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Increased forefoot loading is associated with an increased plantar flexion moment

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ABSTRACT

The aim of this study was to identify the cascade of effects leading from alterations in force generation around the ankle joint to increased plantar pressures under the forefoot. Gait analysis including plantar pressure measurement was performed at an individually preferred and a standardized, imposed gait velocity in diabetic subjects with polyneuropathy ($n = 94$), without polyneuropathy ($n = 39$) and healthy elderly ($n = 19$). The plantar flexion moment at 40% of the stance phase was negatively correlated with the displacement rate of center of pressure ($r = -.749$, $p < .001$ at the imposed, and $r = -.693$, $p < .001$ at the preferred gait velocity). Displacement rate of center of pressure was strongly correlated with forefoot loading ($r = -.837$, $p < .001$ at the imposed, and $r = -.731$, $p < .001$ at the preferred gait velocity). People with a relatively high plantar flexion moment at 40% of the stance phase, have a faster forward transfer of center of pressure and consequently higher loading of the forefoot. This indicates that interventions aimed at increasing the control of the roll-off of the foot may contribute to a better plantar pressure distribution.

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1. Introduction

Plantar foot ulceration is an invalidating co-morbidity of diabetes. Increased plantar pressure during walking is an important risk factor for (recurrent) plantar ulceration (Boulton et al., 1983) and it has been shown that the plantar pressure pattern is altered in patients with diabetes and polyneuropathy (DPN) (Allet et al., 2008; Caselli, Pham, Giurini, Armstrong, & Veves, 2002). Several authors showed that in this population especially the forefoot is prone to higher pressures and consequently at risk for ulceration (Caselli et al., 2002; Mueller, Zou, Bohnert, Tuttle, & Sinacore, 2008).

Diabetes has been associated with several structural and functional alterations in the foot that can result in higher plantar pressures, such as anatomical deformities (claw toes, charcot neuro-osteoarthropathy), limited joint mobility and skin changes (callus formation) (Ahroni, Boyko, & Forsberg, 1999; Boyko et al., 1999; Bus, 2008; Morag & Cavanagh, 1999; Morag et al., 1997; Mueller et al., 2003). Moreover, it has been suggested that changes in gait biomechanics can lead to higher loading of the foot in DPN patients (Andersen, Poulsen, Mogensen, & Jakobsen, 1996; Savelberg, Schaper, Willems, de Lange, & Meijer, 2009). Gait in DPN is characterized by longer stance phases, shorter steps and lower gait velocity (Kwon, Minor, Maluf, & Mueller, 2003; Mueller, Minor, Sahrmann, Schaaf, & Strube, 1994; Sacco & Amadio, 2000; Savelberg et al., 2009). In this context van Deursen, Sanchez, Ulbrecht, and Cavanagh (1998) argued that deficits of the sensory nerve system could lead to alteration of gait in this population. The sensory nerve system provides information on the interaction with the ground and on the tension of the muscles. If the feedback from this system is reduced, it can lead to insecurity of gait or limitation in motor control resulting in abnormal loading of the foot (Richardson, Ching, & Hurvitz, 1992; Sacco & Amadio, 2000; van Deursen et al., 1998).

In addition to its effect on the sensory system, DPN can also affect the motor nervous system. Abboud, Rowley, and Newton (2000) found a delayed activation of the tibial anterior muscle , while others found prolonged activity of this muscle (Savelberg et al., 2010) or premature activation of the calf muscles in DPN (Kwon et al., 2003). Besides possible changes in muscle activation patterns, DPN affects the force generating ability. Several authors found reduced lower extremity muscle strength in people with diabetes (Andersen et al., 1996; IJzerman et al., 2011; van Schie, Vermigli, Carrington, & Boulton, 2004). In addition, Andreassen, Jakobsen, and Andersen (2006) observed weakness of the ankle plantar and dorsiflexors in particular in DPN patients with severe neuropathy. Several authors found a combination of lower extremity muscle dysfunctioning and increased plantar pressures. They suggested that a decrease in motor control due to muscle weakness or limited feedback might be partly responsible for the observed increased plantar pressures in patients with DPN (Abboud et al., 2000; Kwon et al., 2003). In an earlier study we found higher plantar flexion moments from loading response to mid stance and an increase in plantar loading in this population (Savelberg et al., 2009). We suggested that reduced strength of the lower extremity muscles (caused by DPN) resulted in an impaired ability to brake the forward momentum of the body during the first part of the stance phase. This would have led to a faster roll-off of the foot, with a faster forward transfer of the center of pressure (COP) and consequently causing higher plantar forefoot loading. In the current study we tested the hypothesis that via the aforementioned cascade of relations, adaptations in force generation around the ankle joint result in a forwardly displaced plantar pressure pattern during gait. This hypothesis was tested in people with and without diabetes, and with and without polyneuropathy.

2. Methods

2.1. Participants

Three groups of subjects participated, with a total number of 152 (Table 1). A group of 19 healthy elderly subjects (HE, mean age of 68 (*SD* = 5) years) was recruited by means of advertisement or participation in previous research. Subjects with diabetes type 2 were recruited from the diabetes clinics