



Universiteit
Leiden
The Netherlands

Peripheral nerve damage : diagnosis and prediction of recovery

Datema, M.

Citation

Datema, M. (2017, November 29). *Peripheral nerve damage : diagnosis and prediction of recovery*. Retrieved from <https://hdl.handle.net/1887/57509>

Version: Not Applicable (or Unknown)

License: [Licence agreement concerning inclusion of doctoral thesis in the Institutional Repository of the University of Leiden](#)

Downloaded from: <https://hdl.handle.net/1887/57509>

Note: To cite this publication please use the final published version (if applicable).

Cover Page



Universiteit Leiden



The handle <http://hdl.handle.net/1887/57509> holds various files of this Leiden University dissertation

Author: Datema, Mirjam

Title: Peripheral nerve damage : diagnosis and prediction of recovery

Date: 2017-11-29



Tinel's sign has no diagnostic value for nerve entrapment or neuropathy in the legs

Mirjam Datema, Elske Hoitsma, Krista I. Roon, Martijn J.A. Malessy,
J. Gert van Dijk, Martijn R. Tannemaat

Muscle Nerve. 2016 Jun;54(1):25-30



ABSTRACT

Objective: The presence of Tinel's sign in leg nerves has been proposed as a criterion for decompressive surgery in polyneuropathy. We investigated the diagnostic yield of Tinel's sign for nerve entrapment and for distal symmetrical peripheral neuropathy (DSPN).

Methods: We prospectively tested for Tinel's sign at three sites of possible nerve entrapment per leg in 91 patients. Entrapment was defined using nerve conduction data. We also investigated whether the number of sites where Tinel's sign was present identified patients with DSPN.

Results: Sensitivity of Tinel's sign for nerve entrapment was low (29%, 44% and 17%) for the three sites and specificity moderate (86%, 75% and 81%). In the subgroup with DSPN sensitivity was extremely low (0%, 20% and 8%) and specificity was moderate (91%, 79% and 73%). The number of sites with Tinel's sign did not identify patients with DSPN.

Conclusion: Tinel's sign does not reliably indicate nerve entrapment or DSPN.

INTRODUCTION

The provocation of paraesthesia by percussion of a damaged nerve is generally referred to as "Tinel's sign" as it was first described by Tinel and Hoffmann in 1915.¹ They assumed that paraesthesia is elicited by generating sensory nerve activity in unmyelinated axonal endings and that the site where paraesthesia can be elicited corresponds to the location of the most distal sprouts of regenerating axons.¹ Later, Tinel's sign became associated with the diagnosis of nerve compression, specifically in carpal tunnel syndrome (CTS), based on the hypothesis that its presence indicates nerve entrapment. However, the use of Tinel sign both in the assessment of nerve injuries and in the diagnosis of nerve entrapment has been the subject of debate.¹ There is evidence that it has limited or no diagnostic value in diagnosing carpal tunnel syndrome and ulnar nerve entrapment.²

Recently, there has been a resurgence in the use of Tinel's sign as an indicator of nerve compression in the legs, following reports on surgical nerve decompression as a treatment of painful diabetic peripheral neuropathy.³ In 2004 Lee and Dellon reported that Tinel's sign at the tarsal tunnel was an excellent predictor of success of decompressive nerve surgery and they deduced that its presence indicates local nerve compression.^{4,5} The sign has since been used as a criterion for decompressive nerve surgery in diabetic and idiopathic polyneuropathy in a number of studies.^{3,6-9} The underlying hypothesis is that patients with polyneuropathy are more vulnerable to nerve entrapment at sites of known anatomic narrowing.³ Reported sites were the common peroneal nerve at the fibular neck, the deep peroneal nerve over the dorsum of the ankle and the tibial nerve below the medial malleolus.⁸ Except for the study of Lee and Dellon there appear to be no other studies that systematically evaluated the diagnostic yield of Tinel's sign for compression neuropathies in the legs.⁵ Two studies reported the presence of a Tinel sign in patients with clinical nerve entrapment syndromes in the leg, but lacked control subjects without nerve entrapment.^{10,11} The main objective of the present study was to investigate the diagnostic yield of Tinel's sign for nerve entrapment in the legs. We first studied whether Tinel's sign at the three sites described was associated with nerve entrapment defined using nerve conduction data in a population including controls, patients with distal symmetrical peripheral neuropathy (DSPN) and patients with other nerve pathology (objective 1). As earlier studies were performed on patients with diabetic neuropathy,^{3,6-9} we repeated the analysis in a subgroup of patients with DSPN (objective 2). Finally, we investigated whether Tinel's sign occurred more often in the legs of patients with any nerve pathology or in patients with DSPN, than in subjects without overt nerve pathology. If so, it could indicate nerve pathology and not specifically nerve entrapment (objective 3).

MATERIAL AND METHODS

Subjects

The study randomly included subjects referred for nerve conduction studies (NCS) of the legs from October 1, 2011 until February 1, 2013 in two regional hospitals, the Reinier de Graaf Hospital in Delft and the Alrijne hospital in Leiden. The inclusion criterion was suspicion of a peripheral nerve disorder in the legs, manifested by symptoms of pain, paraesthesia, or numbness in one or both legs, with or without muscle weakness. The study was approved by the local medical ethics committees. All participants provided written informed consent. Procedures followed were in accord with the Helsinki Declaration of 1975. Age, sex, body height, weight and the presence or absence of diabetes mellitus (DM), defined by the use of anti-diabetic medication, were recorded.

Nerve conduction testing and assignment of clinical diagnoses

Nerve conduction measurements were performed according to the protocols of the Dutch Society for Clinical Neurophysiology, using a Viking Select EMG machine (Nicolet) at both hospitals.^{12,13} The temperature of the legs was maintained at $\geq 30^{\circ}\text{C}$. The selection of measurements was left to the discretion of local clinical neurophysiologists. The data were analysed in two ways.

The first analyses consisted of a classification in three groups based on the diagnoses reached by the neurologist treating the patient. These diagnoses were based on history, clinical findings, nerve conduction testing and additional investigations (e.g., imaging of the lumbar spine). The three groups were no neurological pathology, distal symmetrical polyneuropathy (DSPN), and neurological pathology other than DSPN such as peroneal nerve entrapment at the fibular head and monoradiculopathy (see Results).

The second analysis of nerve conduction data was performed to define nerve entrapment. We defined a criterion for each site based on published guidelines or reports. For the peroneal nerve at the fibular neck we required that: a) that the conduction velocity over the fibular head was at least 10 m/s lower than in the leg segment, recorded from the extensor digitorum brevis muscle; or b), that the amplitude of the compound motor action potential (CMAP) obtained after stimulation in the popliteal fossa was at least 25% less than that obtained using stimulation at the fibular neck.¹³ For the peroneal nerve at the dorsum of the ankle we used a prolongation of distal motor latency (DML) of > 2 standard deviations (SD) of published data corrected for age and height, obtained after stimulation at the ankle.¹⁴⁻¹⁶ Finally, for the tibial nerve we used a $> 2\text{SD}$ DML prolongation, corrected for age and height.¹⁷ Using these thresholds, each nerve tested was assigned a yes/no label as to whether it met demands for nerve compression. This allocation was also done using 1SD and 3SD thresholds to explore their potential influence on diagnostic yield.

Evaluating Tinel's sign

Prior to performing NCS, i.e. blinded to the outcome of NCS, the presence or absence of Tinel's sign was assessed by a trained technician who followed a standardised protocol. The technician firmly tapped each site three times with the index finger and middle finger while asking the patient: "Do you feel a tingling or electric-like sensation irradiating below the site of tapping?" The technician instructed the patient to answer either 'yes' or 'no'. In case of doubt technicians were allowed to repeat tapping only once at each site. The Tinel sign was considered absent if the patient answered 'no' or if the patient remained uncertain after repeated tapping. The test was carried out not only at the three sites mentioned above (the neck of the fibula, anterior aspect of the ankle over the inferior extensor retinaculum, hereafter labelled the dorsum of the ankle, and below the medial malleolus) but also at one additional site, i.e. below the lateral malleolus overlying the sural nerve. This nerve is not known to be involved in a specific compression neuropathy, but is involved in diabetic and other types of neuropathy. If Tinel's sign represents nerve entrapment it will not be present over the sural nerve, but if it indicates nerve pathology in general it may. For each patient we calculated a Tinel sum score ranging from 0-8, reflecting the number of times Tinel's sign was present at the four sites in both legs.

Analysis

Data were analysed with SPSS (Statistical Package for Social Sciences), version 20. Differences in age, body mass index (BMI) and sex between the three clinical groups were assessed using one way analysis of variance and the Chi-square test respectively.

It is possible that examiners differ in their tendency to label Tinel's sign as present or absent. Thus, we arranged Tinel sum scores in groups of patients investigated by one technician and compared the mean of the sum scores between technicians using one way analysis of variance (ANOVA). The sum score ranges from 0 to 8 in discrete steps and is not a true continuous variable, but the range is large enough to use ANOVA to explore these differences. The significance level was set at $p < 0.05$.

To test the first objective, that is, the diagnostic yield of Tinel's sign for nerve entrapment, we studied whether its presence correlated with nerve entrapment at each of the three sites. Cross-tabulations were made to calculate sensitivity and specificity and the Chi square test was used to calculate statistical significance. Significance was defined as $p < 0.05$. For the second objective, the same analysis was performed in a subgroup of patients with DSPN.

For the third objective, receiver operating characteristics (ROC) analysis was performed to determine the diagnostic value of Tinel's sign. We performed two ROC analyses using the Tinel sum score. The first concerned the group with any nerve pathology (DSPN or other) versus those without nerve pathology; the second concerned those with DSPN versus those without nerve pathology.

The overall performance of the ROC analysis was quantified by computing the area under the curve (AUC). An AUC of 1.0 indicates a perfect discrimination between groups; a value of 0.5 indicates that the test in question works no better than chance.¹⁸

RESULTS

Baseline characteristics

Ninety-one patients were included of whom 46 (52.0%) were female. Mean age was 59.9 years (range 23 – 90 years) and mean BMI was 26.7 kg/m² (range 18.0 – 41.5). Forty-one patients were classified as having no neurological pathology, 27 were diagnosed with DSPN (including 9 with diabetic neuropathy), 17 had polyneuropathy of unknown aetiology at the time and one had a history of alcohol abuse. Twenty-three patients had other neurological pathology: unilateral compression of the peroneal nerve at the fibular head (n = 9), small fibre neuropathy (n=5), lumbar canal stenosis (n=2), radiculopathy (n=2) or other (n=5). There was no difference in age, sex, BMI or the occurrence of diabetes mellitus between these groups (table 1).

Table 1. Demographics.

	Age, years, (SD)	Sex, female (%)	BMI, kg/m ² (SD)	Patients with DM (%)
No neurological pathology n = 41	56.5 (13.7)	24 (58.5)	26.4 (3.9)	9 (22.0)
DSPN n = 27	64.4 (15.1)	12 (44.4)	26.6 (3.7)	9 (33.3)
Other n = 23	60.8 (14.3)	10 (43.5)	26.7 (5.6)	3 (14.3)
p-value	0.085	0.385	0.943	0.231

There were no significant differences in age, sex, BMI or prevalence of diabetes between patients with no neurological pathology, DSPN or other neurological pathology. DSPN: Distal symmetrical peripheral neuropathy. BMI: Body Mass Index. DM: Diabetes Mellitus.

The occurrence of Tinel's sign.

Thirty seven of 91 patients (40.7%) exhibited Tinel's sign at at least one site. The prevalence did not differ between groups (subjects without nerve pathology, patients with DSPN and patients with other nerve pathology; $p = 0.71$). The mean Tinel sum score did not differ between technicians ($p = 0.72$).

Tinel's sign was present 30/182 times (16.5%) below the lateral malleolus overlying the sural nerve. The rate did not differ between groups; Tinel's sign occurred in 13 legs of patients without neurological pathology (15.9%), in 8 legs in the DSPN group (14.8%) and in 9 legs in those with other neurological pathology (19.6%; $p = 0.43$). Furthermore, the prevalence of Tinel's sign over the sural nerve was similar to that at the other sites.

Objective 1. Diagnostic yield of Tinel's sign for nerve entrapment

Of 123 peroneal nerves, 17 met the criteria for nerve entrapment and 20 exhibited Tinel's sign. It did not occur significantly more often in the entrapment group than in the non-entrapment group ($p=0.11$). Sensitivity of Tinel's sign for entrapment was 5/17 or 29.4% and specificity was 91/106 or 85.8% (Table 2). Similar results were obtained for the dorsum of the ankle using the ≥ 2 SD DML criterion: of 139 assessments, entrapment was found in 9 patients. Tinel's sign did not occur more often in the entrapment than the non-entrapment group ($p=0.19$). Sensitivity was 4/9 (44.4%) and specificity was 98/130 (75.4%). For the tibial nerve ($n=85$), also using a ≥ 2 SD DML criterion, Tinel's sign did not occur preferentially in the entrapment group ($p=0.84$). Sensitivity was 4/23 (17.4%) and specificity was 50/62 (80.6%). The exploratory analyses using ≥ 1 SD and ≥ 3 SD criteria for peroneal and tibial DML did not produce a better diagnostic yield.

Table 2. Diagnostic value of Tinel's sign for nerve compression at three sites of possible nerve compression in the leg.

	All subjects (n=91)			Patients with DSPN (n=27)		
	Fibular neck	Dorsum of the ankle	Below medial malleolus	Fibular neck	Dorsum of the ankle	Below medial malleolus
sens (%)	5/17 (29.4)	4/9 (44.4)	4/23 (17.4)	0/2 (00.0)	1/5 (20.0)	1/12 (8.3)
spec (%)	91/106 (85.8)	98/130 (75.4)	50/62 (80.6)	30/33 (90.9)	27/34 79.4)	11/15 73.3)
p-value	0.113	0.189	0.837	0.656	0.976	0.223

Entrapment of the peroneal nerve at the fibular neck, entrapment of the peroneal nerve at the dorsum of the ankle and entrapment of the tibial nerve directly below the medial malleolus. sens = sensitivity, spec = specificity

Objective 2. Diagnostic value of Tinel's sign for nerve entrapment in patients with DSPN

The same analysis was done in the group of 27 patients with DSPN. Sensitivity and specificity for Tinel's sign in diagnosing entrapment neuropathies in patients with DSPN was comparable to values found in the total group of subjects (Table 2). The exploratory analysis using a ≥ 1 SD threshold for tibial nerve entrapment in patients with DSPN yielded a sensitivity of 5/9 (55.6%) and a specificity of 17/18 (94.4%). The analyses using ≥ 1 SD and ≥ 3 SD for peroneal nerve and ≥ 3 SD for the tibial nerve did not produce a better diagnostic yield.

Objective 3. The Tinel sum score as a test to distinguish between different groups of patients .

The first ROC analysis, concerning the group with any nerve pathology (DSPN or other) versus those without nerve pathology, resulted in an AUC of 0.491 (Figure 1). The second, concerning those with DSPN versus those without nerve pathology, yielded an AUC of 0.469 (Figure 1).

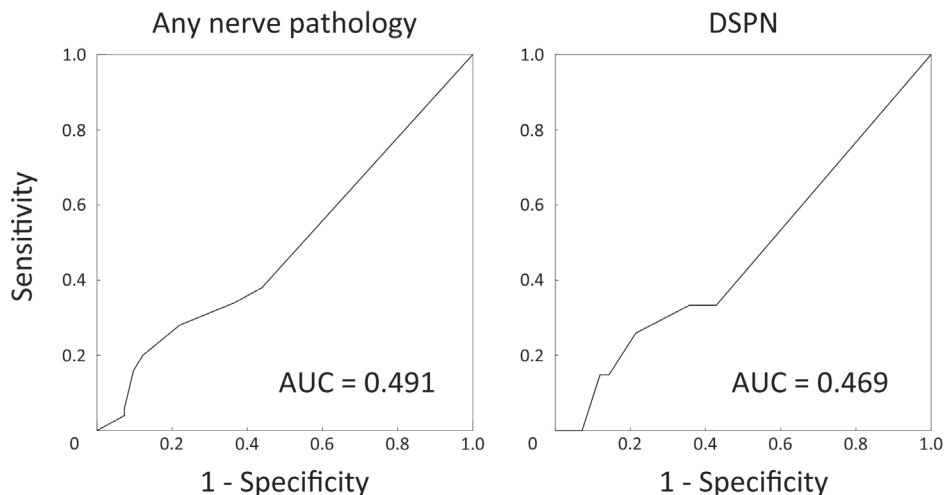


Figure 1. The left panel shows the Receiver operating characteristics (ROC) for the Tinel sum score (range 0-8) of the group with any nerve pathology (DSPN or other) versus those without nerve pathology. The right panel shows the ROC curve of patients with DSPN versus those without nerve pathology.

DISCUSSION

In this study we show that Tinel's sign is a common occurrence and did not indicate nerve entrapment in a heterogeneous group of patients (objective 1) nor in a subgroup of patients with DSPN (objective 2). Tinel's sign does not signify the presence of any nerve pathology, nor the presence of DSPN (objective 3).

Objective 1: diagnostic yield of Tinel's sign for nerve entrapment, and objective 2: diagnostic value of Tinel's sign for nerve entrapment in patients with DSPN

We show that Tinel's sign is not associated with nerve entrapment, as defined by nerve conduction parameters. The only other study reporting sensitivity and specificity for Tinel's sign did not include NCS,⁵ but studied its prognostic value in predicting successful decompressive surgery. Our study was not designed to predict the effect of surgery. Instead, our aim was to examine an assumption that is implicit in the rationale for decompressive surgery: that Tinel's sign can accurately identify nerve entrapment, which it did not. It has been argued that Tinel's sign is particularly useful in patients with such severe diabetic neuropathy that there is no response on standard NCS.³ However, the severity of neuropathy as measured by NCS was not used as a criterion for surgery in published studies on decompressive surgery.⁶⁻⁹

The nerve conduction parameters used here, were selected after a review of the existing literature on nerve entrapment. A drop in conduction velocity or amplitude across the

fibular head is a widely accepted, reliable indicator of peroneal nerve entrapment.¹⁹ We identified 17 subjects with a proven peroneal nerve entrapment of whom only five exhibited a Tinel sign. Nerve conduction criteria for tibial nerve entrapment and deep peroneal nerve entrapment at the dorsum of the ankle are less well defined. Most authors choose to classify these nerve compression syndromes, if at all, using clinical criteria.^{11,16,20} However, it can be difficult to distinguish symptoms of nerve compression from the dysesthesia or pain caused by DSPN. We attempted to circumvent this problem by using published quantitative abnormality thresholds. Sensory NCS are considered to be more sensitive for medial tarsal tunnel syndrome by some,¹¹ but a prolonged tibial nerve DML was increased in 74% of patients with a clinical diagnosis of tarsal tunnel syndrome.¹⁷ This is the parameter used in our study. Similarly, one study and several case reports described prolonged DML of the peroneal nerve in all patients with a compression of the deep peroneal nerve on the dorsum of the ankle ('anterior tarsal tunnel syndrome').^{11,14-16} We therefore believe that the DML prolongation used here is a valid approach to establish nerve entrapment, perhaps not for individual diagnostic use, but certainly for a group-wise statistical approach. In addition, we explored the potential diagnostic value of different cut-offs for the tibial and peroneal nerve DML. These analyses did not lead to better diagnostic values, with the exception of sensitivity of 56% using a $\geq 1SD$ criterion for the tibial nerve in patients with DSPN. This may be a spurious result, but even if it is not, a sensitivity of 56% is insufficient for clinical purposes. It may be thought that investigating nerves with well-defined compression syndromes, such as carpal tunnel syndrome and ulnar neuropathy at the elbow would be better suited to investigate the value of Tinel's sign for entrapment, but it has an established poor diagnostic value for these syndromes.^{2,21} We investigated leg nerves because the Tinel sign is currently used as a criterion for nerve surgery in the legs.

Furthermore, Tinel's sign could be elicited over the sural nerve below the lateral malleolus in a substantial number of our patients, comparable to the rate of occurrence at the other sites. This is additional evidence that it is not a reliable indicator of nerve compression, as the sural nerve is not associated with nerve entrapment.

Objective 3: the Tinel sum score as a test to distinguish between different groups of patients

Our data show that Tinel's sign is not associated with NCS parameters for nerve entrapment, whereas a previous study found that its presence at the tarsal tunnel was an excellent predictor of success of decompressive nerve surgery.⁵ A putative explanation for this discrepancy might be that Tinel's sign is an indicator for nerve pathology in general or perhaps for DSPN, rather than for compression neuropathies. The presence of Tinel's sign over the sural nerve in our patients can be interpreted to support this hypothesis, as the sural nerve is not associated with nerve entrapment. However, this hypothesis was rejected, as the areas under the curve in the ROC analyses were close to 0.5, indicating no value at all in discriminating between the various groups of patients.

STRENGTHS AND LIMITATIONS

The strengths of our study are the prospective design, systematic evaluation of Tinel's sign and the large number of patients. The study population consisted of a heterogeneous group of patients and is likely an accurate reflection of the type of patients generally seen in daily practice by many neurologists. However, the number of patients with both DSPN and focal nerve entrapment was relatively low.

A potential pitfall is that the actual force applied during tapping may have differed between technicians and sites in spite of the pre-defined procedure. The inter- and intra-examiner variability in the force applied when eliciting Tinel's sign is indeed considerable.²² However, the manual tapping procedure was performed prior to the NCS, i.e. blinded to the outcome of NCS. Furthermore, there was no difference in the mean sum scores of Tinel's sign between different technicians. Most importantly, manual examination of Tinel's sign is the current criterion used by many groups for surgical decompression in diabetic neuropathy. We followed the procedure as described in these publications on decompressive surgery as closely as possible.^{3,6-9}

CONCLUSION

The main objective of this study was to investigate the diagnostic yield of Tinel's sign for nerve entrapment in the legs. We found very low sensitivity and moderate specificity in diagnosing nerve entrapment in the legs defined by electrophysiological parameters. We conclude that Tinel's sign does not indicate nerve entrapment nor other nerve pathology in the legs. As stated, we did not investigate whether Tinel's sign accurately predicts a benefit of nerve surgery. We can only conclude that this predictive ability, as stated by Lee and Dellon,⁵ is not based on the presence of nerve entrapment or other nerve pathology. A putative mechanism may be that Tinel's sign somehow predicts the ability of nerves to recover after surgical decompression.

The current treatment strategy used by proponents of decompressive surgery consists of a sequence of steps that includes examination for the presence of Tinel's sign as an indicator of nerve entrapment. To rigorously determine whether surgery is of benefit to patients with diabetic neuropathy, we believe that each of these steps merits a thorough analysis, followed by a randomised blinded study on long-term outcome. As Tinel's sign is not useful as a first step, i.e. the diagnosis of nerve entrapment, the question arises whether the evidence for subsequent steps, i.e. decompressive surgery, is at present strong enough upon which to base treatment decisions, or whether such decisions should be postponed until confirmatory studies are available. We prefer the latter choice.

DISCLOSURE

M.R. Tannemaat is a member of the scientific advisory board of Araim Pharmaceuticals and holds stock options in this company. There are no conflicts of interest for any of the other authors.

There was no funding or financial support for the realisation of this study.

REFERENCES

1. Davis EN, Chung KC. The Tinel sign: a historical perspective. *Plastic and reconstructive surgery* 2004;114:494-9.
2. D'Arcy CA, McGee S. The rational clinical examination. Does this patient have carpal tunnel syndrome? *Jama* 2000;283:3110-7.
3. Dellon AL. Diabetic neuropathy: review of a surgical approach to restore sensation, relieve pain, and prevent ulceration and amputation. *Foot & ankle international* 2004;25:749-55.
4. Dellon AL, Muse VL, Scott ND, et al. A positive Tinel sign as predictor of pain relief or sensory recovery after decompression of chronic tibial nerve compression in patients with diabetic neuropathy. *Journal of reconstructive microsurgery* 2012;28:235-40.
5. Lee CH, Dellon AL. Prognostic ability of Tinel sign in determining outcome for decompression surgery in diabetic and nondiabetic neuropathy. *Annals of plastic surgery* 2004;53:523-7.
6. Aszmann OC, Kress KM, Dellon AL. Results of decompression of peripheral nerves in diabetics: a prospective, blinded study. *Plastic and reconstructive surgery* 2000;106:816-22.
7. Macare van Maurik JF, ter Horst B, van Hal M, Kon M, Peters EJ. Effect of surgical decompression of nerves in the lower extremity in patients with painful diabetic polyneuropathy on stability: a randomized controlled trial. *Clinical rehabilitation* 2015;29:994-1001.
8. Valdivia Valdivia JM, Weinand M, Maloney CT, Jr., Blount AL, Dellon AL. Surgical treatment of superimposed, lower extremity, peripheral nerve entrapments with diabetic and idiopathic neuropathy. *Annals of plastic surgery* 2013;70:675-9.
9. Wood WA, Wood MA. Decompression of peripheral nerves for diabetic neuropathy in the lower extremity. *The Journal of foot and ankle surgery : official publication of the American College of Foot and Ankle Surgeons* 2003;42:268-75.
10. Fabre T, Piton C, Andre D, Lasseur E, Durandeau A. Peroneal nerve entrapment. *The Journal of bone and joint surgery American volume* 1998;80:47-53.
11. Oh SJ. Neuropathies of the foot. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology* 2007;118:954-80.
12. P.A. vD. Richtlijn Polyneuropathie. Dutch Society for Neurology and Dutch Society for Clinical Neurophysiology. 2005.
13. van Dijk J.G SF. Consensus EMG bij compressienuropathieën - deel III compressienuropathie van de n. peroneus bij de fibulakop. Guideline of the Dutch Society for Clinical Neurophysiology. 2015.

14. Andresen BL, Wertsch JJ, Stewart WA. Anterior tarsal tunnel syndrome. *Archives of physical medicine and rehabilitation* 1992;73:1112-7.
15. Borges LF, Hallett M, Selkoe DJ, Welch K. The anterior tarsal tunnel syndrome. Report of two cases. *Journal of neurosurgery* 1981;54:89-92.
16. Logullo F, Ganino C, Lupidi F, Perozzi C, Di Bella P, Provinciali L. Anterior tarsal tunnel syndrome: a misunderstood and a misleading entrapment neuropathy. *Neurological sciences : official journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology* 2014;35:773-5.
17. Mondelli M, Morana P, Padua L. An electrophysiological severity scale in tarsal tunnel syndrome. *Acta neurologica Scandinavica* 2004;109:284-9.
18. Hajian-Tilaki K. Receiver Operating Characteristic (ROC) Curve Analysis for Medical Diagnostic Test Evaluation. *Caspian journal of internal medicine* 2013;4:627-35.
19. van Dijk JG, Reenalda H, Bollen EL, den Heijer JC. Nerve conduction velocity to different muscles in peroneal pressure neuropathy. *Electromyography and clinical neurophysiology* 1991;31:145-50.
20. Ahmad M, Tsang K, Mackenney PJ, Adedapo AO. Tarsal tunnel syndrome: A literature review. *Foot and ankle surgery : official journal of the European Society of Foot and Ankle Surgeons* 2012;18:149-52.
21. Beekman R, Schreuder AH, Rozeman CA, Koehler PJ, Uitdehaag BM. The diagnostic value of provocative clinical tests in ulnar neuropathy at the elbow is marginal. *Journal of neurology, neurosurgery, and psychiatry* 2009;80:1369-74.
22. Lifchez SD, Means KR, Jr., Dunn RE, Williams EH, Dellon AL. Intra- and inter-examiner variability in performing Tinel's test. *The Journal of hand surgery* 2010;35:212-6.

