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Do patients with diabetic neuropathy use a higher proportion of their maximum strength when walking?

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ABSTRACT

Diabetic patients have an altered gait strategy during walking and are known to be at high risk of falling, especially when diabetic peripheral neuropathy is present. This study investigated alterations to lower limb joint torques during walking and related these torques to maximum strength in an attempt to elucidate why diabetic patients are more likely to fall. 20 diabetic patients with moderate/severe peripheral neuropathy (DPN), 33 diabetic patients without peripheral neuropathy (DM), and 27 non-diabetic controls (Ctrl) underwent gait analysis using a motion analysis system and force plates to measure kinetic parameters. Lower limb peak joint torques and joint work done (energy expenditure) were calculated during walking. The ratio of peak joint torques and individual maximum joint strengths (measured on a dynamometer) was then calculated for 59 of the 80 participants to yield the 'operating strength' for those participants. During walking DM and DPN patients showed significantly reduced peak torques at the ankle and knee. Maximum joint strengths at the knee were significantly less in both DM and DPN groups than Ctrls, and for the DPN group at the ankle. Operating strengths were significantly higher at the ankle in the DPN group compared to the Ctrls. These findings show that diabetic patients walk with reduced lower limb joint torques; however due to a decrement in their maximum ability at the ankle and knee, their operating strengths are higher. This allows less reserve strength if responding to a perturbation in balance, potentially increasing their risk of falling.

1. Introduction

Diabetes is a major health concern in many developed countries, in the UK affecting almost 5% of the entire population (Diabetes UK, 2010), and the number is growing. One of the major complications of diabetes is diabetic peripheral neuropathy (DPN), which may be present in 30–40% of patients (Boulton et al., 2004). Diabetic peripheral neuropathy affects sensory and motor nerves, predominantly in the feet and lower limbs. Both sensory and motor aspects of DPN have the potential to impact upon gait. Sensory neuropathy impairs perception of foot-ground contact, therefore influencing sensory feedback and balance control. Motor neuropathy impairs the force producing capabilities of muscles via impaired motor nerve function, and therefore impacts on the torques generated around lower limb joints. However, even before the development of DPN, the effects of non-enzymatic glycation can act on the contractile machinery to impair muscle function (Kindig et al., 1998; Phielix and Mensink, 2008). As a result of these diabetes-related complications there is a marked impact upon gait (Allet et al., 2008; Wrobel and Najafi, 2010), contributing to the fact that diabetes has been shown to increase the risk of falls during daily life (Tilling et al., 2006), with DPN being a particular risk factor (Bonnet and Ray, 2011; Richardson and Hurvitz, 1995). Such falls can often result in injuries, and for the general population of the UK between 2001 and 2003, 16% of accidents occurring at home or during leisure time and resulting in hospital treatment were due to someone falling during walking (DTI and Industry, 2003). Therefore, given diabetes causes a greater risk of falling there is considerable importance in understanding the possible underlying mechanisms, so that intervention strategies can be aimed at preventing future falls.

The majority of previous studies investigating the biomechanics of walking in diabetic patients have focused primarily upon reporting temporal–spatial characteristics or joint angular alterations. The main observations include a shorter stride length, longer stance times, smaller hip joint range of motion, slower walking speed and reduced cadence in diabetic patients compared to controls (Allet et al., 2009;

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Ko et al., 2011; Menz et al., 2004; Mueller et al., 1994; Petrofsky et al., 2005). These gait characteristics, especially a slower gait velocity and shorter stride length are common in other populations with instability such as the elderly (Taylor et al., 2013), and they demonstrate alterations to key movement characteristics. Many studies have investigated the above parameters in patients with DPN (Allet et al., 2009; DeMott et al., 2007; Ko et al., 2011); however some studies have included an additional group of diabetic patients without DPN, showing similar but less pronounced differences than the group with neuropathy (Menz et al., 2004; Mueller et al., 1994). This suggests that DPN may not be the sole factor leading to alterations in gait in diabetic patients.

Although previous studies described above have identified gait pattern alterations in diabetic patients, the parameters reported reveal little of what is actually 'driving' such changes. As all movements start from muscle contraction, investigating joint kinetics allows us to examine how muscles are acting on the major lower limb joints and uncover the mechanisms behind the previously reported pattern alterations. It has been shown that during walking diabetic patients develop lower peak joint torques at all three lower limb joints (Ko et al., 2011; Mueller et al., 1994; Sawacha et al., 2009), indicating an altered muscular gait strategy. Although joint torque developed during gait provides an indication of the 'strength' requirements of the task, we also need to understand how this relates to the physical capabilities of the individual. Maximum muscular strength, which has previously been identified as a key factor for falls in the elderly (Pijnappels et al., 2008a, 2008b), is reduced in diabetic patients with DPN compared to healthy controls (Andersen et al., 2004; Andreassen et al., 2006). This may also mean that diabetic patients are operating closer to the limits of their physical capabilities during walking, a finding already observed in healthy elderly people during stair ascent (Reeves et al., 2009). Operating closer to maximum ability while walking would leave a smaller 'reserve capacity' to meet the increased joint torque demands required to maintain balance and prevent a fall in response to any perturbation, or unexpected challenge to balance therefore this may provide further insight as to why this population is more likely to fall. The present study investigated peak joint torques generated at the lower limb joints during walking and how these torque levels relate to the physical capabilities of the individual. The aim of the study was to investigate the influence of diabetes and DPN on lower limb kinetics relative to maximal capabilities during walking, and consider the implications this has for stability.

2. Methods

2.1. Participants

After receiving ethical approval for the study from all relevant bodies, a total of 80 participants were recruited, who gave their written informed consent to participate. Participants were allocated into one of three groups based upon defined criteria: patients with diabetes and moderate-severe peripheral neuropathy (DPN, $n=20$), patients with diabetes but no neuropathy (DM, $n=33$) and healthy controls without diabetes or peripheral neuropathy (Ctrl, $N=27$).

2.2. Clinical assessment

All participants were assessed to confirm they met the inclusion criteria. Major exclusion criteria included: inability to walk independently of assistance, the presence of amputation, open foot ulcers, history of cerebral injury, neurological disorder (other than diabetic aetiology), musculoskeletal injury and visual acuity poorer than 6/18. The presence of peripheral neuropathy was assessed using the modified Neuropathy Disability Score (mNDS) and the vibration perception threshold (VPT). The mNDS is a semi-quantitative composite score derived from assessment of temperature perception, pain, vibration, and the Achilles tendon reflex (Boulton, 2005) and the VPT is a quantitative assessment performed using a neurosthesiometer (Bailey Instruments Ltd., Manchester, UK; (Boulton et al., 2004)). Patients were considered to have moderate-severe peripheral neuropathy and allocated to the DPN group if they demonstrated a mNDS ≥ 6, or a VPT ≥ 25. Diabetic patients demonstrating a mNDS < 6 and a VPT < 25 were allocated to the DM group without neuropathy. Blood glucose levels were assessed from the Ctrl group to confirm the absence of diabetes and the above neuropathy tests conducted to confirm the absence of neuropathy in the Ctrl group resulting from any aetiology.

2.3. Gait analysis

Kinematics were collected at 100 Hz using a full-body modified Helen-Hayes marker set, and a 10-camera Vicon motion capture system (Vicon, Oxford UK) positioned around an 8-m walkway. Kinetics were simultaneously collected at 1000 Hz from 3 Kistler force platforms embedded into the middle of the walkway. Where possible markers were placed directly onto the skin, to eliminate artefacts resulting from loose clothing all participants wore tight-fitting shorts and tops. All participants wore specialist diabetic shoes (MedSurg, Darco, Raisting, Germany) with a neutral foot-bed, ensuring the diabetes patients with DPN walked with safe, appropriate footwear whilst minimising the effect of footwear and standardising across all participants. Participants were instructed to walk the length of the walkway at their self-selected speed. Participant's starting position was altered by the experimenters to ensure a 'clean' (i.e., no overlap outside the force platform) foot-strike on one or two of the force platforms per walk without alteration to their natural gait. Walking trials were repeated until at least three 'clean' foot contacts with the force platforms were made per limb.

2.4. Gait variables and joint kinetics

Joint torques and temporal–spatial parameters (gait velocity, stance time) were then calculated using Visual 3D software (C-motion Inc., MD, USA), using the process of inverse dynamics to calculate joint torques and powers. Peak joint torques during stance were calculated for each participant from left and right legs for each of the 3 trials. Mean peak torques (ankle, knee, and hip) were calculated taking into account data from both legs, across all three trials, and were subsequently normalised to body mass. This approach (mean across both legs and three trials; kinetics normalised to body mass) was used for all variables presented. Power curves during stance were calculated to assess concentric and eccentric work done; positive (concentric) and negative (eccentric) periods of power during the stance period were isolated to separately define concentric and eccentric work done which was defined as the power-time integral (area under the curve) during these periods. Periods of eccentric (muscle lengthening) and concentric (muscle shortening) power were isolated to calculate eccentric and concentric work done respectively, and were then subsequently normalised to body mass.

2.5. Maximum joint torque reference value assessment

Individual maximal torque reference values were assessed for the ankle plantar flexors and knee extensors using an isokinetic dynamometer (Cybex Norm, USA). These muscle groups were selected because they are the predominant muscle groups contributing to propulsion and weight-acceptance during walking. These maximum effort contractions were performed for the purpose of relating the maximum muscle capabilities to that of the joint torque demands during walking for calculation of what we term from here on as 'operating strength' (see below). Measurements were taken with participants in a seated position on the dynamometer for knee extension, and prone for ankle plantar flexion. Maximal torque was recorded at four different joint angular velocities ($60^\circ\,s^{-1}$, $120^\circ\,s^{-1}$, $180^\circ\,s^{-1}$ and $240^\circ\,s^{-1}$) for both concentric (shortening) and eccentric (lengthening) contractions, in order to cover a range of joint angular velocities that are experienced during normal gait. Individual maximum eccentric and concentric joint torques were also calculated across the speeds in order to compare the variations in maximal abilities between groups.

2.6. Operating strength calculations

Operating strength was defined as the ratio of the peak joint torque developed during gait, to the maximum joint torque reference value produced at the same joint, under matched conditions (i.e., muscle action and joint angular velocity; values presented as percentages). Essentially, this measure (operating strength) expresses the peak demands of walking relative to the participant's maximum muscular capabilities. Operating strength was calculated for 59 of the original 80 participants due to non-completion of assessment for the maximum reference values; this occurred either when a participant failed to return for this data collection session, or were physically unable to perform the tests. The group sizes for the operating strengths are therefore Ctrl: $n=18$, DM: $n=27$, DPN: $n=14$. The starting point for the operating strengths calculation was to identify the peak knee and ankle joint torques occurring during the gait cycle. Peak torque values were identified and normalised to the appropriate maximum joint torque reference value, matched for muscle action (eccentric/concentric) and joint angular velocity