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Carpal tunnel syndrome – a comprehensive review

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With 16 Figures

Contents

"*Hands are an instrument, as the lyre is the instrument of the musician . . . every soul has through its very essence certain faculties, but without aid of instruments is helpless to accomplish what is by nature disposed to accomplish*"

(*Galen*)

Abstract

Purpose. To provide a comprehensive review of the management of carpal tunnel syndrome.

Methods and results. A systematic literature review is provided of the history, anatomy, pathophysiology, epidemiology, diagnostic criteria, investigative surgical techniques, results and complications for carpal tunnel syndrome.

Conclusion. Surgery for carpal tunnel syndrome requires meticulous attention to history-taking, investigation, counseling, training and surgical technique if unsatisfactory results and complications are to be avoided.

Keywords: Carpal tunnel syndrome; median nerve; neurophysiology; magnetic resonance imaging; microsurgery; endoscopy; systematic review.

Median nerve entrapments

Introduction

Entrapment means, "To be caught in a trap". Therefore entrapped nerves involved in nerve compression syndromes are treated by increasing the free space surrounding the nerve with the intention of lowering the presumed pressure on and in the nerve.

One of the most common symptoms when a peripheral nerve is compressed/pinched is paraesthesia. Paraesthesia is unfortunately a purely subjective phenomenon that is only experienced by the patient. Consequently – Medical Doctors (MD) – when patients complain of paraesthesia (tingling) – consult anatomy books seeking for a plausible diagnostic solution to their diagnostic problem. If a nerve pierces a membrane or turns around a muscle in the anatomy book, many surgeons assume that this is "in accordance with the symptomatology". Based on this purely hypothetical and non-valid scientific conclusion, they may suggest to the patient to "decompress" the nerve. This may result in a relief of symptoms but is also often followed either by no change in symptoms (because the nerve was not compressed), or by deterioration due to surgical trauma.

Surgical intervention is a kind of agreement between patient and surgeon. The patient wants to be healthy if surgery is performed, patients tend to be somewhat biased when being questioned regarding the surgical result at follow-up. I have personally met many patients who according to Hospital records

were better after surgery, but when carefully questioned, in reality no change had occurred. If the surgical intervention has introduced a complication, the patient may develop both psychological and social dysfunction. Pain is the most typical complication of most operative procedures, including "simple" median nerve decompression.

Any nerve can be compressed but some nerves are specifically prone to entrapments. The most common nerve with entrapment symptoms is the median nerve at the wrist, resulting in the so-called carpal tunnel syndrome (CTS). [7, 28, 38, 39, 48, 58, 68, 93, 128].

History of peripheral nerve surgery for CTS

Paget mentions CTS in his original paper from 1865 followed by Marie and Foix who in 1913 suggested that the transverse carpal ligament could be the compressive agent resulting in proximal neuromas of the median nerve [48]. Brain discussed in 1947 – immediately after World War 2 – the pathophysiology of CTS [58, 93]. This was the time when standards of peripheral nerve surgery evolved rapidly [48, 58, 117] with the introduction of operative microscopes, microsutures, fibrin glue, intraoperative neurophysiology and nerve grafting whereby the concept of how to treat an injured nerve changed [29, 39].

Peripheral nerve anatomical structure

A peripheral nerve comprises nerve cells e.g. conducting axons, insulated by Schwann cells and surrounded by connective tissue (Fig. 1).

The neuronal structures are bounded together and bundled in fascicles by the perineurium. These fascicles have patterns that vary longitudinally in the nerve [117]. There is a natural sliding between fascicles and nerves can stretch 10–20% before structural damage occurs. Endoneurium encircles each myeli-

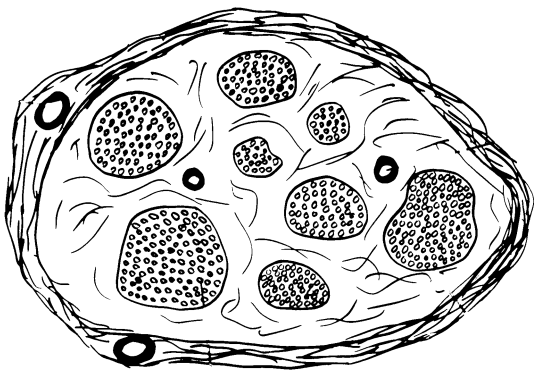


Fig. 1. Cross section of a peripheral nerve

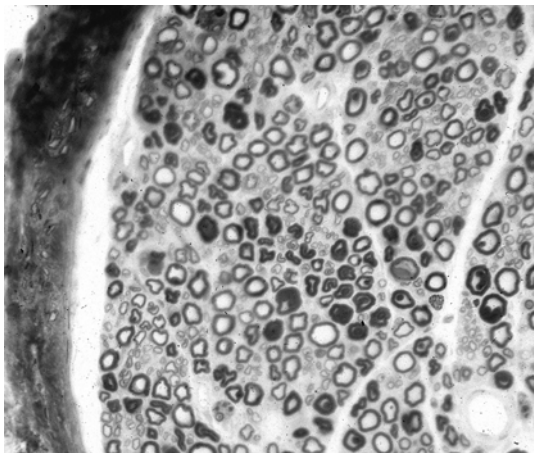


Fig. 2. Peripheral nerve histology

nated axon and groups of non-myelinated axons. Schwann cells provide the axons with lipoprotein coverage and some unknown trophic factors [120]. Outside the Schwann cells a basal lamina layer is found serving as a road for growing nerve fibers [66]. The cross section in Fig. 2 shows the micro histology of a peripheral nerve.

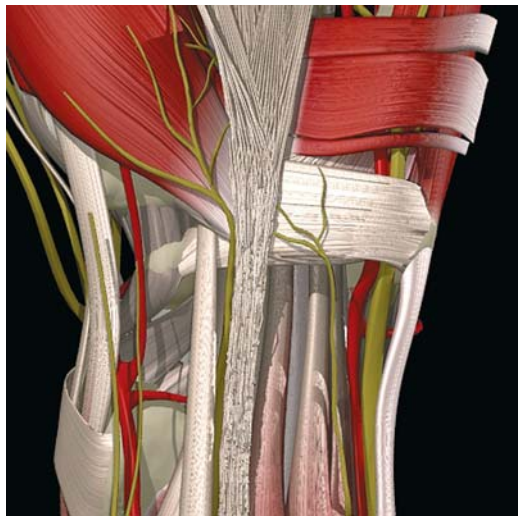
Connective tissue constitutes 50% component of a peripheral nerve. It consists of an external layer – the epineurium and an internal layer called the interstitial epineurium. Nerves close to a joint often have up to 85% connective tissue and only few fascicles. The connective tissue is made up of fibroblasts, fat, macrophages and blood vessels [117]. The vasculature of a nerve comprises both regional vessels entering the nerve obliquely at intervals along its course and an extensive longitudinal network of anastomotic vessels. Vessels traverse the perineurium and their sleeves close to the vascular walls of the nerve and thereby create a connecting channel. The major blood/nerve barrier is through the tight junctions of the perineurium [117, 120].

Carpal tunnel anatomy

Major anatomical variations exists within the carpal tunnel area [63]. Nevertheless, a general conception of this anatomy is important for surgeons.

An illustration of the wrist region is found in Fig. 3, where the skin has been removed. The transverse carpal ligament (TCL), the palmar aponeurosis, the tendon of the different flexor muscles and the nervous structures are outlined.

If we look further into the carpal tunnel it has a floor and walls created by the Navicular, Trapezius, Scaphoid, Hook of Hamate and Pisiform wrist bones as shown in Fig. 4.



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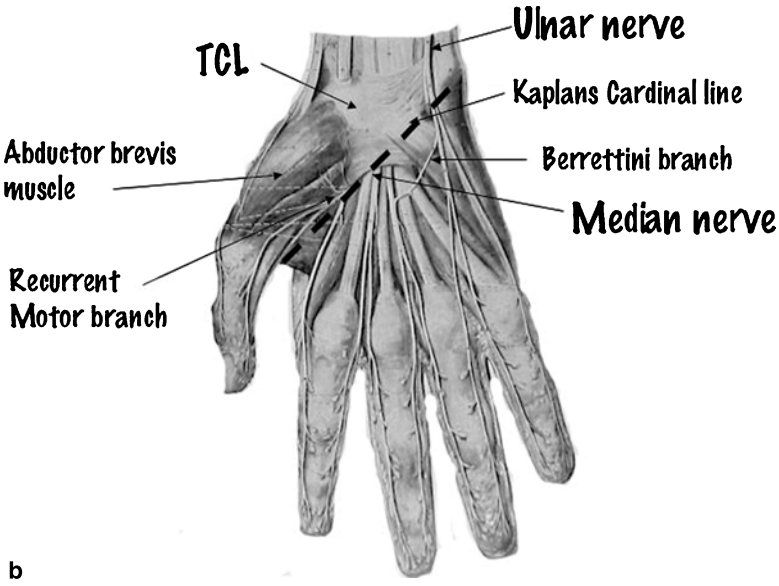

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Fig. 3. View of wrist region with skin removed (a, b). 3D image courtesy of Primal Pictures Ltd., www.primalpictures.com

Transverse carpal ligament (TCL)

From the surface of the hand it is our surgical task to visualize the structures of our surgical target – the transverse carpal ligament (TCL). The TCL attaches to the Pisiform, Hamate, Scaphoid, and Trapezium bones and creates the carpal tunnel. Different lines, and landmarks will guide us to this TCL. One of these

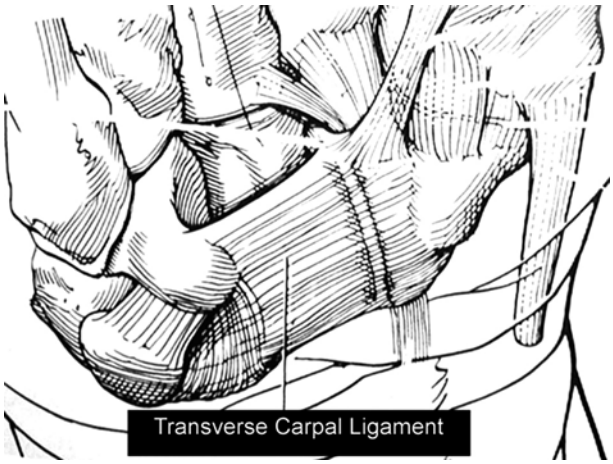

Fig. 4. TCL, wrist bones and ligaments

are Kaplan's cardinal line that is drawn from the apex of the first web space (between the thumb and index finger) toward the ulnar side of the hand, parallel with the proximal palmar crease (Fig. 3). The recurrent motor branch of the median nerve emerges at the intersection of Kaplan's cardinal line and a line through the axis of finger 2. The mean distance from the distal edge of the TCL to the recurrent motor branch of the median nerve is 2.7 mm (range 0–4.1 mm). The palmar cutaneous branch of the median nerve passes over the tubercle of the Scaphoid bone that can be felt in normal hands.

Entrance to the carpal tunnel is proximal and distal by the rim of the TCL. The size of the carpal tunnel can vary from 2 to 5 cm in length and is in a recent cadaver measurement a mean of 41 mm (35–45 mm) [49]. It is 2–3 cm wide and has a roof that is 0.5 cm thick with major variations [32, 33, 47, 49].

The mean distance from the radial aspect of the Pisiform bone to the radial border of Guyon's canal and the ulnar edge of the long palmar muscle tendon is 10.3 mm (range 9–12 mm) and 16.1 mm (range 12–22 mm), respectively. The mean distance from the distal portal of the carpal canal to the superficial vascular palmar arch and the ulnar artery is 10.4 mm (range 5–15 mm) and 7.6 mm (range 4.5–9 mm), respectively. The carpal tunnel has a rather steep descent into the palm as seen in Fig. 5.

The TCL serves as a trolley for the flexor tendons [58, 81]. The median nerve and the nine long flexor tendons of the digits pass through the carpal tunnel in synovial sheets. When we move our fingers or bend the wrist the tendons move up to 5 cm provided they are well lubricated. The eight superficial flexor- and the deep flexor tendons are in the same synovial sheet, whereas the long flexor tendon to the thumb has its own sheet radially placed in the carpal ligament (Fig. 6).

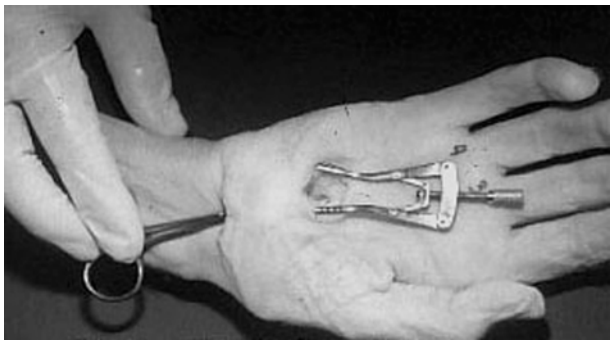

Fig. 5. Carpal tunnel, cadaver demonstration with instrument inside the tunnel

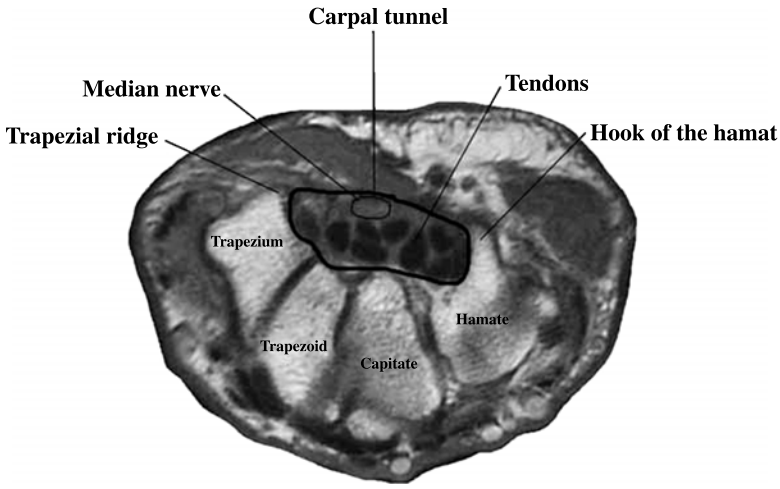

Fig. 6. Carpal tunnel, cross section, wrist bones, tendons and median nerve

Superficial to the TCL lies the palmar aponeurosis. Deeper transverse fibers of this fascial plane (volar carpal ligament), continues into the ante brachial fascia of the forearm that on the ulnar side of the wrist constitutes the roof for Guyon's canal. The radial edge of Guyon's canal contains the ulnar nerve and artery limited by the cooptation of the palmar aponeurosis with the TCL. The superficial longitudinal fibers of the palmar aponeurosis are in proximal continuity with the tendon of the long palmar muscle (Fig. 3). The abductor brevis muscle inserts from the radial side upon the TCL.

The median nerve

Variations in the anatomy of the median nerve at the wrist are of importance to the surgeon preparing to divide the TCL. The median nerve passes in the

forearm radially to emerge between the tendons of the superficial long flexors and the radial flexor carpi (Fig. 6). It enters the carpal tunnel under the radial edge of the long palmar muscle tendon (Fig. 3). The median nerve consists of sensory and motor fibers to the radial part of the hand. The motor fibers are mainly located in one or two fascicles volar located in the median nerve [117]. The nerve passes through the carpal tunnel located volar close to the TCL and radial to the superficial flexor tendons between those to the middle finger and the radial flexor carpi tendon (Fig. 6). The median nerve dips under the TCL usually after having given off a cutaneous palmar branch that lies radial to the tendon of the long palmar muscle. This branch may also pierce the TCL and great variations of this superficial cutaneous branch exist. On the ulnar side of the palmar minimal muscle tendon the ulnar nerve gives off some smaller branches that can be found in the majority of variations.

The median nerve divides into five sensory branches at the distal end of the TCL. These five branches include:

1) A proper digital nerve to radial side of the thumb;
2) A short common digital nerve to the first web space that quickly divides into a proper digital nerve to the ulnar side of the thumb;
3) A proper digital nerve to the radial side of the index finger; and
4, 5) Two common digital nerves to the second and third web spaces [32, 119].

Within the carpal tunnel the median nerve gives off a recurrent motor branch radially and distally to the abductor brevis muscle with many variations. It is in 46% of patients leaving the median nerve freely extra ligamentous distal to the TCL. In 31% it is placed subligamentous and in 23% it passes through the TCL. In these two situations it is easily possible to injure this nerve during

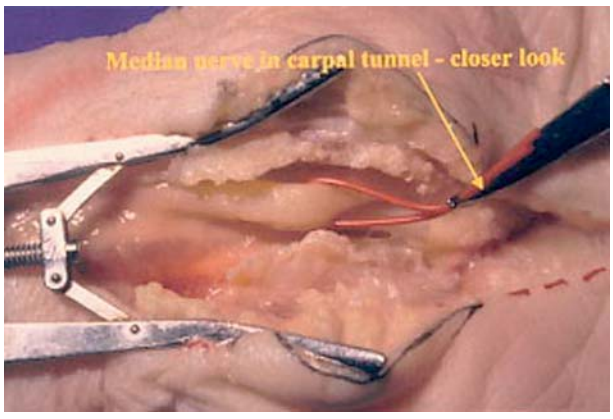

Fig. 7. Cadaver presentation of the median nerve with the TCL cut

surgery [32, 49]. Branches from the median nerve at the same level may also innervate the first interdigital- and lumbrical muscles.

Distal to the TCL the communication branch between ulnar- and median nerves (Berrettini branch) is found among 84%, always closely related with the distal vascular communication vessels [49].

The median nerve changes size and format according to the position of the wrist joint and with movements and elongation of the TCL. It is oval becoming more elliptical at the level of the Hamate bone. With the wrist extended it is found more anterior and deep to the TCL whereas it in flexion is pressed towards the TCL. It may move up to 2 cm during flexion/extension [53]. The median nerve is seen in the carpal tunnel in Fig. 7.

Cutaneous innervations of the palm

The palm derives its cutaneous sensation from branches of both the median and ulnar nerves. The cutaneous branch from the median nerve – already mentioned – is found 3–11 cm proximal to the wrist crease following the radial flexor carpi tendon, with penetration of the superficial layers of the TCL at the level of the Scaphoid bone [119]. The cutaneous branch from the ulnar nerve is found up to 16 cm proximal to the wrist crease. It innervates skin at the thenar eminence in approximately 50% of the patients. Short palmar branches originate proximal to the wrist crease penetrating the fanning fibers of the long palmar muscle tendon and terminate in the TCL [32].

The distal edge of the TCL

The superficial palmar arch lies in soft fat 2–26 mm from the distal edge of the TCL. It consists of the communicating branch between the ulnar and median nerves (Berettini branch), and the common digital nerve to the adjacent long and ring fingers. The superficial vessel connection between radial- and ulnar arteries lies together with these nerves. The hook of the Hamate marks the ulnar edge of the distal TCL, and the deep motor branch of the ulnar nerve passes around this hook (Fig. 6).

Carpal tunnel size – imaging

There is a great difference between measuring anatomical structures on cadavers and in the living [63]. Therefore with the introduction of Magnetic Resonance Imaging (MRI) we have received better information regarding the carpal tunnel. MRI is used to measure the transverse dimension of the carpal tunnel in neutral, flexed and extended positions among 16 normal subjects. In wrist extension the anterior–posterior (A–P) dimension and cross-section area (CA)

decreased compared to neutral. Similar the transverse dimension (TD) increased at the level of the Pisiform bone and CA at the Hamate level. In flexion TD and CA decreased at Pisiform level. With wrist extension, the median nerve may be subject to significant pressure at the Pisiform level [131]. A plain radiographic study of 21 uninjured wrists in 12 volunteers defined the position of the distal wrist flexion crease (DWC) with respect to the hand. In 19 of the 21 wrists the DCW was within 2 mm of the proximal pole of the Capitate bone. Newer studies prove that the distal part of the tunnel is the narrowest in all these situations [49, 94]. The cross-sectional area of the carpal canal proximal and distal to the wrist flexion crease demonstrates on MRI a gradual increase in the area of the carpal canal moving proximal from its distal narrowest point. There is no significant increase in area until approximately 23 mm proximal to the narrowest point [78].

The dimensions of the carpal tunnel are slightly but not significantly smaller among patients with symptoms indicating CTS. These have thus a normal mean distal cross section of the carpal tunnel of 2.857 mm^2 [94].

Important to note is that this volume depends on the wrist position and decreases with wrist extension. The volume of a 400 mm long TCL is thus $2{,}857 \times 400 = 114.280\ mm^3$. The size of CTS symptomatic and abnormal carpal tunnels is a mean of $238{,}9\ mm^2 \times 400 = 95.560\ mm^3$. The difference is $114.280 - 95.560 = 16\%$. When cutting the TCL the size of the carpal tunnel should increase. In one series it changed only 6%, whereas other series have shown up to 24% increase [123].

The ulnar nerve and artery is located ulnar to the hook of the Hamate (Fig. 6). Radial flexion of the wrist joint causes in 30% the ulnar artery to move inside of Guyon's canal whereas ulnar flexion causes it to move radial toward the carpal tunnel [49].

What is a carpal tunnel syndrome?

"*The Danish poet and storyteller Ludvig Holberg used in his play Erasmus Montanus the following circular argument – said to his mother by the clever student (Erasmus) that returned to Denmark from studies in a University abroad: "A stone cannot swim, You – mother – cannot swim – therefore You are a stone*".

Despite what has been written in thousands of papers, we do not have a golden standard for what a CTS is [7, 79, 128].

In a patient with "symptoms" of Diabetes Mellitus, we will monitor blood–glucose levels and find them elevated. We know from pathophysiology that he lacks insulin. Now providing him with insulin he will feel better. His primary symptoms are soon relieved and simultaneously his blood–glucose level is lowered. Then we are now confident that the primary diagnosis was correct.

With CTS it is partly the same. We suspect CTS if patients are having certain "symptoms". Our problem here is that we do not know what parameters to monitor to find the "glucose" of CTS. When we surgically decompress the median nerve, and for example increase the volume of the carpal tunnel, most patients will recover. From that it is deducted that it is because the carpal tunnel volume was too small. We know from experimental science that an irritated median nerve will result in symptoms like paraesthesiae. Then we begin to look for pressure in the canal to prove our point. However it is still the neuropractic dysfunction of the median nerve that is the cause of the symptoms. Instead of monitoring pressure we seek simpler methods such as a better description of symptomatology (Hand-Diagrams). We start also to monitor nerve dysfunction with electrophysiology, and in both case we find many false positive and false negative results. We must evidently be careful in comparing results of treatment of CTS as the majority of papers describe "typical symptoms" of CTS and uses these "typical symptoms" in a circular argument, that when these symptoms are present, the patient harbours CTS – just like Ludvig Holberg did.

We have to add information on the population our patient is living in to decide which diagnostic tests we can rely on in the actual population. How to document an entrapment is therefore still today open to much debate – and often a very heated debate. How can we define CTS? Which test shall be used to diagnose a CTS? How shall we treat CTS? All these questions are some of the major issues for us as neurosurgeons. Most important for the future is that we try to answer our questions in evidence based way, and not just by "personal opinion" [104]. Following a personal screening of >2500 scientific papers dealing with CTS, I believe it is necessary to understand part of epidemiological basic statistics if we are to draw any conclusions regarding the true value of diagnostic and screening tests.

If one is interested, a check-up at the Cochrane Library [18] can be valuable for the understanding of how many unnecessary papers have been written on CTS problems through out the years.

For the remainder of this chapter we will accept that:

"*The most plausible reason is, that the median nerve is compressed in the carpal tunnel and that this is leading to the symptomatology of a CTS*",

and remember that the CTS based on symptoms alone are not just "one" disease, but possibly a combination of many [79, 97].

Pathophysiology of CTS

When a peripheral nerve is compressed this leads to immediate biochemical changes of the nerve and subsequent anatomical changes. The metabolism of a peripheral nerve was thoroughly studied by the Swedish scientist Lundborg

[66] and the deeper understanding of peripheral nerve biology gave a significant input to our understanding of entrapment treatment policies.

The carpal tunnel is to be considered as a closed hydrostatic space. An Italian group proved by continuous pressure monitoring, that daily fluctuating pressures occurs with a peak at 6 am – the time most patients are awakened shaking their hand. Lundborg [66] confirmed in his important work on blood circulation and vessels in peripheral nerves that a pressure of 30 mmHg leads to the first neurophysiologic changes and that a total blocked nerve conduction was found with pressures >50 mmHg. The normal pressure in the carpal tunnel is 2.5–15 mmHg and is highest distally [33]. The most compressed point is 10 mm distal to the wrist crease, with a pressure of 44.9 ± 26.4 mmHg. The correlation coefficient between the highest canal pressure and the latency was 0.393 and between highest canal pressure and duration of symptoms was 0.402. The most compressed part of the median nerve found in the carpal canal is 10 mm distal to the wrist crease and is related to the distal motor latency and to the duration of symptoms [51, 87]. Flexion/extension in the wrist reduces the carpal tunnel volume and increases consequently the pressure to around 30–50 mmHg – even up to 200 mmHg especially by wrist extension. Pressures measured with the wrists in three positions: neutral, full passive flexion and full passive extension showed that at each wrist position, the mean pre-operative pressures in a study group were significantly higher than in the control group. In both groups, the pressures were maximal with full passive extension and minimal in the neutral wrist position [33, 106].

The thick, myelinated fibres are more sensitive to hypoxia than the small thinner fibres [66]. An incremental loss of motor and sensory function of the median nerve occurs with increasing pressure [33] and a direct linear relation is found between electrical nerve conduction velocity and pressure in the tunnel.

This elevated pressure results at the same time in a slowing of the capillary circulation leading to a slight venous engorgement and further to hypoxia of nerve structures [66, 68].

A physical compression of the median nerve results also in further anatomical changes as distal axoplasmic flow is disturbed if the compression is static. Axonal membrane excitability is also altered and if the compression is continued, polarized distortion of the internodes is developed. The myelin lamella slips away from the site of compression and telescope through the node of Ranvier first described by Ochoa [3, 66]. Local demyelisation is found as a result of stretching and increased pressure, whereby blood flow is reduced and ischemia the result, but is reversible and recovery begins within hours. In cases of nerve compression, the proximal axons are distended and a decreased transport of nutritive substances and enzymes are found distally [66, 98]. A pseudoneuroma of the median nerve with distended vessels and cyanosis is developed and in 14% of operated CTS hands an hourglass deformity of the

median nerve has been encountered [3]. The oedematous tissue facilitates proliferation of fibroblasts and in later phases internal long lasting compression of the median nerve even with scarring inside the fascicles. Fibrotic changes of the circumferential and interstitial epineurium are always found later in this process [66]. The longer the duration of/and amount of pressure – the more neural dysfunction is found.

The intermittent compression, stretching or dislocation of the nerve dictated by anatomical factors leads to development of an additional axon cylinder constriction [98]. These fibrotic changes affect the degree of free neural gliding [68]. The restricted nerve sliding may lead to increased strain, and possibly contributing to symptoms. Both among CTS patients and control patients longitudinal movements of the median nerve fascicles in the forearm averaged 2.62 mm. The nerve strain is thus not increased and should not contribute to symptoms. The CTS patients have in contrast to this a 40% reduction in transverse nerve movement at the wrist.

Tensile strength of a peripheral nerve is mainly determined by the perineurium and allows the nerves to stretch up to 20% before structural changes occurs [66].

When Fibrotic changes increases axonal degeneration will occur. Wallerian degeneration of the distal part of the axons is seen in the most severe degrees of compressions [117]. The neurophysiologic correlation to these changes is that only fast nerve conduction velocity will be reduced in the phases of documented clinical presenting CTS e.g. the more severe cases. Sensory fibers predominates in the median nerve, and therefore we find mostly sensory findings but we have no clues to why sensory fibers are more selectively injured following a compression of peripheral nerves [117]. Sensory nerve fiber dysfunction seems to start at larger fibers and gradually extend to smaller fibers [82]. The autonomic median nerve dysfunction not caused by vasoconstriction but increased sweating is often the first signs of CTS [88].

It is thus a combination of ischemic and mechanical factors that are involved in compression neuropathy. This suggests two mechanisms of the nerve entrapment, one with reversible dysfunction in nerve fibers due to simple lipid/myelin changes associated with some ischemia and one with more slowly developed structural changes in nerve fibers due to pressure.

In summary, the peripheral nerve reacts to pressure in a standard scenario. First stage is called neuropraxia being the physiologic, reversible functional disruption of a neuron. This first-degree injury is a reversible local nerve conduction block at the site of compression. In the second degree injury axonotmesis with loss of continuity of some axons is found with the endoneurium sheet being intact. These lesions tend also to recover but in a slower rate with axons sprouting from 0.7–2.7 mm per day. If fibrosis increases, further Wallerian degeneration develops and fixed nerved lesions is the result.

After denervation the muscle fibres undergoes fibrosis that will be maximal and permanent after 2 years [28, 117, 120].

Definition of carpal tunnel syndrome (CTS) for epidemiological purposes

The fact is that it is subjective symptoms that lead us neurosurgeons to validate patients. CTS is currently anticipated to be the most common peripheral nerve compression neuropathy affecting an estimated 1% (or more) of a given population so it seems important to develop a system that can be used for screening/diagnosing [4, 38, 39, 44, 72, 85, 93, 124, 128]. The golden standard for CTS diagnosis exists only if by consensus [38].

This consensus has not yet been achieved despite attempts [97].

How do we diagnose CTS in a scientific way?

Appropriate patient selection is extremely important for a successful outcome and inaccurate diagnosis of CTS is one of the most common causes of treatment failure of CTS [38, 39]. We know from history and experience, that a majority of patients claiming CTS-hand-symptoms will benefit from surgical decompressive treatment. As this treatment is linked with increasing the volume of the carpal tunnel we assume that the volume of the carpal tunnel plays a role. Anything that can decrease the size e.g. volume of the carpal tunnel or increase the contents of the carpal tunnel structures, may thus lead to increased pressure on and in the median nerve [4, 128]. Nervous structure size changes is not related to bodyweight as could be expected. The median nerve cross-sectional area was found to be equal in an obese and a thin groups (9.3 vs. 9.4 mm^2), as was the carpal canal pressure (16.2 vs. 15.5 mmHg), respectively [78].

We must as a consequence of this experience describe precisely how and when reduction of volume and increase of pressure in the carpal tunnel causes a functional reaction in the median nerve. Only then can we to make a firm objective diagnosis of CTS. Therefore we need to be able to screen know the population to be able to discuss prevalence or incidence of a specific disease e.g. CTS.

There are three types of anatomical problems in this context: The carpal tunnel is decreased in size due to:

1) External factors.
2) Intrinsic factors.
3) The carpal tunnel is smaller than "normal".

In all three situations, the possibility of developing symptoms from the median nerve is possible. To this anatomical information, we may add further scientific documentation of an actual median nerve injury at the wrist level using electrophysiology.

Incidence, prevalence and diagnostic epidemiology – important facts for diagnosing CTS

We use the terms specificity and sensitivity when we introduce a diagnostic procedure or disease [75, 79, 85]. These are linked with the terms incidence and prevalence used to compare groups of patients and are often mentioned in scientific papers. In many papers, quoted "incidence rates" are in reality prevalence rates. Thereby misunderstandings can easily occur because they relate to totally different populations.

Specificity and sensitivity of diagnostic procedures

"Description of a disease" is also called nosographic. Therefore a nosographic description of CTS differs from that being used when describing CTS by clinical evaluation. The nosographic specificity and sensitivity are irrelevant for clinical practice, but both terms are too often used in the literature called simply: "specificity/sensitivity". It is only valid to discuss the true value of a clinical test e.g. neurophysiology, if we know the clinical population we are dealing with.

Incidence

The number of new cases of a disease (CTS) that develop within a specified population over a specified period of time. The crude incidence is thus the mean number of cases found per year in e.g. Denmark per 100,000 inhabitants = the number of CTS-entrapment cases divided by number of person-years under risk, multiplied by 100,000. Incidence is therefore obviously dependent on the composition of what is usually a continuously changing population and that of the diagnostic parameters used.

Prevalence

Prevalence is in contrast the ratio (for a given time period) of the number of occurrences of a disease (CTS) to the number of people "at risk" for having the disease in the population. Therefore an entrapment syndrome like CTS must have a higher prevalence among patients with any type of e.g. polyneuropathy.

The prevalence should only be referred to in a well defined, and stable population – and this is usually not the case in our global world. In 1961–65 the prevalence of CTS in Rochester, USA was 88/100,000 and in 1976–80 it rose to 125/100,000 [114]. Either the frequency of the disease has increased in this part of the US or the diagnostics and general interest in the disease have increased or – perhaps (?) – the diagnostic criteria are not the same in the two time periods. An electrophysiological CTS prevalence study in the Netherlands showed that it was 5.8% among women and only 0.6% among males.

Scandinavian figures show almost equal representation with a marginal tendency of having more females harbour CTS than males [4, 68, 128]. Both statements cannot be true demonstrating this basic problem. The influence of diagnostic interest and general knowledge among Physicians may be found in the figures from Denmark. With a population of 5.3 million, we treated in year 2001: 7500, in 2002: 8860 and in 2003 10,700 patients with the diagnosis of CTS.

Diagnostic methods – and their validity

Diagnosing CTS is like asking, "What is a Duck?" My good friend and colleague James Steers often uses the following: We all know that a duck is a bird. If we add that it also has large feet, that it waddles, further that it can swim and finally that it quarks we are very close to have identified a duck and not a sparrow.

Carpal tunnel syndrome (CTS), or compression neuropathy of the median nerve at the wrist, is the one of the most common conditions encountered and is diagnosed with increasing frequency in the general population and among certain occupational groups [4, 85]. CTS is a frequent cause of morbidity in western societies and can have a profound impact on an individual's ability to perform their daily activities. There are a wide variety of predisposing factors and conditions that are associated with the development of CTS symptoms [68].

We may agree that clinical signs of median nerve neuritis e.g. paresthesia, sensory deficits, nightly painful paresthesia ("screening"), often leads to our proposed surgical interventions. Too often Medical Doctors assume that this symptomatology is synonymous with a true compression of the median nerve [104].

From a scientific point of view it is irrelevant to define CTS caused by compression from "clinical subjective symptomatology" alone, unless we know precisely the population structure – and then search for reasons hereafter. However this is generally the case when you review the literature.

The clinical picture alone is not sufficient to predict the diagnosis of CTS. A compressive lesion of the median nerve at the carpal tunnel can be present both among patients with no typical symptoms of CTS (asymptomatic individuals) and among symptomatic patients in which neurophysiologic studies are negative. In a general population a 0.7% prevalence of undiagnosed CTS is found. These patients have all symptom-severity similar to that of patients undergoing surgery. Therefore variable numbers of this group may be drawn into a "medical system" and thus account for variations in the rate of surgery performed [4, 37].

In this review I try to look at the history, anatomy, epidemiology, diagnosis and the key issues in the management of CTS.

CTS are usually diagnosed based on a combination of history taking that includes subjective signs combined with objective findings and objective mea-

surable "imaging" of carpal tunnel and nerve [6, 30, 125]. The predominant classic symptoms are nocturnal painful paraesthesiae of the hand, and sensory disturbances within the distribution of the median nerve, both of which are characteristically relieved by hand movements (hand shaking). Ancillary tests are used to objectively document the problems at the carpal tunnel. These include imaging techniques and nerve conduction studies. Imaging tests (ultrasound/sonography and MRI) are useful for demonstrating the structure of the carpal tunnel. In cases with persistent symptoms following surgical relief of the median nerve they may explain the reasons. These methods are still considered to have a lower diagnostic accuracy than neurophysiology, Neurophysiology with nerve conduction studies are specific less accurate in the early stages of CTS and among younger patients. Development of these imaging modalities and supplementary tests of small nerve fiber function and techniques of measurement the intra carpal pressure, may in the future improve early recognition of CTS [126].

Our present diagnostic methods includes a combination of the following:

1. History taking, e.g. symptomatology or "subjective" signs.
2. Clinical evaluation e.g. "objective" examination by a MD.
3. Diagnostic tests, by tradition but only partly objective and reliable.
4. Imaging of the median nerve and carpal tunnel.
5. Electrophysiology.

1. History taking – symptomatology

The following general medical conditions may often lead to a peripheral nerve symptomatology mimicking CTS. They can both be a part of the syndromes and by reducing the carpal tunnel volume lead to median nerve pressure.

A prominent example is thus rheumatoid arthritis, which reduces the carpal tunnel volume and simultaneously gives structural disturbances inside the median nerve. Obesity results in normal median nerve and carpal tunnel sizes, but endoneural oedema is found together with reduced SNCV [123]. Synovial engorgement is the result of fluid retention seen in pregnancy, and post menopause (PMS) with water retention and through increase of contents causes nerve symptoms simulating CTS. Renal failure and long-term haemodialysis, high blood pressure, and congestive heart failure may be associated with the build up of amyloid in the median nerve and increase in size.

Hormonal disorders such as acromegaly and hypothyroidism are commonly related to CTS for the same reasons.

CTS has been described together with persistent median artery, lipomatosis, gout, anomalous hand muscles, vascular tumors, collagen vascular diseases, inflammatory or septic tenosynovitis, lepra, tuberculosis, Fungal infection, Gout, De Quervain's disease, wrist fractures and dislocations including wrist

bone luxations. Hemorrhages during anticoagulation therapy are another well-known cause of CTS [7, 58, 68, 93, 128].

Peripheral neuropathies (polyneuropathy) associated with Diabetes Mellitus, cancer and alcoholism are diseases that easily mimic CTS symptomatology. 5–25% of diabetic patients with polyneuropathy, may also have a "true" CTS = with a compression of the median nerve. Hereditary neuropathy with liability to pressure palsies (HNPP) is an autosomal-dominant peripheral neuropathy that results from deletion of a 1.5-Megabase pair (Mb) segment of the short arm (p) of chromosome 17. It should increase susceptibility of peripheral nerves to pressure and trauma and can be associated with symptoms at multiple anatomic entrapment sites. There is no evidence for an association between HNPP and patients who have multiple surgical releases for upper-extremity entrapment neuropathies [25, 105].

Significant vascular ischemia usually provoked by cold with subsequent Raynaud's phenomenon also causes paresthesia of the fingers [128].

More proximal upper extremity Median Nerve entrapment can also result in CTS symptoms. This has lead to a discussion on the possibility of a so-called double-crush syndrome. Cervical disc herniation, proximal median nerve entrapment and Dystonia are possibilities of entities with a Double-crush. According to this idea, nerve fibers that are injured proximal close to the spinal cord should become more sensitive to supplementary distal lesions [122]. The frequency and electrophysiological data of CTS analyzed according to cervical radiculopathy level do not support a neurophysiologic explanation of double crush syndrome so that it must be considered a highly hypothetical idea but not an actual existing entity [60]. If palsy of thumb flexion is found on clinical examination it is often caused by affection of the median nerve more proximal than the wrist. In this situation we will find sensory disturbances in the palm. Supplementary entrapments of other nerves exist and an ulnar nerve entrapment in conjunction with CTS is not uncommon.

We also know that patients often complain of nightly painful paresthesia. If this symptom is added to the previous, we are further tempted to conclude that the patient has CTS. Which weight we shall add to each symptom is difficult to assess. This has been tried by expert groups and in situations using Bayes theorem [38, 75, 84].

From a large case-control study using the UK General Practice Research Database [34] the relative contributions of the common risk factors for carpal tunnel syndrome (CTS) in the community were quantified. Cases were patients with a "diagnosis of CTS" and for each, four controls were individually matched by age, sex and general practice. The dataset included 3391 cases, of which 2444 (72%) were women, with a mean age at diagnosis of 46 (range 16–96) years. Multivariate analysis showed that the risk factors associated with CTS were previous wrist fracture (OR = 2.29), rheumatoid arthritis (OR = 2.23),

osteoarthritis of the wrist and hand (OR = 1.89), obesity (OR = 2.06), diabetes (OR = 1.51), and the use of insulin (OR = 1.52), sulphonylureas (OR = 1.45), metformin (OR = 1.20) and thyroxine (OR = 1.36). Smoking, hormone replacement therapy, the combined oral contraceptive pill and oral corticosteroids were not associated with CTS. The results were similar when cases were restricted to those who had undergone carpal tunnel decompression [34].

With history taking we need to know more details as shown in the following:

1) Age and family history?

From literature studies it is concluded that CTS is most common in age group 30–60 years and with preponderance for the female gender [68, 128]. A family history is found among 20% of "proven" CTS patients especially if they have bilateral symptomatology [2]. Other authors have in contrast found an equal distribution among gender suggesting differences in screened populations. Interestingly it is shown that 14.4% of the Swedish population without any other signs of CTS complains of pain, numbness and/or tingling in the median nerve distribution [4, 5]. Increased occurrences of CTS near menopause and during last months of pregnancy emphasizes that secure information of the population screened is needed for a global discussion of gender preponderance. Male gender is not associated with poor outcome but many male patients are involved in heavy manual work activities, which is a poor prognostic indicator [5].

2) Is the history of symptoms short- or long lasting and work-related?

Did the symptoms start abrupt or were they as expected more continuous? Did we find a work history of force-presence, sustained or extreme vibration, awkward posture or repetition in work duties? A combination of personal and occupational risk factors is according to modern ideas the major determinant of CTS [12, 44, 100, 101]. Workers in cold environments e.g. butcher and poultry workers have higher frequencies of CTS than others [4, 101]. If the industrial work situation includes repetition, high force, awkward joint posture, direct pressure, vibration and prolonged constrained posture, including recently prolonged keyboard work, the risk for developing CTS is high. The diagnosis is best established using a combination of history and symptom distribution for screening for CTS in the industrial setting has questionable benefit [124]. Among blue-collar workers psychosocial factors seems more pronounced [5, 12]. By improving the consistency of the diagnosis of CTS these consensus criteria could lead to a more effective treatment and a better understanding of the effect of e.g. work place exposures in the development of this condition [38].

3) Does the history include subjective symptoms like numbness, tingling and/or burning sensations of the hand involving the distal median nerve distribution?

Sensory problems progressing gradually over months should be typical for CTS. They are affecting at least two of the first three digits and not involve dorsal- and palmar aspects of the hand should be typical for CTS [39, 58, 128]. Despite the fact that these first symptoms of a CTS may be pain in the wrist and hand and paraesthesia of the 3–4 radial fingers they can also involve the 5th finger. An explanation today is that extra-median spread of sensory symptoms is associated with higher levels of pain and paresthesia suggesting that central nervous system mechanisms of plasticity may underlie the spread of symptoms [133].

From a clinical history including 8223 patients with suspected CTS these were compared with the results with neurophysiologic findings [7]. Symptoms from the radial part of the hand and nocturnal exacerbation of symptoms showed the strongest individual correlations with positive nerve conduction studies. The regression model derived from the complete questionnaire achieved an overall sensitivity of 79% and specificity of 55% for the diagnosis of carpal tunnel syndrome. A simple regression model for evaluating the history compares favorably with the widely used clinical signs in its ability to predict the findings of nerve conduction studies. Similar fifty-seven clinical findings associated with CTS have been ranked previously in order of diagnostic importance using Delphi as a method of establishing consensus among a panel of expert clinicians. The 8 most highly ranked criteria were then placed into all possible combinations to create 256 unique case histories. Two new panels of experts rated these case histories. One panel made a binary evaluation as to whether the case history did or did not represent CTS. This allowed the development of a logistic regression model that had the probability of carpal tunnel syndrome as the dependent variable and the weighted diagnostic criteria as the independent variables. This model then was validated against the judgments of the second panel of clinicians who estimated the probability of CTS for each of the same case histories. The correlation between the probability of CTS predicted by the model and the panel of clinicians was 0.71. A methodology that emphasizes a rigorous approach to item generation and item reduction through expert consensus, followed by validation, may represent a template for establishing consensus among experts on controversial clinical issues [38].

4) What is the distribution and character of symptoms and are they worse with activity or at night?

Nocturnal painful paresthesia disrupting sleep being relieved by manipulation such as shaking of the hand should be linked with CTS [20, 118]. Patients with arthritis may also have the same symptoms but during daytime. The use of

neurodynamic tests for CTS is perhaps able to differentiate the diagnosis from other wrist and hand pathologies [19].

5) Does the patient feel a weakness of the hand?

The patients note that they often drop items and sense that the hand is swollen. Hand clumsiness is thus a frequent symptom and many females may have stopped knitting or sewing, as symptoms increase with wrist/finger movements [128]. Decreased muscle power e.g. grip strength due to changed long flexor tendon pulley function is seldom described in neurosurgical literature but plays an important role in orthopedic literature [81, 128].

6) Is there a trauma?

If a history includes earlier fractures of the arm, wrist or hand, pain during daytime, other atypical symptoms (polyneuropathy), a Swedish study advocates a supplementary neurophysiologic examination [41].

7) Hand-diagrams – self-assessment

Median nerve neuropathy creates paresthesia (subjective information) that we as physicians try to convert into objective signs. Many attempts have been made to categorize symptoms [55, 62, 118]. Among these are the best known "Katz Self-administered Hand Symptom Diagram" [56]. Similar has been developed "Michigan Hand Outcomes- and Disabilities of the Arm, Shoulder and Hand" (DASH) questionnaire, Patient Evaluation Measure questionnaire (PEM) [46] and the Boston – Self Administered Score System (BO) being a disease-specific questionnaire [96].

"The Katz Hand-Diagram" [56] is a personal patient screening procedure that documents the distributions of (subjective) symptoms for the Physician rather than one used for diagnosing the disease [55]. A correlation between PEM and DASH shows that the combination of subjective information and objective measures are high in PEM and DASH [46]. In a prospective series of 323 hands undergoing surgery for CTS a Boston self-administered questionnaire was used 1 and 6 months after CTS surgery. By grading the clinical and electrophysiological severity, it was found to obtain a valid, precise, reliable, and straightforward comparison of results from different patient series and different operating techniques [96].

Szabo *et al.* [118] validated three groups of patients. One group with CTS based on history, clinical presentation and with improvement following CTS surgical release. Group two included a variety of upper extremity disorders. Group three were normal healthy volunteers. All were submitted for a self-administered Hand-Diagrams, including validation of nocturnal pain, symptom duration, Phalen, sign, Tinel sign, Durkan test and monofilament testing before

and after a Phalen manoeuvre for 5 minutes. A uni-variate analysis of the first two groups showed that the most sensitive symptom predictor was the nocturnal pain. With a multivariate equation the probability to diagnose the CTS was 86% and that if all tests were normal there is only a 0.7% chance of having a CTS [118]. Patients under 40 years of age with normal or questionable Hand-Diagram ratings have thus a low risk of CTS [56].

A prospective study evaluated the DASH questionnaire by comparing it with the disease-specific Boston questionnaire (BQ). To measure responsiveness (sensitivity to clinical change), 57 patients with a clinical diagnosis of carpal tunnel syndrome completed the DASH and BQ preoperatively and again 3 months after open carpal tunnel decompression. A second group of 31 patients completed the questionnaires in the outpatient clinic and again 2 weeks later to assess test-retest reliability. The time to complete all questionnaires was recorded. Responsiveness of the DASH is comparable with the BQ with standardized response means of 0.66, 1.07 and 0.62 for the DASH, BQ-symptoms and BQ-function, respectively. Test-retest data show both questionnaires are reliable. Mean times to complete questionnaires were 6.8 minutes (DASH) and 5.6 minutes (BQ). The DASH questionnaire is thus a reliable, responsive and practical outcome instrument in carpal tunnel syndrome [40]. A combination of symptom diagrams, hypalgesia and thumb abduction strength testing were most helpful in diagnosis of CTS. All other signs (nocturnal paresthesia, Phalen and Tinel signs, thenar atrophy, 2-point-, vibratory- and monofilament sensory test) had little or no diagnostic value [20].

2. Clinical evaluation, ''objective'' signs

The clinical examination is linked with physicians evaluating the objective documentation of median nerve compression. Use of decreased sensibility findings will indeed increase the specificity of the disease CTS, but we will at the same time under diagnose CTS and exclude many patients with moderate CTS, who would benefit from treatment [79].

Clinical examination by an experienced medical doctor seems sufficient for screening purposes, if symptoms of CTS are found. Unfortunately many reports do not include methodology, which makes the results difficult to reproduce and thereby to apply to other populations [73]. It is in this context essential to standardize the clinical diagnostic criteria and develop a consensus for this testing [73, 92].

What are the most important objective signs?

1) Anatomical deformity of wrists and range of motion

Looking at the wrists, we may suggest that they are "square" meaning with an increased antero-posterior to medio-lateral wrist dimension, or long and

thin. None of these mean anything significant for the diagnosis of CTS. Do we see a swelling or masses e.g. tumours, ganglia, arthritis at the wrist? How is the perfusion state of hand? Does the wrist present with visible erythema? When we touch the wrist is it warm, do we feel crepitus [132]? We are now as investigators close to be subjective. We need to define what tests we use for these and how to demonstrate them, otherwise they are only for personal use?

2) How is the sensibility?

Sensibility changes involves sensory loss/reduction to pin prick, light touch, Semmes-Weinstein monofilament evaluation [117] and reduced two-point discrimination [4] are all modalities of interest in this context. How do we compare them is our major problem.

Two statisticians (A and B) were at scientific meeting small talking. A's wife stands 20 meters away. B says to his friend: "What a beautiful wife you have". A responds: Compared to whom?

Bear in mind that distribution of sensory symptoms may vary in many aspects [111] and can have several degrees of seriousness including, intermittent subjective paresthesia in the median nerve territory, to severe lasting sensibility disturbances. The literature suggests that 2-point discrimination has low sensitivity for diagnosing CTS. If a decreased sensation in the median nerve distribution is the most helpful finding in making the diagnosis, it will mainly help to diagnose the more advanced CTS cases. Similar hypalgesia to pin prick is important in the more advanced cases [20]. Sensibility reduction at finger 1–3 is often biased, as the technique of investigation may not be the same from one Physician to the other [117]. It should be stressed that the position of the hand/wrist is essential during the examination. If the sensibility testing is carried out subsequent to a Phalen test with flexion of the wrist it tends to be more abnormal.

We need to customise our sensory sense tests and carry them out with the patients blinded [112]. Many patients with open eyes will try to please the examining Physician by saying: "Yes – I feel a difference" in the area of "paresthesia". The cortical representation of the sensory functions of hands is not symmetric, however a distinct difference between 3rd finger and 5th finger is demonstrable. There may also be differences between left and right (dominant) hand based on cerebral function. Many studies do not have sufficient detail or include methodology to allow repetition of the protocol by other researchers. The sensitivities and specificities reported for each can be compared with the quality criteria ratings they each received [73].

We can thus perhaps increase the specificity, but not the sensitivity of a CTS diagnosis by simple clinical sensibility examination.

3) Muscle function

Thenar atrophy

The abductor pollicis brevis muscle may be congenitally absent and can also be difficult to assess in thick hands. It is a sign only seen in connection with severe long-lasting cases of CTS.

Motor paresis

Thumb abduction strength test is maybe a useful indicator for ruling out CTS [20]. Weakness of abductor pollicis brevis muscle is tested by pressing the palms together and then tests the abductor strength of the thumbs. This is a very biased investigation.

Grip and Pinch strength measurements

Measurement of grip strength may be influenced by pain. Monitoring grip strength is not generally accepted by neurosurgeons as a diagnostic tool. It serves mostly as a method to follow the individual patients course. It is widely used by hand surgeons because they are much focused on the hand movements. For the postoperative evaluation it is essential to know that testing with the wrist in a flexed position decreases the strength further due to tendon bowing. A consensus on how to monitor muscle function is badly needed.

3. Objective diagnostic tests – by tradition – but only partly objective

If we look more detailed into the different objective signs we may receive further information for our personal diagnostic scenario:

Neuro-provocative tests

We also use provocative tests in our clinical work where we try to induce e.g. sensibility disturbances as an objective sign. Through Medline, Current Contents, and related readings a critical review were undertaken [73]. The use of clinical diagnostic tests for CTS was further compared with the results of neurophysiology. Criteria for systematically reviewing the studies were developed, tested for reliability, and applied to the studies. Many studies did not have sufficient detail to allow repetition of the protocol by other researchers. The sensitivities and specificities reported for each could be compared with the quality criteria ratings they each received. This literature review supports that the use of the wrist flexion (Phalen-test) and carpal compression test (Durkan-test) has a low sensitivity for diagnosing CTS [95].

The goal in recommending a clinical examination technique for the diagnosis of CTS is both high specificity and sensitivity. The carpal compression tests (Durkan- or Phalen test) is therefore not a markedly better way to achieve this goal. The more severe the nerve compression is, the less sensitive these provocative tests seem to be [77]. By validating the 5 clinical tests Tinel, Phalen, reverse Phalen, carpal compression and vibration sense none of these had a degree of specificity that they could serve as an indicator of a CTS.

The efficacy of provocative tests for diagnosing CTS was evaluated in a Dutch study. Each test and them all in combinations did not show the probability of patients harbouring CTS. The authors recommends as a consequence of this to use neurophysiologic examination to increase diagnostic specificity [21].

Phalen sign/test

Phalen himself stated: "Positive sign is pain and paresthesia in the median nerve distribution with prompt exacerbation of symptoms when the wrist is held in a flexed position and the production of tingling in the fingers by percussion over the Carpal tunnel" [45, 117]. Is this how all of you have been taught to interpretated this test? I guess is that the answer is: No!!. Phalen-test is positive in among 20% of normal people [4]. One of the reasons may also be that the test is not performed in a standard fashion by different clinicians. It is highly questionable whether a test that does not harbor a strong positive correlation among different investigators can be used for diagnosing a disease – also in our situation with CTS.

Tinel sign

Originally it is called Hoffman/Tinel sign as it was first described by Hoffman [117]. It is elicited by careful percussion on the nerve starting distally and moving the tapping centrally. Almost everyone can have a positive Tinel sign; it only depends on the degree of percussion. It can thus in the literature range from being positive in 8–100% and in control patients it is positive from 6 to 45%. So the same questions as with Phalen sign can be applied here.

Durkan test

Carpal compression test where the examiner applies direct pressure on the carpal tunnel leading to symptoms of pain and paresthesia. Durkan's compression test had a sensitivity of 89% in diagnosing CTS – but it is suggesting rather than diagnosing [118].

Closed fist test

Holding fist closed for 60 seconds reproducing median nerve paresthesia [128].

Positive signs from the contra lateral wrist

Is also considered to suggest CTS in this situation as some 20% have bilateral symptomatology [2].

Complementary findings

Myofascial findings

A common finding among these patients. Many CTS patients have been treated for cervical spondylosis for long periods of time. The pain is believed to be a referred pain from the median nerve and may resemble a C5 or C6 radiculopathy [122].

Psychological evaluation

These testing are used if a result of treatment is not turning out as expected and if the symptomatology and clinical findings are not clearly linked.

The psychological testing must include history of employment, interpersonal relationships, leisure activities current perception of the medical system, results of current treatment, perceive locus of control and childhood history including abuse and family history of disability [2].

Laboratory tests

Serum rheumatoid factor
TSH for hypothyroidism
Fasting glucose and/or loading test
Serum protein electrophoresis
SR
Serum calcium, phosphorus, uric acid, alkaline and acid phosphates
Liver and kidney function profiles

A combination of the above-mentioned self assessment tests (Hand-Diagrams), history taking and a thorough clinical examination and laboratory tests are best to exclude those patients without median nerve compression e.g. for screening.

For categorizing our diagnosed CTS patients, we must use imaging and/or neurophysiology.

4. Imaging of the median nerve and carpal tunnel

Mainly four methods are being used to evaluate the carpal tunnel size and contents using imaging such as: – Radiography/Computerized Tomography, MRI, Bone-scan and Sonography [6, 30, 94, 131].

Radiography and computerized tomography

An additional part of a general evaluation is radiographic imaging of joints. Axial x-ray with the hand in maximal dorsal flexion and with the beam parallel to 4th metacarpal bone and 30 degrees off a perpendicular line of the film have been used to demonstrate the size of the carpal tunnel and signs of previous fractures. Standard X-ray imaging is hardly used routinely anymore in Europe and has widely been replaced by CT scanning techniques [15, 132]. In the hand-surgeons outpatient clinic painful wrists diseases are common and it is therefore in these situations suggested to use a systematic approach including use of imaging modalities such as bone scans and CT imaging, when diagnosing painful wrists [132]. Standard CT scan will not reveal many of the soft tissue abnormalities. Helical CT is in this situation more sensitive and can give us information of minor bony trauma and 3-D models of the carpal tunnel.

These methods have no value for a general "diagnostic" screening for CTS among a normal population. However they are still important in the Hand Surgeons Outpatient clinic.

Magnetic resonance imaging (MRI)

With MRI we can measure the volume of the Carpal Tunnel and visualize intrinsic structures related to it. Pierre-Jerome *et al.* [94] tested two groups of patients with rheumatoid arthritis (thirty one) and carpal tunnel syndrome (sixty two), and a group of asymptomatic controls (fifty four). All underwent bilateral MR axial wrist imaging from the metacarpal bases to the distal radio carpal joint. The imaging techniques included spin echo (SE), turbo spin echo (TSE) and fast field echo (FFE) sequences, using 3 mm-slice thicknesses. Different anatomical variants including hypoplasia of the Hamulus or hook of the Hamate bone (4 cases), anomalous muscles (lumbricals) inside the carpal tunnel (2 cases), unusual location (5 cases) and double branching of the median nerve (14 cases), and aberrant median artery (one case) were detected. These variants, if unfamiliar to MR readers and neurosurgeons, may be misinterpreted as pathological features [94].

Quantitative MRI is a valuable method for assessing the anatomic characteristics of the carpal tunnel [94]. Carpal tunnel areas are largest in neutral and smallest at the distal end with wrist flexion. An extended wrist resulted in the smallest carpal tunnel and content volumes as well as the smallest carpal tunnel content volume to carpal tunnel volume ratios. While men had significantly larger areas and volumes than women for both the carpal tunnel and it contents, there were no differences in ratios between the contents and tunnel size [10].

Earlier papers have concluded that nerve compression in CTS is proximal located in the carpal tunnel. This is in contrast with our present knowledge, where we show that it is in the distal part of the tunnel that the compression

takes place. Twenty-seven female patients with CTS and 28 asymptomatic female controls were examined with MRI of the wrists [94]. On the MRI axial images, the volume of carpal tunnels, the wrists and the thenar muscles were calculated bilaterally in all subjects. The values for the signal intensity of the median nerve from all wrists were also quantified. The carpal tunnel volume (CTV) and the wrist volume (MV)/CTV ratio were almost identical in both groups ($p = 0.36$ and $p = 0.45$, respectively). The focal narrowest point of the tunnel was located at its distal third, about 8 mm from the tunnel distal outlet. The median nerve in the patients were hyper-intense compared with the controls, $p = 0.037$. In another study bilateral MRI axial wrist images were obtained by means of turbo spin echo (TSE) and fast field echo (FFE) sequences. The mean (SD) length of the carpal tunnel, from inlet to outlet was 36.3 mm (SD = 3.4) [94]. The tunnel has a cone shape, with the proximal inlet constantly larger than the outlet distal in all subjects. The mean (SD) cross-sectional area of the tunnel inlet was found to be larger among women >45 years of age, compared to women <45 years of age ($p = 0.029$). The calculated mean (SD) volume of the tunnel also appeared significantly larger in the older group ($p = 0.023$) [10, 94].

MRI findings in 23 cases of CTS demonstrated perfect correspondence with MRI findings and operative views. Misalignment of the tendons was found in 20 cases and fibrous tissue deposits in 20 of a smaller series [13, 94].

What type of MRI images should be used? This is the decision for our Radiologists. The neurosurgeon must nevertheless be familiar with the different options. Inflammation of the Synovial sheets can be seen as low signal intensity on T1-weighted images and increased signal intensity on T2-weighted.

T1-weighted imaging with planes parallel and transverse along the nerve using phased-array coils defines the bony structures and the detailed anatomy of tendons and nerves (Fig. 6). Gadolinium enhanced T1 weighted imaging is used if we suspect nerve tumors. T2-weighted imaging defines pathology such as edema and ischemia of the median nerve [30].

The MR Neurography (MRN) is performed with high-resolution fast spin echo (FSE) and T2 imaging technique and often with suppression of the normal high signal intensity of fat (FLAIR chemical shift selection or inversion recovery). Flow sensitive sequences or dynamic contrast-enhanced MRI can detect circulatory disturbances within the median nerve where marked enhancement of the nerve is found with hypervascular edema and lack of enhancement if nerve ischemia. In later phases of CTS the internal nerve fibrosis will be shown as decreased intensity on both T1 and T2 images. Wrist flexion/extension can alter these patterns due to mechanical obstruction of blood flow. MRI imaging makes it a potential useful diagnostic tool for initial evolution and management but also for postoperative evaluation of patients with CTS [30]. If the patient is still symptomatic, we may see residual increase T2 signals of the median nerve and lack of complete section of the TCL.

MRI shows important changes in synovial tissue, excessive fat and lesions in the abductor pollicis muscle. In muscles that by electromyography have undergone severe denervation changes similar severe changes in the thenar muscles can be found using short tau inversion recovery sequences of MRI [13]. When dealing with apparent significant nerve dysfunction, the T2 weighted imaging of muscle fibers can be of interest, as they will remain normal if the lesion is neuropractic and show atrophy if we are dealing with axonotmesis.

Comparison with the other hand is not valid as bilateral CTS are common.

MRI documented disturbances and movements of tendons and median nerve can be found among patients with CTS. Patients with CTS will show proximal swelling, distal flattening and increased signal intensity of the median nerve combined with palmar bulging of the TCL at the level of the hook of the Hamate and Pisiform bone.

A Scandinavian study discussed the problems of validity and consistency of evaluations of MRI's in CTS. The conclusion was that better validated diagnostic criteria must be used in the future [91]. Modern techniques make it possible to specify location of nerve entrapments and muscle disturbances and it is only a question of time before MRI will be introduced in our diagnostic scenario.

Magnetic Resonance Imaging (MRI) is today less accurate than standard electro diagnostic testing as a diagnostic specificity test and is so far not directly recommended for diagnosing CTS in 2007 [13, 30, 57].

Nuclide bone scans

This includes injection of radionuclide that is absorbed by inflamed tissue e.g. connective/cartilage. It is sometimes used in the more complicated cases, but plays no role for the primary simple diagnosis or screening.

Sonography

The technique is simple, low-cost, non-invasive and easy to use (walker) with update equipment that include a high frequency transducer with a frequency of 7–13 MHz [6]. It may be an excellent adjunct in the diagnostic scenario. It can show exactly where the median nerve is placed which can be of interest for surgeons using endoscope methods for cutting the transverse carpal ligament. Transverse normal elliptical median nerve is visualized becoming more and more flat when moving distally. Sonography and MRI images were compared and it is concluded that sonography is acceptable for screening the carpal tunnel contents especially the shape of the median nerve was found to be equal demonstrable with sonography and MRI [15]. High-resolution ultrasound shows enlargement of the median nerve at the distal wrist crease in symptomatic patients and is a reliable modality for imaging the wrist in patients with CTS.

Nerve flattening in the distal tunnel, nerve swelling at the level of the distal radius and palmar bowing of TCL with a nerve cross sectional area greater than 9 mm^2 proximal is the best criterion for the diagnosis of CTS.

Median nerve cross-sectional areas were found to be larger in arthritic patients with CTS than in RA patients and healthy persons without CTS. This supports previous studies of idiopathic CTS in which increased cross-sectional areas have been found. Thus, as with idiopathic CTS, arthritic patients may be examined by US of the median nerve when CTS is suspected. Still today, sonography is mainly used for detecting space-occupying lesions in the tunnel [6].

Wrist arthroscopy

When the surgeon cannot identify where the wrist pain comes from it is possible to visualize inflammation and cartilage damages, to remove debris, ganglion cysts etc through minor endoscopes. This is a specialist investigation for Hand surgeons, not neurosurgeons.

5. Electrophysiology – neurophysiology

Neurophysiology serves both as a diagnostic categorizing factor and allows a possible prognostic validation for the neurosurgeon. It is a method that solely expresses the functional state of the median nerve alone within CT.

An electro physiologic or neurophysiologic examination consists of

1) Electromyography (EMG) testing muscle cell and end-plate zone function,
2) Electroneurography (ENG) testing nerve conduction and
3) Distal Motor Latency (DML)

For details the reader should consult textbooks on neurophysiology [61, 64].

1) Electromyography (EMG)

Describes the functional state of muscle cells. This is obtained by insertion of fine electrodes into the appropriate muscles. This may reveal a normal muscle cell function or show "Denervation potentials". These small size denervation potentials are normally clear signs of previous or ongoing neurogenic lesions with axonal loss = Wallerian degeneration.

Following relief of nerve compression, a typical reinnervation pattern is found often earlier than that by clinical examination [64].

Muscle fatigue can mimic a CTS. Compound Muscle Action Potentials (CMAP) can occasionally be used when screening between occupational and primary care cases of CTS [5].

2) Electroneurography (ENG)

This is the most important for the CTS diagnosis. It consists of electric stimulation of the median nerve at one site and recording the traveling potential at another place along the nerve across the potential lesion site. Sex, diurnal variations and handedness have no significant influence on conduction velocities [61, 64]. For clinical work, males present with milder symptoms but often more severe electrophysiological changes than females.

This nerve stimulation may be carried out with needles or as today surface electrodes [61] (Fig. 8).

Knowing the distance between the electrodes, the spreading speed of potentials = conduction velocity, can be estimated and measured in m/s [61, 64]. We can monitor both motor nerve conduction velocity (MNCV) and sensory nerve conduction velocity (SNCV). Sensory neurography responses have much smaller amplitudes measured in microvolt compared with the larger motor responses measured in millivolt (mV).

Slowing of median nerve conduction is due to demyelization of neurons. With this focal nerve lesion demyelinization a temporal dispersion of the stimulus response is found. It includes a fall in signal amplitude, an increase in its latency or a drop in conduction velocity. Axonal loss is very likely to be present if the recorded signals have reduced amplitudes. Amplitudes of both sensory and motor responses also tell us something about the number and synchrony of the fibers being tested. Most important for the diagnosis of CTS is measurement of the sensory nerve conduction velocity (SNCV). Our problems here are that in many instances we cannot detect the sensory potentials. Electronic noise may be a problem when recording the small amplitude sensory nerve action potentials (SNAP's). It is heavily influenced by electrode placements, especially if they are too close to each other.

Therefore practicing neurologists/neurosurgeons often rely on DML monitoring. We know that in 25% of cases with abnormal sensory nerve conduction

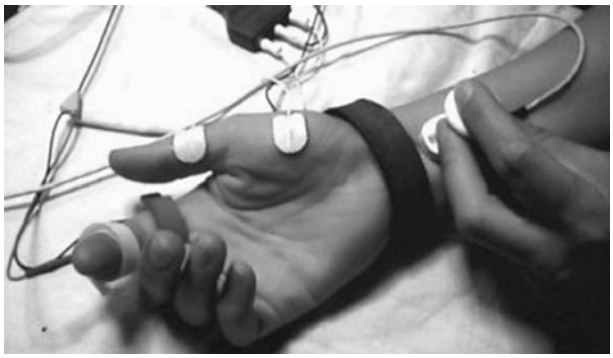

Fig. 8. Surface electrode set up for electrophysiological investigation of CTS

studies the motor studies are normal [61, 64]. It has been suggested that the following should be used as criteria for CTS syndrome: SNAP < 45 ms, however if conduction distance is >8 cm and SNCV is normal turn to new measurement over a shorter distance or compare with radial nerve SNCV or ulnar nerve SNCV [52]. Further the neurophysiology defines the median nerve involvement (A-fibres) more precisely and we can compare our neurophysiology-investigated patients in controlled prospective studies. Hand/arm temperature plays an important role as a change in temperature of 1 degree Celsius may lead to a change in sensory nerve conduction velocity of 1.2–2.4 m/s per degree Celsius [64]. Age also influences the nerve conduction velocity, which decreases after 60 years of age with 0.5–1.8 m/s per decade. Discussions on thresholds for abnormalities are different, but important for consensus [82].

Norwegian scientists uses photoplethysmography and laser Doppler fluxmetry in order to monitor autonomic nerve function [88]. They have showed that the micro vascular perfusion in fingertip skin and skin temperature are significantly reduced among patients with CTS in contrast to normal subjects.

Polyneuropathy is diagnosed by abnormalities in multiple nerves (ulnar nerve) and the presence of late F reflexes. F-waves monitoring occurs when a motor nerve is stimulated and impulses travel from the stimulation point to the neuron and back to the muscle. F-wave measurements reflect conduction along the entire nerve and are of general interest for diagnosing polyneuropathy and proximal lesions [61].

3) Distal motor latency (DML)

Distal motor latency (DML) estimates the traveling time of a stimulus potential from the site of stimulus to an electrode in/on a muscle. Therefore it includes measurement of the delay of impulses in the end-plate zones. Changes in DML can be found already as soon as one hour following operative decompression of a median nerve and it seems therefore to be a sensitive parameter.

In CTS cases DML is measured to the abductor brevis muscle. If DML preoperatively is >6 ms, the surgical results are good and fast. A mild improvement can be expected if DML is between 4–6 ms whereas nothing happens if it is <4 ms [102]. DML values in CTS cases have often been given with upper limits of 4.5 ms/8 cm, but the influence of distance between electrodes gives rise to a problems and interested readers should consult [64, 102] for further information.

Motor fibers are affected in CTS even when conventional electro diagnostic tests show normal motor conduction. Altered recruitment of motor axons could mainly be due to impairment of energy-dependent processes that affect temporal dispersion of the compound volley or axonal conduction block. In mild CTS, motor fibers are more often affected than was originally thought of.

If submaximal stimulus intensities are used, the sensitivity of wrist-to-APB motor conduction studies may be increased and used to document a beginning CTS.

Benefits and critical observations obtained from the use of neurophysiology

Clinical neurophysiologic investigations play a role in describing, defining and document whether a nerve irritation exists or not! – the latter perhaps being the most important. If a patient claims persisting symptoms following CTS decompression, the neurosurgeon can only deal with the question of potential insufficient operation if a preoperative electrophysiological evaluation is present.

Neurophysiology may – depending on who is referring physician – primarily be used for differential diagnostic purposes. When General Practitioners are referring patients it is mainly for diagnostic purposes, while neurological specialists are more interested in whether they may deal with polyneuropathy.

Additionally the correct (consensus) tests should be used [52, 62]. It is suggested that over 1/3 of Medicare patients treated for CTS in Washington State, US had an inappropriate electro diagnostic workup before surgery [115].

Single electrodiagnostic findings are therefore not recommended for routine use [97, 98].

Supporters of routine preoperative neurophysiologic investigations forget their shortcomings: lack of standardization, absence of population-based reference intervals, and lack of sensitivity and specificity. Only controlled trials, in which patients are randomized to receive treatment either with or without nerve conduction studies, will determine whether this investigation improve the outcome in patients with a firm clinical diagnosis of carpal tunnel syndrome. The influence of demographics is huge. The value of electro diagnosis has been challenged by Lee Dellon who stated: "There is no reason to deny surgery to a patient with a normal preoperative electrical study or to require all patients to have such a study, if the history and physical examination of the patient are themselves consistent with . . . . 'The dispute between WF Brown – a devoted neurophysiologist- and Lee Dellon, can be consulted in the discussion about the "WOG" syndrome (Word of God Syndrome) [14]. Therefore, if the neurosurgeon asks the neurophysiologist: "Do we have neurophysiologic indication for "surgery"? – They can only answer: "Yes and No".

In a patient group of normal youngsters we may not need neurophysiology to document a clinical CTS while it must be demanded if we are dealing with an outpatient clinic of hand surgery. 504 people from a "general population" were tested and it was found that 50 (10%) of these were awakening by nocturnal paresthesia in 93 hands (bilateral symptoms) [21]. Only 44 (47%) of these

hands had neurophysiologic signs of CTS. So with these false negative results we have again to accept that we have no precise definition of what the "general population" was.

Why use electroneurography at all the reader may ask faced with series of papers on the great value of Hand-Diagrams etc? The answer is clear; we need to document objectively what the causes of CTS symptoms are.

Our electrophysiological results can only be used in conjunction with the clinical situation [17]. The highly myelinized fibre lesions are rather easy to document. Neurophysiologic changes are definitely not significant nor specific in the milder cases of CTS unless special techniques are used [110, 115].

One of the key features of using neurophysiology is to make it possible to monitor median nerve function. Problems are also that all electrophysiological values obtained are based on local laboratories, local equipment and may occasionally include/exclude monitoring of hand temperature [64].

With this in mind, it must be accepted that a testing of the same person will show a 5% deviation by interpreter bias alone. For a given nerve conduction test value, post-test probability of CTS can be determined from the estimated pretest probability (derived from clinical data), interval likelihood ratios, and Bayes theorem [75].

Neurophysiology is possibly not needed if the chances of harbouring CTS are high. This in contrast to the situation where it is doubtful or low. Electrophysiological test data should be interpreted in a Bayesian context using the output of the diagnostic instrument as an estimate of pretext probability. The ability to report the electro diagnostic test results as a post-test probability may improve their precision beyond the current standard of establishing the CTS diagnosis based on comparison with a threshold for nerve conduction velocity [38]. Nerve conduction (NC) tests, using rigid cut-offs separating normal from abnormal test values, are commonly used to confirm CTS.

The authors [84] studied patients with clinically defined mild CTS and a normal median DML to determine: 1) How much sensory mixed NC test results increase (or decrease) the probability of CTS and 2) The NC test values required to confirm (or exclude) CTS for the range of pretest probabilities of CTS. Palmar, digit 4 (D4), and digit 2 (D2) median NC tests were reviewed in 125 hands with mild carpal tunnel syndrome (CTS) and 100 control hands with musculoskeletal pain. Receiver operating characteristic curves and interval likelihood ratios were plotted for the three tests. Using Bayes theorem, post-test probability of CTS was then determined for the range of pretest probabilities and NC test values. Receiver operating characteristic curves showed that for a set specificity of 97%, palmar and D4 studies had higher electrodiagnostic utility than D2 studies with cut-off test values (sensitivities of 0.3 ms, 64.0%; 0.4 ms, 71.2%; and 50 m/s, 44.8%). However, Bayesian analysis showed that to confirm CTS more conservative cut-off values (palmar 0.5 ms, D4 0.7 ms,

D2 44 m/s) were required for pretest probabilities 50%, whereas borderline abnormal values (palmar 0.4 ms, D4 0.5 ms, D2 48 m/s) sufficed when pretest probabilities were 75%. Conversely, normal test values could exclude CTS only for pretest probabilities <25%. For a given NC test value, post-test probability of CTS can be determined from the estimated pretest probability (derived from clinical data), interval likelihood ratios, and Bayes theorem. Use of rigid cut-off values to confirm CTS is problematic, because more conservative cut-offs are required for low pretest probability. Conversely, NC tests with sensitivity <95% cannot exclude CTS when pretest probability is high [84].

In a British study a scored questionnaire was used and an electrophysiological assessment given by two independent observers assessing a group of patients. The patients with CTS on either one or on both type evaluations were operated upon and the result e.g. symptom relief used as a standard for having treated a "true CTS". The predictive positive value of the questionnaire was 90% and for nerve conduction 92% [55].

Hand-diagram from questionable patients may witch an added positive nerve conduction velocity study, make the diagnosis more likely. Combined with a negative nerve conduction velocity-study there can remain a high percentage of possible minor CTS syndromes as we do not monitor the thin c-fibres [50].

How MRI data are correlated with pressure increase and neurophysiology is not yet completely solved, except for the fact that the more electro diagnostic abnormalities is found in CTS cases, the more severe disease can be expected in the nerve using MRI [30, 94].

Many attempts have been made for classification of types of CTS and include neurophysiologic findings, usually without lasting effect on our global discussion:

A *mild CTS* is often said to include a prolonged median sensory or mixed action potential distal latency. This may be so – but is not to me an absolute indication for surgery. However these are often operated upon because the results will be "good".

A *moderate CTS includes* usually abnormal median sensory latencies and prolongation of median motor distal latency both in both absolute- and relative values, comparing these with the asymptomatic "normal" hand. With clear-cut symptoms this supports the surgical indication because we have documented a nerve lesion.

Severe CTS shows both prolonged median motor and sensory distal latencies with absent sensory or palmar potentials and or low amplitude or absent thenar motor action.

Similar to this, CTS patients were described and combined with neurophysiologic values [59]. It was suggested that four degrees or stadiums of "CTS-disease" exists.

Summarized they are as follows:

Stadium 1

From a pathophysiological point of view, intermittent ischemia of sensory neurons is the basic feature of this initial stage of the disease.

Clinically these patients experience acroparesthesia, nightly painful paresthesia and pain provoked by handwork. By clinical examination and neurophysiologic investigations, we find no abnormalities. These are the so-called symptomatic but neurophysiologic negative cases!

Stadium 2

Clinical it is found that these patients' presents with added minor hypoesthesia. If the decreased capillary flow continues for longer periods of time more permanent changes of the endothelium will develop with secondary protein leakage from the capillaries, resulting in oedema within the nerve fascicles. The intra fascicular pressure will therefore increase, leading to a further internal compression to the nerve structures and a circulus vicious has developed. Chronic ischemia of sensory highly myelinized axons is the bases. This will result in a focal reduction of sensory nerve conduction velocity (SNCV) due to paranodal demyelization.

Direct mechanical distortion is the major factor underlying these more severe long lasting forms also seen following tourniquet paralyses.

In these early stadiums (1 & 2) of entrapment it is thus probably mainly a biochemical lesion caused by a shock like injury that leads to anatomical changes. Recent evidence suggests that ischemia may be primarily responsible for the milder type of reversible entrapment nerve lesions.

These findings suggest a stadium 3 leading into a stadium 4 with more significant structural disturbances inside the median nerve mainly involving the highly myelinized fibres.

Stadium 3

Clinical examinations reveal sensory disturbances and beginning of muscle atrophy and palsy of abductor pollicis brevis muscle and in the thenar muscle denervation potentials in the abductor pollicis brevis muscle is present. The developed axonal degeneration resulting in both decreased amplitude of sensory nerve potentials and a reduced SNCV. The painful paraesthesias are less pronounced in this stadium.

Stadium 4

Clinical examination reveals a persistent hypoesthesia/anesthesia in the distal median nerve distribution. Consistent with this less clinical pain and lack of the

nightly painful paresthesia is found. The loss of many axons and subsequent a significant reduced SNCV may end in a stadium with no functioning fibers at all.

Accepting these suggestions makes it even more confusing and difficult to make comparisons.

Conclusion

The neurophysiological investigation is a concrete attempt to monitor electric function in peripheral nerves. Use of electro diagnosis remains an important part of the objectification and evolution of peripheral nerve injuries [52]. It is not valid for screening patients with clinical symptoms of CTS unless very special information is needed.

We have to very carefully describe our findings and use them based on a universal consensus. Only in that case can we compare different patient groups. If we compare operations among patients with specific documented neurophysiologic disturbances with groups of patients with clinical "typical" symptomatology, it is not two populations defined the same way and it is like comparing apples and pears and no firm conclusions on treatment efficacy can be drawn.

Among patients presenting with hand paresthesia – the most common symptom – they must therefore for scientific purposes be divided in two groups of patients:

1) A neurophysiologic positive-group and
2) A neurophysiologic negative-group.

So when is neurophysiology needed? First and foremost neurophysiology is used for differential diagnostic purposes e.g. to document or exclude polyneuropathy. Secondly it serves for the clinician to be able to grade the degree of nerve affection both pre- and postoperatively.

Combination of electro diagnostic study findings and symptom characteristics provides us the most accurate information for classification of CTS syndromes as it may document the median nerve involvement [97] and thereby compare valid groups of CTS patients in the future.

Treatment of CTS

"*We may discus glioma treatment and aneurysm surgery but whenever you try to discuss the carpal tunnel, however, people get up and start hitting each other over the head with chairs. As always, this kind of emotionalism denotes a lack of data. If there is clear-cut data, there is no need to get emotional*".

Allan Hudson
Toronto Canada

1. Non-operative or conservative treatment

What is "conservative treatment of CTS"?

This is usually not clearly outlined in the literature, nor is the effects proven. From a Dutch Cochrane study it is evident that very little is known about the efficacy of most conservative treatment options and that high quality trials are needed to show their benefits [36]. By adding a conservative treatment such as splinting or rest or "wait and see" to a treatment protocol, the fluctuating symptomatology of CTS may "cure" the patient spontaneously through time. Another problem is that roughly 50% of CTS may be occupational and related to forceful grasping of pinching tools, awkward position of wrist, and vibrating hand-held tools [35, 72, 100, 101, 124].

The most important conservative treatment is therefore perhaps simply to avoid situations that provoke symptoms – prevention. Prevention is important if symptoms evidently are provoked by use of vibrating tools, or other working conditions that should of course be changed first. If a patient stops using e.g. vibrating tools = prevention, and simultaneously add another "conservative treatment modality" e.g. yoga or splinting at the same time, it is difficult to tell which these that is the essential. Among patients treated conservatively only 40% remained free of symptoms after 12 months [33]. Similar among milder cases – but all with an abnormal SNCV – only 35% were symptom free after 6 months.

In a resent Cochrane review [33] it was shown that patients did not obtain effect on their disease course from steroids, splinting, ultrasound, yoga or carpal bone mobilization [86].

Which are the conservative treatment options?

Job site alterations

Occupation with forceful grasping of pinching tools, awkward position of wrist, vibrating hand-held tools relates in approximately 50% of CTS. If vibrating tools is the cause, it is easy to reduce the use of them. If bicycling (Mountain Bike) with compression on the wrist is the cause, change posture so that the wrists are not loaded on the handlebar.

Ergonomics

Many CTS patients claim they have shoulder and arm pain too developed while they perform repetitive work tasks. Therefore it has often been advised that frequent break periods should be benefited but it has never been documented. Among normal people changing the sequence of active muscle fibres carries out these repetitive movements. Some patients cannot change their sequences

as normal persons do and this leads to muscle-fatigue and pain. They are the so-called "Repeaters" and they tend to use the same muscle pattern for the repetitive movements, whereas normal persons the "Replacers" change pattern. Much of the pain these patients develops based on repetitive movements is thus caused by changes in the muscles and not within the median nerve.

The frequency of carpal tunnel syndrome among computer users at a medical facility was 10.5% if they had the clinical diagnosis of CTS, which is almost the same as in a normal population [114]. When discussing CTS this fact may also influence our figures, as those patients that come to evaluation could easily be overrepresented by "Repeaters".

Nerve gliding promotion exercises

Athletes and swimmers seem to have a lower risk for developing CTS. Therefore it has been suggested that exercises may be benefited in preventing symptoms of CTS. These exercises include isometric- and stretching for both hands, and exercises for neck and rest of the upper extremity. They involve wrist circles, finger exercises are thus advocated but randomized trials are lacking and we have no scientific proof of its value.

Manual therapy

Can be used providing there is no contraindication. No controlled series exists.

Orthotics

Immobilization with splinting may have a short-term effect in milder cases of CTS [33, 86]. Splinting including nighttimes splints seems of some initial benefit. However this could also be a documentation of the known fluctuation of symptomatology among patients with CTS.

Yoga techniques

A group of patients treated with yoga were compared with a control group treated with simple splinting. The yoga group improved grip strength, reduced the wrist pain and normalized the Phalen sign whereas nothing happened in the control group. However the set up was not scientifically acceptable and there is still no scientific proof that yoga helps [60].

Medications

Vitamin B6 has often been advocated but no documented benefit is found. NSAID medication is also without scientific proven effect. In a CTS book you can find advises on how cream/jelly may help, just documenting how unscientific much of the actual advises on the Internet are.

Ultrasound

In a prospective trial ultrasound were CTS patients were compared with "sham" patients showed a possible short-term significant benefit. However we need more investigation before we can use ultrasound routinely. The effect postulated can easily be pure placebo function.

Acupuncture

Acupuncture with and without electrical stimulation has been suggested for treating CTS. How acupuncture functions is still not clearly understood. It influences central "pain" perception and this is therefore possibly the reason for some success for this treatment. We need controlled studies in the future for documentation.

Biofeedback

No scientific documentation of its value.

Micro-current TNS

Similar there exists no scientific proof of benefit.

Botulinum A (Botox®, Dysport®) injections

Resent information seems to indicate a direct effect on the muscle function, but a controlled study is needed before it can be recommended. If muscles are tense and painful it may be a choice. However we need again controlled studies to prove this point.

Corticosteroid injections

Local steroid injections are said to be effective and affordable as early treatment of CTS. Best results are obtained among mild cases whereas hands with severe symptoms will not respond sufficiently [33, 38]. With a follow up of 6–26 months, 22% were symptom free at the end after steroid injections and splinting for three weeks at best. Local injection of corticosteroids is better than oral intake and gives a 1–3 month benefit [71]. The major complication is direct injection of corticosteroids into the median nerve leading to severe axonal and myelin degeneration. Avoiding repetitive movements in combination with steroid injections helps less than 50% and relapse rate is 60% e.g. an overall efficacy <20% – but which one is the key to this effect?

Local anaesthetics injections

The most severe complication is direct injection on the nerve, which can lead to nerve necrosis.

2. Surgical – operative treatment

In the US with 285 million inhabitants a total of 400,000 CTS hands are operated upon each year equivalent to figures from Scandinavia [4, 5, 24, 38, 80, 107, 114]. These high figures make it therefore relevant to discuss the surgical techniques, as complications are possible with all types of surgery.

The aim of surgery consist – in our cases of documented median nerve compression – of the following:

"There is only one step in surgery for carpal tunnel syndrome – cut the transverse carpal ligament completely – and do not injure the nerve in any way" [48].

During a trip to Europe Allan Hudson observed that many of the surgical details he thought of as being mandatory was not that in Europe – so he concluded that we are all biased by traditions [48].

This TCL cutting can be obtained by three different methods e.g.

1) Open surgery with and without the use of magnification (OCTR), or by
2) Using an endoscope to cut the TCL (ECTR) or by
3) Performing TCL section blindly/openly.

The major surgical problem is not whether the surgeons use an endoscope or not. Most important to all kinds of surgery is the question: "Will the procedure relieve symptoms and are the complication rate low and insignificant"?

The major difference between an OCTR and ECTR procedure is that with microsurgical techniques we cut the TCL from outside the carpal tunnel viewing all structures in a normal 3-Dimentional (3-D) fashion. This is in contrast to the endoscope method where we cut the TCL from inside the carpal tunnel viewing it in a new 3-D fashion that first must be learned through regular practice.

Fundamentally all surgical methods lead to the same result, but the complications of the methods are different apparently being more severe among the endoscope- or blind methods. The complication rate and severity is essential when evaluating any surgical technique [1, 3, 7, 8, 16, 23, 109, 116, 121, 122]. When carrying out surgery we need some kind of anaesthesia and complications to this must be added to the surgical technique.

In surgery, the surgical-methods and the education of the surgeons are closely linked to each other. If the surgeons have been working with the microscope he/she may suggest the use of microsurgical techniques because he is used to it. If he/she works with endoscopes e.g. for knee surgery, a tendency to test how endoscope techniques apply to CTS is equally natural. Many OCTR series have proven the benefit of with a success rate of close to 95–99%. The general problems of the ECTR techniques are that we do not always visualize the nerve perfectly in the tunnel during surgery, and that we only partly view the TCL. No endoscope surgeon can therefore be 100% confident to cut the ligament completely with these techniques [122].

My biggest concern – being educated in a Scandinavian country – is that if the surgeon is working in a private scenario he/she may be tempted to operate more than if he/she is in a community hospital.

So before we look into the different surgical techniques, we shall focus on the neurosurgeon and his/her level of education.

Surgical training for handling CTS surgery

Although we all may believe that we (personally) do know what a good neurosurgeon is, it cannot be commented further than to: "A healthy, intelligent, tenacious, dynamic, psychologically intact and sincere candidate with manual dexterity, resistant to psychological and psychiatric stress" (Gilsbach, personal note).

Learning curves and the importance of learning surgical techniques

Anatomical knowledge of the region is naturally mandatory for the success of surgical procedures. Primarily the senior experts must therefore first dwell on anatomy and neurophysiology techniques so called declarative knowledge. That mistakes in interpretation of anatomy do occur is clear even from well-established journals (J Hand Surgery (Br)) demonstrating, for example, e.g. a drawing of the TCL erroneously being placed proximal to the hand wrist creases [27].

Secondly the seniors must also teach our residents to obtain the skills of surgery [65]. This includes in most curricula that we seniors supervise this "simple CTS operation" being carried out by our trainees. The trainee fills out a logbook and documents in this way that he/she has performed e.g. 5 or 10 CTS cases.

The serious complications must be regarded as the result of: "Careless or inexperienced surgery and the established principle of surgery under direct vision has provided reliable protection against disaster". In a non-published series from the Department of Neurosurgery, Aalborg University Hospital from 1997 we tried to document our departmental complication rate. We found to our surprise several complications in all operated by junior staff. Among 85 openly operated CTS hands we had 11 complications =13%, with the majority of complications being incomplete sectioning of the distal part of the TCL.

Proper formal training in both open and endoscope techniques must be obtained [27, 43, 65]. Steep learning curves within both microsurgery and endoscopy. The endoscope surgeon needs to become thoroughly familiar with the actual anatomy and anatomical relationships viewed within the carpal tunnel through the endoscope. The 3-D Vision in an endoscope is different from that of obtained from the operative microscope that is like normal 3-D vision.

Part of the endoscope training may take place in a virtual scenario [42] besides using cadavers. Two groups of endoscope surgeons were examined, the one being trained in a virtual environment. The experienced virtual trained group completed the procedures faster ($p > 0.001$). Similar they had less errors ($p = 0.006$) and a higher score on economy of movements ($p = 0.005$) than the inexperienced group. A 17% complication rate for 12 hand surgeons learning the endoscope technique on cadaver specimens illustrates these problems.

"Endoscope surgical technique requires rigorous training in order to avoid dangerous pitfalls: it must be performed by experienced hand surgeons". Chow and Papachristos [16] emphasizes that we need well-trained surgeons to perform these endoscopy operations. Many of the potential complications that occur during CTS surgery can be avoided if the surgeon has a good grasp of the anatomy of the carpal tunnel and its possible anomalies. The learning curve is important and the fact that one great Endoscope surgeon or similar micro neurosurgeon can carry out CTS without a complication does not invariably indicate that the rest of the younger lesser experienced surgeons will do the same.

Surgical techniques are being developed through training and time [43]. For microsurgery fatigue is important and some of us may be able to operate for hours without fatigue, others will not.

The more experienced surgeons "seniors" giving lectures and writing papers telling young trainees about new techniques – because we "invented" them. Many published surgical series are thus "personal" by neurosurgical experts and are thus not suited for generalizing – which is always happening even today. Complication rates for both open (OCTR) and endoscopy carpal tunnel release (ECTR) procedures are usually low in these "expert" papers.

Training courses in endoscope decompression are many – in open surgery few, probably because "it is so simple". Release of the median nerve for the treatment of carpal tunnel syndrome (CTS) can be one of the most straightforward and satisfying procedures performed by a neurosurgeon.

The new UEMS charter [113] emphasizes this and training surgical techniques is soon an integrated part of all surgical programs and is today part of the formal EANS training courses today (www.EANS.org).

Anesthesia used for CTS surgery

Performing surgery to day demands some type of anesthesia.

Anesthesia may be

1) Local infiltration anesthesia (LA),
2) LA or intravenous (IV), with and without use of tourniquet and
3) General anesthesia (GA).

1) Local anesthesia

Where the skin cut is to be made 4–5 cc Lidocain 1% infiltration is used. Be careful not to inject the anesthetics directly into the nerve as this may result in interstitial nerve necrosis [45]. Applying topical anesthetic cream reduces the pain of infiltration if patients are nervous.

During surgery haemosthasis is obtained by the local anaesthetics and by using bipolar coagulation [70]. All bleeding vessels must be carefully occluded whereby postoperative blood oozing is very seldom experienced. Using LA, the patient and surgeon can communicate during surgery and any abnormal feeling from the patient's side alerts the surgeon on a potential complication [16].

Postoperatively a small bandage will be sufficient and active movement of fingers instituted.

It is standard in hand surgery to recommend that the patients should keep the hand high e.g. above the heart for the first days but there is no documented proof of its value.

2) Intravenous anesthesia, or local anesthesia eventually combined with tourniquet

Hand surgeons traditionally use intra venous anesthesia combined with extremity exsanguinations necessary for a bloodless field. An Esmarch bandage is used to empty the extremity of blood, followed by inflation of a tourniquet above systolic pressure. This method is absolutely mandatory for dissecting tendons and synovial tissue. Regarding dissection of nerves, it is – to me – a lesser good idea. Why? The nerve will appear pale during surgery with the same color as the tendons and vessels. Thereby cyanotic color changes of the nerve or distended vessels on the nerves caused by the entrapment cannot be visualized. Mauer *et al.* [74] found by Visual impression of the median nerve among 1.420 open CTS operations that 1.312 (92.4%) had a clear nerve compression and that 85 (34.2%) of these had color changes = cyanosis of the nerve (Fig. 9).

As with the development of a neuropathy, bear in mind that tourniquet also leads to ischemic disturbances in nervous tissue, changes that increase with the time tourniquet is used. It is suggested that a tourniquet should only be maintained for 30–60 minutes = the "safe-time period". If the patient harbors a polyneuropathy this "safe time period" may not apply and reperfusion will not always lead to restoration of nerve damage caused by the tourniquet [76, 83]. Patients with significant increased pressure in the carpal tunnel before the operation and patients with polyneuropathy have therefore a greater chance that tourniquet lead to a secondary iatrogenic nerve lesion.

Postoperative hemorrhages in the operative field are more common following use of tourniquet. If the tourniquet pressure drops during the operation,

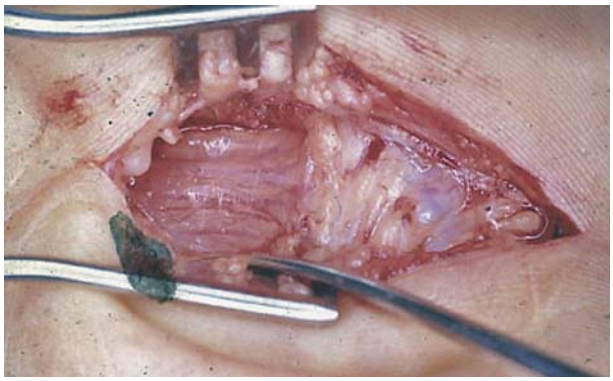

Fig. 9. Cyanosis of a compressed median nerve in the carpal tunnel

venous blood oozing may be a problem especially with endoscope techniques. Comparing blood oozing by the use of tourniquet and local adrenaline infiltrations simple infiltration of skin with adrenalin was superior to tourniquet [11].

So for neurosurgeons – a personal view – please forget all about using tourniquet. Our micro neurosurgical techniques and use of bipolar coagulation are sufficient for all types of open CTS surgery.

3) General anesthesia

"Virgin" operations can be carried out with simple local anaesthesia, but general anaesthesia may be indicated in cases of re-operations with significant scarring or if the patients are very nervous.

It is perhaps wise to begin with general mask anesthesia (Propofol® only) before attempting to perform the procedure with the patient receiving a local anesthetic as a supplement. Dealing with endoscopy surgery, the patients sense a certain degree of discomfort (and occasionally arm movement) when the obturator (endoscope) is inserted into the carpal tunnel. For this reason, many hand surgeons prefer general anesthesia with endoscope approaches.

Surgical techniques

1a) Open surgery (OCTR)

Many types of open surgery exist although they are traditionally thought of and classified as "one" in most clinical papers.

When correlating published surgical series, open surgery is often by mistake considered to consist of only "one procedure" despite that there is significant enough variation to classify open surgery as being several different procedures. Similar we always believe that patients with CTS are a uniform category. As you

understand from the previous this is not the case. Open surgical section of the TCL has been the gold standard surgical treatment for patients with carpal tunnel syndrome over the past 50 years. Cutting the TCL with a scalpel under direct vision produces reliable symptom relief in the vast majority of cases. However – despite this high clinical success rate – transient post-operative symptoms such as "pillar pain," scar tenderness, or hand weaknesses are known to occur [45, 48, 99, 109].

1b) The minimal open technique – ''Safeguard''

Limited open techniques with single and double incisions were introduced in 1993/1994 [134]. They involve both simple types and those with supplementary instruments to secure safe division of the TCL. A prospective study revealed that the single incision method gave better results in respect of grip and pinch strengths, whereas functional and symptom scores were not different. The Safeguard method seems promising. Another type of minimal open technique is the "Carpaltone" method. Basically this is just an OCTR with smaller skin incisions [134].

Before we discuss the different techniques please see my personal open technique description:

1c) The author's personal surgical technique
(learned and developed through a period of 40 years)

The goal for the surgery is viewed in Fig. 10.

The author uses local infiltration of the skin distal to the wrist creases with 4–5 cc of 1% Lidocain with noradrenalin. A 3–4 cm long incision is made with a 15 blade from the distal crease of the hand/wrist towards the interdigital space 3/4 (Fig. 11).

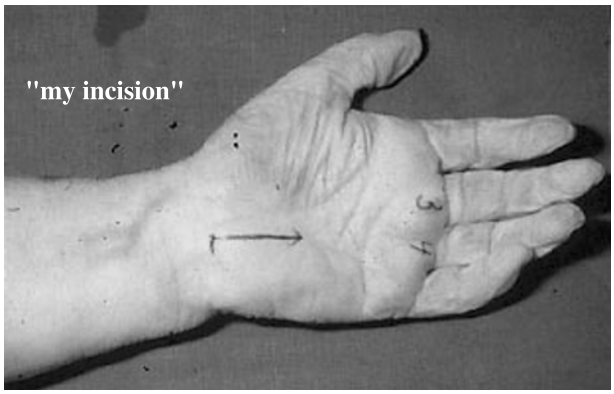

Fig. 10. ''My incision'' for CTS

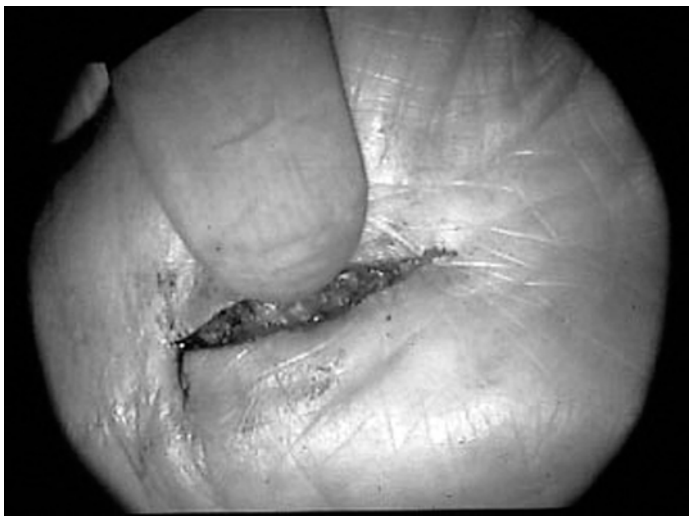

Fig. 11. Size of ''my incision''

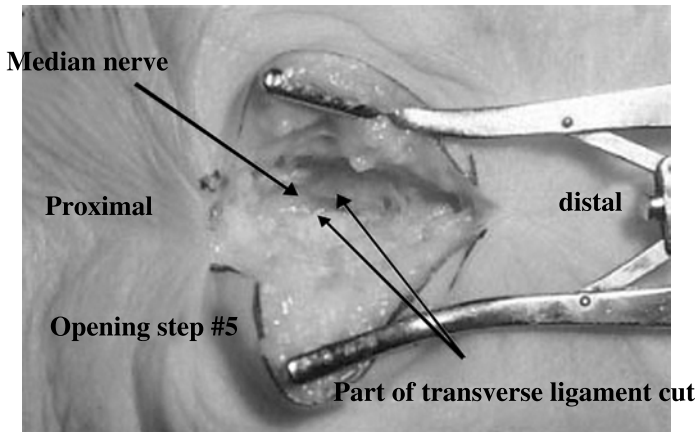

Fig. 12. The cadaver demonstration of the incision for CTS

Using the operative microscope and with a small self-retaining retractor, the palmar aponeurosis is visualized (Fig. 12).

Do not use too much power to spread the skin edges. The "safe zone" for incision of the aponeurosis and TCL is the ulnar part of the aponeurosis as the motor branch to the abductor brevis muscle usually leaves the median nerve from the radial side. The aponeurosis is cut longitudinally at the ulnar area. The

TCL is now found with its white transverse fibers. The TCL is opened with a fresh 15 blade.

When the contents of the carpal tunnel is encountered, the incision is carried further distally to the rim of the ligament whereby the normal fat in the hollow of the hand is visualized (Fig. 12). I do not touch the median nerve and do not try to se the recurrent branch of the median nerve. When the distal rim of the TCL is cut the palmar fat will protrude further and occasionally you may see the transverse vascular arcade pulsating. Then the proximal part of the ligament is cut and eventually part of the ante brachial aponeurosis – still keeping ulnar to the midline. You may lift the skin to observe this part of the cutting better (Fig. 13).

The palmar cutaneous branch of the median nerve is usually never seen with this approach. The palmar cutaneous branches of the ulnar nerve may be found and spared. Cyanosis of the median nerve is often visible where it is compressed (Fig. 9).

The use of internal neurolysis of the median nerve is to be abandoned.

Movements of the flexor tendons and the median nerve are then tested and observed. The end of operation is seen in Fig. 7.

The ligament edges are coagulated with low current bipolar coagulation and the skin closed in one layer with single 5–0 nylon sutures. There is no significant difference between using non-absorbable and nylon sutures for any of our outcome measures at the final follow-up. The wound is covered with a

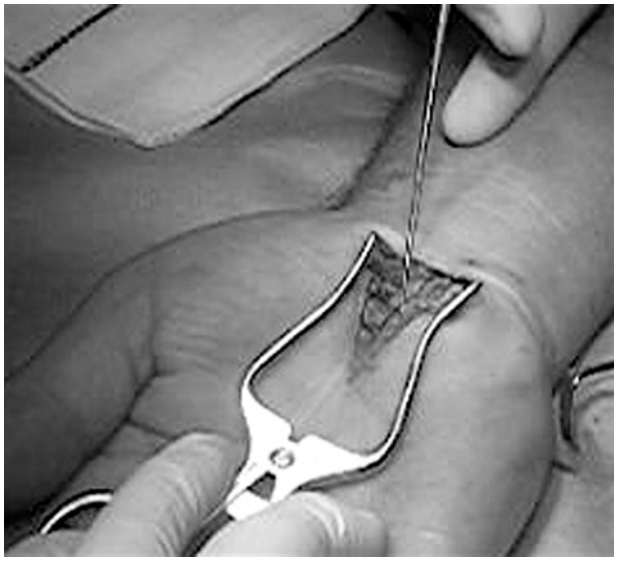

Fig. 13. The end of the operation, a hook is lifting the distal skin edge for better visualization of the antebrachial fascia

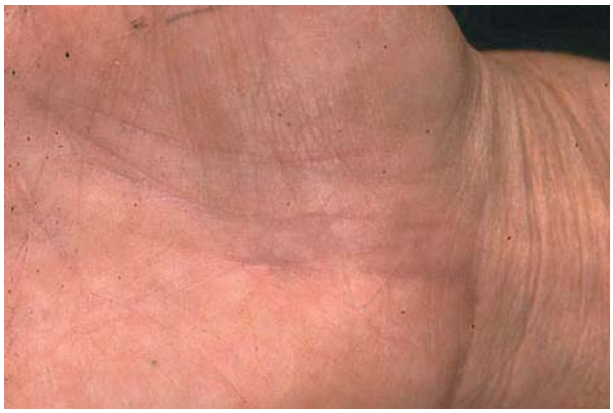

Fig. 14. Scar following OCTR after 6 months

band-aid and the hand bolstered leaving the fingers free for immediate active movements after surgery. The patient is instructed in using finger movements from day one. Skin sutures are removed after 12–14 days and movements of wrist begun after this, as early mobilization seems relevant.

End-result of CTS is not influenced whether immobilization or no-immobilization is used in the immediate post-operative period.

The scar will be minimal after 6 months as demonstrated in Fig. 14.

In a non-published consecutive series of 96 hands operated upon by two senior neurosurgeons at Aalborg University Hospital, Denmark in 1998, no complication was encountered with this technique.

2. Endoscope techniques (ECTR)

Application of endoscopy techniques to CTS treatment has not decreased operative expense, increased operative efficiency, or improved intraoperative visualization (compared with conventional OCTR). 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, 14, 16, 24, 26, 45, 54, 80, 87, 90, 103, 108].

The first endoscope procedure for transecting the TCL was introduced in 1987 and many modifications have been described since that [16, 48].

Single-portal techniques are those in which a single skin incision is made in the proximal wrist crease. Dual-portal techniques are those in which a second small incision is made in the palm when the endoscope/obturator has reached this area. Both methods require some degree of hyperextension and fixation of the hand during surgery.

Visualization of anatomical structure is of course of paramount importance when performing endoscope procedures. Blood obscures vision and extremity

exsanguinations with an Esmarch bandage followed by inflation of a tourniquet above systolic pressure is necessary to obtain a bloodless field. Even with perfectly planned endoscope surgery the surgeon must be prepared to change to an open type of surgery if anatomical landmark identification is not possible. If synovial tissue is prominent it may also hinder endoscope technique. As mentioned previously, the learning curve for endoscope surgery is very steep and it takes relatively long training to obtain mastery of endoscopes. Detailed knowledge of the complex anatomy of the anatomy of the carpal tunnel with respect to the related neurovascular structures is essential to perform safe endoscopy carpal tunnel release. Staying at the ulnar side of the long palmar tendon keeps the superficial palmar branch of the median nerve at a safe distance from the instruments. The "fat drop sign" is also a useful guide for the placement of the distal margin of the transverse carpal ligament, keeping the distal portal away from the superficial palmar arch. Synovial adhesions can usually cover the inferior surface of the transverse ligament, and they need to be removed for clear endoscopy identification of the transverse fibers before the ligament is cut [130].

We are basically dealing with 2 types of endoscope surgery (ECTR):

a) *One-port technique* includes those described by Okutsu, Agee, Menon, Worseg, and Jimenez [1, 16, 54, 80, 90, 121]

The original Okutsu technique was modified in 1989 so that the endoscope was inserted directly in the carpal tunnel on the ulnar side of the long palmar muscle tendon instead of starting extrabursal. A retrograde hook knife is introduced alongside the ulnar aspect and used – under direct visualization – to incise the TCL cutting from distal to proximal. Agee *et al.* [1] introduced in 1990 a technique with a pistol grip endoscope seen in Fig. 15.

Fig. 15. 1 pistol grip one-port endoscope

A window near the tip of the system made it possible to view the undersurface of the transverse carpal ligament through the endoscope when inserting it. A small 2 cm transverse skin incision is used positioned ulnar to the palmar tendon at the distal wrist crease. The ante brachial fascia is incised and the endoscope is passed into the carpal tunnel till the distal edge of the ligament is viewed. A cutting blade is then inserted and cutting of the ligament takes place from distal to proximal viewing the ligament but not the median nerve with this technique, the cutting blade cannot always be visualized when cutting [1].

The success rate of one-port techniques reported in published papers was 96.2%, with a complication rate of 1.83% and a failure rate of 1.44% [90].

b) *Two-port technique*: includes lesser different types

With the two portal techniques the introducer/obturator is passed somewhat blindly through the carpal tunnel through a similar small transverse cut in the skin crease and a supplementary contra incision is made in the hand palm where the tip of this introducer pushes the skin from the inside. The original Chow technique included an extrabursal approach to the carpal tunnel with gentle retracting the flexor tendons. The canal is visualized and it is secured that no tendons or nerve are in the field view. Then the endoscope and a probe are inserted in the inserter from the distal end of the carpal tunnel in order to cut the ligament with a forward movement. The custom designed instrumentation protects the median nerve and flexor tendons, and positioning of the slotted cannula through the two portals ensures a stable surgical environment [16]. This technique was modified so that the surgeon inserts a hook knife via the proximal port and advances it behind the distal end of the TCL. The TCL is caught by the knife and the ligament is cut in a backwards pull [14].

The option for all types of CTS operations is to reduce pressure in the carpal tunnel by increasing its size. In order to obtain e.g. a 6% increase of carpal tunnel volume [94], you introduce an endoscope. The size of an abnormal carpal tunnel (length 4 cm) is $238.9 \times 400 = 95.560\ mm^3$. A 6% change in this volume is $5.734\ mm^3$. The size of this endoscope of 3 mm diameter is: $400 \times 2 \times 3.14 \times 1.50 = 3.768\ mm^3$. So we actually introduce a devise that has a volume close to what we want to obtain in reduction and we do that in a situation where the pressure in the canal is increased. How will pressure in the canal be influenced by ECTR? 20 patients had surgery for idiopathic CTS by one-portal Agee endoscopy section of the TCL (MicroAire, Charlottesville, VA). With a special transducer pressures were measured and were all elevated initially. The pressures were maximal (mean 93 mmHg) with full passive wrist extension. Peaks of high pressures, on average 97 mmHg, were recorded with the Agee endoscopy device in the canal. Release of the endoscopy TCL

resulted in a marked decrease of the pressures [108]. The single-portal ECTR does not seem to influence the median nerve excursion for the wrist positions studied in patients with carpal tunnel syndrome. The results from an in vivo study showed longitudinal gliding of the median nerve being twice as great as in vitro studies.

Although the potential decreased palmar tenderness, better preservation of grip strength, and earlier return to work associated with ECTR are very noteworthy, these advantages may still be negated by the risk of neurovascular and tendon injury. Many surgeons remain skeptical about the safety and reliability of ECTR. Today >5.000 patient hands have been treated with the 2-port technique with an average success rate reported of 98.3%, a complication rate of 1.87% and a failure rate of 1.44%.

If the surgeon is unfamiliar with the actual technique or the anatomy he/she may easily injure the nervous structures and create severe failures [27] According to recent studies, the overall complication rate is in the range of 1–2% in experienced hands for both ECTR and OCTR surgery [122, 127, 129].

For the author it is still philosophically difficult to accept that despite we base our surgical indication on an increased pressure in the tunnel (small space). We add hyperextension e.g. reduces this carpal tunnel volume and simultaneously increase pressure even more performing endoscope surgery.

3. The blind ''Paine'' retinaculum technique

This was introduced with the purpose of cutting the TCL without opening the skin in a broad manner. After opening the ante brachial fascia ulnar to the palmar tendon, a retinaculatome instrument is blindly passed down the carpal tunnel with its foot under the ligament cutting the ligament simultaneously [89]. Its movement leads to a "characteristic sound" telling the surgeon when the ligament is cut. In 0.3% of cases an incomplete divisions of the TCL was found. In another 4 cases a palmar haematoma were discovered. Supplementary technique is – after having cut the ligament – to pass a light source down the carpal tunnel and watch light passing through the skin indicating whether a complete cutting has been achieved. This method is hardly in use to day in Europe.

Complications to surgical treatment of CTS

Complications to CTS surgery are frequent [1, 7, 14, 45, 67, 90, 96, 99, 122]. Despite that the complication rates sited in the literature seems significantly underestimated. Complications are many and rank from simple postoperative wound infections to median nerve laceration. The main advantages of the ECTR techniques are considered to be minor postoperative pain and a more rapid postoperative recovery. The rate of other complications (reflex sympathetic

dystrophy, haematoma, wound problems, etc.) was about the same with endoscopy as with open release [8].

Disadvantages are thought to be the impossibility of a direct median nerve neurolysis and a higher and more severe surgical complication rate, including injury to the median nerve and vascular structures with profound physical and psychological sequel [32, 45].

The frequency and severity of complications have increased since endoscope release began [24]. This is not based on the technique used, but mainly on the lack of education in new surgeons.

Most common complications are incomplete release of the TCL or postoperative scarring with both OCTR and ECTR [23].

A survey of the most common and serious complications to open surgery (OCTR)

The operative mistakes/complications using open surgery are mainly:

1) Recurrent CTS due to inadequate cut of the transverse ligament.
2) Erroneous decompression of the ulnar nerve instead of median nerve.
3) Direct surgical lesion of the median nerve.
4) Direct surgical lesion of the recurrent motor branch of median nerve.
5) Lesion of the palmar cutaneous branch of median nerve.
6) Reflex Sympathetic Dystrophy (RSD).
7) Hypertrophy scar.
8) Hypersensitive scar.
9) Pillar pain.
10) Dysaesthesia.
11) Injury to superficial vascular arch.
12) Wound infection.
13) Decreased grip strength.
14) Decreased wrist movements.

Supplementary complications to the endoscope methods (ECTR)

The same as with the open method +

15) Lesion of the motor branch of ulnar nerve,
16) Lesion of ulnar nerve in Guyon's canal.
17) Lesion of the Berrettini branch between ulnar and median nerves.
18) Pseudo aneurysm on superficial arch.
19) Cutting of median nerve.
20) Significant lesions of digital nerves.
21) Lesions of tendons to the superficial digital flexor 4–5 muscle.
22) Deep hand space infections.

Discussion on surgical techniques

Many reasons for inadequate surgical results are found – incorrect diagnosis, inadequate decompression, iatrogenic compression or direct nerve injury. Most clinical papers on CTS results are unfortunately typical retrospective studies without necessary precise pre- and postoperative information. They simply do not give us a possibility to neither validate results by time nor by precision of the used operative techniques. When we read these papers information is found regarding "good" results.

What is a ''good'' result?

When we discuss results of CTS operative treatment we must decide what we in fact mean by a "good" result?

Most patients will be operated upon due to significant painful paraesthesiae. Relief of these will be "a good result". Shapiro [109] found that the mean time before patients returned to work was 6 weeks e.g. also good results, just a different way of looking upon results.

How do we handle information of transient paraesthesiae as the referral symptom?

If symptoms disappear fast the overall prognosis is "good". However, if this result is based on a lesion of the median nerve with subsequent absent sensibility, it seems to be a "good result" with some reservations. Total relief of pain and nightly paresthesia is generally obtained within the first postoperative 24 hours in close to 90% of cases.

If the patient becomes free of pain very fast, but that these returns 6 months later? So we also need exact information on follow-up periods. Among patients with hard hand labour postoperative problems are found in up to 80% – but it is important to remember that "time heals". After 6 months only 2% had pain and hypersensitive scars and 5% pain in hand and wrist after OCTR. Pagnanelli and Barrer [89] found what he called good results among 90% of patients operated upon but only 81% among a group of diabetic patients. Again we lack information on the demographics in this paper.

What about age? "The surgical results among elderly patients had significant better results among men >70 years of age compared to non-surgical treatment". What shall we do with a statement like this?

If the primary symptom is atrophy of thenar muscles, is it the normalization of muscle function? Patients in later states of CTS with significant loss of sensation due to axonal injury cannot expect full recovery in years [3, 4, 8, 16, 24, 67, 74, 99, 103, 109, 121, 122].

So, we must accept that the multitude of symptoms and dysfunction makes it very difficult to decide what a good result is e.g. we need a consensus, and we need well designed prospective studies in the future to deal with these questions.

Similar we may ask:

What is a surgical ''failure''?

Surgical failure rates are reported to be from 2–31% and in conservative series from 1–50%. These huge variations indicate to me that we have no firm consensus definition of what a failure is.

Major, if not devastating, complications can and do occur with both OCTR and ECTR [90]. Surgically treated CTS complications of endoscopy and open carpal tunnel release over a 5-year period were sent to members of the American Society for Surgery of the Hand to assess and compare major complications of the 2 procedures [24]. The 708 respondents treated a total of 455 major complications from ECTR. These included a total of 100 median nerve lacerations, 88 ulnar nerve lacerations, 77 digital nerve lacerations, 121 vessel lacerations, and 69 tendon lacerations. There were similar 283 major complications from OCTR treated by 616 respondents, including 147 median nerve lacerations, 29 ulnar nerve lacerations, 54 digital nerve lacerations, 34 vessel lacerations, and 19 tendon lacerations. The conclusion was: That there is no difference in complication rate between open and endoscope surgery. Although this is a retrospective voluntary study with resultant methodological flaws, the data support the conclusion that carpal tunnel release, be it endoscopy or open, is not a safe and simple procedure. Only 15% OF CTS operations in this 5-year time periode in New York were ECTR. Never the less they resulted in 46% of the nerve laceration complications in the whole series. This implies that ECTR surgery in fact had more severe complications than OCTR [50].

A surgical failure is it just a situation where an expected result is not reached? Have we in all these cases before treatment was instituted defined the symptoms and the expected result? I doubt it. Is it a so-called "bad result"? or is a failure a case with a direct complication? – Something unexpected happening?

In a prospective series of 378 patients 97% improved their motor weakness and only one patient showed worsening [109]. Is this worsening a failure or a complication? If the recurrent motor branch was cut resulting in the worsening of muscle power it is a surgical complication and not a failure of the operation.

Two large, prospective, randomized, multicenters, clinical trials compared OCTR and ECTR methods emphasize the potential benefits of ECTR [1, 14]. Agee *et al.* [1] showed in a randomized, prospective, multicenter study of 147 hands (65 OCTR patients vs. 82 ECTR patients) that the median time to return to work was 21 days shorter in the ECTR group than the OCTR group. In this series it was said that the best predictors of return to work were lack of incision tenderness and good return of grip strength. In the ECTR group three complications were found: one incomplete release of the ligament and two transient ulnar nerve neuropraxia. In the OCTR group four complications were described: two wound dehiscence, one bowstringing of the flexor tendons, and

one injury to the deep motor branch of the ulnar nerve. Brown *et al.* [14] found in his randomized, prospective, multicenter study of 160 hands (82 OCTR patients vs. 78 ECTR patients) that the median return to work time was 14 days shorter in the ECTR group than the OCTR group [45, 90, 121, 127].

An analysis of 54 publications, reporting a total of 9516 endoscopy and 1203 open releases showed the rate of irreversible nerve damage to be 0.3 and 0.2%, respectively. Reversible nerve problems were more common after ECTR [8].

How can we scientifically compare these results? We lack besides demographics with precise definition of working conditions, information on social structures, and consensus on what we base our "good" results on.

How to avoid the most common and serious complications?

Direct complications can be related to the skin incisions, inadequate sectioning of TCL and injuries to tendon, nervous and vascular structures. There may also develop scar tissue in and around the median nerve. Pain symptoms may develop locally or as part of a RSD.

Problems with skin incisions

What type of skin incision should be used?

The skin incision used in order to reach the TCL, can have many shapes and lengths [45, 48]. Most important is that it allows full visualization of the entire carpal tunnel contents and the whole TCL ligament. The length of the skin incisions may vary between 2 and 8 cm. The skin incision can be located from the forearm crossing the distal wrist crease and end at the midpalm. The incision is positioned along the axis of the third web space, towards the third or fourth finger or along the thenar crease. The most common longitudinal incision is aiming towards the interspace between the 3rd finger, and 4th finger. The shape of the incision may be curved, straight, sigmoid, or any combination thereof. The skin incision should not cross the wrist flexion creases as this may lead to hypertrophy scarring. Six months postoperatively only 2% have hypersensitive scars. If a painful hypertrophic scar should occur a complicated revision with a Z-plasty may be needed.

If the incision is placed towards the ulnar territory of the hand, the surgeon can easily by mistake enter Guyon's canal with the ulnar nerve and artery. If it is placed too radial a lesion of the palmar cutaneous branch of the median nerve may happen and severe neuromas pain and reflex sympathetic dystrophy (RSD) may be the result. The Palmar cutaneous branch of the median nerve emerges some 6 cm proximal to the wrist and runs parallel and on the radial side with the median nerve and the long palmar tendon, some 2 cm proximal to the proximal border of the transverse carpal ligament [119] (See Fig. 3). With

any incision made in the palm it is likely to injure the small terminal cutaneous branches of the palmar cutaneous nerves. Even though an incision based on the axis of the ring finger may reduce the incidence of this nerve lesion, there is no true "inter-nervous plane" that will completely avoid all cutaneous palmer branches, whether of median or ulnar origin. Injury to the large palmar cutaneous branch is probably the second most commonly cited complication in OCTR. After section of this nerve a subsequent neuroma formation occurs. The result is a reduced sensibility in palm and persistent neuromas. We are now faced with inferior treatment options, burying the neuromas in the forearm muscles or to cut it. Neither of these options have had a high success rate [67].

Hands are very different and very large, bulky hands are found among acromegalics and hard working people. In this context, the Kaplan Cardinal Line is used by hand-surgeons for identification of the distal part of the TCL. Testing hand-surgeons knowledge of this line it was shown that it varied where they thought it to be. Kaplan's cardinal line does not locate the deep structures of the hand accurately but may assist in making palmar incisions. Before the surgeon reaches the TCL the palmar aponeurosis and palmar minimal muscle tendon is found and cut longitudinally. Persistent minimal incision tenderness is present in 61% of OCTR patients versus 36% of ECTR patients at 12 weeks follow-up.

Transverse incisions are abandoned as the surgeon will injure the palmar cutaneous branch and give an inferior view of the operative field. It was popular many years ago but forces the surgeon to cut the TCL blindly and injuries to vessels and nerves were common. Analysis of the OCTR CTS literature reveals thus great variability regarding the location, length, and shape of the skin incision [45, 48].

The incisions used for ECTR are as invalidating as the small used for open surgery. The proximal incision for the 1-port techniques may also injure cutaneous branches. Its length is roughly 1.5–2 cm. In the 2-port technique we must ad the distal incision of 1.5 cm so the total length is 3–3.5 cm. The length of the standard incision I use for OCTR is about 3–4 cm (Fig. 11) and it cannot be seen after 6 months (Fig. 14). In an attempt to minimize openings, a short (approximately 2 cm) incision may be used as part of a mini OCTR operation technique [134]. No statistical difference in scar length, scar tenderness, rate of complications, or length of time before return to work was found in a prospective series of 71 patients undergoing OCTR surgery through an average 2.1-cm incision versus 66 patients in whom an ECTR two-portal technique was used. The minimal-incision OCTR technique can thus achieve the same low incidence of incision tenderness [47, 134].

These historical types of incisions have all been discussed in details by Hudson *et al.* [48].

Visualization of carpal tunnel – use of magnification

With a long microsurgical experience I cannot understand why all surgeons performing CTS do not use magnification. Even loupes are better than normal vision. I always use an operative microscope as seen in Fig. 16. The use of visual magnification during the operative procedure is optional to prevent complications occurring due to anatomical variations of the recurrent motor branch. Chances for recognizing a wrong dissection, aberrant nerve structures and vessels are greater using an operative microscope and lesions of larger cutaneous nerves are seldom found with microsurgical techniques.

In addition to general better vision it is far easier to make precise bipolar coagulations and thus obtain perfect haemostasis.

The better visualization has the benefit of demonstrating the TCL far better before we cut it. The main reason for incomplete operations with postoperative pain and unchanged symptoms is from all series incomplete sectioning of the TCL distally. Because of the potential for neurovascular and tendon injury, most endoscopy surgeons agree that if the transverse fibres of the TCL cannot be visualized along its entire length, the endoscopy procedure should be converted into an open one. Inferior visualization of the anatomical structures leads to a similar problem.

Haemostasis

Blood oozing is controlled by bipolar coagulation at a low setting. Postoperative hemorrhage is virtually never seen [70]. Whether this is a problem or not

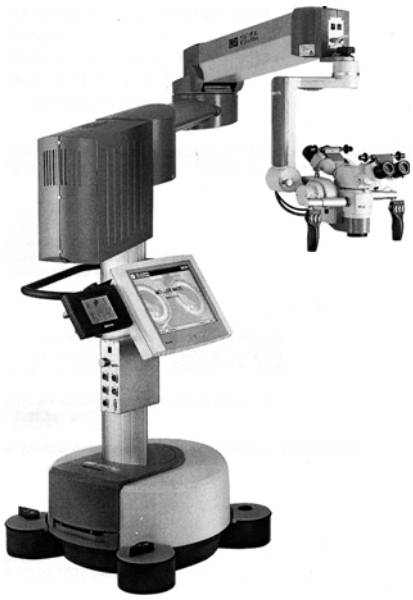

Fig. 16. Möller-Wedel operative microscope HiR 700; www.moeller-wedel.com

can be discussed, but the hand surgical wide use of exanguation working in a bloodless field is not only a tradition but also serves to reduce blood oozing. Braithwaite *et al.* [20] examined the use of tourniquet and local adrenaline infiltrations and found that simple adrenalin was much better than tourniquet to prevent blood oozing from skin. Postoperative hemorrhages are more common following use of tourniquet. If the tourniquet pressure drops during the operation, venous blood oozing may be a problem [11]. A lesion of vascular structures may lead to wound haematomas and in one OCTR series 1.4% haematomas was found [14]. The major problem besides inferior surgical vision is that blood can result in tendon and median nerve adhesions.

TCL cutting

Most common complication in all types of CTS surgery is inadequate cutting of the TCL. The cut in the TCL is usually straight [48]. It is appropriate to transect the ligament over 4 mm apart from the lateral margin of the hook of the Hamate without placing the edge of the scalpel toward the ulnar side. We would also recommend not transecting the TCL in the ulnar flexed wrist position to protect the ulnar neurovascular structure [53]. A cut like a flap with secondary reconstruction of the ligament has been tried but failed. Similar there is no identifiable benefit in lengthening the TCL when decompressing the carpal tunnel. The TCL can then be cut by a knife or by scissors. Many surgeons insert a Mickey probe in the carpal canal and cuts the TCL using this probe as a protection of the median nerve. In some cases an injury to the recurrent motor branch to the abductor muscle can occur especially if the distal part of the ligament is cut "blindly" with scissors [67]. Cut of the vascular arcade by mistake is also possible if the cut is made blindly. If, in an attempt to avoid the median nerve, the surgeon cuts through the ulnar side of the TCL, the motor branch of the ulnar nerve may be injured. Similarly, if the surgeon blindly cuts the distal fibres of the TCL in a radial direction, the third common digital nerve may be injured.

Immediately after surgical release of the TCL, there is a marked decrease of the carpal canal pressure. During the second postoperative month and persisting after 12 months the pressure arises again but stay inside normal ranges. These findings suggest that the TCL reconstitutes by normal scar formation, but with some lengthening [106]. This is also visualized on MRI where the carpal tunnel contents may show significant postoperative alterations including displacement of flexor tendons [94].

The potential for neurovascular and tendon injury, most endoscopy surgeons agree that if the transverse striations of the TCL cannot be visualized along its entire length, the endoscopy procedure should be converted to an open one. This is in accordance with the fact that incomplete section of the TCL is the most common complication to OCTR and ECTR. With OCTR twelve cases of incomplete release of the ligament, constituting 35% of the total 34 com-

plications were found in 186 patients [67]. In another series 67% of patients with persistent symptoms had incomplete section of the TCL. Two hundred patients with recurrent symptomatology were reoperated during a 2 years follow-up period. In 108 cases (54%) the TCL had been incompletely sectioned and among these 46 (43%) the median nerve was fixed by simple scar tissue and with circumferential scarring among 17 (16%) of these [116].

With ECTR we are dealing with three types of incomplete release:

1) release of Guyon's canal,
2) incomplete distal ligament release (as in OCTR), and
3) incomplete central (superficial) ligament release.

It is thus somewhat more complicated to cut the TCL with ECTR than the simple OCTR. Incomplete ligament release in ECTR ranges from 5% to as high as 50% in cadaver studies.

Infection

Superficial skin infections occur as in all types of surgery and should be treated accordingly. They are seldom constituting some 0.5% to 6%. Deep infections is also found especially if surgical drainage is used, with prolonged operative time and if attempts to perform tendon synovectomy is undertaken. In the endoscope surgery, deep infections are serious and have been reported.

Pillar pain

Linked with the skin incision is "Pillar pain" an ill-defined pain in the thenar and hypothenar eminences aggravated by gripping.

Its etiology remains obscure. Cutting of the sensory nerve fibers supplying the palmar brevis fascia and resulting micro neuroma formation is one explanation. Other possible mechanisms include widening of the carpal arch and realignment of the carpal bones. It has been stated that pillar pain is more common if incising openly in the palm but has never been proven in a randomized prospective trial. Moderate or severe pillar and scar pain is common in literature, occurring in 25% of hands after surgery, but only in 4% by the 12th week and 2% by the 25th week of follow-up [96]. Sometimes this pillar pain may hinder functions among workers with heavy hand loading jobs. Among patients with hard hand labour postoperative problems are thus in some series found in up to 80% – but it is important to remember that "time heals".

Tendon adhesions

Tendon adhesions are not common in a neurosurgical practice. Tendon adhesions may result from poor haemostasis or bleeding from tenosynovectomy.

Resection of the synovium is usually only indicated in cases of extremely bulky synovium as we see it with Rheumatoid Arthritis.

Physical therapy with range-of-motion exercises and dynamic splinting rather than tenolysis surgery is the best treatment, but not randomized, prospective studies exists.

Lesion of vascular structures

It is most common with ECTR and among untrained surgeons. The lesions are of the superficial arch distal to the TCL and the ulnar neurovascular bundle injured at the proximal port by inadvertent entry into Guyon's canal. Similar occasional lesion of the ulnar artery has been published by failed OCTR dissection of Guyon's canal. Proper use of open surgery with microscope magnification and avoidance of working in a bloodless field reduces the chances of injuring the vessels during surgery.

Nerve lesions

A skin incision directly over the median nerve rather than toward the ulnar side may result in postoperative nerve adherence to the skin.

Intra- and perineural median nerve scarring may be the results of long term CTS and of surgery. It leads often to disabling dysaesthesias, severe local pain, and hypersensitivity of the skin. Proper haemostasis is as already stated, important to prevent perineural scarring. Internal neurolysis, epineurotomy or epineurectomy is not indicated and no series has proved benefit from such attempts.

Two hundred and seventy-three patients with CTS without advanced neurophysiologic changes (DML below 11 ms) were randomized to treatment by OCTR with – or without – epineurotomy. Patients were examined clinically and by nerve conduction studies preoperatively and at 3, 6 and 12 months postoperatively. No statistically significant difference between simple decompression and decompression combined with epineurotomy with regard to either the clinical or the neurophysiologic outcome were found [9].

If a median nerve adhesion is encountered it may be isolated from the skin using:

1) Rotation of a hypothenar fat-pad flap or
2) Rotation of local muscle pedicle with a pronator quadratus/abductor digiti 5 flap.

A Z-plasty with underlying temporary silicone sheeting to prevent scar adherence has also been advocated, but is not used today.

Direct cutting of the recurrent motor branch of the median nerve results in thenar atrophy and loss of opposition. Due to the many variations of the

median nerve and its branching all surgery performed without perfect visualization carries a risk of nerve-branch lesions. Especially with ECTR lesions of the ulnar nerve and other nerve branches have been reported. Most common nerve branch lesions are: Injury to the: median nerve, ulnar nerve, digital nerves, communicating branch between ulnar and median nerve (Berretini branch). The radial digital nerve of the fourth finger can be injured at the distal port [14, 45]. Lesions of the common digital nerve to the adjacent long and ring fingers have all been reported using ECTR techniques but very seldom with OCTR [32]. Irreversible nerve damage is uncommon in either technique; however, there is an increased susceptibility to reversible nerve injury that is three times as likely to occur with endoscopy carpal tunnel release than with open carpal tunnel release.

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 something that predisposes them to injury during inadvertent release of Guyon's canal.

Grip and Pinch strength

We are dealing with the general "Grip of the whole hand" and the "Pinch grip" where we use our thumb and index finger [128].

One of the drawbacks of normal open technique is that we cut both skin and the palmar aponeurosis in order to reach the transverse carpal ligament. The subsequent volar displacement of the long flexor tendons leads to bow stringing. This may thus change the whole grip strength as the pulley function of the ligament is reduced. In the endoscope technique the palmar aponeurosis is kept intact and prevents perhaps the bowstringing of the flexor tendons to some degree. Bowstringing of the flexor tendons is found with clinical symptoms in a limited frequency. Postoperative evaluation showed that grip strength was reduced to some extent among 35% of patients. Some immediate postoperative loss of grip strength can be anticipated in all patients persisting in up to one-third of patients. Grip strength is most decreased in wrist flexion than in wrist extension. It normalizes fully only in 47%. In another series grip strength was 28% of normal 3 weeks postoperatively. It was 73% by 6 weeks and became as preoperative by 12 weeks. After cutting the TCL we may therefore anticipate a possible changed grip strength that normalizes after at least 12 weeks. Grip strength is less in wrist flexion possible due to prolapsed flexor tendons out of the carpal tunnel. Grip and pinch strengths were measured in different groups with and without ligament transposition. At 6 weeks after surgery in the group that underwent transposition flap repair exceeded preoperative grip strength values. All groups surpassed preoperative grip strength measurements at 12 weeks. By 6 weeks after surgery, all pinch measurements for 3 groups equalled or exceeded preoperative pinch measurements [81]. Grip and thumb key pinch strength were measured pre- and immediately postoperatively

in another 30 patients with CTS. It was estimated both while the wrist was in flexion and when in extension. The CTS was performed under local infiltration with 1% lidocaine. Grip strength decreased postoperatively more in wrist flexion than in wrist extension. No difference was found in thumb pinch strength. The authors conclude that some of the immediate postoperative loss of grip strength in wrist flexion can be attributed to bow stringing of prolapsed flexor tendons out of the carpal tunnel in this position [58]. In these cases reconstruction of the TCL with lengthening may be considered and undertaken.

Pain

Long-term persistent "pain" is a major determinant of the success or failure of the open CTS release. The pain-complications of long-term persistent pain may arise from any of the following causes: hypertrophy 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). It is considered a nociceptive pain in most cases. The median nerve carries approximately 70% of the sympathetic nerves to the hand. With every CTS operation nervefibres (skin, TCL) are cut and will result in some nociceptive pain. RSD is thus a possible complication of CTR but in most series it is very infrequent. In one serie [67] it was surprisingly found in 12% of cases. Here again we miss a precise definition/and common understanding of what we are discussing as RSD has many stages.

RSD develops through three stages. The first stage is characterized by swelling, hyperesthesia, skin that is warm and dry, and movement aggravates persistent pain. In the second stage proximal spread of pain and edema is found and a cool and pale shiny skin with atrophic changes, and joint stiffness. In the third stage a progressive degree of atrophy with joint contractures is found and the patient claims "intractable pain".

Treatment in the first stages is physical therapy and corticosteroids. Many attempts have been made to solve the patient's problem including use of sympatectomy. All papers on sympatectomy are based on a blend of poor quality, lack of evidence, being uncontrolled studies and based on "personal experiences" and have no evidence-based effects [69].

Failed tract – false road

Optimal clinical outcomes are obtained in ECTR when attention is paid to critical technical aspects with correct positioning of the portals, familiarization with the endoscopes and endoscopy carpal anatomy, and maintenance of clear visualization into the carpal canal [16]. The small openings for endoscope surgery carry higher risks of introducing false tracts especially if the patient

has big hands. We can easily reach the Guyon's canal and injure the ulnar nerve/artery by mistake [49, 83]. A 35% complications rate is found with the transbursal endoscope approach compared to 3.7% with the extrabursal endoscope technique [26].

With open surgery it occurs if the surgeon cuts the TCL too ulnar. Hereby the surgeon can easily slip into the Guyon's canal and will eventually release the ulnar artery – by mistake. Again it is benefited to be able to view our operative field with pulsating vessels. The size of the ulnar artery is so large that it will be obvious that you are in the wrong position for the more experienced surgeons.

The neurosurgeon who perform ECTR section of TCL must be aware of all the iatrogenic complications that potentially occur because of inadequate training or experience. Conversion of an ECTR procedure to an open procedure should be done whenever either an anatomic variation or a technical difficulty occurs [16].

Conclusion

For detailed validation of the generally used open surgical techniques, the reader is referred to the review in Surgical Neurology regarding "Carpal Tunnel" [48].

The fact is that populations from which we find our patients vary and the diagnostic methods that should only be used for screening are also used – erroneously – for diagnosing CTS. We can use the Hand-Diagram to validate the subjective symptoms and add our own "objective" tests in order to better categorize the patients.

Most important still is that we need a valid information about the carpal tunnel structure and the degree of nerve involvement. Neurophysiology adds to this latter if used in a scientific way. The simple DML monitoring favoured by many surgeons, as a screening method performed by surgeons is possibly completely unnecessary as the same information is obtained through Hand-Diagram and questioning.

The patients have today access to the Internet. They prepare their visit in the MD's office by consulting the www. The quality of Internet information about CTS obtained e.g. from "Google" is still today rather poor with much non-evidenced information = "Gaff" [31]. When we as neurosurgeons have to decide the operative technique we will be faced with numerous question from the Internet! Commercial views promote newer techniques e.g. use of endoscope and sales techniques and advertisement may have the surgeon feel that it is necessary to use the new method to "keep abreast of modern developments". Further a warning: "We should be wary about yielding to pressure to use endoscope carpal tunnel release from instrument makers, medical supply houses, insurance companies and patients" [22].

Are there situations where endoscope is favored or not? Endoscope technique with wrist extension is neither ideal for 78-year-old women with severe degenerative wrist arthritis nor for a patient with significant synovial reactions or for a patient with polyneuropathy? May be endoscope techniques are not suited for thin wrists or big hands? Similarly the preservation of handgrip is more important for some patients working with heavy handwork than others.

We must validate these problems in the future, instead of fighting between "endoscope versus open surgery" issues.

What about costs of the two methods? The ECTR is more costly if the complication rate of endoscopy surgery exceeds 6.2% (best case estimate, 5.0%). The ECTR is also more costly if the risk of career ending injury exceeds 0.001 (best case estimate, 0.0004) and if the average works absence following a complication exceeds 15.5 months (best case estimate, 12 months) [129].

Return to work is also important for estimation of costs. Return-to-work status followed in 291 cases (199 non-worker's compensation cases and 92 worker's compensation cases) showed that the worker's compensation patients returned to work in an average of 57 days, compared with 22 days for non-worker's compensation patients [80]. In these cases the ECTR technique would be in favor compared to the OCTR? With the endoscope technique [121] and double open incision technique [134], the palmar aponeurosis is left intact and prevents perhaps this bowstringing to some degree? Loss of grip strength, scar tenderness, and persistent pillar pain are late sequel of the OCTR procedure and has provided much of the impetus to switch to the alternative of ECTR. However, long-term satisfaction seems lower in an ECTR group, attributable to a 5% (or more?) rate of re-operation. Potential benefits of ECTR predominate in the 1st several postoperative weeks but diminish significantly beyond this time period.

In many series social function e.g. compensation is a strong indicator for the development of complications. Patients treated with endoscope techniques seem to recover strength faster. It is also stated that following endoscope decompression the time until return to work is shorter than with the open techniques. However no controlled series exist to prove that statement. In a recent review the problems of return was found related to type of work and eventual workers compensation [72]. In a US series no differences could be found while in Scandinavia workers tend to stay longer out of the work than blue-collar people [5]. This may indicate a difference in society cultural structure that makes it difficult to compare series from different countries.

The increasing very high number of operative procedures carried out in the US and Scandinavia may indicate that our diagnostic criteria are uncertain and used without critically accepting the epidemiological factors. As we assume that changes in the carpal tunnel are responsive for some of the CTS we

need to agree on the final diagnostic methods for evaluation of the tunnel, which include MRI and sonography. Neurophysiology is in this scenario used to confirm the degree of neuronal degeneration and for differential diagnostic purposes.

It seems unethical to accept a complication rate above 1% with these operations – whatever technique being used. Proper endoscope or microsurgical training in techniques is a must. It is likely that most of the nerve injuries incurred during endoscope release have remained unreported, but sooner or later the medical defense associations will become aware of them [30].

We can now conclude that carpal tunnel release seems to be a widely underestimated procedure and revision surgery could be largely avoided by reducing technical errors during the primary operation [116].

What we need for the future is a protocol with a systematic prospective validation of symptoms and tests – a protocol to be used universally.

The optimal CTS operative 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. According to a Cochrane review there is no evidence to support that endoscope surgery (ECTR) is better that open surgery (OCTR) [107].

The Year 2005 was the year of H. C Andersen, the famous Danish storyteller. Every one knows the story about the ugly duckling that turned into a beautiful swan. The ducks treated the ugly duckling badly despite they thought it was one of their own and only because it looked "ugly" e.g. different. They did not realise their mistake until very late when the young unhappy swan saw its brothers and sisters and understood that he himself was a swan and not a duck and suddenly he felt great and happy.

It is close to the same story that has happened with neurophysiology contrary to hand-diagrams or endoscopy surgery contrary to open surgery.

The goal of this chapter was to give the readers the possibility to achieve better results in the future remembering that this – for us neurosurgeons – simple operation for the patient is still a major event in life. The neurosurgeon treating these patients should be as cautious as if it were patients with complicated intracerebral aneurysms being treated.

The statement

"*I have had to cope with damage, inexperienced surgeons have caused by doing, what I consider to be an unnecessary operation" (Allan Hudson, Toronto, Canada)*",

tells me how we should treat these patients in the future. It also encourages us to take training of surgical techniques up to a review and increase global collaboration in designing prospective studies.

References

1. Agee JM, McCarroll HR, Tortosa RD, Berry DA, Szabo RM, Peiner CH (1992) Endoscopic release of the carpal tunnel: a randomized prospective multicenter study. J Hand Surg (Am) 17: 987–995
2. Alford JW, Weiss AP, Akelman E (2004) The familial incidence of carpal tunnel syndrome in patients with unilateral and bilateral disease. Am J Orthop 33: 397–400
3. Arons JA, Collins N, Arons MS (1999) Results of treatment of carpal tunnel syndrome with associated hourglass deformity of the median nerve. J Hand Surg (Am) 24: 1192–1195
4. Atroshi I (1999) Carpal tunnel syndrome: prevalence, electrodiagnosis and outcome instruments. Thesis Lund University, Sweden
5. Atroshi I, Larsson G-U, Ornstein E, Hofer M, Johnsson R, Ranstam J (2006) Outcomes of endoscopic surgery compared with open surgery for carpal tunnel syndrome among employed patients: randomized controlled trial. BMJ 332: 1473–1478
6. Beekman R, Visser LH (2003) Sonography in the diagnosis of carpal tunnel syndrome: a critical review of the literature. Muscle & Nerve 27: 26–33
7. Bland JDP (2005) Carpal tunnel syndrome. Curr Opin Neurol 18: 581–585
8. Boeckstyns MEH, Sorensen AI (1999) Does endoscopic carpal tunnel release have a higher rate of complications than open carpal tunnel release. J Hand Surg (Br) 24: 9–15
9. Borisch N, Haussmann P (2003) Neurophysiological recovery after open carpal tunnel decompression: Comparison of simple decompression and decompression with epineurotomy. J Hand Surg (Br) 28: 450–454
10. Bower JA, Stanisz GJ, Keir PJ (2006) An MRI evaluation of carpal tunnel dimensions in healthy wrists: Implications for carpal tunnel syndrome. Clin Biomechanics 21: 816–825
11. Braithwaite BD, Robinson GJ, Burge PD (1993) Haemostasis during carpal tunnel release under local anaesthesia: a controlled comparison of a tourniquet and adrenaline infiltrations. J Hand Surg (Br) 18: 184–186
12. Braun RM, Doehr S, Mosqueda T, Garcia A (1999) The effect of legal representation on functional recovery of the hand in injured workers following carpal tunnel release. J Hand Surg (Am) 24: 53–58
13. Britz GW, Haynor DR, Kuntz C, Goodkin R, Gitter A, Kliot M (1995) Carpal tunnel syndrome: correlation of magnetic resonance imaging, clinical, electrodiagnostic and intraoperative findings. Neurosurgery 37: 1097–1103
14. Brown WF, Lee Dellon A, Campbell WW (1995) Electrodiagnosis in the management of focal neuropathies: the "Wog" syndrome. Muscle & Nerve 17: 1336–1342
15. Buchberger W (1997) Radiologic imaging of the carpal tunnel. Eur J Radiol 25: 112–117
16. Chow JCY, Papachristos AA (2006) Endoscopic carpal tunnel release: Chow technique. Techn Orthopaedics 21: 19–29
17. Chung KC (2003) Commentary: severe carpal tunnel syndrome. J Hand Surg (Am) 28: 645–646
18. Cochrane: www.cochrane.com
19. Coppieters MW, Alshami AM, Hodges PW (2006) An experimental pain model to investigate the specificity of the neurodynamic test for the median nerve in the differential diagnosis of hand symptoms. Arch Physical Med Rehab 87: 1412–1417
20. D'Arcy CA, McGee S (2000) The rational clinical examination. Does this patient have carpal tunnel syndrome? JAMA 283: 3110–3117

21. De Krom MCTFM, Knipschild PG, Kester AD, Spaans F (1990) Efficacy of provocative tests for diagnosis of carpal tunnel syndrome. Lancet 335: 393–395
22. De Smet L, Fabry G (1995) Transection of the motor branch of the ulnar nerve as a complication of two-portal endoscopic carpal tunnel release: a case report. J Hand Surg (Am) 20: 18–19
23. Dodds SD, Trumble TE (2006) Management of complications related to carpal tunnel release. Techn Orthopaedics 21: 75–83
24. Duncan KH, Lewis RC Jr, Foreman KA, Nordyke MD (1987) Treatment of carpal tunnel syndrome by members of the American Society for Surgery of the Hand: results of a questionnaire. J Hand Surg (Am) 73: 384–391
25. Elstner M, Bettecken T, Wasner M, Anneser F, Dichgans M, Meitinger T, Gasser T, Klopstock T (2006) Familial carpal tunnel syndrome: further evidence for a genetic contribution [1] Clin Genetics 69: 179–182
26. Erdmann MWH (1994) Endoscopic carpal tunnel decompression. J Hand Surg (Br) 19: 5–13
27. Evans D (1994) Endoscopic carpal tunnel release – the hand doctor's dilemma (Editorial). J Hand Surg (Br) 19: 3–4
28. Fernandez E, Pallini R, Lauretti L, Scogna A, La Marca F (1997) Carpal tunnel syndrome. Surg Neurol 48: 323–325
29. Fernandez E, Pallini R, Lauretti L (1997) Neurosurgery of the peripheral nervous system: injuries, degeneration and regeneration of the peripheral nerves. Surg Neurol 48: 446–447
30. Filler AAG, Kliot M, Howe FA, Hayes CE, Saunders DE, Goodkin R, Bell BA, Winn HR, Griffiths JR, Tsuruda JS (1996) Application of magnetic resonance neurography in the evaluation of patients with peripheral nerve pathology. J Neurosurg 85: 299–309
31. Fricke M, Fallis D, Jones M, Luszko GM (2005) Consumer health information on the Internet about carpal tunnel syndrome: indicators of accuracy. Am J Med 118: 168–174
32. Friedman AH (1997) Surgical anatomy of the carpal tunnel. Neurosurg Focus 15: 31–41
33. Gelberman RH, Rydevik BL, Pess GM, Szabo RM, Lundborg G (1988) Carpal tunnel syndrome. A scientific basis for clinical care. Orthop Clin North Am 19: 115–124
34. Geoghegan JM, Clark DI, Bainbridge LC, Smith C, Hubbard R (2004) Risk factors in carpal tunnel syndrome. J Hand Surg 29: 315–320
35. Gerritsen AA, de Vet HC Scholten RJ, Bertelsmann FW, de Krom MCTFM, Bouter LM (2002) Splinting vs surgery in the treatment of carpal tunnel syndrome: a randomized controlled trial. JAMA 288: 1245–1251
36. Gerritsen AA, de Krom MC, Struijs MA, Scholten RJ, de Vet HC, Bouter LM (2002) Conservative treatment options for carpal tunnel syndrome: a systematic review of randomized controlled trials. J Neurol 249: 272–280
37. Gomes I, Becker J, Arthur Ehlers J, Bocchese Nora D (2006) Prediction of the neurophysiological diagnosis of carpal tunnel syndrome from the demographic and clinical data. Clin Neurophysiol 117: 964–971
38. Graham B (2006) The diagnosis and treatment of carpal tunnel syndrome. BMJ 332: 1463–1464
39. Grant GA, Goodkin R, Kliot M (1999) Evaluation and surgical management of peripheral nerve problems. Neurosurgery 44: 825–840

40. Greenslade JR, Mehta RL, Belward P, Warwick DJ (2004) DASH and Boston Questionnaire assessment of carpal tunnel syndrome outcome: what is the responsiveness of an outcome questionnaire? J Hand Surg 29B: 159–160
41. Gunnarsson LG, Amilon A, Hellstrand P, Leissner P, Philipson L (1997) The diagnosis of carpal tunnel syndrome. Sensitivity and specitficityspecificity of some clinical and electrophysiological tests. J Hand Surg (Br) 22: 34–37
42. Haase J, Musaeus P, Boisen E (2004) Virtual reality and habitats for learning microsurgical skills. Virtual applications. Applications with virtual inhabited 3D worlds. In: Andersen P, Qvortrup L (eds) Springer, Berlin Heidelberg New York, pp 29–48
43. Haase J (2007) Learning surgery. Surg Neurol (in press)
44. Harris I, Mulford J, Solomon M, van Gelder JM, Young J (2005) Association between compensation status and outcome after surgery: a meta-analysis. JAMA 293: 1644–1652
45. Henkin P, Friedman AH (1997) Complications in the treatment of carpal tunnel syndrome. Neurosurg Focus 15: 10–16
46. Hobby JL, Watts C, Elliot D (2005) Validity and responsiveness of the patient evaluation measure as an outcome measure for carpal tunnel syndrome. J Hand Surg 30: 350–354
47. Huang JH, Zager EL (2004) Mini-open carpal tunnel decompression. Neurosurgery 54: 397–400
48. Hudson AR, Wissinger JP, Salazar JL, Kline Dg, Yarzagary L, Danoff D, Fernandez E, Field EM, Gainsburg DB, Fabri RA, Mackinnon SE (1997) Carpal tunnel syndrome. Surg Neurol 47: 105–114
49. Hung JT, Lee SW, Han SH, Son BC, Sung JH, Park CK, Park CK, Kang JK, Kim MC (2006) Anatomy of neurovascular structures around the carpal tunnel during dynamic wrist motion for endoscopic carpal tunnel release. Operative Neurosurg 58: 127–133
50. Idler RS (1996) Persistence of symptoms after surgical release of compressive neuropathies and successive management. Ortop Clin North Am 27: 409–416
51. Ikeda K, Isanyra B, Tomita K (2006) Segmental carpal canal pressure with carpal tunnel syndrome. J Hand Surg 31: 925–929
52. Jablecki CK, Andary MT, Floeter MK, Miller RG, Quartly CA, Vennix MJ, Wilson JR (2002) Practice parameter: electrodiagnostic studies in carpal tunnel syndrome: report of the American Association of Electrodiagnostic Medicine. Neurology 58: 1589–1592
53. Jae TH, Sang WL, Seung HH, Byung CS, Jae HS, Choon KP, Chun KP, Joon KK, Moon CK (2006) Anatomy of neurovascular structures around the carpal tunnel during dynamic wrist motion for endoscopic carpal tunnel release. Neurosurgery 58 Suppl 1: ONS-127–ONS-13
54. Jimenez DF, Gibbs SR, Clapper AT (1998) Endoscopic treatment of carpal tunnel syndrome: a critical review. J Neurosurg 88: 817–826
55. Kamath V, Stothard J (2004) A clinical questionnaire for the diagnosis of carpal tunnel syndrome. J Hand Surg (Br) 29: 95–99
56. Katz JN, Simmons BP (2002) Clinical practice. Carpal tunnel syndrome. N Engl J Med 346: 1807–1812
57. Kleindienst A, Hamm B, Hildebrand G, Klug N (1996) Diagnosis and staging of carpal tunnel syndrome: comparison of magnetic resonance imaging and intra-operative findings. Acta Neurochir (Wien) 138: 228–233
58. Kline D, Hudson A (eds) (1995) Nerve injuries. WB Saunders, Philadelphia

59. Kremer M, Gilliatt RW, Golding JSR, Wilson TG (1953) Acroparaesthesia in the carpal-tunnel syndrome. Lancet 265: 590–595
60. Kwon HK, Hwang M, Yoon D-W (2006) Frequency of carpal tunnel syndrome according to level of radiculopathy. Double crush syndrome? Clin Neurophys 117: 1256–1259
61. Lehman RM (2004) A review of neurophysiology. Neurosurg Focus 16: 1–16
62. Levine DW, Simmons BP, Koris MJ, Daltroy LH, Hohl GG, Fossel H, Katz JN (1993) A self-administered questionnaire for the assessment of severity of symptoms and functional status in carpal tunnel syndrome. J Bone Joint Surg (Am) 75: 1585–1592
63. Lindley SG, Kleinert JM (2003) Prevalence of anatomic variations encountered in elective carpal tunnel release. J Hand Surg 28: 849–855
64. Liveson JA, Ma DM (1992) Laboratory reference for clinical neurophysiology. F. A. Davis Company, Philadelphia
65. Long D (2004) Competency based training in neurosurgery; the next revolution in medical education. Surg Neurol 61: 5–14
66. Lundborg G (2000) A 25-year perspective of peripheral nerve surgery: evolving neuroscientific concepts and clinical significance. J Hand Surg (Am) 25: 391–414
67. MacDonald RI, Lichman DM, Hanlon JJ, Wilson JN (1978) Complications of surgical release for carpal tunnel syndrome. J Hand Surg (Am) 3: 70–76
68. Mackinnon SE (2002) Pathophysiology of nerve compression. Hand Clin 18: 231–241
69. Mailis A, Furlan A (2002) Sympatectomy for neuropracthic pain. The Cochrane Database Syst Rev CD002918
70. Malis LI, Apuzzo ML (2006) Electrosurgery and bipolar technology. Neurosurgery 58 Suppl 1: ONS-1–ONS-11
71. Marshall S, Tardif G, Askwarth N (2002) Local corticosteroid injection for carpal tunnel syndrome. Cochrane Database Syst Rev CD001554
72. Masear VR, Hayes JM, Hyde AG (1986) An industrial cause of carpal tunnel syndrome. J Hand Surg (Am) 11: 222–227
73. Massy-Westropp N, Grimmer K, Bain G (2000) A systematic review of the clinical diagnostic tests for carpal tunnel syndrome. J Hand Surg 25: 120–127
74. Mauer UM, Raath SA, Richter HP (1993) Intraoperative anatomic and pathologic findings in 1.4200 initial operations in carpal tunnel syndrome. Handchir Mikrochir Plast Chir 25: 124–126
75. McNeil BJ, Keeler E, Adelstein SJ (1975) Primer on certain elements of medical decision making. New Engl J Med 293: 211–215
76. Mohler LR, Pedowitz RA, Myers RR, Ohara WH, Loopez MA Gershuni DH (1999) Intermittent reperfusion fails to prevent post-tourniquet neuropraxia. J Hand Surg (Am) 24: 687–693
77. Mondelli M, Passero S, Giannini F (2001) Provocative tests in different stages of carpal tunnel syndrome. Clin Neurol Neurosurg 103: 178–183
78. Morimoto KW, Budoff JE, Haddad J, Gabel GT (2005) Cross-sectional area of the carpal canal proximal and distal to the wrist flexion crease. J Hand Surg 30: 487–492
79. Myers KA (2000) Utility of the clinical examination for carpal tunnel syndrome. CMAJ 163: 605–610
80. Nagle DJ, Fischer TJ, Harris GD (1996) A multicenter prospective review of 640 endoscopic carpal tunnel releases using the transbursal and extrabursal Chow techniques. Arthroscopy 12: 139–143

81. Netscher D, Mosharrafa A, Lee M, Posen C, Choi H, Steadman AK, Thornby J (1997) Transverse carpal ligament: its effect on flexor tendon excursion, Morphologic changes of the carpal canal, and on pinch and grip strengths after open carpal tunnel release. Plast Rec Surg 100: 636–642
82. Nishimura A, Ogura T, Hase H, Makinodan A, Hojo T, Katsumi Y, Yagi K, Mikami Y, Kubo T (2004) A correlative electrophysiological study of nerve fiber involvement in carpal tunnel syndrome using current perception thresholds. Clin Neurophys 115: 1921–1924
83. Nitz AJ, Dobner JJ (1989) Upper extremity tourniquet effects in carpal tunnel release. J Hand Surg (Am) 14: 499–504
84. Nodera H, Herrmann DN, Holloway RG, Logigian EL (2003) A Bayesian argument against rigid cut-offs in electrodiagnosis of median neuropathy at the wrist. Neurology 60: 458–464
85. Nora DB, Becker J, Ehlers JA, Gomes I (2005) What symptoms are truly caused by median nerve compression in carpal tunnel syndrome? Clin Neurophysiol 116: 275–283
86. O'Connor D, Marshall S, Massy-Wentropp N (2003) Non-surgical treatment (other than steroid injection) for carpal tunnel syndrome. The Cochrane Database of Systematic Rev CD003219
87. Okutsu I, Ninomiya S, Hamanaka I (1989) Measurement of pressure in the carpal canal before and after endoscopic management of carpal tunnel syndrome. J Bone Joint Surg (Am) 71: 679–683
88. Orlin JR, Stranden E, Slagsvold CE (2005) Effects of mechanical irritation on the autonomic art of the median nerve. Eur J Neurol 12: 144–149
89. Pagnanelli DM, Barrer SJ (1991) Carpal tunnel syndrome: surgical treatment using the Paine retinaculatome. J Neurosurg 75: 77–81
90. Palmer AK, Toivonen DA (1999) Complications of endoscopic and open carpal tunnel release. J Hand Surg (Am) 24: 561–565
91. Pasternack II, Malmivaara A, Tervahartiala P, Forsberg H, Vehmas T (2003) Magnetic resonance imaging findings in respect to carpal tunnels syndrome. Scan J Work Environ Health 29: 189–196
92. Peabody JW, Luck L, Glassman P, Dresselhaus TR, Lee M (2000) Comparison of vignettes, standardized patients and chart abstraction a prospective validation of 3 methods for measuring quality. JAMA 283: 1715–1722
93. Pécina MM, Krmpotic-Nemanic, Markiewitz AD (eds) (2001) Tunnel syndromes, 3rd edn. CRC Press
94. Pierre-Jerome C, Bekkelund SI, Mellgren SI, Nordstrom R (1997) Quantitative MRI and electrophysiology of preoperative carpal tunnel syndrome in a female populations. Ergonomics 40: 642–649
95. Priganc VW, Henry SM (2003) The relationship among five common carpal tunnels syndrome tests and the severity of carpal tunnel syndrome. J Hand Ther 16: 225–236
96. Reale F, Ginanneschi F, Sicurrelli F, Mondelli M (2003) Protocol of outcome evaluation for surgical releases of carpal tunnel syndrome. Neurosurgery 53: 343–351
97. Rempel D, Evanoff B, Amadio PC, de Krom M, Franklin G, Franzblau A, Gray R, Gerr F, Hagberg M, Hales T, Katz JN, Pransky G (1998) Consensus criteria for the classification of carpal tunnel syndrome in epidemiologic studies. Am J Public Health 88: 1447–1451
98. Rempel D, Dahlin L, Lundborg G (1998) Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. J Bone Joint Surg (Am) 81: 1600–1610
99. Rodner CM, Katarincic J (2006) Open carpal tunnel release. Techn Orthopaedics 21: 3–8

100. Roquelaure Y, Mechali S, Dano C, Fanello S, Benetti F, Bureau D, Mariel J, Martin YH, Derriennic F, Penneau-Fontbonne D (1997) Occupational and personal risk factors in industrial workers. Scand J Work Environ Health 23: 364–369
101. Rossignol M, Stock S, Patry L, Armstrong B (1997) Carpal tunnel syndrome: what is attributable to work? The Montreal study. Occup Environ Med 54: 519–523
102. Rotman MB, Enkvetchakul BV, Megerian JT, Gozani SN (2004) Time course and predictors of median nerve conduction after carpal tunnel release. J Hand Surg (Am) 29: 367–372
103. Russell SM (2006) Dual-portal endoscopic release of the transverse ligament in carpal tunnel syndrome: results of 411 procedures with special reference to technique, efficacy, and complications. Commentary Neurosurg 59: 3–8
104. Sackett DL, Straus SE, Richardson WS *et al* (2000) Evidence-based medicine. How to practice and teach EBM, 2nd edn. Churchill Livingstone, Edinburgh
105. Sander MD, Abbasi D, Ferguson AL, Steyers CM, Wang K, Morcuende JA (2005) The prevalence of hereditary neuropathy with liability to pressure palsies in patients with multiple surgically treated entrapment neuropathies. J Hand Surg 30: 1236–1241
106. Sanz J, Lizaur A, Sánchez del Campo F (2005) Postoperative changes of carpal canal pressure in carpal tunnel syndrome: a prospective study with follow-up of 1 year. J Hand Surg 30: 611–614
107. Scholten R, Gerritsen A, Uitdehaag B, Geldere D, Vet H, Bouter L (2004) Surgical treatment options for carpal tunnel syndrome. Cochrane Database Syst Rev 18: CD003905
108. Schuind F (2002) Canal pressures before, during, and after endoscopic release for idiopathic carpal tunnel syndrome. J Hand Surg 27: 1019–1025
109. Shapiro S (1995) Microsurgical carpal tunnel release. Neurosurgery 37: 66–70
110. Sheu JJ, Yuan RY, Chion HY, Hu CJ, Chen WT (2006) Segmental study of the median nerve versus comparative tests in the diagnosis of mild carpal tunnel syndrome. Clin Neurophys 117: 1249–1255
111. Skre H (1972) Neurological signs in a normal populations. Acta Neurol Scand 48: 575–606
112. Spindler HA, Dellon AL (1982) Nerve conduction studies and sensibility testing in carpal tunnel syndrome. J Hand Surg (Am) 7: 260–263
113. Steers J, Reulen H-J, Lindsay KW (2004) UEMS charter on training of medical specialists in the EU – the new neurosurgical training charter. Acta Neurochir Suppl 90: 3–11
114. Stevens JC, Sun S, Beard CM, O'Fallon WM, Kurland LT (1988) Carpal tunnel syndrome in Rochester, Minnesota 1961–1980. Neurology 38: 134–138
115. Storm S, Beaver SK, Giardino N, Kliot M, Franklin GM, Jarvik JG, Chan L (2005) Compliance with electrodiagnostic guidelines for patients undergoing carpal tunnel release. Arch Phys Med Rehab 86: 8–11
116. Stütz NM, Gohritz A, van Schoonhoven J, Lanz U (2006) Revision surgery after carpal tunnel release – analysis of the pathology in 200 cases during a 2 year period. J Hand Surg 31: 68–71
117. Sunderland S (1969) Nerve and nerve injuries. Livingston ES, London
118. Szabo RM, Slater RR Jr, Farvr TB, Stanton DB, Sharman WK (2000) The value of diagnostic testing in carpal tunnel syndrome. J Hand Surg (Am) 25: 183–184
119. Taleisnik J (1973) The palmar cutaneous branch of the median nerve and the approach to the carpal tunnel. An anatomical study. J Bone Joint Surg (Am) 55: 1212–1217

120. Taniuchi M, Clark HB, Schweitzer JB, Johnson EMJ (1988) Expression of nerve growth factor receptors by Schwann cells of axonotomized peripheral nerves: ultrastructural location, suppression by axonal contact and binding properties. J Neurosc 8: 664–681
121. Trumble TE, Diao E, Abrams RA, Gilbert-Andersen MM (2002) Single – portal endoscopic carpal tunnel releases compared with open release – a prospective randomized trial. J Bone J Surg (Am) 84: 1107–1115
122. Urbaniak JR, Desai SS (1996) Complications of nonoperative and operative treatment of carpal tunnel syndrome. Hand Clin 12: 325–335
123. Werner RA, Jacobson JA, Jamadar DA (2004) Influence of body mass index on median nerve function, carpal canal pressure, and cross-sectional area of the median nerve. Muscle & Nerve 30: 481–485
124. Werner RA (2006) Evaluation of work-related carpal tunnel syndrome. J Occup Rehab 16: 207–222
125. Wiesler ER, Chloros GD, Cartwright MS, Smith BP, Rushing J, Walker FO (2006) The use of diagnostic ultrasound in carpal tunnel syndrome. J Hand Surg 31: 726–732
126. Wilder-Smith EP, Seet RCS, Lim ECH (2006) Diagnosing carpal tunnel syndrome – clinical criteria and ancillary tests. Nature Clin Pract Neurol 2: 366–734
127. Wong KC, Hung LK, Ho PC, Wong JM (2003) Carpal tunnel release, a prospective, randomized study of endoscopic versus limited-open methods. J Bone Joint Surg (Br) 85: 863–868
128. Wright PE (1998) Carpal tunnel syndrome. In: Canale TS (ed) Campbell's operative ortopaedics, 9th edn. Mosby, St Louis
129. Vasen AP, Kuntz KM, Simmons BP, Katz JN (1999) Open versus endoscopic carpal tunnel release: a decision analysis. J Hand Surg 24: 1109–1117
130. Vasiliadis HS, Tokis AV, Andrikoula SI, Kordalis NV, Beris AE, Xenakis T, Georgoulis AD (2006) Microsurgical dissection of the carpal tunnel with respect to neurovascular structures at risk during endoscopic carpal tunnel release arthroscopy. J Arthroscop Rel Surg 22: 807–812
131. Yoshioka S, Okuda Y, Tamai K, Hirasawa Y, Kodda Y (1993) Changes in carpal tunnel shape during wrist joint motions. MRI evaluation of normal volunteers. J Hand Surg (Br) 18: 620–623
132. Young VL, Higgs PE (1996) Evaluation of the patient presenting with a painful wrist. Clin Plast Surg 23: 361–368
133. Zanette G, Marani S, Tamburin S (2006) Extra-median spread of sensory symptoms in carpal tunnel syndrome suggests the presence of pain-related mechanisms. Pain 122: 264–270
134. Zyluk A, Strychar JA (2006) Comparison of two limited open techniques for carpal tunnel release. J Hand Surg (Br) 31: 466–472