Ontario

Carpal Tunnel Syndrome

Discussion paper prepared for

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WSIAT literature search reviewed by Dr. D. Rowed in 2011, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence. See *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

Definition

Carpal tunnel syndrome is most broadly defined as a condition characterized by an abnormality of median nerve function due to compression of the nerve within the carpal canal.

Epidemiology

Carpal tunnel syndrome is widely held to be a very common clinical condition. Most physicians would agree that, among the compressive nerve lesions that may affect the upper extremity, carpal tunnel syndrome is, by a substantial margin, the most commonly diagnosed. However, the exact prevalence of this condition in industrialized economies like Ontario, has not been reliably established. In fact, studies of self-reported symptoms of carpal tunnel syndrome indicate that the prevalence in the general population of North America is approximately 1% -- about the same as that observed for rheumatoid arthritis. Recent studies of local populations in Scandinavia documenting symptoms confirmed by physicians as due to carpal tunnel syndrome, indicate that the prevalence is no higher than between 2 and 4%. The literature also indicates that the prevalence among working individuals is somewhat lower, about 0.5%. This suggests that the majority of cases occur in the non-working population and this reflects the experience of most experienced clinicians.

These findings contrast sharply with those of studies which estimate the prevalence of carpal tunnel syndrome among individuals in specific occupations such as workers in the grocery and meatpacking industries, clerical workers performing data entry at computer keypads, and individuals engaged in other repetitive or machine paced work. Most of these studies indicate that carpal tunnel syndrome is extremely prevalent, even epidemic in some cases. However, the majority of these studies have utilized flawed methods in obtaining their results and, in most instances, it is difficult to ascertain the validity of their conclusions.

While it is possible that different work activities may expose workers to a variable risk of developing symptoms of carpal tunnel syndrome, an assessment of the independent effect of the workplace as an etiologic factor is hampered by wide variations in the diagnostic criteria used to identify carpal tunnel syndrome. In fact, there is so much variation in the manner in which carpal tunnel is diagnosed, that comparison between studies of the workers of different industries is difficult and sometimes impossible.

These variations also extend beyond studies of the condition and really reflect different concepts of the condition held by doctors and other health professionals who care for patients with these symptoms.

Pathophysiology

The pathophysiologic basis for carpal tunnel syndrome is, in most instances, unknown. The carpal canal normally contains only the median nerve, the flexor tendons of the digits and the synovial lining of the tendons. The median nerve is compressed when the space available to it is decreased within the finite volume of the carpal canal. Conditions that cause synovial swelling are known to be associated with carpal tunnel syndrome. Pregnancy and rheumatoid arthritis are two well-known examples. Conditions like acromegaly and hypothyroidism may also be associated with carpal tunnel syndrome but these conditions are relatively rare, especially in the context of work-related symptoms of carpal tunnel syndrome.

Studies of biopsy specimens taken from the flexor tendon synovium at the time of surgery to perform a carpal tunnel release, show edema of this tissue even in patients not known to have a condition which is associated with swelling. This observation suggests that flexor tendon synovial edema may be the cause of median nerve compression but the immediate cause of the swelling in these cases is not known.

Intrinsic abnormalities of the median nerve itself may also act to lower the threshold for symptomatic compression. Pressures within the carpal canal that might not otherwise cause symptoms of carpal tunnel syndrome may do so if the nerve is rendered particularly sensitive to pressure by some other disease or condition. A common example of this is diabetes mellitus, which frequently affects peripheral nerve function. Peripheral nerves, including the median nerve, are a target of diabetes and in individuals with this condition, carpal tunnel syndrome may occur even where the pressure in the carpal canal is insufficient to cause these symptoms in a non-diabetic individual. In this sense, diabetes may be considered a pre-existing condition that predisposes the median nerve to symptoms of compression under circumstances where this might not otherwise occur. Other diffuse peripheral nerve diseases may also play a role in the development of symptoms of carpal tunnel syndrome but these are also very rare conditions which will usually be known to be present in an individual before symptoms of hand numbness are attributed to carpal tunnel syndrome.

A much less clear, but related concept is that of “double crush”. In the double crush syndrome it is thought that sub-clinical compression of the median nerve at several points in its course between the spinal cord and the carpal tunnel, lowers the threshold for symptomatic compression at level of the carpal canal. While this idea fits in with some of what is known about peripheral nerve function, it is a largely unproven hypothesis. Furthermore, it can rarely be shown to be present in clinical cases of carpal tunnel syndrome and should seldom, if ever, play a role in the diagnosis or management of carpal tunnel syndrome.

Diagnosis

By far, the most common cause of failure in the treatment of carpal tunnel syndrome is an inaccurate diagnosis leading to therapy which may be appropriate for carpal tunnel syndrome but which is inadequate for the condition actually causing the symptoms.

While the relative importance of the physical examination and electrodiagnostic testing remains controversial, most clinicians agree that the cardinal symptom of carpal tunnel syndrome is a sensory disturbance, specifically numbness or tingling, in the anatomic distribution of the median nerve. That area includes the thumb, index, middle and ring fingers of the hand. The presence of numbness or tingling somewhere in this area must be clearly identified before the diagnosis of carpal tunnel syndrome can be considered. Carpal tunnel syndrome should not be diagnosed unless this symptom can be definitely established to be present. The absence of a symptom of this nature militates very strongly against the diagnosis.

A significant problem for clinicians is the manner in which the patients express the nature of their complaint. Clearly, numbness and tingling will usually be perceived as an uncomfortable sensation and may be, in many instances, described as pain, especially in a patient with a limited knowledge of English. Nonetheless, it is incumbent on the physician to definitely establish the true nature of the symptoms by repeated and probing questioning.

Symptoms may be reported outside the median nerve area as well. The relationship between carpal tunnel syndrome and symptoms of pain or a sensory disturbance related to the wrist, forearm, arm, shoulder and neck is unclear. These symptoms should be explored when the diagnosis of

carpal tunnel syndrome is being considered but they cannot be used to make this diagnosis. While they may be useful in indicating an alternative diagnosis, in the absence of the key symptom of numbness in the median nerve distribution, they have no role in ruling in the diagnosis of carpal tunnel syndrome.

A nocturnal symptom of a sensory disturbance is a classic manifestation of carpal tunnel syndrome. Relief of these symptoms by splinting is also a compelling finding for the diagnosis of carpal tunnel syndrome. Similarly, the response of the symptoms to an intervention like injection of the carpal canal with a steroid medication may also be helpful in confirming the diagnosis. The reliability of these measures as aids to the clinical diagnosis of carpal tunnel syndrome is certainly imperfect but they may be helpful as pieces of collateral clinical data obtained as a consequence of the initial management of the problem.

The relationship between sensory symptoms and strenuous hand use is less well defined but may be prominent. The literature indicates that the hand activity must be repetitive and forceful. Activities characterized by a high frequency but low force, such as computer key pad use, have not been shown to be an important precipitating factor despite the overwhelming volume of information in the lay media to the contrary. The fact is that actual evidence of this relationship, by valid medical or epidemiological studies, is lacking. Where the relationship between exposure to repetitive hand use and carpal tunnel syndrome has been carefully studied, no significant increase in the risk of developing this condition can be identified. In rare circumstances, where a clear temporal linkage between the development of symptoms and their relief, in relation to a given exposure, can be reliably and repeatedly identified, then a major criterion for causality may be met. Other issues which should have an impact on establishing causality include a dose response relationship and a plausible biologic basis, both of which are largely lacking in most, though not all, instances where there is held to be a work-related etiology for carpal tunnel syndrome.

A large number of physical examination findings have been described for the diagnosis of carpal tunnel syndrome. These include provocative tests for median nerve compression including the Phelan test[1], Tinel sign[2] and

[1] reproduction of numbness or tingling in the distribution of the median nerve by placing the wrist into a position of flexion

[2] radiating numbness or tingling in the distribution of the median nerve with percussion, through the skin, over the nerve at, or just above the carpal tunnel

tenderness to palpation over the median nerve. The neurologic examination may include signs of median nerve denervation such as atrophy and weakness of the abductor pollicis brevis muscle or a loss of two-point discrimination in the distribution of the median nerve. Other tests of sensory function, like the perception of light touch, pinprick and joint position are not generally used in the diagnosis of carpal tunnel syndrome may be relevant in ruling out other causes of hand numbness or tingling.

Generally, some or of all of these findings are present in patients with carpal tunnel syndrome and, when this is the case, the diagnosis is clear and usually unequivocal. However, in a minority of cases, there will be no physical evidence of median nerve compression despite a convincing history of carpal tunnel syndrome symptoms. In these cases, the diagnosis should not depend solely on the demonstration of physical signs of median nerve compression. Nonetheless, the absence of physical findings may mitigate against the diagnosis if the symptoms do not fit a classic pattern. Generally, most clinical content experts emphasize the reporting of characteristic symptoms much more heavily than the demonstration of physical findings.

The role of electrodiagnostic testing in the diagnosis of carpal tunnel syndrome remains controversial. Although this investigation is frequently considered a gold standard for the diagnosis, the assumptions underlying this concept are flawed. There is overwhelming evidence to indicate that there are clearly frequent cases in which this investigation is inaccurate both in incorrectly diagnosing patients as having carpal tunnel syndrome and in failing to identify carpal tunnel syndrome as the cause of the presenting symptoms. Furthermore, the results of electrodiagnostic tests are not consistently predictive for most outcomes of treatment for carpal tunnel syndrome, especially those related to return to work. There has been little study of the reproducibility of this investigation so that it remains unknown how the result of testing might change in a given individual over two separate examinations even if the symptoms are the same. It is entirely likely that the reported outcome of electrodiagnostic testing of an individual would vary significantly between two separate laboratories, even if they review the same data.

The criteria by which a diagnosis of Carpal Tunnel Syndrome is made, on the basis of electrical testing, also varies from laboratory to laboratory. It is arguable whether or not the results of testing should be reported as “positive” or “negative” since there are gradations of normal nerve function in the normal population.

The most appropriate role for electrodiagnostic testing is as an *adjunct* to the clinical assessment in instances where the diagnosis is not clear. For example, when the clinical impression of carpal tunnel syndrome is equivocal because the history is difficult to obtain due to a language barrier or is in some way atypical, there are conflicting findings on the physical examination, or there is an indifferent response to nonsurgical treatment, electrodiagnostic testing may be helpful in increasing or decreasing the probability of carpal tunnel syndrome as the correct diagnosis. In this sense, the test is used as an aid to the clinical assessment by representing another piece of data which must be interpreted in the context of the individual's symptoms and what physical findings may, or may not, be present. Any other way of utilizing this information assumes that the clinical assessment has no value, if it can be completely superceded by the findings of electrical testing. Obviously, this is entirely inconsistent with the experience of experts caring for this problem.

Where the clinical findings are clearly indicative of carpal tunnel syndrome or strongly suggest another diagnosis, there is no role for electrical testing. Electrodiagnostic tests should definitely not be used as confirmatory evidence in these cases because there is a significant risk that the result will conflict with the clinical findings. When this occurs, the electrodiagnostic tests are usually given an inappropriate emphasis in establishing the final diagnosis when, in fact, experience has shown that the clinical judgment of a content expert is much more likely to be correct.

Finally, there are a number of conditions which may produce symptoms like those of Carpal Tunnel Syndrome and it is essential that alternative explanations for these symptoms be sought, especially when standard and adequate treatment for Carpal Tunnel Syndrome has failed to result in improvement. It is important, when reviewing a case, to determine whether or not these diagnoses have been considered as a plausible alternative explanation for the symptoms. Some examples would include: conditions affecting the joints such as the various types of arthritis, peripheral nerve diseases, and abnormalities of the nerves in the upper part of the limb, the neck or even in the brain.

Treatment

The management of carpal tunnel syndrome should generally begin with non-operative measures. Splinting the wrist in neutral or slight extension,

with a standard, prefabricated cock-up splint should be the mainstay of treatment initially. The patient should wear this splint when symptoms usually occur. This will usually be during sleep but might include daytime wear as well if there are significant symptoms with activities.

A steroid injection into the carpal canal may also be considered in the non-operative management of carpal tunnel syndrome. This should be considered if splinting is partially, but not completely, successful in reducing the symptoms or if there is some type of contra-indication to splint use. An injection of 0.75 cc of methylprednisolone is recommended. The effect of an injection is usually noted within two weeks and may be long lasting although it is often transient. Where there has been a satisfactory response followed by a relapse of symptoms, the prognosis for subsequent surgery to release the carpal tunnel is satisfactory.

The role of diuretics, anti-inflammatories and vitamin supplements such as vitamin B6, is unproven. There is also little evidence for other modalities such as physical therapy, yoga, chiropractic or acupuncture in the routine care of carpal tunnel syndrome.

Modifications to the work place may appear to be necessary in certain circumstances although, in most instances, the work-related of the symptoms is difficult to definitively establish. When the symptoms have a close temporal relationship to both attendances and absences from work, and where the work consists of both high force, high repetition activity, there may be a need to address the workplace in the treatment of carpal tunnel syndrome. Generally speaking, there is little valid published evidence to support this approach to carpal tunnel syndrome except for a few specific industries. The lack of adequate evidence is at least partially due to the inconsistencies in diagnostic practice and case definitions, but the fact remains that successful treatment of established carpal tunnel syndrome by modifying the workplace is only anecdotally reported.

The two main indications for surgical management of carpal tunnel syndrome are the failure, or expected failure, of nonsurgical treatment and evidence of denervation in the hand as manifest by a loss of two-point discrimination or thenar muscle atrophy. The manner in which the procedure is done varies widely among surgeons, especially with respect to the anesthetic technique and the use of a tourniquet. The most important consideration is the surgical incision, which must be adequate to allow complete division of the transverse carpal ligament and the distal antebrachial fascia. The only reliable way in which this requirement can be met is through a longitudinal wound made between the thenar and hypothenar eminences, directly over the transverse carpal ligament.

Endoscopic carpal tunnel release is currently performed in only a few centres. Well-designed randomized trials, comparing this treatment to conventional open carpal tunnel release, have proven that it has no special benefit in terms of outcome, and it may be associated with a substantially increased risk. It is associated with a substantial risk of significant complications including injuries to the median and ulnar nerves, lacerations of the flexor tendons and incomplete release of the carpal tunnel. These complications are especially likely to occur where the procedure is only carried out occasionally.

The need for postoperative immobilization is also controversial. It is widely accepted that early active digit flexion should be encouraged to avoid adhesions between the nerve and flexor tendons, although in practice, this complication is rare. It may be advisable to splint the wrist in slight extension to avoid flexion of the wrist and a risk of volar displacement of the carpal tunnel contents including the median nerve. Physical therapy is not routinely required postoperatively.

The response to treatment, as a retrospective indication of the validity of the diagnosis should be considered with caution. Normally, a satisfactory response to treatment would indicate that the diagnosis was accurate however, in conditions other than carpal tunnel syndrome, where this has been studied, a clear placebo effect of up to 30% has been reported even for surgical procedures. It has been repeatedly shown that a poor outcome from treatment does not necessarily indicate inadequate treatment or an inaccurate diagnosis. The challenge for clinicians, researchers and insurers is to evaluate responses to therapy that can be measured objectively.

Prognosis

Without question, the prognosis for a complete relief of symptoms correctly attributed to carpal tunnel syndrome and skillfully treated is ordinarily excellent. Recurrences should be rare. The interaction between a return to the workplace and continued control of symptoms is unknown but should be a focus for further study. So far, there has not been adequate study of this issue to allow generalizations to be made regarding the return to work following carpal tunnel release, especially in those cases where the workplace is thought to have played an etiologic role in the development of carpal tunnel syndrome symptoms.

Some special considerations

Trauma to the area of the wrist

Where there has been a significant injury to the area of the carpal tunnel and subsequent symptoms of numbness attributable to carpal tunnel syndrome, the main consideration is that an alternative diagnosis of a direct median nerve injury should be considered. Although this may appear to be an artificial distinction, in fact, it is an important one because the treatment that should ensue and the expected prognosis may vary significantly.

For example, a direct closed injury to the median nerve would be expected to have a satisfactory prognosis and to clear spontaneously, although this may take many months or even as long as a year. A carpal tunnel release in this context rarely, if ever, changes the rate of recovery. Normally, these injuries are manifested by the immediate onset of a sensory disturbance that does not vary except to slowly resolve as the nerve recovers. A good example of a context in which this occurs is a fracture of the distal radius. The median nerve often suffers a direct injury due to displacement of the fracture fragments but almost invariably recovers completely without any further treatment, even if the fracture remains unreduced.

A less severe injury to the wrist, like a contusion, would be distinctly unlikely to cause either a direct median nerve or to predispose the patient to developing symptoms of carpal tunnel syndrome due to compression of the median nerve.

Exposure to vibration

The role of vibratory exposure in the etiology of carpal tunnel syndrome is unclear. Certainly reports in the literature suggest a relationship between activities of this nature and both sensory and vascular disturbances in the hands. Carpal tunnel syndrome would not usually be considered to synonymous with other diagnostic labels like “vibration white finger”. As described above, the establishment of causality requires that there be demonstration of temporal and dose-response relationships as well as a biologically plausible explanation. The available literature does not satisfy these criteria and so a definitive relationship between exposure to vibration and the development of carpal tunnel syndrome has not been proven. However, there is evidence to suggest that a plausible biologic link between exposure to vibration and carpal tunnel syndrome does exist and therefore, given a reasonable duration and extent of exposure, there is reason to consider this possible etiologic connection in certain cases. What remains unknown is how much exposure constitutes a threshold beyond which this relationship should be held to exist.

Repetitive movements and exacerbation of a pre-existing or intermittently symptomatic state of carpal tunnel syndrome

The role of repetitive movements has been alluded to above. The data available on this subject suggests little if any relationship between this type of exposure and carpal tunnel syndrome. The exception would be in instances where the repetitive activity requires both frequent and forceful movements. Guidelines for defining a critical frequency and degree of force can be inferred from these reports.

Where there is a pre-existing diagnosis of carpal tunnel syndrome which is claimed to be exacerbated by a work activity, the same issues in establishing causality pertain as in establishing work-relatedness in general. Carpal tunnel syndrome is known to be a condition that is characterized by both exacerbations and remissions and so the effect of modified work, absences from work and ergonomic modifications to the workplace are difficult to measure. Similarly, the status of an individual who has apparently been successfully treated for carpal tunnel syndrome and is contemplating a return to employment that may be thought to be a risk factor for carpal tunnel syndrome is unclear.

Tenosynovitis and carpal tunnel syndrome

Although an inflammatory condition affecting the tendons is frequently diagnosed, in most cases, there is little or no evidence to support the presence of this type of condition and it essentially represents a diagnosis of exclusion. It is difficult to link the diagnosis of carpal tunnel syndrome to an inflammation of the tendons except in the context of conditions known to cause an extreme degree of inflammation like rheumatoid arthritis. This is rare in the context of carpal tunnel syndrome encountered in the workplace and is usually unambiguous when it does occur.

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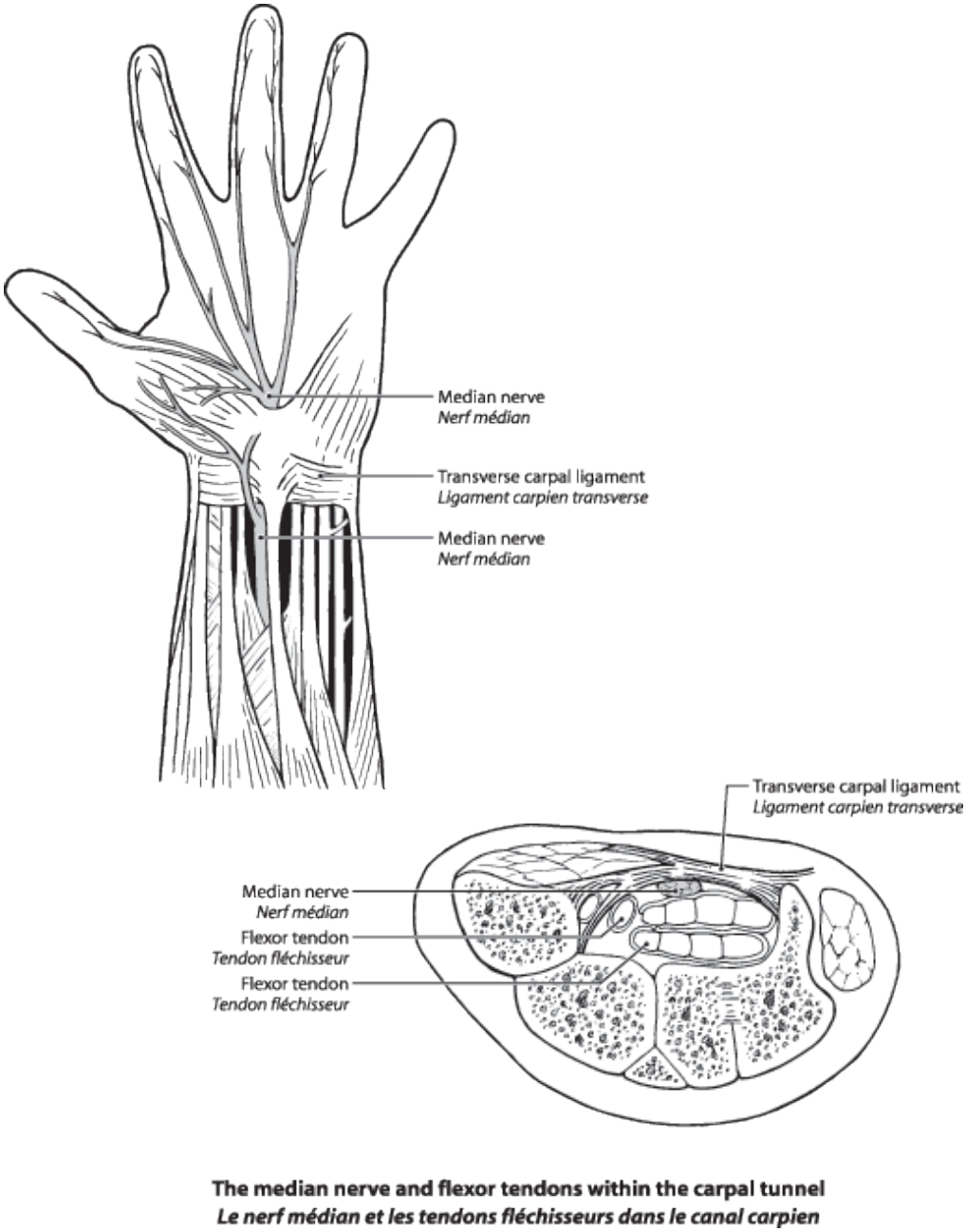
Appendix

These diagrams have been commissioned by the Tribunal in order to help the reader understand the anatomy of the carpal tunnel and the median nerve.



The median nerve and flexor tendons within the carpal tunnel
Le nerf médian et les tendons fléchisseurs dans le canal carpien

Figure 1 - The median nerve and flexor tendons within the carpal tunnel

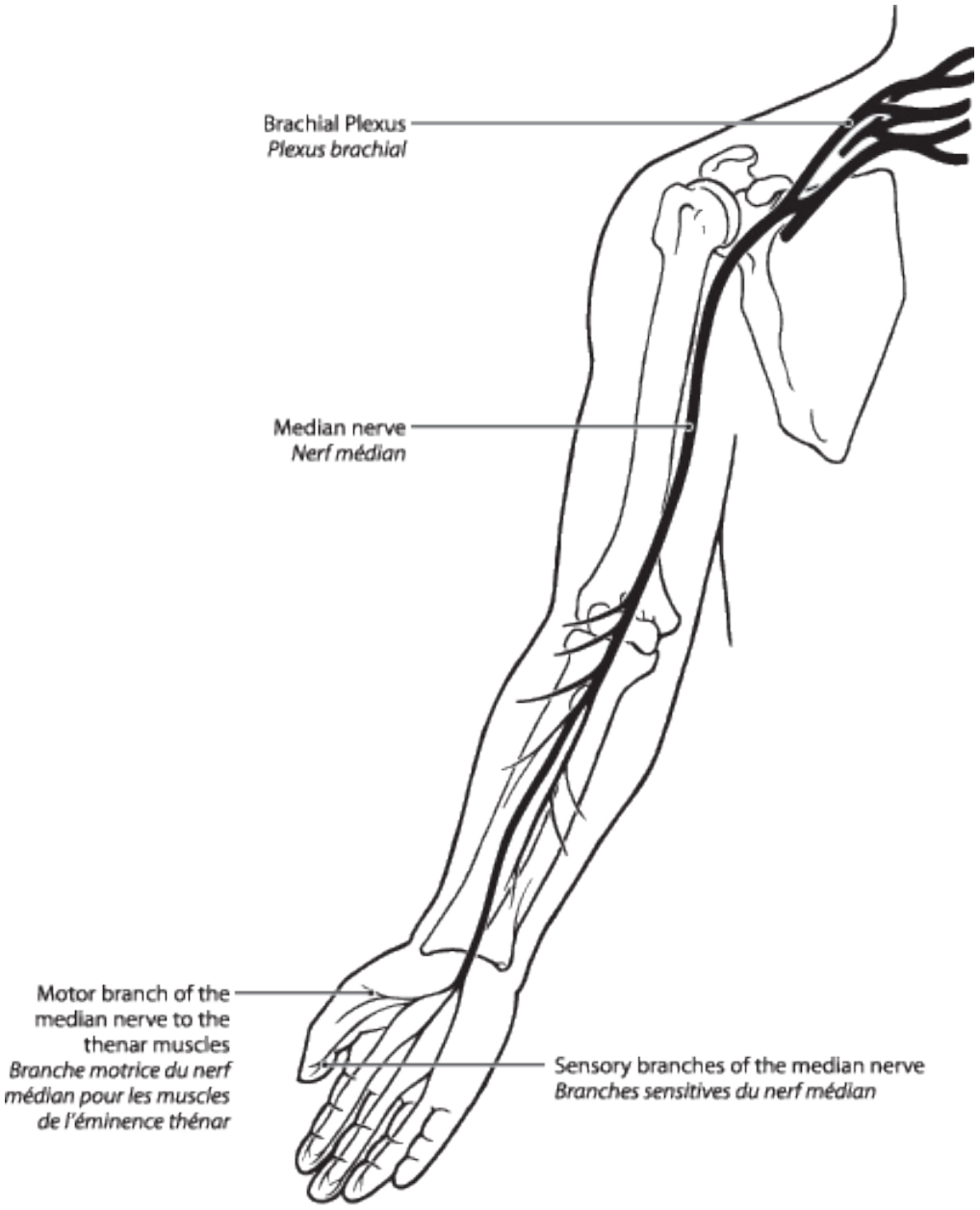
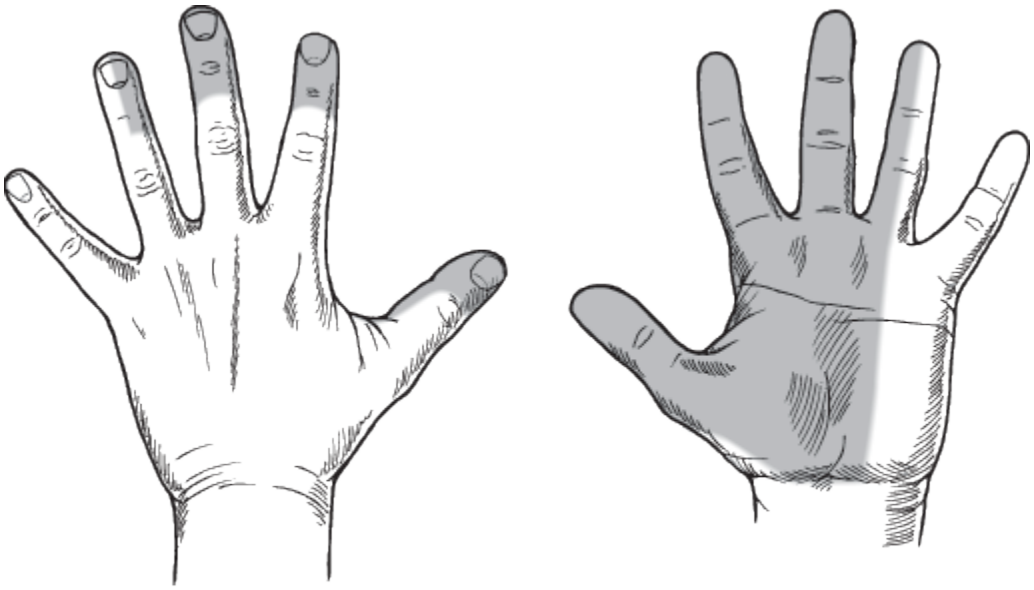


Figure 2 - The median nerve



Area of the skin supplied by the media nerve
Régions de la peau parcourues par le nerf médian

Figure 3 - Area of the skin supplied by the media nerve