

Slower Nerve Conduction Velocity in Individuals with Functional Ankle Instability

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Key words

- NCV
- FAI
- peroneal
- nerve

Abstract

▼ The purpose of this study is to quantify nerve conduction velocity differences in individuals with functional ankle instability compared to a “healthy” population. 38 participants ages 18–30 were recruited from a large university with approximately 43 000 students. 19 subjects (9 men and 10 women; age = 21.0 ± 1.4 years; height = 172.0 ± 9.3 cm; mass = 74.4 ± 12.4 kg) with symptoms of functional ankle instability were in the functional ankle instability group. 19 subjects (10 men, 9 women; age = 22.0 ± 2.6 years; height = 169.8 ± 9.1 cm; mass = 69.0 ± 14.8 kg) with “healthy” ankles were in the control group. Nerve conduction velocity was conducted using one trial at 2 different sites: posterior to the fibular head (fibular), and 10 cm superior/posterior of

the first site (popliteal). Nerve conduction velocity (m/sec) was assessed using a SierraWave II system (Cadwell Laboratories; Kennewick, WA). A MANCOVA was performed on the two dependent variables (fibular and popliteal). Covariates included surface temperature of the leg, body mass index, and age. The independent variable was group (functional ankle instability and control). The effect of group was significantly related to nerve conduction velocity at the fibular site ($F_{(1, 27)} = 16.49$, $p = 0.01$) and popliteal site ($F_{(1, 27)} = 4.51$, $p = 0.01$), with responses significantly faster for individuals in the control group than the functional ankle instability group. These results indicate that patients with functional ankle instability might have damage to the peroneal nerve which results in slower peroneal nerve conduction velocity.

Introduction

▼ The purpose of nerve conduction velocity (NCV) is to describe the status of the peripheral innervation [27] as well as to identify pathophysiological variations including polyneuropathy and neuropathies [35,59]. NCV was often evaluated to determine the presence of nerve compression, entrapment syndromes and other injuries in both the upper and lower limbs [10]. Specifically, when assessing the ankle, stabilizing reflexes from the superficial and deep peroneal nerves as well as static ligaments provide stability to the joint. However, after sustaining a lateral ankle sprain these reflexes may become damaged, which can disrupt normal stability and mobility of the joint [43,48].

Ankle sprains are one of the most common injuries with approximately 785 000 ankle sprains a year in the United States [60]. Nearly half of all ankle sprains (49.3%) occur during athletic activity with basketball (41.1%), football (9.3%), and soccer (7.9%) having the highest percentages

[60]. Approximately 85% of all ankle sprains occur to the lateral ankle ligaments as a result of inversion stress [16,60,61]. In 1965 functional ankle instability (FAI) was first described as a disability in which individuals sustain an ankle sprain and continue to report episodes of giving way for weeks, months or years after the initial injury [12]. This was later refined by Hertel [20], who proposed a paradigm in which chronic ankle instability was described as a condition which includes repeated bouts of lateral ankle instability. This recurrent instability could occur as a result of mechanical instability, functional instability or a combination thereof. Mechanical instability involves excessive inversion laxity of the rearfoot or excessive laxity of the talocrural joint using instrumented or manual stress testing [20], whereas, functional instability involves episodes of “giving way” of the ankle and sensations of ankle joint instability [20]. The Hertel model [20], which included 3 subgroups, was later expanded in 2011 by Hiller, Kilbreath, and Refshauge [21] to at least 7 subgroups. This new

accepted after revision
October 28, 2013

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DOI <http://dx.doi.org/10.1055/s-0033-1361183>
Published online:
February 27, 2014
Int J Sports Med 2014; 35:
731–736 © Georg Thieme
Verlag KG Stuttgart · New York
ISSN 0172-4622

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model clearly shows the array of recurrent instabilities that can occur following a lateral ankle sprain. In epidemiological studies some researchers have reported that 30–45% of people who sustain an ankle sprain have long-term residual symptoms [2,57]. Regardless of the model adopted, neuromuscular damage has repeatedly been speculated as a contributing factor for these residual symptoms. Muscle reaction time has been researched in individuals with FAI. The theory behind slower muscle reaction time in individuals with FAI is based on the concept that an ankle ligament injury often causes proprioceptive deficits in the ankle and its reflex, leading to an impaired postural control, and possibly causing ankle instability [15]. Numerous studies [5, 12, 26, 31, 37] have reported delayed response of the peroneal muscle during a sudden inversion perturbation. While this delayed reaction has been described as a potential cause of functional instability following a lateral ankle sprain, several studies have found conflicting results [3, 24, 25, 46]. NCV complements muscle reaction time studies. Peripheral nerves contain many nerve fibers of different diameters, degrees of myelination, and afferent or efferent connections [41]. NCV studies the fastest 20% of these fibers, and the purpose of the investigation is to document focal or continuous abnormalities in the length of mixed, motor, or sensory nerves [41]. A recent literature search resulted in no studies specifically evaluating the possible presence of nerve compression, traction injury to the nerve, or an entrapment syndrome assessed by NCV in participants with FAI. In the ankle, joint-stabilizing reflexes play an important role [48]. Following inversion trauma there is evidence that peroneal nerve palsy does occur [48]. However, this deficiency has primarily been documented in case presentation in participants with acute inversion injuries [43]. With this limited information it is easy to speculate that long-lasting neural disturbance and, consequently, an inhibited ability to regulate the temporal and spatial characteristics of muscle activation may lead to residual instability of the ankle joint.

With the absence of literature documenting peroneal palsy in individuals with FAI, the purpose of this study is to quantify NCV differences in individuals with FAI compared to a healthy population. Completing this study will provide us with a better understanding of persistent instability of the ankle as well as identify an objective way of quantifying the effect of FAI.

Materials and Methods



Participants

All subjects were over the age of 18 and had no recent (within 6 weeks) acute lower extremity injury. Participants were excluded if they had a history of ankle, hip or knee surgery, fracture of the leg or foot, or if a subject had any general medical conditions that could create sensory deficits. Subjects were assigned to either the FAI group or the control group based on their history of previous ankle injuries. FAI was determined by the use of 2 disease-specific questionnaires: the Cumberland Ankle Instability Tool (CAIT) and the Ankle Instability Instrument (AII).

The CAIT is a 9-item questionnaire intended to identify and grade ankle instability. Each question is assigned a point value. Point values can range from 0 to 5 depending on the question, and subjects separately score left and right limbs [22]. Individual limb scores of 24 or less are likely to have FAI [51, 53]. Founding authors of the CAIT reported excellent test-retest reliability (ICC=0.96) [22].

The AII was designed specifically for the detection of FAI. The AII is a 16-item questionnaire that consists of 9 Yes/No questions, 6 multiple-choice questions and 1 open-ended question [9]. Each question was designed to fit into one of 3 categories: Factor 1, severity of initial ankle sprain, Factor 2, history of ankle instability, and Factor 3, instability during activities of daily life [9]. Subjects who answered yes to 5 or more Yes/No questions were considered to be individuals with FAI. This criterion is based on previous articles published that have used the AII as their FAI inclusion criteria [8, 42, 52]. The AII was proven to have good test-retest reliability (ICC=0.70–0.98) [9]. Subjects had to meet the cut-off score on both questionnaires to be deemed as having FAI. To be assigned to the FAI group, only one of the participant's ankles had to be identified as functionally unstable. To be assigned to the control group, subjects had to have no history of injury to either ankle. 38 participants between the ages of 18 and 30 were recruited from a large university with approximately 43000 students. 19 subjects (9 men, and 10 women; means and standard deviations: age=21.0±1.4 years; height=172.0±9.3 cm; mass=74.4±12.4 kg) with symptoms of ankle instability were placed in the FAI group with a CAIT score of 16.5±4.2, and AII score of 6.3±0.9. All individuals in the FAI group had previously sprained their ankle and experienced repeated episodes of giving way. Subjects reported the last time they experienced the sensation of giving way: <1 month (15 people) or 1–6 months (4 people); experienced instability while walking on a flat surface: yes (10 people), no (9 people); experienced instability walking on an uneven surface: yes (19 people), no (0 people); experienced instability during recreation or sports: yes (18 people), no (1 person). 19 subjects (10 men, 9 women; means and standard deviations: age=22.0±2.6 years; height=169.8 cm±9.1 cm; mass=69.0±14.8 kg) with “healthy” ankles were placed in the control group with a CAIT score of 30±0.0, and AII score of 0±0.0. Individuals in the control group indicated never experiencing an ankle sprain or symptoms associated with instability. The University's Institutional Review Board Ethics Committee approved the study, and all subjects read and signed the informed consent prior to participating in the study. The study meets the ethical standards of the International Journal of Sports Medicine [19].

Procedures

To complete NCV testing, subjects were asked to lie on their non-FAI side on a treatment table. For individuals in the control group the evaluated limb was matched side-for-side to the FAI group. NCV was then assessed using a SierraWave II system (Cadwell Laboratories; Kennewick, WA). The procedure used an electroneuromyograph equipped with an oscilloscope, amplifier and stimulator. All equipment was calibrated using the procedures provided by the manufacturer. The test was performed in the same room for all participants, and room temperature was monitored during each testing session. Each participant's temperature was also monitored using a temperature probe. Next, the participant was positioned so that the nerve adopted its natural resting length. This was achieved by having the participant lie down with hip and knees flexed to approximately 90° and placing a pillow between the participant's legs and under his or her head. Amplifier settings were 2K gain (uV/Div), 10k hicut (Hz), 10 locut (Hz) and 10.0 sweep speed (ms/Div) [6]. Surface electrodes were used to provide information about the whole muscle stimulated. Surface electrodes provide data for the time taken for the fastest axons to conduct an impulse to the

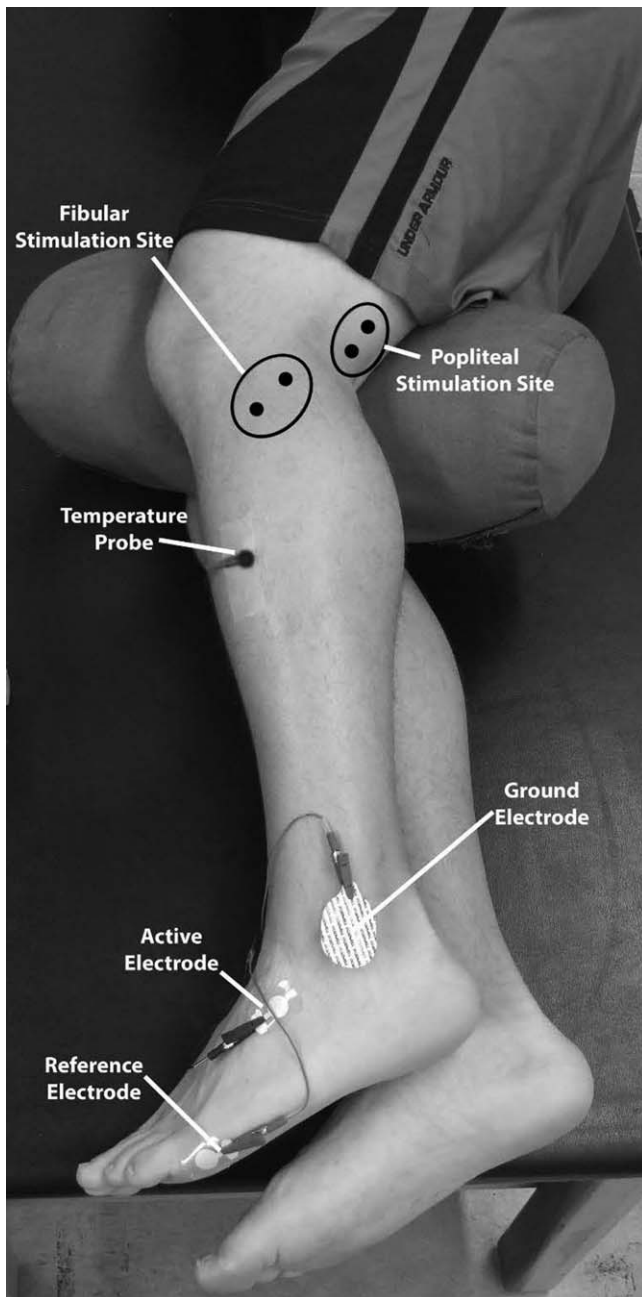


Fig. 1 Nerve conduction stimulation sites (fibular and popliteal).

muscle [6,41]. This is termed latency and is produced in milliseconds [41]. The ground electrode was placed on the lateral malleolus, the reference electrode on the subject's 5th toe and the active electrode 6 cm distal and anterior to the ground electrode on the dorsum of the foot [6]. The stimulator was set to produce a direct current (square wave) stimulus of one millisecond at each test site [41]. When the nerve was stimulated a stimulus artifact (compound action potential) appeared at the moment of stimulation on the computer screen [41]. 2 sites along the superficial peroneal nerve were stimulated (► Fig. 1). The first site was posterior to the fibular head (fibular, latency 1), and a dot was marked on the skin with a permanent marker to calculate velocity. The procedure was repeated in order to attain a second latency (popliteal) which was located 10 cm superior/

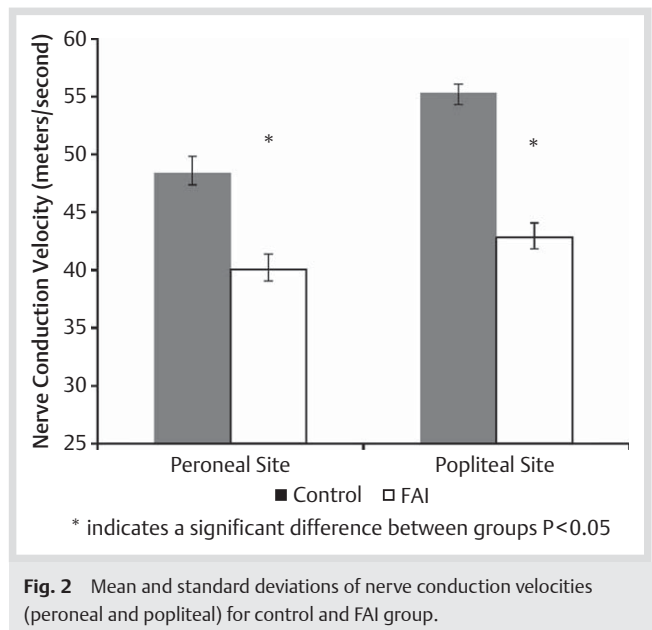


Fig. 2 Mean and standard deviations of nerve conduction velocities (peroneal and popliteal) for control and FAI group.

posterior to latency 1 [41]. Both responses were stored by pressing the store key on the Sierra Wave unit. For all of the stimulations the cathode (-) was oriented closest to the recording electrodes [6]. Lastly, distance was measured (meters) to calculate velocity [41]. Distance for the first and second stimulation sites (fibular and popliteal) was measured from the active electrode to the respective cathode permanent marker spot [41]. Each site was analyzed separately for statistical analysis.

Statistical analysis

A multivariate analysis of covariance was performed on 2 dependent variables (fibular and popliteal NCV (meters/second)). Adjustment was made for 3 covariates: surface temperature of the leg, BMI and age. The independent variable was group (FAI and control).

Results

Results of evaluation of assumptions of normality, homogeneity of variance-covariance matrices, linearity and multicollinearity were satisfactory. With the use of Wilks' criterion, the combined dependent variables were significantly related to the combined covariates, approximate $F_{(10,27)}=3.58$, $p=0.01$, $\eta_p^2=0.37$, $1-\beta=0.79$, and to group $F_{(4,24)}=9.77$, $p=0.01$, $\eta_p^2=0.62$, $1-\beta=0.99$. After adjusting for differences on the covariates, none of the 3 covariates made a significant contribution to the dependent variables. Univariate analyses for the effect of group are significantly related to NCV at each stimulus site. At the fibular site individuals with FAI had slower NCV compared to controls with a mean difference of 8.32 m/sec (95% CI 7.2–9.4 m/sec, $F_{(1,27)}=16.49$, $p=0.01$, $\eta_p^2=0.38$, $1-\beta=0.98$). Similarly, at the popliteal site individuals with FAI had slower NCV compared to controls with a mean difference of 12.48 m/sec (95% CI 11.1–13.9 m/sec, $F_{(1,27)}=4.51$, $p=0.01$, $\eta_p^2=0.14$, $1-\beta=0.54$). Overall, the responses were significantly slower for individuals with FAI than individuals with no FAI (► Fig. 2).

Discussion

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The overall finding of this study was that individuals with FAI had significantly slower NCV of the superficial peroneal nerve compared to individuals without FAI. Specifically, this was seen at both the fibular site and popliteal site of the peroneal nerve. It is documented in the literature that the mechanism of stabilizing joints is achieved through an intact nerve [12–14,30]. Several authors have applied this theory to the ankle and have proposed that stability of the ankle is dependent on an intact reflex mechanism [12–14,30]. These authors suggested that FAI is predominantly caused by injured joint receptors [12–14,30]. However, the possibility of slower nerve conduction velocities as an explanation for FAI has not been the focus of previous researchers. This may be an important omission, since slower velocities may contribute to the deficits seen in individuals with FAI. For instance, slower nerve conduction velocities can explain the prolonged peroneal muscle reaction time reported by many researchers in individuals with FAI [7, 11, 23, 24, 29, 30, 49, 56].

In the present study motor NCV was collected to evaluate the presence of a peroneal nerve lesion. We found that the motor NCV of the superficial peroneal nerve was slower in individuals with FAI. While normal ankle inversion does not damage the peroneal nerve because of the inherent properties of the nerve [39,44,54,55], we speculate that the slower nerve conduction velocities in individuals with FAI may be due to a mild to moderate traction lesion that occurred during the initial inversion injury or following continued repetitive bouts of giving way. Stretching a peripheral nerve beyond its physiological limits will lead to acute and long-term deficiencies [38,40,54]. Permanent functional deficits such as motor and sensory impairment as well as pain have been shown after strain of more than 12% of the *in situ* strain of the rabbit tibial nerve [33]. However, the question remains whether repetitive bouts of inversion stress is able to induce slower motor NCV found in the present study.

First, the length of the nerve and the amount of displacement needed to create damage should be evaluated [44]. For example, if the total length of the peroneal nerve is assumed to be 100 cm, a 3-cm displacement of the nerve at the lateral side of the foot would result in an elongation of 3%. However, the peroneal nerve has an interesting location just distal to the head of the fibular [32], which creates the elongation of the peroneal nerve to almost 20 centimeters with an elongation of 15% [32]. Nerve injury can occur with as little as 6% elongation, as reported by Liu, Benda and Lewey [36], thus making it possible that this relatively small nerve excursion may be sufficient to produce nerve damage in the peroneal nerve. Second, the total amount of movement in the foot can contribute to nerve damage. For example, when the entire weight of an individual's body is exerted on an inverted ankle, a larger inversion angle can be expected [54,55]. This will consequently create a larger nerve displacement and may cause further traction on the peroneal nerve. In addition, the velocity of the traction forces must be considered. During daily activity nerve dysfunction is unlikely to occur because the traction force is exerted gradually and slowly [54,55]. However, during inversion trauma and repetitive ankle sprains an abrupt, fast traction will be exerted on the nerve which can cause damage [45]. The last factor simultaneously takes into consideration traction and compressive forces. When the foot is moved medially the fibular head becomes a pulley to the peroneal nerve. The fibular head exerts a transverse force resulting in compression of the nerve [45].

In the work completed by Nitz, Dobner and Kersey [47] it was reported that peroneal nerve injury was associated with grade II and III lateral ankle sprains. The values reported by Nitz, Dobner and Kersey for their control group are similar to our values for the control group. However, our FAI group had much slower NCV than either of their groups with Grade II or III ankle sprains. Other work completed by Kleinrensink testing individuals who had suffered a recent lateral ankle sprain revealed that 4–8 days following an ankle sprain, peroneal nerve motor NCV was reduced when compared to their contralateral leg and the control group [28]. However, after 18 days and 36 days post-initial trauma nerve conduction velocities had returned to normal [28]. These individuals had experienced an initial inversion ankle sprain with no residual symptoms associated with FAI. In this current study, we evaluated individuals with FAI. Specifically, our subjects had experienced at least one ankle sprain and continued to have bouts of “giving way” or episodes of repetitive inversion. These repetitive bouts of inversion stress could have caused the additional trauma to the peroneal nerve creating the difference between these 2 studies. It appears that this recurring trauma in individuals with FAI creates a slower motor nerve conduction velocity of the peroneal nerve which might even perpetuate the instability.

Covariates in NCV Research

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In numerous NCV studies [1,4,17,18,34,50] there has been much discussion of the use of covariates in human peripheral nerve function. A systematic investigation of covariates of nerve conduction measures was performed on data collected by the United States Centers for Disease Control. NCV was obtained for the median motor, median sensory, ulnar sensory, peroneal motor and sural sensory nerves for 4462 subjects [34]. Covariates collected were skin temperature, height, body mass index, age, race, place of any military service, smoking status, alcohol consumption, examiner, and income. This investigation concluded that the major covariates were skin temperature, height, and different examiner [34]. Based on this research skin temperature, BMI and age were used as covariates. BMI was used in this investigation to examine the effects of height and weight as there were major covariates in previous research [34]. The addition of skin temperature was also to account for skin temperature being a major covariate in previous research [34]. Even though age was not a major covariate in the previous research, it was important to account for the large variances in age in our study. One examiner was used for this study, making it unnecessary to evaluate the effect of different examiners. After adjusting for differences on the covariates, none of the 3 covariates made a significant contribution to the dependent variables.

Limitations

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One limitation is that only surface electrodes were used to evaluate the activation pattern of the lower leg musculature. More accurate results might have been produced by needle EMG evaluation. However, using surface electrodes to estimate NCV is the accepted method in the literature, [58] and invasive needles can be strongly influenced by electrode placement because of the different fiber orientation (i.e., bipennate structure), which furthermore introduces movement artifacts. In addition, NCV was

only performed on the superficial peroneal nerve. If there was damage to a different nerve or contributing nerve root, the effect thereof may not have been apparent in the testing. Measurement error is a limitation of the measure. In an attempt to control this, only one investigator was used to evaluate NCV. Participant tolerance of the one millisecond electrical pulse as well as fear of pain may have affected measurement accuracy.

Future Research

There are many considerations for future research. One possible area for future research is to investigate the effect of a 6-week intervention program to improve NCV in individuals of FAI. A longer study (6 weeks of treatment) is important, because in clinical practice a change is not always apparent after a single treatment. In addition, NCV should be performed on individuals across the spectrum of FAI. This would include evaluating individuals who are deemed “copers”. Traditionally, “copers” are defined as individuals who sustain an initial lateral ankle sprain but exhibit no symptoms of FAI such as giving way. This would provide additional information to the FAI literature.

Conclusion

The peroneal nerve is an important part of the ankle joint, providing a stabilizing reflex and therefore functional stability. The results of this study support the hypothesis that an injury to the peroneal nerve might be present in individuals with FAI. Following an initial ankle sprain, damage to the peroneal nerve might occur. This injury may lead to FAI, and future giving way episodes may further damage the peroneal nerve, causing repetitive damage and preventing the nerve from ever healing. This damage could jeopardize the functional stability of the ankle joint and put patients at greater risk of future injuries.

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