Effects of ankle fatigue on functional reflex activity during gait perturbations in young and elderly men

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ABSTRACT

There is growing evidence that aging and muscle fatigue result in impaired postural reflexes in humans. Therefore, the objective of this study was to examine the effects of ankle fatigue on functional reflex activity (FRA) during gait perturbations in young and elderly men.

Twenty-eight young (27.0 ± 3.1 years, $n = 14$) and old (67.2 ± 3.7 years, $n = 14$) healthy active men participated in this study. Fatigue of the plantarflexors and dorsiflexors was induced by isokinetic contractions. Pre and post-fatigue, subjects were tested for their ability to compensate for decelerating gait perturbations while walking on a treadmill. Latency, FRA of lower extremity muscles and angular velocity of the ankle joint complex were analysed by means of surface electromyography and goniometry.

After the fatigue protocol, no significant main and interaction effects were detected for the parameter latency in m. tibialis anterior (TA). For both groups, a significant pre to post-test decrease in FRA in TA ($p < .001$) was observed coming along with increases in antagonist coactivity ($p = .013$) and maximal angular velocity of the ankle joint ($p = .007$). However, no significant group $\times$ test interactions were found for the three parameters.

Ankle fatigue has an impact on the ability to compensate for gait perturbations in young and elderly adults. However, no significant differences in all analysed parameters were detected between young and elderly subjects. These results may imply that age-related deteriorations in the postural control system do not specifically affect the ability to compensate for gait perturbations under fatigued condition.

Keywords:

Seniors

Aging

Postural control

Neuromuscular fatigue

Isokinetic fatigue protocol

1. Introduction

The number of senior citizens aged 65 and older has substantially increased in societies of western industrial countries. A serious problem of the aging population is the susceptibility of sustaining a fall. Twenty-eight to 35% of individuals over the age of 65 years experience at least one fall over a 1-year period [1]. Furthermore, it was reported that slips and trips account for 30–50% of falls in community-dwelling older adults [2]. Numerous epidemiological studies have identified a multitude of risk factors for falling. In fact, deficits in postural control substantially contribute to the increased fall incidence in seniors [3]. Age-related degenerative changes in muscle mass, sensory function,

and/or neural processing may impede the ability to control posture effectively [4].

There is evidence in literature that balance impairments, greater stride-to-stride variability, the inhibition of gait quality and thus the increased occurrence of falls is not only caused by biologic aging but also by fatigue of the lower leg muscles [5,6]. Parijat and Lockhart [6] observed that localised muscle fatigue of the quadriceps affected various kinematic and kinetic gait parameters that are linked with a higher risk of slip-induced falls in young healthy adults. Helbostad et al. [5] reported that a repeated sit-to-stand task affected gait control in older persons in terms of an increased variability in step width and length.

From a functional point of view, it is of interest to know, whether muscle fatigue has similar or different effects on dynamic stability control during postural perturbations in young and elderly subjects. Recently, Mademli et al. [7] investigated the effects of muscle fatigue on the dynamic stability control of young and older adults after inducing a sudden perturbation from a fixed

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Table 1
Characteristics of the study cohort.

Characteristic	Young men, <i>n</i> = 14	Elderly men, <i>n</i> = 14
Age (years)	27.0 ± 3.1	67.2 ± 3.7
Body height (cm)	178.4 ± 6.5	175.0 ± 4.8
Body mass (kg)	74.5 ± 6.3	77.0 ± 5.5
Body mass index (kg/m ²)	23.5 ± 0.8	25.1 ± 2.1
Activity level (h/week)	13 ± 8	13 ± 3

Note: Values are mean ± SD.

forward inclined position. The authors were able to demonstrate a lower margin of stability (boundary of the base of support) at touchdown after the forward fall in old compared to young adults. In addition, muscle fatigue of the knee extensors and flexors did not influence the margins of stability in both experimental groups [7].

Since chances of sustaining a fall are particularly high during slipping or tripping situations in fatigued condition [2,6], the objective of this study was to examine the effects of ankle fatigue, as established by standard criteria, on functional reflex activity (FRA)¹ during gait perturbations in young and elderly men. We hypothesized that fatigue of the muscles encompassing the ankle joint would decrease FRA of muscles compensating for gait perturbations in young and elderly men, with the elderly subjects showing larger decreases than the young.

2. Methods

2.1. Study population

Fourteen young and fourteen elderly men with no statistically significant anthropometrical differences in body mass, body height, and BMI provided written informed consent to participate in the study after experimental procedures were explained (Table 1). The subjects were healthy with no previous lower extremity trauma and no history of serious muscular, neurological, cardiovascular, metabolic and inflammatory diseases. Before the tests of the study started, participants were asked to complete the validated "Freiburg questionnaire for everyday and sports activities"SM [10]. The young and elderly subjects were classified as physically active. According to the lateral preference inventory [11], all subjects were right leg dominant. All experiments were conducted according to the latest revision of the declaration of Helsinki.

2.2. Testing protocol

In order to analyse effects of ankle fatigue on FRA during gait perturbations in young and elderly men, all subjects were tested for treadmill perturbation impulses. Thereafter, they performed an ankle fatigue protocol on an isokinetic device and were subsequently re-tested for treadmill perturbation impulses.

2.2.1. Treadmill perturbation

Before the actual testing started, subjects were provided with standardized low-cut athletic footwear (Adidas[®], Spezial, Herzogenaurach, Germany) to minimize variability and to control for shoe type and design. Thereafter, participants were accustomed to walking on a treadmill for a 5 min warm-up period. Decelerating perturbation impulses were randomly induced at distinct phases of the gait cycle while subjects were walking on a treadmill (Woodway[®], Weil am Rhein, Germany). In this way, an everyday stumbling situation was transferred into a laboratory condition. The application of decelerating perturbation impulses at heel strike while walking on a treadmill induces an everyday stumbling stimulus which moves the body's center of mass closer to the edge of the base of support. As a reactive response, the prime mover TA is activated to bring the body's center of mass back over the base of support [12]. For safety reasons, subjects wore a chest harness system. The treadmill was powered by a DC motor with a gear unit, a tachometer generator, and a transistor-servo-amplifier in a four-quadrant system. The impulse capacity of the amplifier (MTR-7025) produced 12.750 W for 1 s. This allowed rapid decelerating impulses of the treadmill from 3.5 km/h to a backward velocity of 0.6 km/h within 0.4 s. Therefore, the treadmill was not only decelerated to zero, but the direction of the belt movement was also reversed. Electrical switches implemented into inlays of the right shoe (underneath the heel and the ball of

Fig. 1. Electromyographic (averaged and rectified) and goniometric data of the stance phase of a regular unperturbed gait cycle (dotted line) are synchronized at heel contact (dashed vertical line) with EMG and goniometric data of a decelerating impulse (thick line). The analysed time interval was 120 ms after the first response in the goniometer-signal to the perturbation impulse (functional reflex activity: grey rectangle). The first part of the latency between heel strike and the onset of perturbation (beginning of the analyzed time interval) can be explained by mechanical delay due to the transmission and procession of the trigger signal coming from electrical switches implemented in the inlays of the right shoe and the deceleration of the treadmill. Modified from Granacher et al. [8].

the foot) were used as a trigger for decelerating the treadmill precisely at heel contact and for determining the stance and the swing phase of a regular gait cycle. FRA was time analysed 120 ms after the first response to the perturbation impulse. More specifically, the first response to the perturbation impulse (trigger signal) was indicated by a goniometer-signal (plantar/dorsiflexion of the ankle joint) (Fig. 1). Therefore, an electronic two-axial goniometer (Penny&Giles[®], type XM180, Gwent, UK) was firmly attached, with double-sided adhesive tape, along the Achilles tendon at the ankle joint. The goniometer-signal was filtered by a Butterworth low-pass filter, using a cut-off frequency of 10 Hz. With regard to the decelerating impulse, the inflection point of the goniometer-signal for plantar/dorsiflexion of the ankle was calculated and taken as a trigger for further data analysis. Maximal angular velocity of the ankle joint was determined in the time interval 120 ms following the first response in the goniometer-signal to the perturbation impulse (Fig. 1). Furthermore, latency defined as the time that elapses between the first response in the goniometer-signal and the first electromyographic (EMG) activity was analysed. Altogether, a total of 10 decelerating impulses on the treadmill were applied and averaged for further data analysis. Besides the perturbation situations, the normal walking pattern (during unperturbed walking) was recorded at a speed of 3.5 km/h. Pijnappels et al. [13] investigated EMG modulation in anticipation of a possible trip during walking in young and older adults. The authors reported that anticipatory effects did not jeopardize the validity of experiments in which subjects were perturbed more than once. Further, intraclass correlation coefficients (ICCs) were calculated for FRA in TA (ICC = 0.85), latency in TA (ICC = 0.92), and maximal angular velocity of the ankle joint (ICC = 0.88) indicating excellent test-retest reliability. This protocol has recently been described in detail elsewhere [8,9,14].

2.2.2. Isokinetic fatigue protocol

Right after the initial perturbation impulses on the treadmill, the participant's right foot was positioned and firmly fixed in an isokinetic device (Cybex[®] Orthodron, Medway, USA) while the subject was lying on a massage table in prone position with the waist fixed. Before the protocol started, subjects became accustomed to the isokinetic device by doing a warm-up consisting of five submaximal dynamic actions in a concentric-concentric mode at an angular velocity of 60°/s (plantar-dorsiflexion). Thereafter, each subject performed four maximal contractions of the plantar- and dorsiflexors at 60°/s. For each trial, subjects were thoroughly instructed to act as forcefully as possible. The best trial was taken as maximal torque (F_{max}). The fatigue criteria were determined by examining the subjects' F_{max} during each exercise. No limitations were placed on the number of repetitions to reach 50% of F_{max} . Once three consecutive repetitions below 50% F_{max} were obtained the subject was immediately unfixed from the isokinetic device and led to the treadmill where the decelerating perturbation impulses were applied. During the fatigue protocol, subjects were instructed to avoid forced respiration during maximal efforts.

¹ FRA refers to reflexive (involuntary) activations of muscles of the lower extremities for the purpose of compensating for gait perturbations while walking on a treadmill [8,9].

2.2.3. Electromyography (EMG)

Circular bipolar surface electrodes (Hellige[®], type 44008347 Ag/AgCl, Freiburg, Germany) (diameter 10 mm, center to center distance 25 mm) were placed over m. tibialis anterior (TA), m. soleus (SO), m. peroneus (PE), and m. vastus medialis (VM) of the right leg. The longitudinal axes of the electrodes were in line with the direction of the underlying muscle fibers. The reference electrode was attached to the patella. Interelectrode resistance was kept below 5 k Ω by shaving, slightly roughening, degreasing and disinfecting the skin. EMG signals were sampled at 500 Hz, amplified and bandpass filtered (10–1000 Hz) and were carefully monitored for artifacts, noise and cross-talk. EMG data were quantified by integrating and time normalising the full-wave rectified EMG signals (mean amplitude voltage (MAV)) over the time interval of 120 ms. In addition, standardized reference contractions (muscle activity during 10 averaged regular and unperturbed gait cycles) were used to normalise MAV of TA, SO and VM during the decelerating impulse (functional reflex activity). Thus, FRA during 10 decelerating impulses was expressed in per cent of muscle activity during 10 regular gait cycles. Antagonist coactivation was expressed for gait perturbations as the ratio between normalized MAV of SO and normalized MAV of TA, measured during the same period of time.

2.3. Statistical analysis

Data are presented as group mean values $\pm$ standard deviations (SD). A multivariate analysis of variance (MANOVA) was used to detect differences between study groups (young, elderly) in all baseline variables. In case of significant baseline differences, deltas (post-fatigue value–pre-fatigue value) were calculated for the respecting parameters and differences between young and elderly men were computed by a multivariate ANOVA. If baseline differences between young and elderly men were not present, parameters of the gait perturbation tests were analysed in separate 2 (groups: young, elderly) $\times$ 2 (tests: pre-fatigue, post-fatigue) ANOVA with repeated measures on test. In addition, the classification of effect sizes was determined by calculating partial eta square (η_p^2). The significance level was set at $p < .05$. All analyses were performed using Statistical Package for Social Sciences (SPSS) version 16.0.

3. Results

Table 2 displays means and standard deviations for all variables. At baseline, the elderly men showed a significantly lower normalized FRA in TA during gait perturbations compared to the young men ($p = .043$). For all other collected variables, no significant differences were detected at baseline between young and elderly subjects. Further, the number of repetitions to reach 50% of $F_{\max}$ was not significantly different between young and elderly men.

After the fatigue protocol, both groups (young, elderly) showed slightly longer latencies in TA in response to the decelerating perturbation impulse (Table 2). However, the analysis failed to indicate statistically significant main and interaction effects.

For both groups, a significant pre to post-test decrease in normalized FRA in TA (Fig. 2; $p < .001$) was observed coming along with an increase in antagonist coactivity (Fig. 3; $p = .013$). However, no significant group $\times$ test interaction was found for either parameter. The analysis of normalized activity in SO, PE, and VM during the compensation of gait perturbations failed to indicate significant main and interaction effects.

There was a significant pre to post-test increase in maximal angular velocity (plantar–dorsiflexion) of the ankle joint for both groups ($p = .007$; Fig. 4), but no group $\times$ test interaction ($p = .374$).

Fig. 2. Functional reflex activity of m. tibialis anterior analysed in the time interval 0–120 ms and normalized on muscle activity of the m. tibialis anterior during the swing phase of 10 regular gait cycles, expressed in % are presented pre and post-fatigue as mean $\pm$ standard deviation.

Fig. 3. Antagonist coactivity (m. soleus and m. tibialis anterior) analysed in the time interval 0–120 ms and normalized on muscle activity of the m. tibialis anterior during the swing phase and the m. soleus during the stance phase of 10 regular gait cycles, expressed in % are presented pre and post-fatigue as mean $\pm$ standard deviation.

Fig. 4. Maximal angular velocity in the ankle joint (plantar–dorsiflexion) analysed in the time interval 0–120 ms, expressed in °/s is presented pre and post-fatigue as mean $\pm$ standard deviation.

Table 2
Effects of ankle fatigue on functional reflex activity during gait perturbations in young and elderly men.

Parameters	Young men		Elderly men	
	Pre-fatigue	Post-fatigue	Pre-fatigue	Post-fatigue
Latency in m. tibialis anterior (ms)	75.1 $\pm$ 9.0	79.6 $\pm$ 17.2	77.7 $\pm$ 13.0	79.4 $\pm$ 11.6
FRA in m. tibialis anterior (%)	306.1 $\pm$ 144.9	253.7 $\pm$ 146.2	215.7 $\pm$ 66.4	174.7 $\pm$ 79.7
Activity in m. soleus (%)	67.8 $\pm$ 20.5	74.3 $\pm$ 27.6	63.7 $\pm$ 11.6	68.9 $\pm$ 31.0
Coactivity (SO/TA) (%)	25.1 $\pm$ 8.7	32.3 $\pm$ 15.2	31.3 $\pm$ 8.4	44.1 $\pm$ 29.6
Activity in m. peroneus (%)	148.9 $\pm$ 59.2	141.1 $\pm$ 82.4	111.3 $\pm$ 39.9	115.1 $\pm$ 51.8
Activity in m. vastus medialis (%)	189.2 $\pm$ 134.1	182.9 $\pm$ 93.0	167.4 $\pm$ 49.3	175.3 $\pm$ 62.0
Maximal angular velocity (°/s)	113.3 $\pm$ 51.9	152.7 $\pm$ 46.9	95.6 $\pm$ 35.3	116.5 $\pm$ 30.2

Notes: Values are mean $\pm$ SD. FRA = functional reflex activity during gait perturbations; SO = m. soleus; TA = m. tibialis anterior.

4. Discussion

From a functional point of view, the most interesting findings of the present study were that fatigue of the muscles encompassing the ankle joint had an impact on the ability to compensate for gait perturbations in both young and elderly men in terms of a fatigue-induced decrease in FRA of TA accompanied by an increase in coactivity of SO and TA. However, no significant differences between young and elderly subjects were detected regarding the impact of muscle fatigue on the ability to compensate for gait perturbations.

In the present study, elderly men showed smaller magnitudes of FRA in TA (prime mover) than young men when postural control was threatened by gait perturbations. The impaired ability of the elderly men to compensate for gait perturbations complies with literature [16,15]. Tang and Woollacott [15] found smaller magnitudes of postural responses and higher levels of antagonist coactivation to unexpected slips during walking in older adults. Lin and Woollacott [16] determined postural muscle response characteristics following various sizes of support surface perturbations in young, stable older, and unstable older adults. Slower onset latencies, smaller magnitudes of postural responses, and longer maintenance of postural muscle activation were found in response to platform perturbations in both stable and unstable older subjects compared to young adults.

It has been speculated that age-related changes in reflex activity are attributed to a desensitization of muscle spindles, a demyelination of sensory axons, with a subsequent decrease in number of sensory neurons, changes in presynaptic inhibition, and a reduction in number of inter- as well as alpha- and gamma-motoneurons [17]. In particular, the reduced afferent input via muscle spindles in old age could affect the control of the muscle's length and velocity of contraction and therefore the individual's ability to adequately respond to balance threats [18].

In the present study, fatigue of the muscles encompassing the ankle joint resulted in a decrease in FRA of TA during gait perturbations in both experimental groups. Furthermore, increases in coactivity and mean angular velocity in the ankle joint complex were observed in young and elderly men. These results suggest that fatigue may impair an individual's ability to correct for an unexpected decelerating perturbation impulse while walking on a treadmill. Our observations are in line with similar findings of previous investigators [19]. Jackson et al. [19] investigated the effects of fatigue and habituation on the stretch reflex of the ankle musculature in response to rapid inversion perturbations in a cohort of young healthy female and male adults. Post-fatigue, the authors observed a significant decrease in reflex amplitude of the m. peroneus longus and brevis and concluded that the diminished reflex amplitude may suggest reduced dynamic stability after fatigue. In a recent study including young and older adults, Mademli et al. [7] examined the control of dynamic stability after inducing a sudden perturbation from a fixed forward inclined position before and after submaximal fatiguing knee extension contractions. The authors reported that at baseline, the older adults showed a lower margin of stability at touchdown as well as at the end of the main stance phase. Post-fatigue, no significant effects of fatigue were identified in both groups regarding the margin of stability after the forward falls. In contrast to Mademli et al. [7], we found that fatigue of the muscles encompassing the ankle joint has an effect on dynamic stability in young and elderly men. This inconsistency may be due to the rather variable methodologies applied in the two studies. Mademli et al. [7] carried out their fatigue protocol on a leg extension weight machine. Subjects performed two-legged submaximal knee extension- flexion contractions at 25% of their

maximum lifted weight until they could not lift the given weight through the whole range of motion anymore. According to Mademli et al. [7], strength of the knee extensors should be reduced by 20–25% after this protocol. In the present study, an isokinetic fatigue protocol at an angular velocity of 60°/s was applied. Thus, mode of contraction, the fatigued muscle groups, as well as the intensity of the fatigue protocol differed substantially in the two studies. It seems likely that the intensity of the fatigue protocol in the study of Mademli et al. [7] was too low to induce changes in balance recovery mechanics due to fatigue. In addition, Mademli et al. [7] examined balance recovery mechanics during forward falls from a biomechanic perspective by means of kinematic and dynamographic methods, whereas we investigated decelerating perturbation impulses from a neuromuscular perspective by means of electromyographic and goniometric methods.

In the present study, ankle fatigue diminished FRA in TA in young and elderly men. There is preliminary evidence that reflex responses contribute to joint stiffness and thus joint stability [20]. Thus, it could be argued that the observed decreases in FRA reduced joint stiffness and impaired joint stability. This seems to be supported by the fatigue-induced increase in maximal angular velocity of the ankle joint complex during the compensation of gait perturbations in young and elderly men in the current study. Thus, it can be speculated that as a result of fatigue and in an effort to compensate for fatigue, our subjects increased coactivation of the TA (agonist) and the SO (antagonist) to enhance joint stiffness and consequently joint stability. This complies with literature, since other researchers have also found increases in agonist/antagonist coactivation following fatigue [21]. Furthermore, an association between alterations in coactivation and changes in ankle stiffness have been established [20].

The observed decrease in FRA of TA following fatigue could stem from small afferents of groups III and IV reacting to the products of muscle metabolism [22]. From animal studies, it is known that these afferents project on inhibitory interneurons [23], reduce the discharge rates of gamma motoneurons and modify Renshaw inhibition [24]. Other researchers propose that the dominant inhibitory effect from group-III and IV afferents in fatigue involves a reduction in excitatory input by presynaptic inhibition of the group Ia afferents [25]. However, it has been reported that the compensation of foot displacements during gait is most likely provided by polysynaptic spinal reflexes via group-II muscle spindle afferents [26]. Thus, fatigue-induced presynaptic inhibition of the Ia terminals is unlikely to account for the observed decrease in FRA of TA during gait perturbations. Other mechanisms have to be considered. Since Rudomin and Schmidt [27] reported that presynaptic inhibition does not only affect primary afferents, but also group-II afferents, it can be speculated that the metabolically induced activity of the small myelinated and unmyelinated group-III and IV afferents causes presynaptic inhibition of group-II afferents. Therefore, this mechanism might be responsible for the observed decreases in FRA of TA following fatigue. However, future studies have to elucidate this issue.

Our second hypothesis is still unresolved. Based on literature [28,15], we speculated that the impaired ability to compensate for different perturbation impulses in old compared to young adults in an unfatigued condition can also be transferred to the fatigued condition. At baseline, we found significant differences between young and elderly men regarding FRA in TA during gait perturbations. Post-fatigue, we could not detect these differences anymore. There is controversy in literature regarding the effect of muscle fatigue on voluntary muscle activation. McNeil and Rice [29] suggest that older adults exhibit greater fatigability during isotonic contractions than young adults. Other investigators have

demonstrated similar fatigability in young and old subjects during isometric contractions [30], whereas still others have observed that older adults fatigue less than young during isometric contractions [31]. These inconsistencies may be due to the rather variable methodologies implemented to quantify fatigue. To the authors' knowledge, there is no other study available which investigated the effects of fatigue of the muscles encompassing the ankle joint on FRA during gait perturbations in young and elderly subjects. Thus, comparable data are missing. However, based on the age-related motor unit remodeling theory [32], it can be speculated that elderly subjects are more fatigue resistant than young subjects. In fact, it is reported that muscles containing a high percentage of type I fibers are more fatigue resistant and show higher reflex excitability than muscles containing high percentages of type II fibers [33]. This might explain why we did find differences at baseline, mainly caused by deficits in the proprioceptive system, but not post-fatigue.

Three potential limitations of this study warrant discussion. First, one might argue that isokinetic fatigue protocols cannot simulate functional fatigue that occurs during activities of daily living. However, the advantage of an isokinetic fatigue protocol lies in the high standard criteria level associated with this type of fatigue protocol. Furthermore, specific fatigue protocols for young and elderly adults could be applied in future studies to avoid misinterpretations of results [34]. Second, since there is contradictory information in literature regarding the impact of fatigue on reflex activity in males and females [35,36], future studies should incorporate female subjects in a similar design. Third, the question remains unanswered regarding the time frame that is necessary to return the fatigue-induced deficits in dynamic postural control to baseline values. Therefore, future studies should involve additional post-fatigue testing times to elucidate this important issue. Despite the limitations presented, results of the study provide strong evidence that isokinetic fatigue impairs the ability to compensate for gait perturbations in young and elderly men.

In summary, the use of a muscle fatigue protocol with standardized fatigue criteria has been shown to decrease FRA in TA, to increase coactivation of muscles encompassing the ankle joint complex, and to increase maximal angular velocity in plantar-dorsiflexion direction of the ankle joint during gait perturbations in young and elderly men. However, no significant differences between young and elderly subjects were detected regarding the impact of muscle fatigue on the ability to compensate for gait perturbations. These results may imply that age-related deteriorations in the postural control system do not specifically affect the ability to compensate for gait perturbations under fatigued condition.

Funding

This work was supported by LGFG Grant (Landesgraduiertenförderung), Baden-Württemberg, Germany. Our sponsor did not have any influence on the design, methods, subject recruitment, data collections, analysis and preparation of this paper. The endorsement involved a scholarship (monthly salary) for the corresponding author from a state program.

Conflict of interest statement

None of the authors have any conflicts of interest.

Acknowledgment

The authors would like to thank Dr. Phil. Harald Seelig for his statistical advice.

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