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Altered leverage around the ankle in people with diabetes: A natural strategy to modify the muscular contribution during walking?

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ABSTRACT

Diabetes patients display gait alterations compared to controls including a higher metabolic cost of walking. This study aimed to investigate whether differences in external moment arm (ExtMA) and effective mechanical advantage (EMA) at the ankle in diabetes patients could partly explain the increased cost of walking compared to controls. Thirty one non-diabetic controls (Ctrl); 22 diabetes patients without peripheral neuropathy (DM) and 14 patients with moderate/severe diabetic peripheral neuropathy (DPN) underwent gait analysis using a motion analysis system and force plates. The internal Achilles tendon moment arm length was determined using magnetic resonance imaging during weight-bearing and ExtMA was calculated using gait analysis. A greater value (P < 0.01) for the EMA at the ankle was found in the DPN (0.488) and DM (0.46) groups compared to Ctrl (0.448). The increased EMA was mainly caused by a smaller ExtMA in the DPN (9.63 cm; P < 0.01) and DM (10.31 cm) groups compared to Ctrl (10.42 cm). These findings indicate that the ankle plantarflexor muscles would need to generate lower forces to overcome the external resistance during walking compared to controls. Our findings do not explain the previously observed higher metabolic cost of walking in the DM and DPN groups, but uncover a new mechanism through which patients with diabetes and particularly those with DPN reduce the joint moment at the ankle during walking: by applying the ground reaction force more proximally on the foot, or at an angle directed more towards the ankle, thereby increasing the EMA and reducing the ankle joint moment.

1. Introduction

Diabetes presents a global health challenge with an international prevalence between 2% and 24% [1,2]. Diabetic peripheral neuropathy (DPN) is a major complication occurring in 30–50% of all patients, causing dysfunction of peripheral nerves [3], with implications for not only sensory but also motor nerves, causing movement dysfunction [4,5]. People with diabetes walk more slowly, take shorter strides and generate lower knee and ankle joint moments during walking [6,7]. We have recently shown a higher metabolic cost of walking (CoW) across a range of matched walking speeds in patients with diabetes and particularly in those with DPN compared to controls [12]. This higher CoW in people with diabetes may underpin their lower physical activity levels, contributing towards a negative spiral where there is a greater perception of difficulty for walking, which causes less engagement in physical activity, leading to poorer metabolic control and worsening of the diabetic condition. To allow interventions to break this negative cycle, it is therefore important to understand the factors that contribute to increasing the CoW in diabetes.

One potential factor contributing to the increased CoW is a lower effective mechanical advantage (EMA), caused by a greater external moment arm (ExtMA) of the resultant ground reaction force (GRF) around the ankle. The EMA around the ankle is given by the ratio of the internal moment arm of the plantarflexors (IntMA) to the ExtMA, with lower values reflecting a relatively greater contribution from the plantarflexor muscles towards the joint moment required to overcome the external resistance [8,9]. Many diabetes patients have some level of foot deformity such as high arch, or toe deformities [11], which may
result in applying force to the ground more distal on the foot, thereby decreasing the EMA due to the increased ExtMA around the ankle. One consequence of this leverage alteration is that the plantarflexor muscles would need to produce more active force to generate the ankle moment required for propulsion. The EMA around the ankle in diabetes patients could also be affected by altered use of the lower limb and foot caused by sensory deficits and plantarflexor muscle weakness. A relative increase in the contribution from ankle plantarflexor muscles during walking may contribute to the increased CoW in diabetes patients.

The aim of this study was to establish whether there are differences in the ExtMA and EMA at the ankle in patients with diabetes and DPN compared to controls at a range of matched walking speeds, as a potential mechanism contributing to the increased CoW recently observed in diabetes patients [12]. We hypothesized that the ExtMA will be higher and the EMA will be lower in diabetes patients compared to controls.

2. Methods

2.1. Participants

After receiving ethical approval for the study from all relevant bodies, a total of 67 participants were recruited, who gave 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 (n = 14, 12 men, 2 women), patients with diabetes but no neuropathy (n = 22, 12 men, 10 women) and healthy controls without diabetes or peripheral neuropathy (n = 31, 19 men, 12 women). Major exclusion criteria included: disorders of the vestibular system, musculoskeletal injury, recent surgery affecting gait, foot or lower limb amputation and open foot ulcer.

2.2. Clinical assessment of peripheral neuropathy

A clinical evaluation was undertaken to quantify neuropathy in diabetes patients. Peripheral neuropathy was assessed by using the modified Neuropathy Disability Score (mNDS) and the vibration perception threshold (VPT) [13]. A random blood glucose test was performed in 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

Kinematic data were collected at 100 Hz using a full-body modified Plug-In-Gait marker set [5] with 54 markers and a 10-camera Vicon motion capture system (Vicon, Oxford, UK) positioned around the 10-m walkway. Ground reaction forces were measured at 1000 Hz synchronously with motion capturing using three force platforms (Kistler, Zurich, Switzerland) embedded into the walkway. Where possible markers were placed directly onto the skin; to minimise movement artefacts resulting from loose clothing. All participants wore tight-fitting shorts and t-shirts. Participants were instructed to walk the length of the walkway at different walking speeds performed in a specific order (0.6, 0.8, 1.0, 1.2, 1.4 and 1.6 m/s). Walking speed was controlled by measuring the velocity of a marker attached to the sacrum after each trial from the motion analysis data and providing immediate verbal feedback for participants as to whether they needed to walk more quickly or slower on the next trial to achieve the required speed. Although this approach involved a systematic, rather than randomised order of walking speeds, it was deemed the most optimal approach to achieve the required speeds while retaining a natural gait, compared to alternatives such as a metronome that restricts cadence. Furthermore, given that walking is not an unusual or unaccustomed task, there is little reason to expect any learning or order effects. Walking trials were repeated to obtain three ‘clean’ foot contacts with the force platforms per limb, per speed condition. All participants wore the same standardised shoes (MedSurg, Darco, Raisting, Germany).

2.4. MRI scanning and analysis

Magnetic resonance imaging (MRI) was used to quantify the IntMA length as the Achilles tendon moment arm length at the ankle, as previously described [14]. IntMA was defined as the perpendicular distance from the centre of rotation on the talus to the Achilles tendon line of action (Fig. 1a) [15,16]. The IntMA lengths were determined with participants standing upright (i.e., full weight-bearing) in a 0.25T MRI scanner (E-Scan, Esato Biomedica, Genoa, Italy). Weight-bearing scans were acquired across the predominant range of ankle joint angles (10 deg dorsiflexion, neutral position, 10 deg plantarflexion) experienced during walking, to relate these measurements as closely as possible to the conditions of walking. The ankle joint instant centre of rotation was located following the graphical approach described by Reuleaux [17] for ankle angle rotations from −10 to 10 deg. Instant centre of rotation was determined by measuring the rotation of the talus, which was considered to represent the whole rotating foot, relative to the tibia. IntMA was measured on the neutral ankle scan. All images were analysed using a custom-script written in MATLAB software.

2.5. Measurement of the ExtMA at the ankle during walking and foot length

Foot length was measured in the standing position as the distance between the end of the big toe and the heel. ExtMA length around the
ankle during walking was defined as the perpendicular distance between the resultant GRF vector in the sagittal plane and the ankle joint centre of rotation. The ankle joint centre of rotation was defined from markers positioned on the lateral and medial malleoli. ExtMA length was quantified throughout the stance phase on every motion analysis frame from integration of the kinematic data with the GRF data.

2.6. Calculation of the EMA

EMA around the ankle joint (Fig. 1b) was calculated as:

\[ EMA = \frac{\text{IntMA}}{\text{ExtMA}} \]

2.7. Gait biomechanical analysis

Gait variables were calculated using Visual 3D software (C-motion Inc., MD, USA): ExtMA, joint moments, GRFs and ankle, knee and hip joint angles. Joint moments and GRFs were normalised to body mass. Data for the ExtMA, joint moments and GRFs were collected during the stance phase, while ankle, knee and hip joint ranges of motion (RoM) were analysed throughout the gait cycle. Means across both legs and three trials were used for all variables presented.

2.8. Statistics

A one-way analysis of variance (ANOVA) was performed for all variables to assess between group differences. If the ANOVA was significant, a Fisher’s least significant difference (LSD) post-hoc test was used to test for differences between the diabetes groups (DM and DPN) and the control group. An analysis of covariance (ANCOVA) was performed for the external moment arm at peak ankle joint moment using foot length as the covariate. All values presented are means and standard deviation. Significance was set at \( p < 0.05 \).

3. Results

3.1. Participant characteristics

Significant differences existed between the groups in age, body mass and BMI (\( p < 0.01 \)), with the DPN group being older and heavier with a greater BMI compared to controls (DPN: age 66 ± 14 yr, body mass 91.5 ± 18 kg, BMI 31 ± 4 kg/m²; DM: age 51 ± 9 yr, body mass 80.5 ± 12 kg, BMI 28 ± 4 kg/m²; Ctrl: age 56 ± 10 yr, body mass 76 ± 10 kg, BMI 26 ± 3 kg/m²).

3.2. Diabetic peripheral neuropathy

As expected, patients with DPN displayed significantly higher mNDS and VPT than the Ctrl and the DM groups (DPN: mNDS 7 ± 2 Score/10, VPT 27.4 ± 9.1 V; DM: mNDS 2 ± 1 Score/10, VPT 8.2 ± 3.4 V; Ctrl: mNDS 1 ± 1 Score/10, VPT 6.1 ± 3.4 V). There were no differences (\( P > 0.05 \)) in the mNDS or VPT between the Ctrl and the DM groups, underlining that this diabetes group had no neuropathy.

3.3. Temporal–spatial gait parameters

The DPN group displayed significantly longer single limb stance times and shorter step lengths in all given speeds compared to Ctrl group (Table 1).

3.4. ExtMA length at peak ankle joint moment during walking & gait parameters

ExtMA length at peak ankle joint moment was significantly smaller...
(P < 0.01) in the DPN group compared to the Ctrl group at walking speeds of 0.6; 1.0 and 1.4 m/s and for the mean across all speeds (Table 1). Significant differences (P < 0.01) were also observed in ExtMA length between the DM and Ctrl groups at a walking speed of 1.4 m/s. Foot length was not significantly different between the three groups (DPN: 25.81 ± 2.4 cm; DM: 24.77 ± 2.1 cm; Ctrl: 25.43 ± 1.7 cm).

3.5. IntMA and EMA during walking

There were no differences (P > 0.05) in the IntMA length in the DPN group and the DM groups compared to the Ctrl group (DPN: 4.98 ± 0.21 cm; DM: 4.73 ± 0.30 cm; Ctrl: 4.72 ± 0.27 cm). EMA at the ankle was significantly (P < 0.01) higher in the DPN group compared to the Ctrl group at walking speeds of 0.6; 1.0; 1.2; 1.4, 1.6 m/s and for the mean across all walking speeds (Table 1). EMA was also significantly (P < 0.01) higher in the DM group compared to the Ctrl group at walking speeds of 0.6; 0.8; 1.4 m/s and for the mean across all walking speeds.

3.6. Ground reaction forces during walking

Ground reaction forces were significantly higher (P < 0.01) in the DPN group compared to the Ctrl at walking speeds of 0.6; 0.8; 1.0; 1.4 and 1.6 m/s and for the mean across all walking speeds (Table 1). Significantly higher (P < 0.01) GRF values were also found in the DM group compared to the Ctrl group at the walking speed of 1.6 m/s and for the mean across all speeds.

3.7. Peak ankle joint moments & lower limb kinematics during walking

Peak ankle plantarflexion joint moments were significantly lower (P < 0.01) in the DPN compared to the Ctrl group for all walking speeds including the mean across all speeds (Table 1), with the exception of values at 1.2 m/s. Peak ankle plantarflexion joint moments were also significantly lower (P < 0.01) in the DM compared to the Ctrl group at walking speeds of 1.2, 1.4 and 1.6 m/s and for the mean across all speeds.

A significantly (P < 0.01) smaller ankle, knee and hip joint RoM was observed in the DPN group compared to the Ctrl group across all walking speeds (Table 2). Joint RoM was also significantly (P < 0.01) reduced in the DM group compared to the Ctrl group at the ankle (1.0 and 1.2 m/s), knee (0.8; 1.0; 1.2 m/s and for the mean values) and hip (all speeds except 0.6 m/s). Between group differences (range of motion) for the DPN and Ctrl groups were in the range 11–15% for the ankle, 4–6% for the knee and 9–11% for the hip across the range of speeds examined. Smaller percentage differences were found when the Ctrl group was compared to the DM group across the range of speeds (1–5% for the ankle, 1–3% for the knee and 4–8% for hip).

Table 2

<table>
<thead>
<tr>
<th>Speed (m/s)</th>
<th>RoM Ankle (deg)</th>
<th>RoM Knee (deg)</th>
<th>RoM Hip (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ctrl</td>
<td>DM</td>
<td>DPN</td>
</tr>
<tr>
<td>0.6</td>
<td>23.1</td>
<td>22.7 (7.8)</td>
<td>20.8 (9.5)**</td>
</tr>
<tr>
<td>0.8</td>
<td>23.7</td>
<td>23.5 (8.5)</td>
<td>21.1 (7.8)**</td>
</tr>
<tr>
<td>1.0</td>
<td>25.6</td>
<td>24.4 (8.3)**</td>
<td>22.5 (9.4)**</td>
</tr>
<tr>
<td>1.2</td>
<td>26.4</td>
<td>25.2 (8.7)*</td>
<td>23.7 (8.4)**</td>
</tr>
<tr>
<td>1.4</td>
<td>26.8</td>
<td>26.0 (9.1)</td>
<td>23.4 (9.2)**</td>
</tr>
<tr>
<td>1.6</td>
<td>27.3</td>
<td>26.9 (8.6)</td>
<td>24.3 (9.0)**</td>
</tr>
<tr>
<td>Mean</td>
<td>25.4</td>
<td>24.7 (8.5)</td>
<td>22.4 (8.8)**</td>
</tr>
</tbody>
</table>

Healthy controls (Ctrl, n = 31), diabetic patients with no neuropathy (DM, n = 22) and diabetic patients with moderate/severe neuropathy (DPN, n = 14). Significant differences from the Ctrl group are denoted by * (P < 0.05) or ** (P < 0.01). Values are means (standard deviations). RoM – range of motion.

4. Discussion

We have recently shown that patients with diabetes, and especially those with DPN, have a higher CoW compared to controls [12]. In the present study we investigated whether differences between diabetes
patients and healthy controls in the ExtMA and EMA around the ankle joint could be a potential mechanism contributing to the higher metabolic cost of walking. In contrast with our hypothesis, we established that patients with diabetes, and especially those with DPN have a smaller ExtMA and a higher EMA around the ankle joint compared to controls (Fig. 2; Table 1).

The smaller ExtMA at the ankle in patients with diabetes and particularly those with DPN was evident across all walking speeds (Table 1) and means that either the resultant GRF was applied closer to the ankle joint centre, or the angle of application was more towards the ankle, thereby reducing the ankle joint moment (Fig. 1c). This is consistent with the finding of reduced ankle plantarflexor joint moment in patients with DPN compared to controls across all matched walking speeds (Table 1).

It has previously been shown that diabetes patients reduce joint moments by taking shorter strides with less flexed joints [10,18]. Our findings reveal a new mechanism through which people with diabetes and particularly those with DPN reduce the joint moment at the ankle during walking. No differences in foot length existed to explain the smaller ExtMA found in patients with DPN and further, this parameter (foot length) was also entered as a covariate in the statistical analysis of variance. Our finding of a smaller ExtMA and greater EMA around the ankle does not appear to explain an increased CoW, as the plantarflexor muscles would need to produce not higher, but smaller contractile forces to rotate the foot and propel the body forward (Fig. 3). However, the consequent reduction in the force applied to the Achilles tendon would result in smaller ROM, reduced tendon elongation and therefore a reduced amount of elastic strain energy stored, which could impact upon the CoW, but this requires further investigation.

The joint kinematics from the present study provides insight as to how patients with DPN might have been able to execute this natural strategy of reducing the ExtMA around the ankle and thereby minimising the ankle joint moment. The ankle joint RoM over the gait cycle was reduced in patients with DPN compared to controls as a result of a reduced peak dorsiflexion angle. This reduced dorsiflexion suggests that patients with DPN were not able to allow the tibia to rotate over the foot to the same extent as controls during the mid-stance phase, further evidenced by the reduced knee flexion (Table 2), thereby applying force to the ground more proximally on the foot and reducing the ExtMA around the ankle as a result. We should also consider that walking with this restricted joint range of motion may not be a natural strategy of choice, but rather a walking strategy of necessity due to specific limitations, which in turn may bring about the alterations in the ExtMA discussed.

Whilst the total hip joint RoM during walking was reduced in diabetes patients and especially those with DPN compared to controls (Table 2), patients with diabetes and to the greatest extent those with DPN flexed the hip more than controls. This kinematic strategy fits very well with the ‘hip strategy’ previously reported in other studies [10,19], whereby diabetes patients ‘drag’ the leg forwards into the swing phase from the hip, rather than ‘propelling’ the leg off from the ground using the ankle plantarflexors. Whilst greater knee and hip RoM occurs with increasing walking speeds in all groups, a consistently smaller RoM at the knee and hip in the DPN group (Table 2) underlies the shorter step length reported in the present study and is comparable with a number of previous studies conducted in diabetes patients [11,19,20,10,12].

Whilst previous studies have consistently reported lower ankle joint moments in diabetes patients during walking, this has typically been at the self-selected speed, which is consistently lower in diabetes patients as they seek to minimise the demands of the task. Here we show that when walking speed is matched, joint moments are consistently lower in the DM and particularly in the DPN group compared to controls (Table 1). The reduction in the ExtMA and increase in the EMA at the ankle in the diabetes groups is relatively independent of walking speed, whereas ankle joint moments and the vertical GRF increase with increasing walking speed (Table 1). It is also noteworthy that although
the peak ankle joint moments were significantly lower in the diabetes groups compared to controls, the vertical GRF was significantly higher especially in the DPN group (Table 1). This higher GRF seems to underline the importance of the strategy in diabetes patients for reducing the ExtMA around the ankle to lower the joint moment substantially below that of controls. What remains unclear is whether this represents a natural strategy to lower the demands, or whether they have no other possibility to walk differently because of musculoskeletal limitations.

In terms of study limitations, the mean body mass was significantly different between groups (being higher in the DPN group), however, this should not affect the ankle ExtMA, EMA, or the joint moments since these were normalised for body mass. Furthermore, the higher body mass of patients with DPN is a well-known characteristic of this clinical population described by previous studies [21,22]. Although only a mean of 10 years difference, patients in the DPN group were significantly older than controls (66–56 years, respectively), which might be a confounding factor for some of the variables examined, but unlikely to affect the main variables of interest: the ankle ExtMA and EMA. Whilst a number of diabetes patients will have had foot deformity to varying degrees, we did not objectively assess this, which should be acknowledged as a limitation. However, within our hypothesis we did not anticipate discriminating between foot deformities, but rather testing the concept that any diabetic foot deformity would serve to shift the centre of pressure under the foot. This aspect could be more thoroughly investigated by future work using pedobarography.

Conflicts of interest statement

None of the authors had any financial or personal conflict of interest with regard to this study.

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Author agreement

All authors were fully involved in the study and preparation of the manuscript and that the material within has not been and will not be submitted for publication elsewhere.

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