:					
Positive	101	(81.5)	102	(87.9)	
Negative	10	(8.0)	9	(7.8)	
Not available	13	(10.5)	5	(4.3)	$p = 0.19$
Electrodiagnostic Tests					
Electromyography:					
Positive	42	(33.9)	28	(24.1)	
Negative	65	(52.4)	47	(40.5)	
Not available	17	(13.7)	41	(35.4)	$p = 0.001$*
Nerve conduction:					
Positive	94	(75.8)	65	(56.0)	
Negative	17	(13.7)	15	(12.9)	
Not available	13	(10.5)	36	(31.1)	$p = 0.003$*

*See Table 5 for these results when unknowns were omitted.

TABLE 5. **Adjusted Values for Specific Findings for Hands in the Control and Experimental Groups after the Omission of Unknowns**

	Control		*Experimental*		*Significance of Difference*
	No.	*(%)*	*No.*	*(%)*	
Semmes–Weinstein threshold test:					
Positive	26	(34.7)	41	(47.7)	
Negative	49	(65.3)	45	(52.3)	$p = 0.13$
Splinting:					
Positive	114	(100.0)	116	(100.0)	
Electromyography:					
Positive	42	(39.3)	28	(37.3)	
Negative	65	(60.7)	47	(62.7)	$p = 0.92$
Nerve condition:					
Positive	94	(84.7)	65	(81.2)	
Negative	17	(15.3)	15	(18.8)	$p = 0.53$

There were, however, a large number of unknowns for this clinical sign (32% of the total number of hands) which, when included in the analysis, resulted in a significant difference between the two groups ($p = 0.02$). This is also true of the two diagnostic tests: 24% of hands did not have electromyographic tests and 20% did not have nerve conduction tests, and when these hands were excluded from the analysis, the difference was not significant —i.e., for electromyography $p = 0.92$, and for nerve conduction $p = 0.53$ (Table 5). When the unknowns were included, approximately 66% of affected hands (76% in the control group compared with 56% in the experimental group) had nerve conduction findings that documented CTS, and 29% (34% in the control group compared with 24% in the experimental group) had a positive electromyographic findings. This represents a significant difference ($p = 0.0001$) between the two groups for both these tests. This difference is due to the large number that were not tested, especially in the experimental group. The reason a large number were not tested (58 for electromyography, 49 for nerve conduction) was that the tests were ordered only if a patient failed conservative treatment and surgery was considered. In addition, a significant number of false-negative electromyographic and nerve conduction results have been reported in the literature.[29] In certain cases in our study, these tests were ordered by a referring physician prior to the patient's presentation for treatment.

Table 4 also compares traditional conservative treatments. Nearly all patients' hands were splinted, half had cortisone injections, and most were given non-steroidal anti-inflammatory medication. No significant difference was found between the two groups in use of anti-inflammatory medication or local cortisone injection. There was a significant difference for splinting ($p = 0.1$); however, when the unknowns (ten hands) were omitted from the analysis, there was no difference between the two groups (Table 5). It is probable that splinting was performed on all hands, but for ten hands in the control group splinting was not recorded.

TABLE 6. Physical Findings, Treatment Regimens, and Results of Electrodiagnostic Tests on Hands in the Surgery and No-surgery Groups (N = 240)

	Surgery (n = 130) No.	(%)	*No Surgery (n = 110)* No.	(%)	*Significance of Difference*
Physical Findings					
Phalen's sign:					
Positive	113	(86.9)	86	(78.2)	
Negative	13	(10.0)	22	(20.0)	
Not available	4	(3.1)	2	(1.8)	$p = 0.08$
Tinel's sign:					
Positive	95	(73.1)	72	(65.5)	
Negative	27	(20.8)	37	(33.6)	
Not available	8	(6.1)	1	(0.9)	$p = 0.01$*
Two-point discrimination test:					
Positive	22	(16.9)	13	(11.8)	
Negative	97	(74.6)	86	(78.2)	
Not available	11	(8.5)	11	(10.0)	$p = 0.52$
Semmes–Weinstein threshold test:					
Positive	42	(32.3)	26	(23.6)	
Negative	43	(33.1)	51	(46.4)	
Not available	45	(34.6)	33	(30.0)	$p = 0.1$
Treatment Regimens					
Splinting:					
Positive	120	(92.3)	110	(100.0)	
Negative	0		0		
Not available	10	(7.7)	0		$p = 0.01$*
Cortisone injection:					
Positive	62	(47.7)	52	(47.3)	
Negative	68	(52.3)	58	(52.7)	$p = 1.0$
Non-steroidal anti-inflammatory medication:					
Positive	106	(81.5)	97	(88.2)	
Negative	8	(6.2)	11	(10.0)	
Not available	16	(12.3)	2	(1.8)	$p = 0.01$*
Electrodiagnostic Tests					
Electromyography:					
Positive	49	(37.7)	21	(19.1)	
Negative	72	(55.4)	40	(36.4)	
Not available	9	(6.9)	49	(44.5)	$p = 0.00001$*
Nerve conduction:					
Positive	109	(83.8)	50	(45.4)	
Negative	14	(10.8)	18	(16.4)	
Not available	7	(5.4)	42	(38.2)	$p = 0.0001$*

*See Table 7 for these results when unknowns were omitted.

Patients who underwent surgery were compared with those who did not in an attempt to determine whether there was a variable that resulted in these patients failing conservative treatment. Table 6 compares physical findings, treatment regimens, and results of electrodiagnostic tests in these two groups. There was no significant difference between the two groups for most of the clinical signs: Approximately 82% had a positive Phalen's sign, 15% had positive two-point discrimination results (>5 mm) and approximately 28% had positive Semmes–Weinstein results (>3.61). The only clinical sign for which there was a significant difference was a positive Tinel's sign ($p = 0.01$). Table 7 shows that this significance disappeared if the unknowns were omitted from the analysis ($p = 0.06$). As in the comparison of hands in the control group with those in the experimental group, comparison of hands in the surgery and no-surgery groups revealed differences in electromyographic and nerve conduction findings ($p = 0.00001$ for electromyographic findings, and $p = 0.0001$ for nerve conduction findings). There were, as expected, a large number of hands in the no-surgery group that were not tested. Table 7 shows that, when the unknowns were omitted from analysis, the difference between electromyographic findings for the two groups disappeared ($p = 0.53$), but a difference between nerve conduction findings remained ($p = 0.01$). This could be because of the large discrepancy in numbers between the two groups.

Table 6 also compares traditional conservative treatments between the surgery and no-surgery groups. In these two groups more than 92% of hands were splinted, 47% received cortisone injections, and more than 80% were treated with nonsteroidal anti-inflammatory medication. There was no difference between the two groups for cortisone use, but there was a significant difference for both splint use and use of anti-inflammatory medication ($p = 0.01$ for both). However, when the unknowns were omitted from the analysis (Table 7), the differences between the two groups disappeared ($p = 0.55$ for anti-inflammatory medication).

A three-way frequency analysis (log linear model statistical analysis) was done for the 197 patients to assess the multi-level interactions among patients in the control and experimental groups, in the surgery and no-surgery groups, and in the four occupational categories.

The overall three-way ($2 \times 2 \times 4$) *chi*-square result was statistically significant (*chi*-square 174.3, df 15, $p < 0.0001$), suggesting disparate frequency patterns among the 16 cells. A multi-way frequency table analysis is similar to a factorial analysis of variance (ANOVA). Any omnibus test compels closer inspection to uncover underlying causes. The highest order interaction—the three-way control/experimental by surgery/no-surgery by occupational status—was statistically not significant (*chi*-square 0.67, df 3, $p = 0.88$) as was the two-factor interaction surgery/no-surgery by occupational status (*chi* square 6.2, df 3, $p = 0.10$). This latter result indicates that there was no statistically significant difference in the percentage patterns of surgery/no-surgery across the four occupational groups.

The other two two-factor interactions were statistically significant. Control/experimental by surgery/non-surgery had a *chi*-square of 15.7 (df = 1, $p < 0.0001$). Seventy-one percent of patients in the control group underwent surgery, whereas only 43% of those in the experimental group did so.

The second statistically significant two-factor interaction was control/experimental by occupational status (*chi*-square 17.8, df 3, $p < 0.0005$). Of occupations of patients in the control group, 42.3% were clerical, 38.5% were manual, 15.4% were homemaker/retired, and 3.8% were unknown. In the experimental group the percentages were 71%

clerical, 16.1% manual, 10.8% homemaker/retired, and 3% unknown. It was the disparity between the clerical and manual groups that contributed predominantly to the chi-square statistic.

Follow-up. Of 53 patients (with 71 affected hands) in the experimental group who did not have surgery, only 6 patients (11.3%; with 8 affected hands [11.3%]) could not be located. Of the 47 patients contacted, 33 patients (62.3%; with 44 affected hands [62.0%]) reported a good or excellent result, whereas 9 patients (17.0%; with 12 affected hands [16.9%]) felt that their recovery was only fair or poor. Five patients (9.4%; with 7 affected hands [9.9%]) were non-compliant, that is, they did not perform the exercises once they were released. None of these 47 patients had yet undergone carpal tunnel surgery.

DISCUSSION

The results of this study indicate that at initial four-month follow-up there was a significant improvement in symptoms related to the patient's carpal tunnel syndrome in a large percentage of patients in the experimental group. This improvement was long-term, in that even at 23 months average, none of these patients went on to have surgery. The number of patients who did not undergo carpal tunnel surgery in the nerve and tendon gliding group compared with the control group was statistically significant. Both the control and experimental groups were well matched with respect to patient demographics, clinical presentation, and the traditional methods employed in the management of their cases.

Although predictive statements cannot be made as to which patients would eventually require surgery in either the control or the experimental group, it is possible that a combination of factors might predict which patients will go on to surgery. The decision to operate on these patients was based solely on their continuing symptoms, which closely matched those on presentation. Expansion of this study to include a multivariate analysis, prospective double-blind model, and validated outcome instruments is planned. A limitation of this study is that not all clinical signs, test results, and treatments were recorded for every patient, and the groups were treated at different times. However, the study suggests that use of the nerve and tendon gliding exercises may significantly decrease the number of patients going on to surgery at an average of 23 months' follow-up. Further follow-up will determine whether the number of patients in the experimental group who do not go on to surgery holds up over a longer period of time.

During the course of the long-term evaluation of these patients, it has become clear that rather than being a steadily progressive disorder, carpal tunnel syndrome is a condition characterized by remissions and exacerbation. In many cases, the symptoms can be managed non-surgically. It is probable that a certain percentage of patients will have resolution of their symptoms with traditional treatment methods, however, there is a subset of patient whose disease becomes progressive and whose symptoms become chronic. These patients may benefit from surgical release.[30]

TABLE 7. **Adjusted Values for Specific Findings for Hands in the Surgery and No-surgery Groups after the Omission of Unknowns**

	Surgery (*n* = 130)		*No Surgery* (*n* = 110)		*Significance of Difference*
	No.	*(%)*	*No.*	*(%)*	
Tinel's sign:					
Positive	95	(77.9)	72	(66.1)	
Negative	27	(22.1)	37	(33.9)	$p = 0.06$
Splinting:					
Positive	120	(100.0)	110	(100.0)	
Non-steroidal anti-inflammatory medication:					
Positive	106	(91.4)	97	(91.5)	
Negative	10	(8.6)	9	(8.5)	$p = 0.55$
Electromyography:					
Positive	49	(40.5)	21	(34.4)	
Negative	72	(59.5)	40	(65.6)	$p = 0.53$
Nerve conduction:					
Positive	109	(88.6)	50	(73.5)	
Negative	14	(11.4)	18	(26.5)	$p = 0.01$

Recent studies of carpal tunnel syndrome have focused on several aspects of the disorder. These include epidemiologic features,[2] new ways of delineating the disease by electrophysiologic methods,[29] the study of endoscopic carpal tunnel release,[7] modification of environmental and occupational factors,[31,32] pressure measurement,[33] advanced imaging of the carpal tunnel[34,35] and, most recently, outcome analysis following treatment for carpal tunnel syndrome.[36]

Treatment of carpal tunnel syndrome by conservative methods (splinting, non-steroidal anti-inflammatory medications, steroid injection, and workstation modification) all too frequently fails.[11,12] To date, there is no recognized, standardized consistent, conservative treatment program for its management. In addition, no recognized hand exercise program has been shown to affect the natural course of the disease and patient's symptoms.

Classical models of compressive neuropathy are static and do not take into account the movement of the compressed median nerve through the point of stenosis.[21] In addition, studies have not looked at the effect of stretching on the median nerve as it is tethered at the point of maximal compression in vivo. Stretching of the nerve may occur because of compression from the overlying transverse carpal ligament and adhesions between the median nerve and surrounding structures, as flexor tendon excursion is nearly five times greater than that of the nerve.

Guiding the wrist and the fingers through a program of nerve and tendon gliding exercises may help maximize the relative excursion of the median nerve in the carpal tunnel and the excursion of the flexor tendons relative to one another. When the exercises are performed, remodeling and stretching of the investing, adhesive tenosynovium around these structures may occur, thus diminishing their adherence to the structures inside the canal. In addition, by bringing the nerve through its maximal excursion, there may be redistribution of the point of maximal compression on the median nerve inside the carpal canal by allowing varying points of the nerve to be exposed to the maximal compressive area underneath the transverse carpal ligament. This "milking" effect would promote venous return from the median nerve, thus decreasing the pressure inside the perineurium. In time, increasing the motion of the median nerve may restore the linear relationship between median nerve and flexor tendon displacement.

The results of this study suggest that a program of nerve and tendon gliding exercises coupled with traditional non-surgical approaches reduces symptoms. A significant number of patients who would have otherwise undergone surgery, having failed traditional approaches to non-surgical management, were spared the morbidity and expense of surgical carpal tunnel release. Longer follow-up analysis is in progress and will be reported on completion. Nerve and tendon gliding exercises may also be useful in patients with occupational carpal tunnel syndrome who have "classic" symptoms with negative electrodiagnostic results. Additional randomized prospective studies are underway to verify these hypotheses.

More effective non-surgical treatment could have a significant effect on the total number of patients undergoing carpal tunnel release nationwide. This in turn may engender enormous cost savings with respect to surgical and hospital fees and time lost from work. In our study, patients performing these nerve and tendon gliding exercises were encouraged to continue working during participation in the non-surgical program. It is estimated that more than 200,000 carpal tunnel releases are done annually in the United States, at an estimated cost of $3.5 billion. Since much of this cost is due to surgical treatment of CTS[2] and the attendant loss of productive work time, we need to develop more efficient methods of non-surgical treatment for this ever-spreading epidemic.

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