Complications in the treatment of carpal tunnel syndrome

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Complications may result from every facet of the management of carpal tunnel syndrome. The authors review the common errors in diagnosis, nonoperative management, and operative treatment, with emphasis on prevention and resolution of complications. In general, surgeons can minimize complications by taking a thorough patient history, performing a comprehensive physical examination, and possessing a precise knowledge of the appropriate anatomy. Endoscopic techniques appear to offer some advantage over conventional open techniques with regard to the patient's postoperative incision pain, preservation of grip strength, and time to return to work; however, these advantages may be potentially negated by the risk of injury to neurovascular structures and tendons.

Key Words * carpal tunnel syndrome * carpal tunnel release * transverse carpal ligament

Release of the transverse carpal ligament (TCL) has become the most commonly performed peripheral nerve operation. The widespread popularity of the procedure is largely a consequence of the ubiquitous nature of the syndrome, the pervasive awareness of the syndrome by clinicians and patients, and the excellent response in most patients to surgical treatment. Since surgical decompression of the TCL was first performed by Learmonth in 1933[49] many authors have reported a high success rate performing the procedure in several large series of patients.[2,12,13,23,37,44,72,82] Concomitant with the increased volume of carpal tunnel releases (CTRs), complications have become more prevalent. MacDonald, et al.,[57] reported 34 complications in 22 patients (12%) undergoing 186 operations for carpal tunnel syndrome (CTS). With the advent of endoscopic CTR (ECTR), the potential for neurovascular and tendon injuries has increased, especially when the surgeon performing the procedure is at the beginning of the learning curve.[79] Furthermore, the practice of many academic hand surgeons includes more patients managed for complications of CTR than patients presenting with the initial symptoms of the syndrome.[97] Based on a 1987 survey of members of the American Society of Hand, this experience is typical for many hand surgeons. The survey revealed that 70% of the 467 respondents performed one to five reoperations per year for CTS and 16% performed more than six reoperations per year.[14] Although few surgical procedures are associated with such low morbidity rates, complications from CTR surgery can still be quite disabling to the patient. We will facilitate this review by arbitrarily dividing the complications into four categories: 1) errors in diagnosis; 2) errors in nonoperative management; 3) complications in open CTR (OCTR) surgical treatment; and 4) complications in ECTR treatment.

ERRORS IN DIAGNOSIS

Although the most commonly cited complications in carpal tunnel decompression are incomplete release
of the TCL and injury to the palmar cutaneous branch of the median nerve,[11,47,54,57] the incomplete or incorrect diagnosis of CTS is truly the most prevalent management error. The physician's failure to recognize comorbidities will result in incomplete resolution of the patient's symptoms after surgery. Likewise, the physician's inability to differentiate CTS from the many maladies that may mimic its symptoms will result in minimal improvement because of inappropriate treatment. A thorough history and physical examination of the entire upper extremity and cervical spine is essential for the diagnosis of CTS. The classic patient presentation for CTS is a middle-aged woman with the gradual onset, over months, of nocturnal paresthesias in the median nerve distribution, which later progresses to pain, numbness, and clumsiness in the hands. In a review of 1016 patients, nocturnal symptoms, considered by some clinicians to be a sine qua non of the diagnosis, were found to be the second most common symptom complex (71% of patients) after paresthesias in the median nerve distribution (100% of patients).[91] Physical findings in CTS are surprisingly mild compared with the magnitude of the patient's complaints. With the frequent paucity of other physical findings, provocative tests can be useful diagnostic adjuvants.

The classic provocative tests, Phalen's sign and Tinel's sign, are subject to subtle variations in test technique, which probably account for the large discrepancies in reported prevalence. The reported prevalence of Phalen's sign ranges from 10%[75] to 88%[31] and that of Tinel's sign ranges from 8%[75] to 69%.[31] A review by Slater and Bynum[88] of 3707 patients reported by six authors,[26,31,40,73,75,78] yields an average prevalence of 45% for positive Tinel's sign and 52% for positive Phalen's sign in patients with CTS. From the same review, 361 control subjects without CTS exhibited an average prevalence for positive Tinel's sign of 35% and 19% for positive Phalen's sign.[27,29,46,85] Because the sensitivity and specificity of the two classic signs are less than optimal, investigators have attempted to develop other noninvasive tests.

Threshold tests such as vibrometry and Semmes-Weinstein monofilaments and innervation-density tests such as two-point discrimination have shown some potential as noninvasive screening tests for CTS. Szabo, et al.[92] demonstrated the increased sensitivity of threshold tests over two-point discrimination for detecting early sensory changes in nerve compression. Spindler and Dellon[89] compared the sensitivity of sensibility testing (vibratory stimuli, two-point discrimination) with nerve conduction studies. Results from their study of 74 symptomatic hands indicated that nerve conduction studies were more sensitive (81%) than sensibility testing (66%) in confirming the diagnosis of CTS, but the combination of the two studies was more sensitive than either test alone (92%). Although noninvasive threshold tests such as vibrometry and Semmes-Weinstein monofilaments demonstrated promise in the early detection of nerve compression in limited clinical trials,[92] larger clinical screening trials provided contrary evidence that nerve conduction studies were much more sensitive to the early sensory changes in CTS.[89,99]

Electrodiagnostic studies may not be necessary in all cases, but they are clearly indicated when the diagnosis is in doubt. The principle purpose of nerve conduction studies is to provide evidence to support the diagnosis, quantify the severity of the diagnosis, exclude other diagnoses, and provide a baseline for patients with persistent symptoms. Many surgeons are reluctant to operate in the face of a normal electromyographic study, even though false-negative rates have been reported to be in the range of 8%[34] to 13%.[55] An even greater risk of excessive reliance on nerve conduction studies is the performance of CTR in normal patients. Nathan, et al.,[68] demonstrated that the false-positive rate for nerve conduction studies may be as high as 6% in a study in which they reported positive nerve conduction studies in 13 of 213 asymptomatic patients.
Many systemic diseases are associated with CTS and treatment of these underlying diseases may resolve the CTS-like symptoms. Thickening of the tenosynovium occurs with rheumatoid arthritis,[65] deposition of gouty tophi,[33,70] deposition of amyloid,[22] or tuberculosis.[42] Acromegaly produces actual thickening of the TCL in addition to hypertrophy of the synovium. Synovial engorgement may result from physiological states that cause retention of fluids such as pregnancy,[30,61] hypothyroidism,[76] and congestive heart failure.[90] Renal dialysis probably causes synovial edema due to increased intravascular flow.[81] Peripheral neuropathies associated with diabetes mellitus, chronic renal failure, and alcoholism alter peripheral nerves so that they are more susceptible to the compressive effects of CTS.

Other more proximal neuropathies may mimic and even coexist with CTS. Because CTS can produce referred pain to the elbow, shoulder, and neck, it may clinically resemble a C-5 or C-6 radiculopathy. Thoracic outlet syndrome, idiopathic brachial plexitis (Parsonage-Turner syndrome), pronator syndrome, and median nerve constriction at the ligament of Struthers may mimic some of the symptoms of CTS. Median neuropathies can occur as part of a more widespread peripheral neuropathy such as mononeuritis multiplex. In elderly patients, CTS may manifest predominantly as hand clumsiness and loss of dexterity. Several disorders of the central nervous system may produce some symptoms similar to CTS. Mass lesions of the foramen magnum, parietal infarctions, cervical spondylosis, and syringomyelia may all produce hand numbness, clumsiness, and slowness of movement.

The "double-crush" syndrome was the term coined by Upton and McComas[96] to describe the simultaneous compression of a single nerve by two compressive lesions. According to this theory, the proximal lesion lessens the ability of that nerve to withstand a more distal compression by impairing axonal flow. This scenario is well represented by tandem cervical radiculopathy secondary to spondylosis coexisting in a patient with CTS. The results of most studies investigating this phenomenon demonstrate that these patients benefit from CTR surgery but the success rate is lower than in patients with isolated CTS.[10,15]

ERRORS IN NONOPERATIVE MANAGEMENT

Injection of the carpal tunnel with corticosteroid agents and wrist splinting are the principle components of nonoperative treatment. Although most clinicians would affirm that corticosteroid injection into the carpal canal produces symptomatic relief, its effectiveness is often short-lived. Green[32] injected 122 wrists and attained good results (more than 50% relief of symptoms) in 81% of patients. In most patients, symptoms began to recur at an average of 3.3 months, but in only 46% were the symptoms severe enough to require surgery. In a prospective study, Gelberman, et al.,[25] treated 50 patients by using a single injection of a steroid agent and splinting, and they achieved complete resolution of symptoms in 76% of their patients at 6 weeks. Unfortunately, the percentage of patients free of symptoms had declined to 22% at 18 months. Using a strict definition of success as "complete resolution of symptoms," Weiss, et al.,[98] reported a success rate of only 13% at an average follow-up time of 11 months in 76 wrists treated with splinting and steroid injection. In light of the transient response to steroid injection and splinting, its most successful application is in the setting of pregnancy. Ekman-Ordeberg, et al.,[16] reported complete resolution of CTS symptoms in 80% of 56 pregnant women treated by splinting of the wrist at night. Only four of 56 patients required surgery because of persistent symptoms.

Direct intrafascicular injection of corticosteroid agents can cause widespread axonal and myelin degeneration, as demonstrated by Mackinnon, et al.[58] Both median and ulnar nerve injuries resulting
from corticosteroid injection have been reported.[53,63,94] Direct injection of the median nerve may produce persistent dysesthesias for weeks or months. Direct injection of flexor tendons may cause tendon rupture, especially in patients suffering from rheumatoid arthritis. Accumulation of the vehicle in which the steroid agents are suspended may produce a synovitis and thus may need to be removed by microsurgical technique during an OCTR procedure.

Use of the proper injection technique will generally prevent intraneural injection. The injection technique described by Green[32] is recommended. A 25-gauge needle is placed 1 cm proximal to the distal flexion crease, ulnar to the palmaris longus tendon or in line with the ring finger. The needle is introduced at a 30º angle to the skin and advanced just past the TCL. If paresthesias are elicited, the needle is withdrawn and angled closer to the skin before the second advance.

**COMPLICATIONS IN OPEN SURGICAL TREATMENT**

Most surgical complications in OCTR surgery result from inadequate or inappropriately placed skin incisions. Although there is considerable variability in the incisions chosen by particular surgeons, three general principles govern the design of the incision. First, the incision should allow adequate exposure under direct vision to section the TCL completely throughout its length. Blind cuts of the ligament will obviously place neurovascular structures in jeopardy. Second, an incision placed too radially will predispose to injury of the distal median nerve branches and one centered far toward the ulna will predispose to injury of the ulnar neurovascular bundle. Third, the incision should not cross the flexion crease at a right angle because a hypertrophic scar will often result. The transverse incision has been condemned because of the risk of injury to the palmar cutaneous branch of the median nerve.[45] In addition, the transverse incision predisposes to blind distal cuts of the TCL and subsequent injury to the digital nerves,[82] superficial palmar arch, median nerve, and flexor tendons.

Several studies cite incomplete sectioning of the TCL as the most common complication in OCTR.[47,57] MacDonald, et al.,[57] reported 12 cases of incomplete release of the ligament, constituting 35% of the total 34 complications found in 186 patients. Langloh and Linscheid[47] found incomplete resection of the TCL in 21 of 34 wrists investigated for persistent symptoms following surgery. Most of the 21 partially intact ligaments remained in continuity distally, most likely resulting from an attempt to avoid the the superficial palmar arch and the digital nerves. Other authors have stressed the importance of releasing the distal forearm fascia a few centimeters proximal to the wrist crease.[19]

Injury to the palmar cutaneous branch (PCB) of the median nerve is probably the second most commonly cited complication in OCTR surgery.[47,54,57] Transection of the nerve with subsequent neuroma formation was recognized in 1972 by Carroll and Green.[6] When transection of the PCB with subsequent neuroma formation occurs, the two recommended treatment options are to sever the PCB at its origin from the median nerve[6,54] or to bury the neuroma in the forearm muscles.[97] Taleisnik[93] investigated the anatomy of the nerve in a study of 12 cadavers and concluded that the best incision to avoid the terminal fibers of the PCB of the median nerve is a curved longitudinal one located on the ulnar side of the axis of the ring finger ray. Engber and Gmeiner[17] encountered two patients with neuromas in the hypothenar area following CTR surgery and subsequently performed a study of 21 cadavers to define the PCB of the ulnar nerve more clearly. Based on their anatomical findings, they concluded that the optimal incision was one in line with the axis of the ring finger. Martin, et al.,[60] conducted another cadaveric study of the cutaneous innervation of the palm in response to the continued occurrence of
painful incisions following OCTR; presumably the pain was caused by injury to cutaneous nerves with subsequent neuroma formation. They demonstrated that such an incision centered on the axis of the ring finger resulted in injury to ulnar-based cutaneous nerves in 16 of 25 cadavers and injury to median-based cutaneous nerves in three of 25 cadavers. Thus, even though an incision based on the axis of the ring finger may lessen the incidence of palmer cutaneous nerve injury, there is no true "internervous plane" that will completely avoid all cutaneous palmer branches, whether of median or ulnar origin. The findings of Martin, et al., intimate that shorter incisions with subcutaneous release of the ligament under direct vision or ECTR may be the only methods of decreasing incision pain from neuromas of cutaneous nerves.

Besides injury to the cutaneous branches of the ulnar nerve, other reported ulnar nerve injuries include laceration of the nerve in Guyon's canal,[20] transection of the deep motor branch of the ulnar nerve just distal to the hook of the hamate in the midpalmar space,[95] and division of the sensory ramus communicans between the ulnar and median nerves.[62] The ulnar nerve and artery lie radial to the hook of the hamate and volar to the ulnar aspect of the TCL in 15% of individuals,[97] which predisposes them to injury during inadvertent release of Guyon's canal.

Injuries to the superficial palmar arch,[57] the ramus communicans between the ulnar and median nerves,[62] and the common digital nerve to the adjacent long and ring fingers,[82] have all been reported. Their anatomical proximity to the distal edge of the flexor retinaculum is described in detail in the surgical anatomy section of the paper by Friedman in this issue.

Severance of the thenar motor branch (TMB) of the median nerve results in thenar atrophy and loss of opposition. Lilly and Magnell[52] reported good results in delayed repair, as late as 14 months, following initially unrecognized TMB injuries. Poisel[74] and Lanz[48] described several anatomical variants that are particularly susceptible to injury. In a study of 100 cadavers, Poisel found that the TMB becomes recurrent and exits from the median nerve distal to the TCL in 46% of cases. He documented an early ramification of the TMB under the ligament in 31% of cases and through the ligament in 23%. The transligamentous variant is probably more vulnerable because transection of the radial aspect of the ligament can produce TMB injury. Lanz[48] delineated the median nerve anatomy in 246 hands explored at operation and reported additional anatomical variations that may predispose to injury, including accessory branches of the median nerve at the distal carpal tunnel and origination of the TMB from the ulnar side of the median nerve.

Long-term persistent pain is a major determinant of the success or failure of the OCTR. The complication of long-term persistent pain may arise from any of the following causes: hypertrophic skin scarring, intra- and perineural scarring, adherence of the nerve to the skin, subcutaneous tender nerve secondary to superficial position, adhesions between flexor tendons and the median nerve, pillar pain at the thenar and hypothenar eminences, and reflex sympathetic dystrophy (RSD).

Hypertrophic scars are often the result of an incision that transverses the flexion crease at a right angle. If a painful hypertrophic scar should occur despite all attempts at prevention, scar revision should be performed. Although the skin of the palmer side of the wrist is thin and immobile, a Z-plasty after scar revision is usually possible and is the easiest remedy.

Intra- and perineural scarring sometimes produces dysesthesias, pain, and hypersensitivity. Neurolysis is seldom a successful remedy and can potentially produce further scarring as well as direct mechanical injury to nerve fascicles during extensive dissection. In fact, internal neurolysis at the initial CTR
procedure has never demonstrated any benefit over CTR without internal neurolysis;[26,59] thus, most hand surgeons would agree that internal neurolysis at the initial surgery is seldom indicated. Proper hemostasis is important to prevent perineural scarring. If intra- and perineural scarring should develop despite the surgeon's best efforts to prevent it, the hypersensitivity and dysesthesias may respond to coverage of the nerve with a fat graft or abductor flap.[97]

Superficial position of the median nerve and adherence of the nerve to the skin are usually consequences of an improper skin incision directly over the nerve, rather than toward the ulna. Splinting the wrist in a slightly dorsiflexed position for the first 3 to 5 postoperative days may lessen the likelihood of superficial nerve position. Three common methods of insulating the nerve from the skin surface include: rotation of a hypothenar fat-pad flap; rotation of local muscle pedicle flaps, such as the pronator quadratus and abductor digiti minimi; and Z-plasty with underlying temporary silicone sheeting to prevent scar adherence.[97]

Tendon adhesions may result from poor hemostasis during conventional OCTR surgery or from bleeding associated with tenosynovectomy. Resection of the synovium is usually indicated only in cases of extremely bulky synovium, such as those associated with rheumatoid arthritis, because of the propensity of tenosynovectomy to cause bleeding and scar formation with subsequent adhesions between tendons or between tendons and the median nerve.[97] Surgical drains may also diminish the incidence of adhesions but have been associated with a higher rate of infection. If postoperative splints are used, removal of splints by postoperative Day 3 lessens the risk of adhesions by allowing early mobilization of the tendons. Physical therapy with range-of-motion exercises and dynamic splinting rather than tenolysis surgery is the best treatment.[97]

Because the median nerve carries approximately 70% of the sympathetic nerve supply to the hand, RSD may result from carpel tunnel decompression. MacDonald, et al.,[57] reported four cases of RSD among 34 complications. Additionally, they described three stages of RSD in which the first stage is characterized by swelling, hyperesthesia, skin that is warm and dry, and persistent pain aggravated by movement. Progression to the second stage includes proximal spread of pain and edema, shiny skin that is cool and pale with atrophic changes, and joint stiffness. The third stage manifests as progressive atrophy with joint contractures and intractable pain. Early recognition and treatment are essential in the management of RSD. Initial therapy includes a 1-week course of oral corticosteroid and Stelazine medications accompanied by physical therapy.[97] This treatment usually resolves the syndrome, obviating the need for stellate ganglion blocks.

Pillar pain is an ill-defined, aching discomfort in the thenar and hypothenar eminences aggravated by gripping. Its etiology remains obscure but many attribute it to transection of the sensory nerve fibers supplying the palmaris brevis fascia and the resulting neuroma formation.[18] Other possible mechanisms include widening of the carpal arch and realignment of the carpal bones.[24] Seradge and Seradge[84] attributed persistent hypothenar eminence pain in five of 500 patients to an abnormal piso-triquetral joint, which resulted from an intercarpal alignment change after ligament release.

Deep wound infection was reported in 17 patients at the Mayo Clinic. Statistically significant risk factors for the infections included intraoperative instillation of steroid agents into the carpal canal, flexor tendon synovectomy, prolonged operative time, and the use of a surgical drain.[36] Treatment consists largely of surgical debridement with a primary closure or delayed primary closure when wound conditions permit. A suboptimal result occurred in seven of 17 patients at final follow-up evaluation. Superficial infection
rates are generally low compared with other surgical procedures. Gainer and Nugent[23] documented 26
"minor" infections in 430 operations. Phalen[73] reported one superficial infection in 212 cases.

Bowstringing of the flexor tendons is a rare, readily reparable complication in OCTR, cited in two of 34
complications by MacDonald, et al.[57] Bowstringing can usually be prevented by immobilizing the
wrist in slight extension for 3 to 5 postoperative days. If bowstringing should occur, it can be easily
corrected by reconstructing the TCL. Jakab, et al.[39] sectioned the ligament, reconstructed it with a
lengthened ligament, and demonstrated resolution of CTS symptoms in 93% of patients who experienced
no loss of grip strength.

Immediate postoperative loss of grip strength occurs in all patients and persists in up to one-third of
patients undergoing OCTR surgery. Kluge, et al.[43] evaluated grip strength at a minimum of 10 months
after surgery in 66 patients (89 hands) by subjective patient self-assessment. Grip strength was judged to
be normal in only 47%. Gellman, et al.[28] measured the time course of recovery of grip and pinch
strength using a dynamometer. Grip strength was expressed as a percentage of the preoperative ipsilateral
grip strength. Grip strength was 28% of the preoperative level by 3 weeks; 73% by 6 weeks, and returned
to the preoperative level by 3 months. Loss of grip strength, scar tenderness, and persistent pillar pain are
late sequelae of the OCTR procedure and have provided much of the impetus to switch to the alternative
of ECTR.

**COMPLICATIONS IN ENDOSCOPIC TREATMENT**

Endoscopic CTR techniques were first reported in 1989.[9,71] Since their introduction, rapid adoption of
these techniques has transpired. In contradistinction to the application of endoscopic techniques to other
surgical procedures, such as abdominal surgeries, ECTR has not decreased operative expense, increased
operative efficiency, or improved intraoperative visualization (compared with conventional
OCTR).[5,35] Despite these shortcomings, ECTR has many proponents who cite the potential benefits of
faster patient recovery time, less incision pain, and improved grip strength recuperation.[1,5,7,8] Two
large, prospective, randomized, multicenter, clinical trials comparing OCTR and ECTR methods
emphasize the aforementioned potential benefits of ECTR.[1,5]

Agee, et al.,[1] reported a randomized, prospective, multicenter study of 147 hands (65 OCTR patients
vs. 82 ECTR patients) in which the median time to return to work was 21 days shorter in the ECTR
group than the OCTR group. The best predictors of return to work were incision tenderness and return of
grasp strength. There were three complications in the ECTR group: one incomplete release of the ligament
and two transient ulnar nerve neuropraxias. There were four complications in the OCTR group: two
wound dehiscences, one bowstringing of the flexor tendons, and one injury to the deep motor branch of
the ulnar nerve. In the other study, Brown, et al.[5] reported a randomized, prospective, multicenter
study of 160 hands (82 OCTR patients vs. 78 ECTR patients) in which the median return to work time
was 14 days shorter in the ECTR group than the OCTR group. Persistent incision tenderness was present
in 61% of OCTR patients versus 36% of ECTR patients at 12 weeks. There were no complications in the
OCTR group and four complications in the ECTR group: one partial transection of the superficial palmer
arch, one digital nerve contusion, one ulnar nerve neuropraxia, and one wound hematoma. Despite the
faster recovery time in their study, Brown, et al., expressed the concern that "the greater rate of
complications indicates that intraoperative safety must be improved before ECTR is performed on a
widespread basis." In addition to these two studies, several other studies comparing OCTR and ECTR
procedures have been published.[1,3,5,18,35,38,83,87] Table 1 summarizes some of the pertinent
findings. The faster return to work, decreased incidence of incision pain, and increased preservation of grip strength during the first 2 to 3 postoperative months associated with ECTR have been affirmed by several authors.[1,4,18,83] In general, these potential benefits of ECTR predominate in the 1st several postoperative weeks but diminish significantly beyond this time period.

Although the decreased palmar tenderness, better preservation of grip strength, and earlier return to work associated with ECTR are very noteworthy, these advantages may be negated by the risk of neurovascular and tendon injury. Many surgeons remain skeptical about the safety and reliability of ECTR. According to recent studies, the overall complication rate is in the range of 1 to 2% in experienced hands for both ECTR and OCTR surgery. Despite a comparable complication incidence, the quality of the complications is vastly different.[4] A complete laceration of the median or ulnar nerve is a devastating injury. These types of injuries are exceedingly rare in OCTR but have been reported not infrequently in ECTR. Endoscopic carpel tunnel release is a technically demanding procedure in which there is a steep learning curve. Rowland and Kleinert[79] reported a 17% complication rate for 12 hand surgeons who were learning the technique on cadaveric specimens. Injury to the following structures has

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of OCTRs</th>
<th>No. of ECTR</th>
<th>Results</th>
<th>Complications</th>
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<tbody>
<tr>
<td>Agee, et al., 1992</td>
<td>65</td>
<td>82</td>
<td>ECTR: better grip &amp; pinch strength 3 wks postop; less scar tenderness at 9 wks</td>
<td>OCTR: deep motor branch ulnar nerve (1), bowstringing of flexor tendon (1), wound dehiscence (2); ECTR: incomplete TCL resection (1), transient ulnar neuropathia (2), persistent symptoms (1)</td>
</tr>
<tr>
<td>Brown, et al., 1993</td>
<td>82</td>
<td>78</td>
<td>ECTR: quicker return to work, improved pinch strength, less scar tenderness, more complications; no difference in patient satisfaction or relief of numbness &amp; paresthesias</td>
<td>OCTR: no complications; ECTR: superficial palmar arch transection (1), hematoma (1), digital nerve contusion (1), ulnar neuropathia (1)</td>
</tr>
<tr>
<td>Bande, et al., 1994</td>
<td>58</td>
<td>44</td>
<td>postop questionnaire; no significant difference in return to work time or relief of symptoms</td>
<td>OCTR: none; ECTR: none</td>
</tr>
<tr>
<td>Erdmann, et al., 1994</td>
<td>52</td>
<td>53</td>
<td>ECTR: quicker return of grip &amp; pinch strength; return to work time (ECTR 14 days, OCTR 34 days)</td>
<td>OCTR: hypertrophic scar (5), scar tethering (1), wound infection (1); ECTR: ulnar nerve paresthesia (1), incomplete TCL resection (1)</td>
</tr>
<tr>
<td>Skoff &amp; Sklar, 1994</td>
<td>20</td>
<td>20</td>
<td>no difference in return to work time, relief of paresthesias, or grip strength</td>
<td>OCTR: none; ECTR: digital nerve neuropathia (1)</td>
</tr>
<tr>
<td>Hallock &amp; Lutz, 1995</td>
<td>71</td>
<td>66</td>
<td>no statistical difference in return to work time, trend to earlier return with ECTR; no statistical difference in scar length or rate of complications</td>
<td>OCTR: none; ECTR: digital nerve neuropathia (1)</td>
</tr>
<tr>
<td>Sennwald &amp; Benedetti, 1995</td>
<td>22</td>
<td>25</td>
<td>ECTR: superior grip strength at 3 mos; return of function (ECTR 24 days, OCTR 42 days)</td>
<td>OCTR: hypertrophic scar (1), RSD (1); ECTR: neuropathia 3rd common digital nerve</td>
</tr>
<tr>
<td>Jacobsen &amp; Rahme, 1996</td>
<td>16</td>
<td>16</td>
<td>no difference in return to work time or improvement in median nerve electrophysiological values</td>
<td>OCTR: none; ECTR: digital nerve neuropathia (3)</td>
</tr>
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</table>
been reported with ECTR: median nerve,[5,21,66,77] ulnar nerve,[67] digital nerve,[5,64] superficial palmar arch,[5,66] wound hematoma,[5] flexor tendon injury,[35] RSD,[86] and incidental release of Guyon's canal.[56] Two of these injuries are often the direct result of endoscopic cannula placement. The radial digital nerve of the fourth finger can be injured at the distal port,[5,39] and the ulnar neurovascular bundle can be injured at the proximal port by inadvertent entry into Guyon's canal.[50,67] Because of this potential for neurovascular and tendon injury, most endoscopic surgeons agree that if the transverse striations of the TCL cannot be visualized along its entire length, the endoscopic procedure should be converted to an open one.

Incomplete ligament release ranges from 5% to as high as 50% in cadaveric studies.[50,51] Lee, et al.,[50] reported three types of incomplete release: 1) release of Guyon's canal, 2) incomplete distal ligament release, and 3) incomplete central (superficial) ligament release. The most common difficulties were penetration of the ligament with the cannula and inability to distinguish the proximal and distal ends of the ligament. Schwartz, et al.,[80] demonstrated incomplete release of the TCL in four of 13 cadavers and incomplete sectioning of the fascia connecting the thenar and hypothenar muscles in four more of the 13 cadavers in which an ECTR procedure had been performed by a surgeon trained to use the Agee 3M Inside Job device (3M Corp., St. Paul, MN). The true incidence of incomplete release of the ligament in noncadaveric studies is difficult to determine. Kelly, et al.,[41] reported five incomplete releases in 83 hands in which the Chow technique of ECTR had been used. Only two incomplete releases were reported in the 384 hands undergoing ECTR in Table 1.

In the final analysis, the optimal CTR technique would be one which incorporates the decreased incision tenderness, increased preservation of grip strength, and earlier return to work provided by ECTR with the lower incidence of serious neurovascular and tendon injuries found in OCTR. A technique that might fit this description is that of OCTR using a short (approximately 2 cm) incision, as reported by Hallock and Lutz[35] and Nathan, et al.[69] In contrast to the two comparative studies by Brown, et al.,[5] and Agee, et al.[1] Hallock and Lutz[35] reported no statistical difference in scar length, scar tenderness, rate of complications, or length of time before return to work in a prospective series of 71 patients undergoing OCTR surgery through an average 2.1-cm incision versus 66 patients in whom a two-portal technique was used. There were no iatrogenic injuries in the OCTR group and one possible digital nerve injury in the ECTR group. The average incision made by Hallock and Lutz was 2.1 cm compared with a 3.5- to 4.5-cm incision made by Brown, et al. Hallock and Lutz's data support their contention that the minimal-incision OCTR technique can achieve the same low incidence of incision tenderness and fast recovery as ECTR, without the potential complications. The benefits of increased recovery of function and a short average return-to-work interval of 17 days were demonstrated in another study incorporating a short-incision OCTR technique with intensive physical therapy.[69]

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Manuscript received June 3, 1997.

Accepted in final form June 10, 1997.

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