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Gait characteristics of people with diabetes-related peripheral neuropathy, with and without a history of ulceration

Anita Raspovic *

Department of Podiatry and Lower Extremity Gait Studies Program, School of Allied Health, Faculty of Health Sciences, La Trobe University, Bundoora, Victoria 3086, Australia

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ABSTRACT

Biomechanical alterations in diabetes are believed to contribute to plantar neuropathic ulceration. This exploratory study documents clinical measures of flexibility and strength, alongside three-dimensional biomechanical gait data of the lower limb, in 10 patients with a history of neuropathic ulceration (DNU; $n = 10$). Comparative data is presented from age and gender matched groups with; diabetes peripheral neuropathy and no ulcer history (DWN; $n = 10$), diabetes and no peripheral neuropathy (DNN; $n = 10$) and a non-diabetes reference group (NOND; $n = 10$). Biomechanical data were collected at a comfortable walking speed with a Vicon motion analysis system. Clinical measures showed a non-significant trend toward decreased static range of motion at the ankle and first metatarsophalangeal joints, with worsening neuropathy status. Of the diabetes groups, knee and ankle strength was significantly lower in those with an ulcer history ($p = 0.01$–0.03), with the exception of knee extension. In the DNU group, walking speed was on average 0.17 ms slower compared to NOND ($p = 0.04$). The DNU group demonstrated a lower range of motion than NOND at the: hips (frontal plane, by 25%: $p = 0.03$); hips and knees (transverse plane, 31%: $p = 0.01$ and 32%: $p < 0.01$); ankles (sagittal plane, 22%: $p < 0.01$) and first metatarsophalangeal joints (sagittal plane, 32%: $p = 0.01$), with less foot rotation (24%: $p = 0.04$). Kinetic alterations in DNU included lower: ankle maximum power (21%: $p = 0.03$) and vertical ground reaction force 2nd peak (6%: $p < 0.01$). The study findings identified gait alterations in people with clinically severe peripheral neuropathy and related plantar foot ulcer history. Further research is needed to explore potential casual pathways.

1. Introduction

Biomechanical alterations of the lower limb in diabetes are believed to be important in the etiology of plantar foot ulceration, although the mechanisms for this are not well understood [1,2]. Diabetes-related foot ulceration is a major medical, social and economic problem, with an estimated 15% of people with diabetes likely to develop a foot ulcer during their lifetime [3]. An estimated 85% of all diabetes-related amputations are preceded by a foot ulcer, therefore great emphasis is placed on prevention and targeted management of this problem [4].

It is well known that chronic hyperglycaemia causes a heterogeneous mix of neuro-motor and connective tissue changes affecting the lower limb. These are expressed clinically in a number of ways, including loss of sensation, reduced strength, altered motor control, reduced static joint range of motion and thickening of soft tissues [5–7]. It has been hypothesized that these elements interact to alter biomechanical function and create damaging patterns of plantar loading although the pathways for this are unclear. Important questions remain regarding the exact cause and nature of diabetes-related biomechanical changes and how they might be implicated in the development of foot ulceration.

Two recent reviews of gait characteristics in diabetes concur that the adoption of conservative strategies are typically employed by those affected by peripheral neuropathy, including slower walking speeds, wider base of gait, prolonged double support time and greater step variability [1,2]. Elevated plantar pressures, a moderate risk factor for ulceration, occur in diabetic neuropathy although the exact reasons behind this have been somewhat elusive [8,9]. Research into kinematic gait changes in this clinical population has produced inconsistent results. One study reported no difference in gait ankle motion in participants with diabetic peripheral neuropathy [10] and another concurred when the confounding issue of walking speed was controlled for [11]. In contrast, reduced ankle motion during gait has been shown by others when consistency of walking speed was maintained [12].

Reports from gait and ulceration studies have also been inconsistent, with findings ranging from reduced dynamic motion,

* Tel.: +61 3 9479 5835.
E-mail address: a.raspovic@latrobe.edu.au

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peak power and peak moment at the ankle [13], lower knee joint angle, vertical ground reaction force and anteroposterior force [14] and reduced gait dorsiflexion at the first metatarsophalangeal joint [15], to reports of no changes in gait motion at the ankle [15]. Research investigating gait biomechanics and ulceration is limited, however, in that; only a small number of studies nominate a group with an ulcer history [13–15], older motion analysis systems have been superseded by more recent technological advances [13,14] and only a few studies, with relatively small and potentially underpowered sample sizes, have evaluated the entire lower limb [13,14].

With recent developments in motion analysis technology, including improved multi-segment foot models, studies are emerging that support links between peripheral neuropathy and reductions in gait excursions of the ankle complex and sub-segments of the foot [16,17]. Given the importance of biomechanical alterations in the development of diabetic neuropathic foot ulceration, and the inconsistency of the available literature, further research is required. This current study is exploratory in nature, aiming to document comprehensive lower limb three-dimensional biomechanical data in patients with a history of neuropathic ulceration. Data on groups of participants with and without neuropathy, and without diabetes is reported for comparative observation.

2. Methods

2.1. Participants

Forty individuals (31 males and 9 females) were recruited via flyers placed around a university health sciences clinic and through advertising in a local diabetes group newsletter. Participants were recruited into one of four groups: diabetes with peripheral neuropathy and a history of related plantar foot ulceration (DNU, $n = 10$); diabetes with peripheral neuropathy and no foot ulcer history (DWN, $n = 10$); diabetes with no history of peripheral neuropathy or ulceration (DNN, $n = 10$); and a non-diabetes reference group (NOND, $n = 10$). Diabetes status and foot ulcer history were self-reported. Peripheral neuropathy was confirmed if the Vibration Perception Threshold (VPT) was >25 V [18] in combination with a positive Neuropathy Deficit Score (NDS) [19]. Exclusion criteria, based on self-reported medical history, were the presence of any orthopedic, visual, neurological or other disturbance that might affect gait, including current pain, injury, active ulceration or amputation other than of a toe.

Participants were age-matched to within 5 years. Participant characteristics according to study group are shown in Table 1. Ethical approval was granted by the Institutional Ethics Committee and written informed consent was obtained from all participants.

2.2. Clinical measures

Static range of dorsiflexion available at the ankle joint complex (i.e. the talocrural and talocalcaneal joints) was measured with the knee flexed, using the technique described by Bennell et al. [20]. Good reliability has been reported for this test (ICC 0.95) [20]. The same test was also performed with the knee extended to assess the effect of posterior lower leg musculature on ankle range of motion. In the frontal plane, range of inversion and eversion at the ankle joint complex was assessed using a previously published goniometric technique which is used clinically [21]. Passive range of dorsiflexion at the first metatarsophalangeal joint was also measured using goniometry, with a reliability coefficient of 0.88 [22].

Maximal isometric muscle strength of knee flexors, knee extensors and ankle dorsiflexors was assessed due to the importance of these muscles during walking [23]. The test protocol is described in full elsewhere with data indicating good validity and reliability [24]. Ankle plantarflexor strength was assessed as a modification to the protocol, with the participant's heel on the ground, the loop of the strain gauge under the forefoot and the measuring device secured vertically. With the knee stabilized and the ankle in a neutral position, the participant was asked to push their foot down against the forefoot strap with maximal force for 2–3 s. Conducted on the dominant limb, the highest of three attempts was recorded.

2.3. Gait analysis equipment and protocol

Three-dimensional motion analysis was conducted using a Vicon 512 Motion Analysis System (Oxford Metrics Ltd., Oxford, England) with six cameras operating at a sampling frequency of 100 Hz. A force plate (Kistler, Switzerland) embedded into a 10 m walkway operating at a sampling frequency of 400 Hz was used to collect kinetic data. The same investigator placed retroflective markers onto each participant according to the Vicon Plug-in Gait (PIG) model [25]. Additional markers were placed medial to the navicular bones, lateral to the fifth metatarsophalangeal joints and medial to the interphalangeal joints of the halluces, to allow estimation of first metatarsophalangeal gait dorsiflexion thorough a simple model created in Bodybuilder (Oxford Metrics Ltd., Oxford, England).

All calibration steps were conducted according to the manufacturer's guidelines. A knee alignment device was used during static subject calibration to determine the location of the knee joint axis. After a period of familiarization with the instrumentation and environment, data were collected with participants walking a 10 m distance at a self-selected comfortable speed. Six successful walking trials were conducted for each participant; three where the left foot and three where the right foot, landed entirely within the force plate with clear vision of all retroreflective markers throughout gait. Starting position was adjusted to facilitate force plate targeting without the participant's knowledge and they were not made aware of the presence of the plate to avoid gait modification.

2.4. Data analysis

Six walking trials were selected for each participant. Each trial underwent reconstruction, markers were labeled and gait events identified using the Vicon software. The Plug-in Gait (PIG) model was applied to each trial using the Vicon software, then the first metatarsophalangeal joint model was run using Bodybuilder. Kinematic data were calculated using Euler angles [25]. Data for pelvic motion and foot progression were calculated relative to laboratory axes, all other kinematic variables were calculated relative to a proximal segment.

Trials were averaged in Polygon and exported into excel where data were extracted on key variables of interest:

i. Temporospatial data – cadence, walking speed and stride length.
ii. Kinematic data – stance phase range of motion: at the pelvis, hip and knee in three planes; at the ankle and first metatarsophalangeal joint in the sagittal plane; and for foot rotation and foot progression. Initial contact angle of the hip, knee and ankles was also recorded. Foot rotation represents the foot position relative to the lower leg in the horizontal plane and foot progression measures foot position relative to the laboratory axis in the frontal plane.
iii. Kinetic data – maximum power and maximum moment at the hip, knee and ankle and the magnitude of the vertical ground reaction force peaks.

2.5. Statistical analysis

Initial data screening was performed to ensure completeness of the data set and accuracy of data entry. The data were explored for normality of distribution prior to

Table 1
Demographic and diabetes characteristics by study group.

Variable	Diabetes neuropathic ulceration group (DNU)	Diabetes with neuropathy group (DWN)	Diabetes no neuropathy group (DNN)	Non-diabetes reference group (NOND)
Age (years)*	64 (7.3)	64 (6.4)	59 (10.5)	63 (8.4)
Gender (male:female)	8:2	9:1	8:2	6:4
Height (cm)*	173 (6.4)	175 (10.9)	172 (6.9)	168 (5.0)
Weight (kg)**	88 (14.7)	99 (18.6)	93 (21.1)	77 (11.2)
Diabetes duration (years)	16 (10.7)	12.9 (9.4)	8.3 (7.7)	N/A
VPT (V)	49 (3.7)	43 (9.8)	20 (8.8)	20 (9.6)
NDS	18.8 (4.0)	14.3 (6.5)	0.5 (1.6)	0 (0)

Values are mean ± standard deviation (SD) except for gender.
VPT, Vibration Perception Threshold; NDS, Neuropathy Deficit Score; 0 = no neuropathy, 1–5 = mild neuropathy, 6–16 = moderate neuropathy, 17–28 = severe neuropathy [19].
* No statistically significant difference between groups; $p = 0.55$ for age and $p = 0.18$ for height.
** Statistically significant difference between DWN and NOND; $p = 0.03$.