PROLONGED ACTIVY OF KNEE EXTENSORS AND DORSAL FLEXORS IS ASSOCIATED WITH ADAPTATIONS IN GAIT IN DIABETES AND DIABETIC POLYNEUROPATHY

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INTRODUCTION

People with diabetes, and in particularly diabetic polyneuropathy, often experience limitations in mobility and gait. These limitations are believed to be related to maladapted muscle control. Abboud et al. [1] found in patients with diabetes a reduced and delayed activation of tibialis anterior. Kwon et al. [2] suggested that in people with diabetic polyneuropathy the triceps surae was prematurely activated. Unfortunately these studies did not control walking velocity. Walking velocity affects most gait characteristics. In people with diabetes gait velocity is lower than in age-matched healthy controls.

The present study evaluated independently the influence of gait velocity and diabetes and diabetic polyneuropathy on lower limb kinematics and muscle activation patterns.

METHODS

In this study ten healthy elderly controls (HC; 72.4±years), ten people diagnosed with diabetes without polyneuropathy (DC; 60.5±years) and eight people with diabetic polyneuropathy (DPN; 68.9±years) participated.

To analyse their gait pattern, all subjects were asked to walk at their preferred velocity and at a standard velocity of 1.4m/s. Using 50Hz-video, 2D marker positions on the subjects' limb were recorded. Joint angle patterns and spatiotemporal gait characteristics were calculated from these recordings. Bipolar surface ElectroMyoGraphy (EMG) was applied to determine activation patterns of *m. tibialis anterior* (TA), *m. gastrocnemius medialis*, *m. soleus*, *m. vastus medialis* (VM), *m. rectus femoris* (RF), *m. biceps femoris* and *m. gluteus maximus*. Raw EMG-data were rectified and filtered (4^{th} order 10Hz Butterworth), subsequently the active period of a muscle was assessed as the time were the activity was higher than 20% of the maximal activity.

RESULTS AND DISCUSSION

HC-participants displayed a non-significant trend to walk faster than both diabetic groups in the preferred velocity condition. All subjects walked slower in the preferred velocity condition than in the standard condition. Spatiotemporal characteristics did not differ between groups. In all groups stride duration was shorter and step length longer in the imposed condition. Also differences in joint angle pattern occurred only as a function of gait velocity, and did not differ between the groups. This suggests that previously reported differences in gait kinematics between people with and without diabetes or diabetic polyneuropathy result mainly from differences in gait velocity.

Muscle activation patterns did differ significantly between the groups. Under the imposed velocity condition VM-activity lasted shorter in HC than in both diabetes groups (p=0.035), RF activation was prolonged in the DPN group compared to DC and HC (p=0.012) and TA ceased activity earlier in DC than in HC and DPN (p=0.057; Fig1). In a previous analysis of the joint moments and plantar pressure patterns of the same subjects [3] we concluded that people with diabetic polyneuropathy have problems to control their forward velocity at heel strike and that consequently the centre of pressure travels faster to the front of the foot. The muscle groups that were found in this study to differ between groups of subjects are the muscles that are involved in braking and controlling the forward velocity at heel strike.

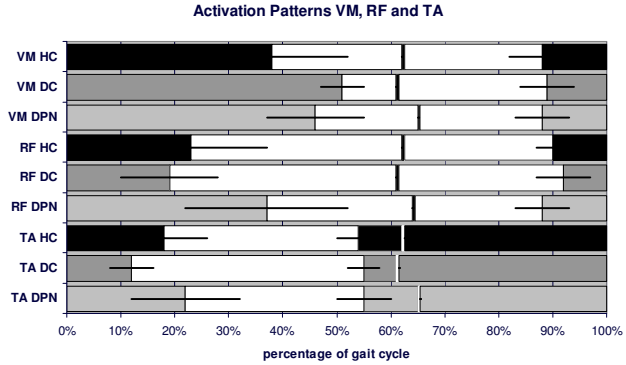

Figure 1: Activation patterns for VM, RF and TA in three groups of participants. The horizontal axis represents the percentage of gait cycle, from one heel strike (0%) to the next heel strike of the same limb (100%). Dark areas indicate phases of gait when a muscle was active. Black horizontal lines in the bars give the standard deviation for the moment of switching on or off of a muscle. The vertical line at about 65% (black for VM and RF, white for TA) indicate the end of stance phase.

CONCLUSIONS

If gait velocity is controlled differences in joint kinematics between people with and without diabetes or diabetic neuropathy do not seem to exist.

Muscle activation patterns in diabetic groups do not differ with respect to the onset of activation, but diverge with respect to cessation of activity. The prolonged activation that was found suggests that gait in diabetes is not disturbed due to hampered sensory information causing a delayed activation. Rather (rate of) force generation at activation seems to be not optimal and thus prolonged activation is required to ensure satisfying impulse generation.

REFERENCES

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