INCREASED FORE FOOT LOADING IN DIABETIC POLYNEUROPATHY PATIENTS AS A RESULT OF RELATIVELY HIGHER ANKLE JOINT MOMENTS AT 40% OF STANCE PHASE.

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INTRODUCTION
Elevated plantar pressures are an important risk factor for the development of plantar ulceration for people with diabetic polyneuropathy (DPN). Redistribution towards higher forefoot plantar pressures especially seems to be associated with development of ulcers [1]. DPN can lead to diminished muscle force of the lower legs [2]. If the anterior tibial muscle is affected, it may lead to a diminished ability to control the roll off of the foot, resulting in a quicker forward translation of the centre of pressure (COP). If this leads to a quicker loading of the fore foot, it may explain the higher plantar pressures in that area. A previous study by Savelberg et al. suggested that changes in joint moments could be the cause of this inability to brake the forward momentum just after heel strike. In DPN, ankle joint moments were relatively higher at 40% of the stance phase [3]. Therefore, it is hypothesised that, in people with DPN, increased fore foot loading is a result of a quicker forward transfer of the COP, caused by a higher plantar flexion moment during the first part of the stance phase. This higher plantar flexion moment could be a result of a diminished ability to generate a dorsal flexion moment.

METHODS
DPN patients (n=14, age 68±7.1), diabetes patients without DPN (DC) (n=7, age 62±6.9) and healthy controls (HC) (n=16, age 68±6.0) volunteered for gait analysis of the right leg, including 2D kinematics, ground reaction forces and plantar pressures. Walking speed was standardised at 1.2 m/s. Step length was monitored using accelerometry. Level of DPN was determined using standardised clinical neurological examination. Foot loading was measured as forefoot to rear foot (F/R) pressure time integral (PTI) ratio. COP displacement was measured as a percentage of total contact time the COP needed to travel towards the metatarsal area of the foot. Ankle joint moment was calculated using inverse dynamics. A ratio (A40/Amax) of ankle joint moment was calculated at 40% of the stance phase (A40) and the maximum (Amax).

RESULTS AND DISCUSSION
The A40/Amax ratio was higher (p=0.092) for DC (0.51±0.14) and significantly higher (p=0.028) for DPN (0.54±0.13), compared to HC (0.45±0.13). This supported previous findings, indicating a diminished ability to reduce the forward momentum just after heel strike in DPN, resulting in relatively higher plantar flexion moments at 40% of the stance phase. [3]. However, possibly due to the fact that the subjects of the DPN group had only light to moderate neuropathic symptoms, F/R PTI ratios and COP displacement did not differ between groups. Regression analysis showed that COP displacement could explain 78% of the variance of the F/R PTI ratio, showing that COP displacement is a relative good indicator of PTI ratios (Fig 1a). In addition, the A40/Amax ratio explains 46% of the variance of COP displacement, indicating that A40/Amax ratios are linked to COP displacement and thereby to F/R PTI ratios (Fig. 1b). Evidence is provided for the link between higher fore foot loading (expressed as F/R PTI ratio) and higher plantar flexion moments at 40% of the stance phase (expressed as A40/Amax ratio). If a diminished ability to generate a dorsal flexion moment is the cause of higher plantar flexion moments and thereby of the higher fore foot loading, training of dorsal flexion muscles could play a role in preventing higher fore foot loading.

CONCLUSIONS
As a result of a higher plantar flexion moment during the first part of the stance phase, the centre of pressure will travel faster to the fore foot, leading to higher forefoot loading. It is concluded that evidence was provided that a diminished ability to generate a dorsal flexion moment can play an important role in the development of higher fore foot loading.

REFERENCES