The Hoffmann-Tinel sign: Historical Background and Clinical Significance

Sinal de Hoffmann-Tinel: contexto histórico e significado clínico

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Abstract

The Hoffmann-Tinel sign is well-known to professionals dealing with nerve lesions and is widely used as a provocative test. It was described by Paul Hoffman and Jules Tinel in the same year (1915), independently. In the present article, a biographical sketch of both authors is presented and the method for eliciting the sign and the sometimes controversial information of its results are discussed.

Keywords

- hoffman-tinel sign
- tinel sign
- tingling sensation
- paul hoffmann
- jules tinel
- nerve regeneration

Resumo

O sinal de Hoffmann-Tinel é bem conhecido pelos profissionais que lidam com lesões de nervos, sendo amplamente utilizado como um teste provocativo. Foi descrito por Paul Hoffmann e por Jules Tinel no mesmo ano (1915), de forma independente. No presente artigo, é apresentado um esboço biográfico de ambos autores e são discutidas a forma de obter o sinal e as informações, por vezes controversas, fornecidas por seus resultados.

Palavras-chaves

- sinal de Hoffmann-Tinel
- sinal de Tinel
- sensação de formigamento
- paul hoffmann
- jules tinel
- regeneração de nervos

Introduction

The Hoffman-Tinel sign (HTS) is a well-known and widely used provocative test in the clinical evaluation of regeneration of an injured nerve and to localize the level of nerve injury. This simple test does not require any instrument and can be easily performed by an examiner using only his finger.[1] A positive HTS is interpreted as paresthesia elicited across the area that corresponds to the location of the most distal sprouts of regenerating axons. Several years after it

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was described, the HTS is also useful for the diagnosis of nerve compression. Although this sign was described much earlier, it was merely considered a phenomenon; its usefulness was acknowledged only after its clinical application in 1915.[2–4]

Definition of the Hoffmann-Tinel Sign

A "positive" HTS refers to paresthesia (tingling sensation) elicited along the distal sensory distribution of an injured nerve or at the site of the injury, provoked by a mechanical stimulus (percussion or pressure). The sensation, which is comparable with that produced by weak electrical stimulation, radiates peripherally from the point where it is triggered to the cutaneous distribution of the nerve. Individuals experience a brief unpleasant sensation but not pain.[5] The peripheral reference of the sensation differs from that elicited by striking a healthy nerve. It is stimulated more easily and persists longer (10 to 15 seconds) after cessation of the stimulus.[6]

The HTS indicates the level of compression or regeneration of peripheral nerve fibers and is also useful to trace the path of recovery or peripheral nerve regeneration along the course of a nerve and across the site of injury from proximal to distal. The most peripheral point at which the tingling sensation is experienced is considered the site to which the fibers have regenerated.

The HTS first becomes evident $\sim$ 4 to 6 weeks after suture or injury.[7] Typically, only $\sim$ 30 centimeters of a nerve is sensitive to percussion at a particular time, which indicates advancement of regenerating axons over the distance and myelination of the proximal part, although these may not necessarily be functionally mature axons.[6]

The neurophysiological or pathophysiological processes underlying a positive HTS is unknown. Demyelination and partial remyelination, accompanied by axonal degeneration and regeneration in chronic nerve entrapment, render the peripheral nerve mechanosensitive.[8]

The HTS undergoes alterations during the course of nerve compression, probably associated with the degree of pathological changes present at the time of evaluation. The HTS tends to be positive during the course of chronic nerve compression and may subsequently show a negative result with further progression of compression.[9]

Overall, a strongly positive HTS elicited immediately postinjury indicates nerve rupture or severance. A centrifugally moving HTS is persistently stronger than that elicited at the suture line suggests the possibility of successful nerve repair, and an HTS that remains stronger than the suture line than that at the growing point suggests the possibility of failure of nerve repair.[10] Failure of distal progression of HTS in a closed lesion indicates rupture or other lesions that may interfere with successful regeneration.[11]

History of the Hoffmann-Tinel sign

Many descriptions of the currently recognized HTS are available in the literature before the classical 1915 publications. However, these early descriptions were purely physiologic in nature, and practical application and clinical interest in the HTS were highlighted only after 1915.

Ibn Sina, one of the most eminent Persian physicians, best known to the West as Avicenna (980–1037 AD), is credited with the first description of compression neuropathy and its clinical examination. In *The Canon of Medicine* (al-Qanun-fi al-Tibb), one of the oldest and most influential historical texts of medicine, Avicenna states '…manual compressing of the hardened nerves produces numbness'.[12]

In his essay (1819) titled *A Dissertation on the Treatment of Morbid Local Affections of the Nerves*, Joseph Swan wrote that '…when a nerve is pressed against a bone for a short time, an uneasy sensation is produced and the parts to which it is distributed feel benumbed'.[13]

Jean Joseph Emile Létiévant, a French surgeon and professor of physiology described a similar sign in many cases of median nerve lesions in his book *Traité des Sections Nerveuses* [Treatise on Nervous Sections], published in 1873.[14]

In 1905, Henry Head observed 'a curious widespread formication' produced by a von Frey hair aesthesiometer, '…that radiates widely over the affected area'.[15]

Wilfred Trotter and Hugh Morriston Davies mentioned '… a large number of sensations elicited from a recovering area referred to distant parts of the area or to the point of the nerve section' in two publications (1909 and 1913).[16,17]

Paul Hoffmann (1884–1962) (►**Fig. 1**) was a German neurophysiologist who performed significant research in human reflexes. He was born in Dorpat (presently in Estonia), which previously belonged to Russia but was inhabited by many German families after the 13th century.[18] Following medical education in Berlin, Marburg and Leipzig, Hoffman graduated from Leipzig University in 1908. After graduation, he worked as a research assistant at the Physiological Institute in Berlin. In 1911, he assisted Professor Max von Frey at the Institute for Physiology of the University of Würsburg. He was primarily involved with research on the action potentials of muscles and the electrophysiology of reflexes and was

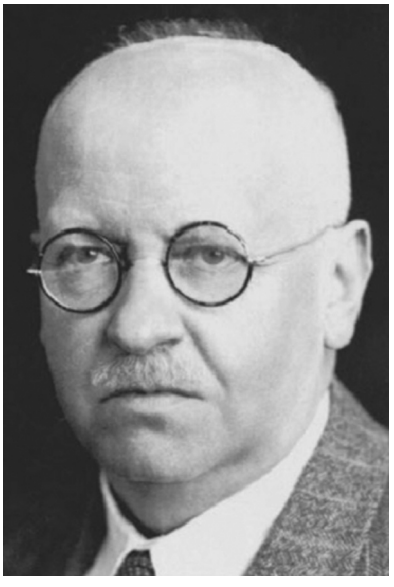

Fig. 1 Paul Hoffman (1884–1962).

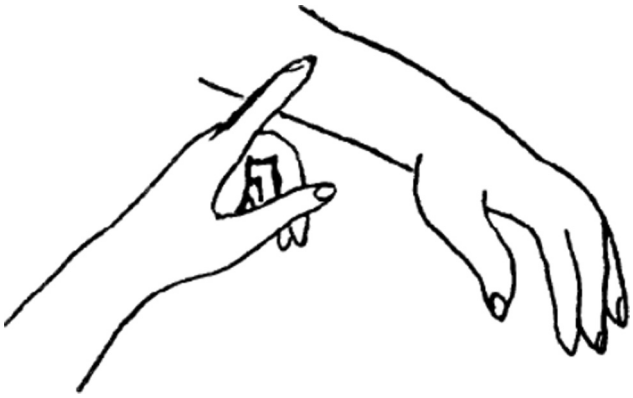

Fig. 2 Illustration from Hoffmann's paper[3] demonstrating the position of the hand during percussion over the radial nerve. Public domain.

recognized as a prolific researcher and writer. Hoffmann published 32 articles before the outbreak of the First World War.[1]

Hoffmann served in the German Army at several field hospitals in France and later at a military hospital in Würzburg during the First World War. He observed that percutaneous percussion of injured and regenerating peripheral nerves elicited a tingling sensation that radiated along the sensory distribution of that nerve in wounded soldiers. He wrote two articles about this sign; the first article published in *Medizinische Klinik* (28 March 1915) described the phenomenon,[2] and the second article in the same journal (1 August 1915) described details of the percussion method used to elicit this sign (►**Fig. 2**).[3] He interpreted the sign as evidence of newly formed, extremely sensitive regenerating nerve fibers.

Between 1912 and 1924, Hoffman rose through the academic ranks from Privatdozent (private lecturer) to Professor at the medical school in Würzburg. In 1924 he joined the faculty of the Physiology Institute at the University of Freiberg-im-Breisgau Medical School as Chair of Physiology.[19] In 1924, he was appointed Director of the Institute for Physiology, where he worked until his retirement in 1954.[1]

At the end of the First World War, Germany was expelled from all international scientific forums. Faced with the boycott of the winners, Germany collaborated with countries that remained neutral in the contest, such as Spain. Paul Hoffmann delivered lectures in Physiology at the School of Medicine, University of Santiago de Compostela, Spain, in 1923 (3 months) and in 1924 (one and a half month) (►**Fig. 3**).[20]

In 1932, Hoffman protested against the Nazi government that restricted him from teaching Jewish students. He nearly lost his position as Chief of Physiology. In November 1944, the Physiology Institute was demolished by errant Allied Forces' aerial bombs,[19] and Hoffman was compelled to relocate to other buildings until the construction of a new institute, where he remained until retirement. According to some historians, Hoffmann had an ambivalent relationship with the Nazi regime.[21] He died in 1962, at the age of 77 years old.[1]

Fig. 3 Paul Hoffmann (1), the Dean of the Faculty (4) and members of the Department of Physiology at the School of Medicine, University of Santiago de Compostela, Spain in 1941. (Reproduced from reference [20] by permission of Cuadernos de Estudios Gallegos).

Fig. 4 Jules Tinel (1879–1952).

Initially, Paul Hoffmann was not widely recognized. It was only through retrospective accounts by medical historians that Hoffmann has begun to receive credit for introducing the sign that he first described in detail.[1]

Jules Tinel (1879–1952) (►**Fig. 4**) was born in Rouen, France, into a family that included five generations of surgeons and physicians. He completed his medical studies in 1906 in Paris and, influenced by Joseph Jules Dejerine, a prominent clinical neurologist and one of his tutors, Tinel began to specialize in neurology and neuropathology.[22,23] In 1911, Tinel became "Chef de Clinique" [Clinic Director] and in 1913 he worked as Chief of the Laboratory at the Neurological Department at the Pitié-Salpetrière Hospital in Paris (►**Fig. 5**). By the end of 1913, Tinel had authored more than 40 publications that, for the most part, were associated with neurological issues.[24] However, these activities were suddenly interrupted by the war.

Following the outbreak of the First World War, Tinel served as an auxiliary physician for an infantry regiment. In March 1915, he was appointed second class assistant physician and was entrusted the responsibility for the Neurology Centre of the 4th military region in Le Mans, France.[25] The numerous traumatic lesions of the peripheral nerves captured his attention, and he was interested in gaining deeper insight into the consequences of ballistic trauma-induced peripheral nerve injuries,[26] which he extensively studied for 3 years. Tinel's exhaustive research led to the understanding of an effect in which compression of an injured nerve led to paresthesias similar to a tingling sensation ['sign du fourmillement'].[4] Tinel also discovered that following progressive regeneration of an injured nerve, the HTS tended to shift to a more peripheral location.

During the Second World War, when he worked at the Boucicaut Hospital, Tinel was actively involved in the French

Fig. 5 Members of the Neurological Department at the Pitié-Salpetrière Hospital in Paris (1912). 1. Jules Tinel; 2. Joseph Jules Dejerine; 3. Augusta Dejerine- Klumpke. Public domain.

Resistance Movement. He belonged to a network referred to as Comete that provided shelter in his home to allied wounded pilots whose planes were shot down over occupied Europe until his son, Jacques Tinel, could drive these men to Spain.[25,27] His son was arrested and transferred to the concentration camp in Mittelbau-Dora, where he died.[26] Tinel spent 3 months in prison in Bordeaux, and his wife and another son spent a year in prison in Fresnes, in the southern suburbs of Paris. The Comete network was completely disabled in 1944.[27]

Following his retirement in 1945, Tinel continued to work in Paris at the Boucicaut Hospital. In 1947, he developed cerebral ischemia and aphasia. He recovered within some weeks and maintained an active research laboratory until his health declined, and he died of heart failure in 1952 at the age of 73 years old.[26,28]

Description of the Hoffman-Tinel sign

In his first paper (March 1915), Hoffman described the phenomenon in two cases of radial nerve lesions during the war and in the second (August 1915) he discussed details regarding the method of percussion to elicit the sign.[2,3,25] In his first paper, Hoffman described the case of a 22-year-old officer who sustained a gunshot wound to the right humerus accompanied by radial nerve transection and sensory loss along distribution of the radial nerve in the hand. The nerve was repaired 2 months later, and moderate finger pressure on the area of the lesion/site of surgery produced a tingling sensation in the indicated area of sensory loss in the hand corresponding to the radial nerve distribution, 3 months postoperatively. More distal stimulation produced no sensation. The patient demonstrated weak extension of the hand, 4 months postoperatively.

Hoffman designated this test 'klopfversuch' [a tapping trial].[2] He emphasized the importance of gentle percussion to avoid an inaccurate result considering that paresthesia may be elicited even in healthy nerves. Hoffmann proposed that a single tap on a ruptured nerve is usually sufficient to provoke paresthesia that could continue for minutes in the innervated area.

In an article titled 'Le signe du fourmillement dans les lesions des nerfs périphériques' [The tingling sign in peripheral nerve damage], published in *Presse Médicale* (October 1915), Jules Tinel reported the conclusions of his clinical experience of war as follows: 'The pressure of a wounded nerve trunk produces very often an impression of tingling, exteriorized by the subject at the periphery of his nerve, and localized by him to a precise territory. It is important to differentiate this tingling from the pain that sometimes also occurs in traumatic nerve lesions. Pain is a sign of neuritic irritation; tingling is a sign of regeneration or, more accurately, tingling reflects the presence of young, growing axons'. Tinel called it "le signe du fourmillement" [the tingling sign].[4]

Tinel carefully distinguished between "formication" generated by pressure over a nerve and referred distally and "sensibility of nerve on pressure" observed in cases of neural irritation, which is invariably experienced as a local sensation.[29] Based on his observations, Tinel stated that the sign is usually seen 4 to 6 weeks after trauma and that the formication sign may disappear over 8 to 10 months or may require an even longer interval following the process of regeneration.[4,30] He was of the opinion that the total absence of the sign below the lesion was pathognomonic of a complete interruption (rupture) if sufficient time had elapsed since injury for the onset of regeneration.[4] Owing to the wartime information blockade, neither author was aware of the other's scientific work.[31]

Method to Elicit the Hoffman-Tinel Sign

Many different descriptions of stimuli to trigger the HTS in an injured nerve have been described, including a 'gentle stroking touch with the finger', 'tapping', 'tapping with a straight finger', 'gentle tapping with a finger', a 'von Frey hair tapping repeatedly', 'pressure', 'percussion', 'pressure with a foam instrument 2–3 mm in width', or even 'gentle percussion with a reflex hammer', or 'a home-made instrument of a rubber eraser fixed at the end of a pencil'.[5]

Tinel originally mentioned that pressure should be applied to the injured nerve to elicit the sign,[4] whereas Hoffman proposed that light percussion was more accurate,[3] and Hoffman's maneuver is more frequently used in clinical practice. Stronger percussion or firmer pressure is necessary to test deeper nerves.

Light percussion by the examiner's finger over a small area along the course of the injured nerve elicits the HTS. A tingling sensation experienced by a patient along the cutaneous distribution of the nerve is interpreted as a positive HTS. Depending on the clinical scenario, the point at which symptoms are provoked indicates nerve compression or regeneration. The nerve is usually stimulated using the examiner's fingertip or the finger in extension (►**Fig. 2**). Subsequent examination may indicate centrifugal progression of the HTS, which becomes progressively stronger at the distal compared with the proximal level of the HTS and indicates nerve regeneration.[11]

Tinel versus the Hoffman-Tinel sign

Ideally, the sign should be referred to as the eponymous 'Hoffman-Tinel Sign'; however, it is currently more commonly known as the Tinel sign in clinical practice, which is perhaps attributable to the fact that Hoffman and Tinel belonged to opposite sides of the war frontline, and neither author was aware of the other's work owing to the wartime blockade of international communication.[31] Although both described an equivalent sign in the same year, Hoffmann belonged to the losing side, and consequently his work was not valued within the scientific community.[18] Furthermore, the lower rank of the German periodical and censorship that affected German publications perhaps contributed to the wider popularity of the Tinel sign.[26] Notably, Tinel's closer ties to the western world, better writing skills, and a keen sense of observation favored rapid translation of his work into English.[1] Tinel's work

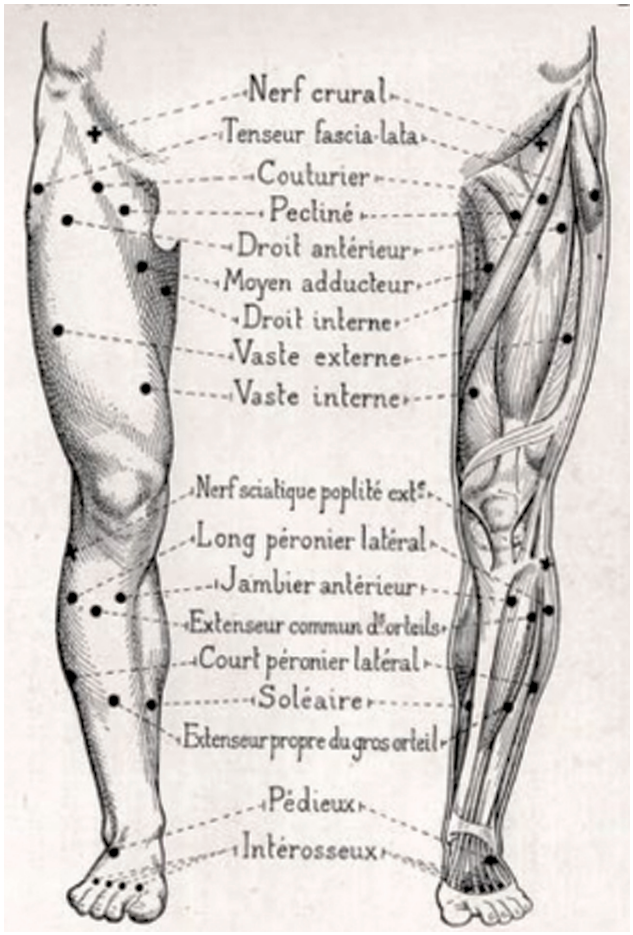

Fig. 6 High-quality illustrations from Tinel's book (1916).[30] Public domain.

includes more comprehensive descriptions, and he pursued research on nerve injuries and published an extensive and renowned reference book titled *'Les Blessures des Nerfs. Sémiologie des Lesions Nerveuses Périphériques par Blessures de Guerre'* [Nerve Injuries. Semiology of Peripheral Nerve Lesions by War Injuries] (1916)[30] based on his personal experience (►**Fig. 6**). The book was translated into English in 1918 and was instrumental in contributing to the wider fame achieved by Tinel and the widespread use of 'Tinel sign' in non-German-speaking medical communities. The 'Hoffmann sign' remained popular in Germanic countries.

Both authors hypothesized that a positive HTS indicated the development of young axons during the process of regeneration[2–4,30]; however, fundamental differences between their observations include the following: Hoffmann observed that the percussion test indicates regeneration of sensory fibers and not motor fibers; however, Tinel did not address this limitation. A positive sign could indicate the possibility of restoration of motor function, but it is not a guarantee of motor regeneration.[1] Hoffmann proposed that regenerating nerve fibers have an extremely low stimulation threshold compared with healthy nerves and therefore recommended the use of light pressure to perform the test because an intact nerve can be stimulated with much higher pressure intensity. However, Tinel virtually did not comment on the method used to elicit the tingling sign. Similarly, Tinel presented his own original ideas. Although Hoffmann described the use of the sign in a more cursory manner, Tinel discussed the application of the sign in greater detail in his book.[30] Tinel has extensively described the syndromes of complete nerve interruption, compression, irritation, and regeneration. In all these cases, he outlined when to expect the formication sign and used the sign to evaluate a patient's condition and to determine the indication for surgical intervention. Tinel addressed the difference between pain and tingling, both of which may be observed upon stimulation of the injured nerve trunk. Tinel explained that pain implies nerve irritation, whereas tingling suggests nerve regeneration. Based on his observations, Tinel was of the view that the sign is usually observed 4 to 6 weeks postinjury; when the nerve is in the process of regeneration, the formication sign may disappear in 8 to 10 months or in an even longer period, to disappear.[4,30]

Clinical Significance of the Hoffman-Tinel sign

The clinical value of the HTS remains controversial. Initially, Hoffman and Tinel's research received negative feedbacks and reactions, and the clinical information obtained from application of the HTS was frequently misunderstood, particularly by clinicians who observed that a positive HTS did not ensure a favorable outcome. Although Tinel emphasized that a positive sign predicted future nerve recovery, some patients recovered full neurologic function without showing a positive HTS. Therefore, many physicians questioned this finding, and the sign was virtually neglected for nearly 30 years.[27] It was only by the end of the Second World War that the significance and usefulness of the HTS was fully appreciated.

In 1948, Henderson,[32] a British neurosurgeon and prisoner-of-war, published his observations based on repeated attempts to elicit HTS in over 400 patients with nerve injuries in field hospitals across Germany, at locations in which surgical treatment was not possible. Henderson observed that the HTS is clinically important $\sim$ 4 months after injury; he was of the view that a strongly positive sign at the level of the lesion with gradual weakening with peripheral movement of the response and a stronger sign in the distal part of the nerve indicated satisfactory progress of regeneration.

Researchers have emphasized that paresthesia phenomenon alone does not predict nerve regeneration. It is important to confirm gradual distal progress of the tingling elicited in a patient for accurate prediction of nerve regeneration. However, if the HTS remains static for several consecutive weeks or months, it is indicative of a likely obstacle to the growth of the nerve fibers[4] or may indicate nerve rupture,[11] and surgical exploration is usually warranted in such cases.[29,33] Nerve regeneration is never associated with a painful sensation[34]; patients invariably compare the vague disagreeable sensation with that caused secondary to an electric current.

Clinicians should be mindful of the following points when interpreting the results of nerve percussion[11]:

(1) The HTS is elicited over a site of nerve fiber regeneration, even in areas where these fibers may grow aimlessly.
(2) The nerve fibers that enter endoneural tubes do not necessarily enter the tubes in which they were originally present or in tubes that lead to the former areas of distribution of the fibers.
(3) The HTS does not accurately indicate the number of regenerating nerve fibers.[35]
(4) The HTS may be elicited even without apparent nerve recovery.
(5) The HTS was shown to be absent throughout the period of regeneration in areas with deep-seated nerves; however, good recovery of nerve function was observed even in such cases.[36]

In light of the current knowledge, Birch interpreted a positive HTS as follows[11]:

1. A strongly positive HTS over a lesion soon after injury indicates ruptured axons or a severed nerve;
2. Centrifugal movement of the HTS that is persistently stronger than that at the suture line suggests a strong likelihood of successful repair.
3. The HTS at the suture line that remains stronger than that shifting distally suggests a strong likelihood of failure of repair.
4. Failure of distal progression of the HTS in a closed lesion indicates rupture or some other injury that is not likely to show natural recovery;
5. A positive HTS indicates a degenerative lesion (not a conduction block) because the injured nerve contains a significant number of axons.
6. A positive HTS result should not be confused with hypersensitivity observed in some cases of neuralgia.

Accurate interpretation of the HTS in brachial plexus lesions is challenging. Landi et al. observed the following responses after tapping in the posterior triangle of the neck[37]: (1) A complete lack of response implies preganglionic injury to the nerve root; (2) Local pain implies a recovery of an underlying cervical plexus lesion; (3) A pure HTS indicates a lesion in anatomic continuity with progressive recovery documented on sequential recordings and sequential recordings can demonstrate progression of recovery. The C5 and C6 nerve roots are most superficial, and the HTS can easily be elicited at this level. C7, C8 and T1 are deep-seated and therefore difficult to evaluate; (4) The neuroma sign is positive in patients in whom pain is elicited along the distribution of the nerve when tapped. This sign indicates disruption of continuity of the nerve.

Birch reported that radiation up to the level of the elbow following percussion in the posterior triangle of the neck suggests rupture of C5 nerve root, radiation that extends to the lateral forearm and thumb suggests C6 nerve root rupture, and radiation that extends to the entire hand, particularly over the dorsum, suggests C7 nerve root rupture.[11]

At the annual meeting of the American Society for the Peripheral Nerve in 2004, Millesi reported the results of HTS testing in 42 consecutive patients with brachial plexus lesions and compared these with surgical findings.[38] He concluded that an absence of HTS supports root avulsion, and an HTS that radiates into the territory of a spinal nerve strongly suggests that at least one root remains intact.

True HTS is experienced as a tingling sensation elicited by stimulation of nerve branches that contain growing touch fibers. In a mixed nerve, touch fibers account for only $\sim$ 10% of all of the fibers; therefore, only a few touch fibers need to remain intact to produce a positive HTS, and the sign provides information regarding regeneration of touch and not of other fibers.[34]

The HTS is consequent to an increased mechanoreceptor sensitization and seems to indicate the presence of young axis cylinders in the process of regeneration; however, the significance appears questionable because this sign is often absent and only sometimes positive even in those with complete nerve division.[39] The absence of the HTS is perhaps of no diagnostic value; however, a positive HTS that advances peripherally from a nerve lesion suggests a strong likelihood of regeneration.[39] However, this sign represents only sensory nerve fiber regeneration; therefore, a positive test provides physiological evidence of nerve fascicle regeneration but does not predict restoration of voluntary movement. Furthermore, some of the regenerating axons are not on their way to any target.

The HTS is often utilized for diagnosis of entrapment neuropathies, particularly median nerve compression within the carpal tunnel or the ulnar nerve compression in the postcondylar groove. Changes during nerve compression are associated with the degree of pathology present at the time of evaluation; a positive HTS elicited during the course of nerve compression changes to a negative sign with further progression of the pathology.[9] The popularity of the HTS for the diagnosis of carpal tunnel syndrome is largely attributable to Phalen, who reported that the Tinel sign showed sensitivity of 73% in 452 patients in whom the clinical presentation was used as the diagnostic standard.[40,41]

The HTS-like elicited by percussion over schwannoma or over nerves in the early stages of entrapment neuropathy does not indicate ruptured axons and suggests sensitization of nerve fibers secondary to focal demyelination and changes in the expression of voltage-gated ion channels at the level of lesion.[11]

Studies have reported a wide range in the sensitivity of the HTS (23–62% [mean 42.4%]), although, usually, it is observed to be highly specific (64–93% [mean 76.4%]).[42–48] Absence of the HTS does not necessarily rule out the diagnosis.[49]

Conclusion

Increased mechanoreceptor sensitization is considered the mechanism underlying the HTS, which suggests the presence of young axis cylinders in the process of regeneration within an injured nerve. However, the significance of this sign appears questionable; it is often absent and only occasionally positive

even in patients with complete division of a nerve. An absent HTS is perhaps of no diagnostic value; however, a positive HTS that advances peripherally from a nerve lesion is strongly suggestive of a regenerative process. However, this sign is associated only with sensory nerve fibers; therefore, a positive test provides physiological evidence for regeneration of nerve fascicles and does not predict restoration of voluntary movement. Furthermore, some regenerating axons are not on their way to any target. If a nerve repair is going to be successful centrifugal moving of the HTS that is persistently stronger than that at the suture line suggests a strong possibility of successful nerve repair, and a HTS that remains stronger at the suture line than that at the growing point is highly suggestive of failure of repair. Failure of distal progression of the HTS in a closed lesion indicates rupture or other lesions that interfere with regeneration. Currently, the HTS is widely used clinically; however, there is lack of standardization, its grading is rarely used, and its reliability or validity is scarcely mentioned in the literature. We recommend that too much should not be expected of the sign, which must be interpreted only in conjunction with other clinical findings.

Conflict of Interests

The authors have no conflict of interests to declare.

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