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ScienceDirect
Clinical Biomechanics 23 (2008) 584–592
CLINICAL BIOMECHANICS
www.elsevier.com/locate/clinbiomech

The effect of diabetic neuropathy and previous foot ulceration in EMG and ground reaction forces during gait

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Received 9 August 2007; accepted 23 November 2007

Abstract

Background. We aimed at investigating the influence of diabetic neuropathy and previous history of plantar ulcers on electromyography (EMG) of the thigh and calf and on vertical ground reaction forces during gait.

Methods. This study involved 45 adults divided into three groups: a control group ($n = 16$), diabetic neuropathic group ($n = 19$) and diabetic neuropathic group with previous history of plantar ulceration ($n = 10$). EMG of the right vastus lateralis, lateral gastrocnemius and tibialis anterior were studied during the stance phase. The peaks and time of peak occurrence were determined and a co-activation index between tibialis anterior and lateral gastrocnemius. In order to represent the effect of the changes in EMG, the first and second peaks and the minimum value of the vertical ground reaction force were also determined. Inter-group comparisons of the electromyographical and ground reaction forces variables were made using three MANCOVA (peaks and times of EMG and peaks of force) and one ANCOVA (co-activation index).

Findings. The ulcerated group presented a delayed in the time of the lateral gastrocnemius and vastus lateralis peak occurrence in comparison to control's. The lateral gastrocnemius delay may be related to the lower second vertical peak in diabetic subjects. However, the delay of the vastus lateralis did not cause any significant change on the first vertical peak.

Interpretations. The vastus lateralis and lateral gastrocnemius delay demonstrate that ulcerated diabetic neuropathic patients have a motor deficit that could compromise their ability to walk, which was partially confirmed by changes on ground reaction forces during the push-off phase.

Keywords: Biomechanics; EMG; Diabetic neuropathies; Gait

1. Introduction

Diabetic neuropathy is the most common chronic complication associated with Diabetes Mellitus, affecting 20–50% of diabetic patients 10 years after their diagnosis (Cavanagh et al., 1993). Diabetic neuropathy leads to a progressive loss of somatosensory sensitivity, proprioception and distal muscle function (Pirart, 1979; Pickup and Williams, 1981; Yavuzer et al., 2006), especially in the lower limbs, which may cause an alteration of the motor control during gait and during static posture (Mueller et al., 1994; Shaw et al., 1998; Katoulis et al., 1997; Beek et al., 1998; Abboud et al., 2000; Sacco and Amadio, 2000, 2003; Yavuzer et al., 2006). These alterations may increase the risk of falling in diabetic neuropathic patients (Cavanagh et al., 1992; Simmons and Richardson, 2001).

The sensory and motor diabetic neuropathy modifies the amount and quality of sensory information necessary for motor control. Consequently, there is a higher instability during gait and static posture (Richardson et al., 1992), that previously has been considered to be due to muscular weakness (Courtemanche et al., 1996). Gait in humans is

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0268-0033/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.clinbiomech.2007.11.015

considered the result of harmonic interplay neural and muscular actions coordinated with skeletal function (Katoulis et al., 1997), and changes in this harmonic relationship will cause important alterations in the locomotor pattern. These alterations are considered the result of a loss of sensitivity and may result in biomechanical alterations of gait, such as plantar pressures changes, usually with higher pressures on the forefoot (Caselli et al., 2002; Gefen, 2003; Perry et al., 2002), differences in kinematic patterns (Fernando et al., 1991; Mueller et al., 1994; Katoulis et al., 1997; Shaw et al., 1998; Sauseng and Kastenbauer, 1999; Sacco and Amadio, 2000; Kwon et al., 2003; Menz et al., 2004; Petrofsky et al., 2005; Yavuzer et al., 2006), differences in kinetic patterns with modified ground reaction forces (Katoulis et al., 1997; Shaw et al., 1998; Sacco and Amadio, 2000), and altered muscle activity (Abboud et al., 2000; Sacco and Amadio, 2003; Kwon et al., 2003).

As diabetic neuropathy develops, the somatosensory inputs diminish, and the motor outputs become progressively more impaired, which may accentuate the changes in the locomotor pattern of diabetic neuropathic subjects. The biomechanical changes during the gait of diabetics, in conjunction with diabetic autonomic impairments, may lead to foot ulcerations. In this way, we may conclude that the presence of foot ulceration may be an indicator of the worsening of diabetic neuropathy. Neuropathic ulceration is the most prevalent type of long-term chronic injury for diabetic subjects (Piaggesi, 2004).

So far, the main findings from the study of electrical muscle activity in diabetic neuropathic subjects, are that the electrical muscle activity present a delayed activation of the gastrocnemius, soleus, peroneus brevis and longus muscles, and most importantly, of the tibialis anterior muscle, during gait, which may be associated with an earlier forefoot contact with the ground (Abboud et al., 2000; Sacco and Amadio, 2003). Still, a delayed activation of the vastus lateralis is mentioned during the initial contact of gait that may be indicative of the presence of a deficiency in the shock attenuation mechanisms (Sacco and Amadio, 2003). Furthermore, it has been found that there is a premature activation of the soleus and medial gastrocnemius, and a prolonged tibialis anterior activity, leading to a co-contraction of these muscles during mid-stance in a possible attempt to improve foot stability. This also causes an earlier contact of the forefoot, and a decrease of shock absorption at the time of heel strike, which may lead to an altered plantar pressure distribution, and may also be related to plantar ulceration on the forefoot (Kwon et al., 2003).

Changes in muscle activity could lead to other alterations during gait, besides the plantar pressure distribution. It is already described that diabetic neuropathic subjects present modified ground reaction forces (GRF) during gait (Shaw et al., 1998; Sacco and Amadio, 2000; Santos and Barela, 2002). These alterations could be related to the slower walking velocity they adapt (Katoulis et al., 1997). What is still unknown is if the diabetic neuropathy itself is in fact leading to significant GRF changes, in addition to a reduction in gait velocity.

Investigations of muscle activity alterations during gait in diabetic neuropathic subjects are still very scarce. The results in literature are controversial; and it is not yet clear how peripheral diabetic neuropathy affects muscle activity and the kinetic responses during gait. Whether muscle activity patterns change along with the evolution of the disease and what consequences altered muscle activity has on the kinetics of diabetes gait function in regard to ground reaction forces, remains unclear. Previous studies have not distinguished between the degrees of neuropathy in their experimental groups; therefore, it has not been possible to identify differences in gait patterns between the early and advanced stages of the disease. The hypothesis of the present study was that late stages of diabetic neuropathy, which is represented here by at least one occurrence of plantar ulceration in the patient's clinical evolution, would lead to greater biomechanical alterations during gait.

So, the purpose of this study was to investigate and compare the electromyographical activity of the thigh and calf muscles during gait among non-diabetic subjects and diabetic neuropathic patients in two stages of the disease: those with and those without previous experience of ulcers in their clinical history. This study also attempted to verify if the changes in EMG do, in fact, cause any alteration in GRF during gait. Within these descriptions of gait characteristics, we speculate about the influence of diabetic peripheral neuropathy and its progression in gait as well as the possible dynamic mechanisms developed to compensate for sensory and motor deficits.

2. Methods

2.1. Subjects

This prospective study involved 45 volunteer adult males and females divided into three groups: the control group (CG) ($n = 16$), a diabetic neuropathic group (DG) ($n = 19$) and a diabetic neuropathic group with previous history of plantar ulceration in the last two years (UDG) ($n = 10$). Ethics approval was obtained from the local institution (Protocol No. 1268/05).

All neuropathic subjects (DG and UDG) were diagnosed by physicians. The following inclusion criteria were adopted for patients: at least 5 years post-onset of Type 2 diabetes; presentation of at least two plantar areas with tactile insensitivity to the 10-g monofilament (Frykberg et al., 1998; Armstrong and Lavery, 1998; Perry et al., 2002), and a minimum score of 6 on the Michigan Neuropathy Screening Instrument-questionnaire (MNSI-q) (Feldman et al., 1994). The MNSI-q is a validated instrument for screening the symptoms related to diabetic neuropathy. In addition, the UDG subjects had at least one previous plantar ulcer in the last two years. The exclusion criteria adopted for all experimental groups included: age over 65 years, partial or total amputation, Charcot arthropathy