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The association between premature plantarflexor muscle activity, muscle strength, and equinus gait in patients with various pathologies

Katrin Schweizer [a,b], Jacqueline Romkes [a], Reinald Brunner [a,c,*]

[a] Laboratory for Movement Analysis, University Children's Hospital Basel (UKBB), Spitalstrasse 33, Postfach, CH-4031 Basel, Switzerland
[b] Clinical Morphology and Biomedical Engineering, University of Basel, c/o Biozentrum Pharmazentrum, Klingelberstr. 50-70, 4056 Basel, Switzerland
[c] Neuro-Orthopaedic Unit, University Children's Hospital Basel (UKBB), Spitalstrasse 33, Postfach, CH-4031 Basel, Switzerland

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ABSTRACT

This study provides an overview on the association between premature plantarflexor muscle activity (PPF), muscle strength, and equinus gait in patients with various pathologies. The purpose was to evaluate whether muscular weakness and biomechanical alterations are aetiological factors for PPF during walking, independent of the underlying pathology. In a retrospective design, 716 patients from our clinical database with 46 different pathologies (orthopaedic and neurologic) were evaluated. Gait analysis data of the patients included kinematics, kinetics, electromyographic activity (EMG) data, and manual muscle strength testing. All patients were clustered three times. First, patients were grouped according to their primary pathology. Second, all patients were again clustered, this time according to their impaired joints. Third, groups of patients with normal EMG or PPF, and equinus or normal foot contact were formed to evaluate the association between PPF and equinus gait. The patient groups derived by the first two cluster methods were further subdivided into patients with normal or reduced muscle strength. Additionally, the *phi* correlation coefficient was calculated between PPF and equinus gait. Independent of the clustering, PPF was present in all patient groups. Weak patients revealed PPF more frequently. The correlations of PPF and equinus gait were lower than expected, due to patients with normal EMG during loading response and equinus. These patients, however, showed higher gastrocnemius activity prior to foot strike together with lower peak tibialis anterior muscle activity in loading response. Patients with PPF and a normal foot contact possibly apply the plantarflexion–knee extension couple during loading response. While increased gastrocnemius activity around foot strike seems essential for equinus gait, premature gastrocnemius activity does not necessarily produce an equinus gait. We conclude that premature gastrocnemius activity is strongly associated with muscle weakness. It helps to control the knee joint under load independent from the underlying disease, and it is therefore a secondary deviation. If treated as primary target, caution should be exercised.

* Corresponding author at: University Children's Hospital Basel (UKBB), Spitalstrasse 33, Postfach, CH-4031 Basel, Switzerland. Tel.: +41 61 704 12 48; fax: +41 61 704 12 49.
E-mail address: reinaldbrunner@sunrise.ch (R. Brunner).

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1. Introduction

Three dimensional (3D) gait analysis is applied to prescribe treatment interventions in patients with different pathologies. The range spans from patients with orthopaedic impairments to patients with flaccid muscles as well as to patients with spasticity (Davis, 1997). Due to anatomical and functional restrictions, patients typically reveal gait deviations, such as premature plantarflexor muscle activity (PPF) during loading response of walking (Brunner & Romkes, 2008; Brunt & Scarborough, 1988; Higginson et al., 2006; Neptune, Burnfield, & Mulroy, 2007; Perry, 1992; Perry, Burnfield, Gronley, & Mulroy, 2003; Romkes & Brunner, 2007).

In the literature, abnormal plantarflexor timing is mainly described in association with initial forefoot contact (Neptune et al., 2007; Perry et al., 2003; Thomas, Moore, Kelp-Lenane, & Norris, 1996) and/or neurological impairment (Brunt & Scarborough, 1988; Higginson et al., 2006; Romkes & Brunner, 2007). For years, PPF was thought to result from spasticity or poor neuromuscular control in neurological patients (Gage, 1991; Perry, 1992). More recent studies, however, have claimed PPF to be a secondary deviation (Brunner & Romkes, 2008; Davids, Foti, Dabelstein, & Bagley, 1999; Romkes & Brunner, 2007; Thomas et al., 1996). According to Schmid, Schweizer, Romkes, Lorenzetti, and Brunner (2013), secondary deviations are either passive secondary effects that follow as a physical mechanism to the primary deviation, or active compensations. The named studies came to this conclusion, as muscular weakness has been shown to provoke abnormal electromyographic (EMG) activity in orthopaedic patients with different impairments (Brunner & Romkes, 2008). Hereby, no anatomical relationship between a specific weak muscle and a muscle showing abnormal EMG timing was possible. The medial gastrocnemius muscle was the most frequently involved muscle with abnormal EMG timing in orthopaedic patients (Brunner & Romkes, 2008). This conforms to the findings of Goldberg and Neptune (2007) where the plantarflexors were able to compensated for weakness in most of the major muscle groups in a forward dynamics simulation.

Subsequently, PPF can result from biomechanical alteration alone, given that similar abnormal muscle activity patterns were observed in healthy subjects when mimicking the walking pattern of patients (Davids et al., 1999; Romkes & Brunner, 2007; Thomas et al., 1996).

Although PPF can be observed in patients with various pathologies (Neptune et al., 2007), the prevalence across different patient groups, such as in patients with neurological or orthopaedic impairments, is still unknown. Muscle weakness and biomechanical alterations, as aetiological factors for PPF, were only examined in orthopaedic patients or healthy subjects. Therefore, it remains indistinct whether these are aetiological factors for all patients independent of the primary pathology.

The objective of the present study was to provide an overview on the association between initial equinus foot contact, muscle strength, and PPF during walking in patients with various pathologies. We hypothesised that PPF is present in all patient groups, and that muscle weakness and equinus gait are aetiological factors for PPF independent of the pathology. The outcome is of clinical relevance, as it will assist in interpreting PPF as primary or secondary deviation. For clinicians this distinction is crucial. Whereas a primary deviation requires treatment, secondary deviations resolve spontaneously once the primary abnormality is treated (Goodman et al., 2004; Stebbins, Harrington, Thompson, Zavatsky, & Theologis, 2010). Consequently, the results of this study may improve treatment planning and therapy outcome.

2. Methods

We retrospectively examined our 3D gait analysis database that has been collected in our laboratory for movement analysis in the context of everyday clinical practice. The parameters included were spatiotemporal parameters, lower body kinematics and kinetics, a clinical examination including manual muscle strength testing, and EMG recordings for all patients.

2.1. Subjects

All 1144 patients of the consecutive clinical gait analysis database from 2001 till 2012 were considered for this study. The database comprised patients with various orthopaedic and neurologic pathologies, mainly children and adolescents, but also adults. Selected for this study were all patients who walked barefoot without assistive devices, providing complete EMG, kinetic and kinematic data of at least three trials, as well as complete manual muscle strength testing. Finally, 716 patients with 46 different primary pathologies and 102 healthy controls were included. All subjects signed an informed consent at the time of the gait analysis. The study was approved by the local ethical committee.

2.2. Patient group clustering

All patients were clustered three times according to different aspects. Subsequently, two subgroups for each group derived by the second and third clustering method were separated according to mean manual muscle strength (MMS). Subgroup ‘‘almost normal MMS’’ has a MMS equal/above 4.5, and subgroup ‘‘reduced MMS’’ has a MMS of less than 4.5.

Table 1
The seven pathology groups.

Group abbreviation	Type of impairment	Muscle tone	Topographical description of the impairment	Included pathologies
OUni	Orthopaedic	–	Unilateral	All problems of foot, knee, hip, including true diseases such as Morbus Perthes, as well as solely pain, and unilateral torsional malalignment
OBi	Orthopaedic	–	Bilateral	Spinal disorders, Arthrogryposis Multiplex Congenita, leg length discrepancy, torsional malalignment
NflaUni	Neurologic	Flaccid	Unilateral	Poliomyelitis, palsy of single nerves
NflaBi	Neurologic	Flaccid	Bilateral	Spina bifida, paraplegia, muscle dystrophy, bilateral poliomyelitis, developmental retardation, trisomias
NspUni	Neurologic	Spastic	Unilateral	Hemiparesis of various aetiology
NspBi	Neurologic	Spastic	Bilateral with adequate trunk control	Diplegia
NspBiNTC	Neurologic	Spastic	Bilateral without adequate trunk control	Tetraparesis of various aetiologies

First clustering strategy. Patients were grouped according to their diagnoses, referred to as pathology groups. Here, the primary source of the problem was of interest: orthopaedic impairments, neurologic spasticity with trunk control, neurologic spasticity without trunk control, and neurologic flaccid patients. Further, it was distinguished whether the impairment was uni- or bilateral. Therefore, seven groups were formed which are described in Table 1. A more detailed composition of the patient groups can be found in the supplementary data to this article in the online version.

Second, independently of the first clustering, the entire patient population was clustered according to the impaired joint level, namely impairment groups, to avoid bias by the second clustering. A joint was defined to be impaired if the kinematic deviation in sagittal plane was above the 97.5 percentile of the Gait Variable Score (GVS) (Baker et al., 2009) for our controls. The thresholds were for the hip 10.9°, knee 11.0°, and for the ankle 7.2°. This resulted in eight impairment groups: (1) patients with abnormal hip; (2) patients with abnormal knee; (3) patients with abnormal ankle; (4) patients with abnormal hip and knee; (5) patients with abnormal hip and ankle; (6) patients with abnormal knee and ankle; (7) patients with abnormal hip, knee, and ankle; (8) patients with normal hip, knee, and ankle joints.

Third, again the whole patient population was divided in patients with normal EMG or PPF and equinus or normal foot contact was formed to evaluate the association between PPF and equinus gait.

2.3. Clinical gait analysis

Three dimensional gait analysis data were collected and pre-processed by a VICON motion analysis system (years 2001–2002: six-camera system 370, 60 Hz, marker diameter 25 mm; VICON Clinical Manager software; years 2003–2010: six-camera system 460, 120 Hz, marker diameter 14 mm, VICON Workstation software; since 2011: twelve-camera system MXT20, 200 Hz, VICON Nexus software; VICON, Oxford, UK). Controls and patients walked at a self-selected speed on a 10 m level ground walkway. Kinetic data were acquired by two force platforms at a sampling rate of 2520 Hz (2001–2007) and of 2400 Hz since 2007 (KISTLER Instrumente AG, Winterthur, Switzerland).

For the kinematics, fifteen passive reflective markers were fixed to specific anatomical landmarks bilaterally on the subject's legs and pelvis according to the protocol of Kadaba et al. (1987). Height, weight, leg length, width of ankles and knees, and tibial torsion were measured clinically for appropriate anthropometric scaling. A knee alignment device was used for the static trial (Motion Lab Systems Inc., Los Angeles, USA).

Surface EMG was recorded simultaneously. Bipolar Ag/AgCl surface electrode pairs (10 mm diameter, 22 mm inter-electrode spacing) were placed bilaterally on the gastrocnemius medialis muscle (GM) and tibialis anterior muscle (TA) according to the SENIAM guidelines (Hermens et al., 1999). The ground electrode was placed over the tibial tuberosity. The electrodes were connected to single differential amplifiers with integrated band-pass filters at 10–700 Hz (Biovision AG, Wehrheim, Germany). The pre-amplifiers and electrodes remained the same for all measurements. Between 2001 and April 2007, pre-amplified EMG signals were collected using a Zebris System (Zebris, Tübingen, Germany) and sampled at a rate of 2520 Hz. Since May 2007 signals were collected by a Neurodata System (Neurodata, Vienna, Austria) at a sampling frequency of 2400 Hz.

Gait events, i.e. foot strike and toe-off, were set manually, and the kinematic and kinetic data were filtered by the Woltring filter (mean squared error set to 10) in the VICON software pipeline.

During the clinical examination, a physiotherapist assessed muscle strength for the lower extremity muscles of each patient according to the manual muscle strength scale described in Hislop and Montgomery (1999) (scale 0 = paralysed muscle to 5 = strong). The muscle groups assessed were: hip flexors/extensors/abductors and in-/external rotators, knee flex-/extensors, plantar-/dorsi flexors. The average on all leg muscles formed the MMS.

2.4. Data processing

The entire post-processing and all calculations were done using the MATLAB software (MathWorks, Inc. Version R2010a, Natick, USA). Kinematic and kinetic data were normalised to a fixed amount of 51 data points per gait cycle (0–100%). A gait cycle was defined as the time between two consecutive foot strikes of the same leg. Subsequently, one trial (gait cycle) for each patient and control subject was selected using the SMaRT method (Schweizer et al., 2012). Hereby, the distance of each principal component score to the median of all trials was calculated for each angle, and the trial which is closest to the median across all angles was then selected (Schweizer et al., 2012).

Raw EMG signals were visually inspected for artefacts and noise, before they were filtered with a 4th order Butterworth band-pass filter with a cut-off frequency of 20–500 Hz (Clancy, Morin, & Merletti, 2002; Moritz, Greene, & Farley, 2004). Subsequently, the signal was full-wave rectified, and a moving average was calculated with a 39.8 s time window (similar to Romkes & Brunner, 2007). The EMG signal was further normalised for stance (31 data points) and swing phase (20 data points), delivering together a gait cycle of 51 data points. Finally, the EMG was amplitude-normalised to the average value of each cycle.

2.5. Parameter definition

Walking speed, cadence, and step length were evaluated in non-dimensional values according to Hof (1996). The GVS were calculated for all patients as a quantity of the kinematic gait deviation by using our own normative data.

A muscle was defined to be abnormally active if the normalised EMG signal was above a certain percentage of its peak value which was set according to the walking speed (Schwartz, Rozumalski, & Trost, 2008). The thresholds were 28%, 23%, and 31% for the non-dimensional walking speeds (Hof, 1996) of <0.227, 0.228–0.363, and >0.363. PPF was specified as activity of the GM above the threshold during loading response phase of gait (i.e. 0–10% of the gait cycle). Validity of this method was examined by checking how many of our control subjects show "abnormal" EMG when applying this method, the less the better.

Equinus at initial contact was defined as 5° of plantarflexion or more at initial contact. This corresponded to approximately two standard deviations (1 SD = 2.8°) below the mean (1.2°) of the norm. Additionally, to exclude drop feet, plantarflexion had to increase by at least 5° over the last 10% of the gait cycle. If the latter was not fulfilled, but the ankle angle stayed 5° or more in plantarflexion during the entire gait cycle, it was still defined as equinus gait. The ankle position was termed as "normal foot contact" when the sagittal ankle angle did not meet any of the criteria for equinus gait.

The variables and gait phases of interest were: MMS, mean GM activity during loading response and terminal swing (87–100% of the gait cycle (Perry, 1992), mean TA activity during terminal swing and peak activity in loading response, equinus gait at initial contact, and mean ankle power during loading response.

2.6. Statistics

In unilateral impaired patients the involved leg side was analysed, and in those with bilateral impairments one leg was selected randomly. Randomisation was achieved by creating a binary vector of 716 rows with the "randi" function in MATLAB.

The *phi* correlation coefficients for each pathology group were calculated between the two dichotomous variables PPF and equinus gait. As the majority of the kinetic and EMG data were not normally distributed according to Shapiro–Wilk test, non-parametric statistics were applied to reveal significant differences. Mann–Whitney *U*-tests were conducted for the GM activity of patients with normal EMG and equinus versus patients with normal ankle for the mean of loading response phase and terminal swing separately. The same patient groups were compared by Mann–Whitney *U*-tests on the mean difference of their TA activity during terminal swing, as well as on their peak difference of this muscle in loading response. The level of significance was set at 5% for all tests.

Prevalence of PPF in the different pathology and impairment groups is given in percentage of the total number of patients in this group. The influence of muscle weakness on PPF was qualitatively assessed.

3. Results

Characteristics of the control and patient groups are specified in Table 2. PPF was identified in 38.7% (277/716) of all patients. It was equally distributed for both genders, with 38.8% (124/320) in females and 38.6% (153/396) in males. Abnormal EMG was unevenly distributed but present in all pathology groups (Fig. 1a). In none of the control group subjects PPF was observed.

All following results are visual trends derived by Fig. 1. For all pathology groups, except for the NflaUni group, the percentage of patients with PPF increased from the subgroups "almost normal MMS" to "reduced MMS" (Fig. 1a). When grouping the patients according to their impaired joints derived by the GVS (Fig. 1b), PPF was observed in all patient groups again. Patients with normal GVS values; hence with normal sagittal plane kinematics for all joints, have the smallest incident rates, followed by patients with abnormal kinematics for one of the joints. Patients with two or all three joints impaired showed the highest prevalence of PPF. Furthermore, PPF is more frequent in weak patients than in patients with normal muscle strength.

Table 2
Characteristics of the subject groups.

Patient group	N	Age (years)	Sex (f/m)	BMI (kg/m^2)	Normalised step length	Normalised walking speed	Normalised cadence
Controls	102	25.1 (±12.0)	51/51	21.7 (±3.4)	0.77 (±0.07)	0.45 (±0.05)	35.42 (±2.05)
OUni	93	20.9 (±13.7)	48/45	21.8 (±4.4)	0.75 (±0.09)	0.43 (±0.07)	34.76 (±2.93)
OBi	176	15.7 (±8.7)	81/95	20.5 (±4.0)	0.76 (±0.09)	0.44 (±0.06)	35.12 (±2.77)
NflaUni	12	21.8 (±16.3)	4/8	19.6 (±3.5)	0.76 (±0.10)	0.41 (±0.09)	33.09 (±4.58)
NflaBi	83	19.4 (±12.9)	41/42	21.5 (±5.6)	0.66 (±0.13)	0.36 (±0.09)	32.03 (±4.45)
NspUni	176	16.7 (±10.0)	80/96	20.8 (±5.1)	0.72 (±0.10)	0.41 (±0.08)	33.31 (±3.98)
NspBi	119	15.8 (±7.9)	46/73	20.0 (±3.9)	0.67 (±0.12)	0.37 (±0.09)	33.36 (±4.33)
NspBiNTC	57	19.1 (±9.5)	20/37	20.3 (±4.5)	0.61 (±0.16)	0.34 (±0.12)	32.01 (±6.60)

The number of subjects (*N*), the mean (±one standard deviation) age in years, the sex (female/male), as well as mean (± one standard deviation) Body Mass Index (BMI), step length, walking speed, and cadence are reported for the healthy controls and each patient group. The last three gait parameters are reported as non-dimensional parameters. The patient groups are decoded as follows: O/N = orthopaedic/neurologic, Uni/Bi = unilateral/bilateral involvement, fla/sp = flaccid/spastic muscles, NTC = no thoracal control.

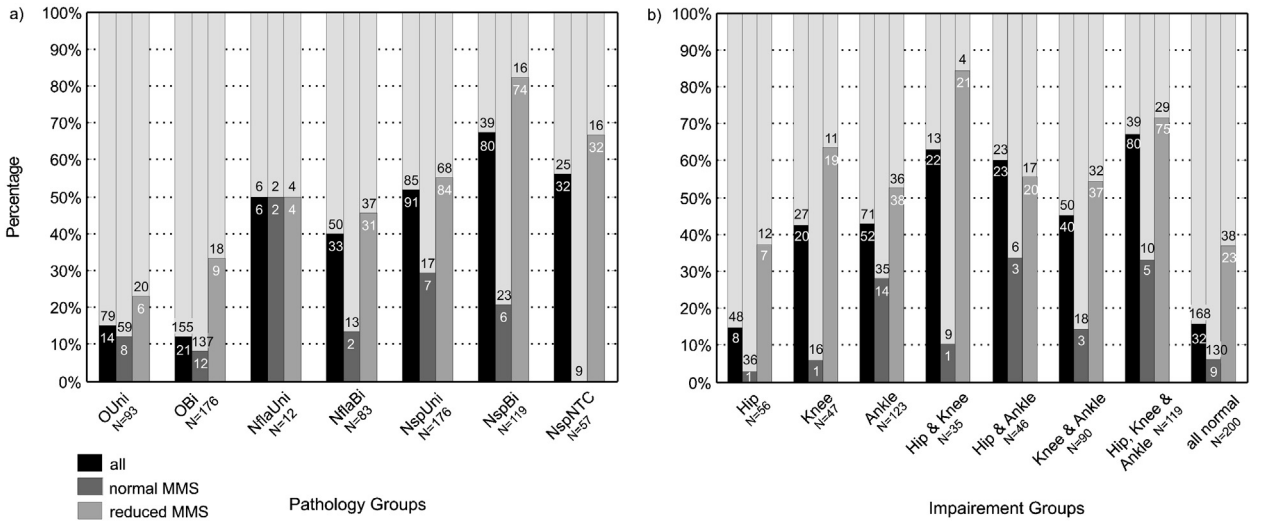

Fig. 1. Prevalence of premature m. gastrocnemius medialis activity. The figure shows the number of patients in each pathology group (a)/impairment group (b) with abnormal (bottom part) and normal EMG (upper part) expressed in percentage (*y*-axis) of the total number (in bars) of patients in this group. The first column in each figure represents the distribution across all patients within this group (all). The second column displays the distribution in the subgroup with mean manual muscle strength (MMS) ≥ 4.5 (almost normal MMS), and the third (reduced MMS) shows the distribution in the subgroup with MMS <4.5. The patient groups in (a) are decoded as follows: O/N = orthopaedic/neurologic, Uni/Bi = unilateral/bilateral involvement, fla/sp = flaccid/spastic muscles, NTC = no thoracal control. In (b) the patients are grouped according to the impaired joint, e.g. the group 'hip' includes patients with abnormal Gait Variable Score (GVS) of the hip.

The correlations between equinus gait and PPF were low to moderate, and they were merely significant for all patients (total), OUni, OBi, and NspBi (Table 3).

Across all patients with normal EMG, 79.0% (347/439) did not show an equinus gait and 21.0% (92/439) did. Of all patients with PPF, a normal foot contact was present in 56.0% (155/277) patients and equinus foot contact in 44.0% (122/277).

Table 3
Correlation between premature GM activity and equinus gait.

Group	*Phi*	*p*
Total	**0.246**	**0.000**
OUni	**0.388**	**0.000**
OBi	**0.262**	**0.001**
NflaUni	−0.333	0.248
NflaBi	0.085	0.440
NspUni	0.113	0.133
NspBi	**0.227**	**0.013**
NspBiNTC	−0.008	0.952

The *phi* correlation coefficients of premature GM activity and equinus gait are presented for the different pathology groups in the second column. The *p* values for the correlations are listed in column three, values in bold are significant at $p < 0.05$. The patient groups are decoded as follows: O/N = orthopaedic/neurologic, Uni/Bi = unilateral/bilateral involvement, fla/sp = flaccid/spastic muscles, NTC = no thoracal control.

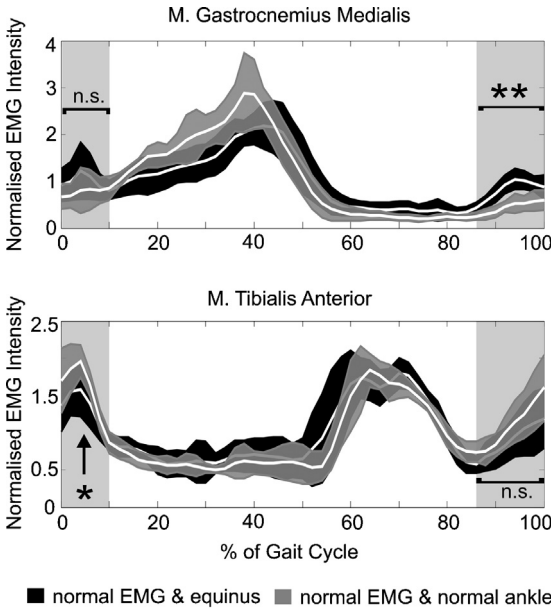

Fig. 2. Normal foot contact vs. equinus gait in patients with normal EMG. The EMG of patients with a normal foot contact in grey and of patients with equinus gait (black) is plotted for an entire gait cycle. Grey shaded areas are the gait phases of interest, loading response and terminal swing. Presented are the mean and one standard deviation of the enveloped EMG signal for the respective groups. The EMG signals was normalised to the mean amplitude of the signal before they were averaged. Asterisks indicate statistical significant differences ($^{*}p < 0.05$, $^{**}p < 0.01$). n.s., non-significant.

Patients with equinus gait but without PPF during loading response, showed significantly ($p = 0.001$) higher mean GM activity during terminal swing than patients with a normal foot contact (Fig. 2). Additionally, they had significantly lower peak TA activity during loading response ($p = 0.026$) than patients with a normal foot contact (Fig. 2). Both, mean GM activity during loading response and mean TA activity during terminal swing, did not differ significantly within these two groups (GM: $p = 0.209$, TA: $p = 0.318$). Patients with a normal foot contact despite PPF revealed a higher mean foot absorption power ($p = 0.007$) during loading response compared to patients without PPF (Fig. 3).

4. Discussion

This study focused on the association between equinus gait, the muscle strength of patients, and their EMG pattern across a variety of different pathologies. It was hypothesised that muscle weakness is among the causes for PPF, and that PPF correlates with equinus gait. Knowledge on the interrelations between these parameters can find clinical implication in the interpretation of gait deviations across different patient groups.

In order to prevent overestimation of the number of patients with abnormal muscle timing, the criteria for PPF activity during loading response were set according to walking speed. Thereby we took into account that the EMG amplitude differs depending on the walking speed (Schwartz et al., 2008). In addition, the activity had to be constantly above the threshold for

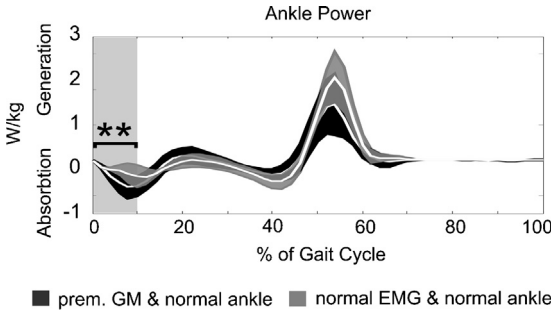

Fig. 3. Premature gastrocnemius activity vs. normal EMG in patients with normal foot contact. The ankle power of patients with a normal foot contact and premature m. gastrocnemius medialis (GM) activity (black) and of patients with normal EMG (grey) is plotted for an entire gait cycle. The grey shaded area is the loading response phase. Presented are the mean and one standard deviation of the ankle power for the respective groups. The bold black lines indicate statistical significant differences.

the entire loading response phase. The detection method for PPF was considered as valid since none of the healthy controls had an abnormal EMG according to this method.

PPF was present across all pathological groups; hence, PPF is not dependent on a solely neurological component. It remains unclear whether in spastic patients the neurological disease itself is another factor for PPF or whether the higher incidence of PPF is only due to poorer neuromuscular control. Except for the NflaUni group, the number of patients with PPF increased drastically between the subgroups "almost normal MMS" to "reduced MMS". Although it is difficult to quantify, there exists at least a qualitative relation between EMG signal and the force of the muscle (Nigg & Herzog, 2007). Consequently, an explanation for these findings could be that weak patients might need higher muscle activity to produce the same or at least a sufficient force to control the joints under load. The results in the NflaUni might have been biased by the small patient numbers. Especially in the subgroup with normal muscle strength, there were only four patients.

The formation of patient groups according to their pathology obviously is a limitation. When clustering according to the pathology, inevitably some groups comprised patients with very different diagnoses such as OUni, OBi, NflaBi, whereas other groups, such as NspUni or NspBiNTC, were rather homogenous. The less homogenous groups were composed of patients with very different diagnoses as the total number of individuals with a given pathology was small. To account for that, we also grouped the patients according to their impaired joints. Similar to the grouping according to the pathology, weak patients showed PPF more frequently than patients with good muscle strength. Considering that for both different grouping strategies the main results were the same, we are confident that the patient group clustering did not bias our work.

The correlations between equinus gait and PPF were lower than expected and often not significant. In the patient groups NflaUni, NflaBi, NspUni, NspBiNTC there seems to exist no such correlation. Even in the patient groups where the correlations were significant (OUni, OBi, NspBi) the *phi* values were low. This fact shows, that equinus gait is a predictor for PPF in these patients, however, only a weak one. Similarly, PPF and equinus gait are significantly associated for all patients (total) but only to a low extend. These results can be explained by the unexpected high number of patients with a normal foot contact despite PPF and by the also unexpected high number of patients with equinus not showing PPF. In patients without PPF and equinus, the higher GM activity just prior to foot strike, together with lower TA activity, could promote equinus with lower GM activity needed during loading response phase of walking. Further, it is possible that these patients use their soleus muscle more to keep the equinus upright. Unfortunately, there were no EMG data on the soleus muscle available. Patients with normal EMG and equinus may still have a higher activity of the GM in loading response, but this activity is not constant or above the thresholds to be detected as PPF. Looking at the mean EMG of these patients this seems to be the case. Patients visually show more GM activity during loading response than patients with a normal foot contact but the difference is not significant. Higher GM activity around foot strike in equinus gait is in accordance with the literature (Davids et al., 1999; Romkes & Brunner, 2007; Thomas et al., 1996). Increased activity is supposed to be essential in order to keep the same force generating capacity of the plantarflexor muscles while they act on a less-optimal force-length condition (Neptune et al., 2007). Patients with PPF and a normal foot contact produced higher ankle absorption power than patients with normal EMG and normal foot contact in loading response. An explanation could be that patients with PPF possibly use the muscle activity to prevent the tibia to translate forward; hence, they might control the knee by PPF during loading response. When this hypothesis can be verified by muscle modelling, this would hold evidence that the plantarflexion–knee extension couple does not only act in mid stance (Gage, 1991) but can be used also during loading response.

5. Conclusion

This study indicates that muscle strength is an aetiological factor for PPF independent of the primary pathology. Even in neurological patients it is not only spasticity which leads to PPF, but also muscle weakness. In consequence, we conclude that PPF should be regarded as a secondary gait deviation with clinical relevance in all patients. While for equinus gait increased GM activity just prior to foot strike or during loading response seems essential, PPF does not necessarily produce an equinus gait. Rather it can also be used to control the knee through the plantarflexion–knee extension couple in loading response.

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