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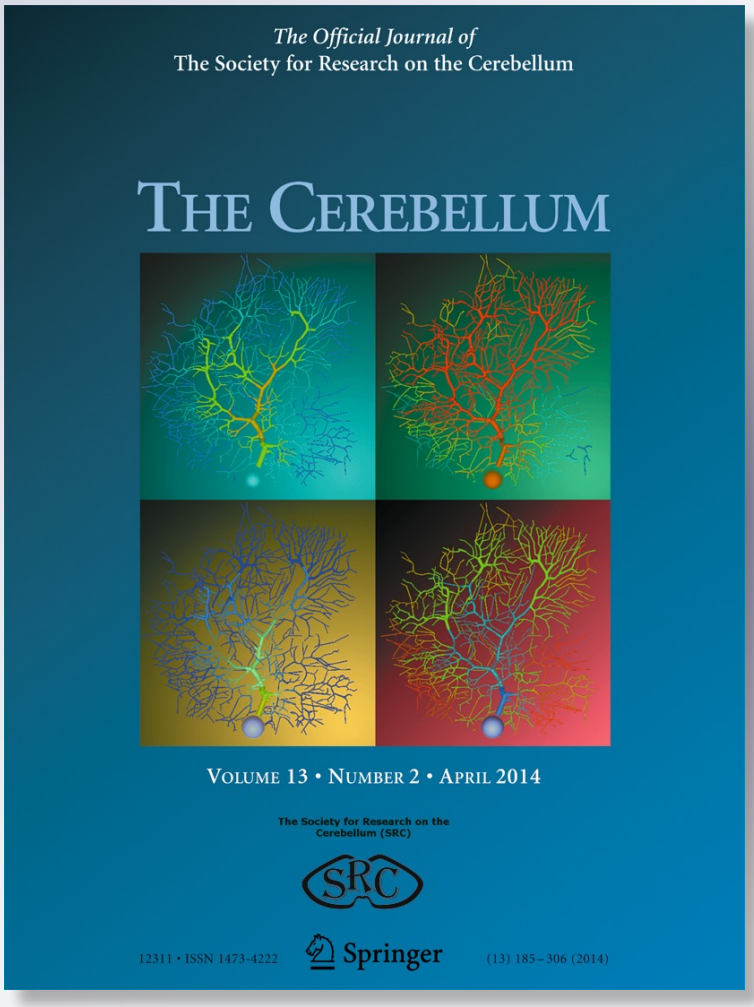

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ORIGINAL PAPER

Lower Limb Antagonist Muscle Co-Activation and its Relationship with Gait Parameters in Cerebellar Ataxia

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Abstract Increased antagonist muscle co-activation, seen in motor-impaired individuals, is an attempt by the neuromuscular system to provide mechanical stability by stiffening joints. The aim of this study was to investigate the co-activation pattern of the antagonist muscles of the ankle and knee joints during walking in patients with cerebellar ataxia, a neurological disease that strongly affects stability. Kinematic and electromyographic parameters of gait were recorded in 17 patients and 17 controls. Ankle and knee antagonist muscle co-activation indexes were measured throughout the gait cycle and during the sub-phases of gait. The indexes of ataxic patients were compared with those of controls and correlated with clinical and gait variables. Patients showed increased co-activity indexes of both ankle and knee muscles during the gait cycle as well as during the gait sub-phases. Both knee and ankle muscle co-activation indexes were positively correlated with disease severity, while ankle muscle co-activation was also positively correlated with stance and swing duration variability. Significant negative correlations were observed between the number of self-reported falls per year and knee muscle co-activation. The increased co-activation observed in these cerebellar ataxia patients may represent a compensatory strategy serving to reduce gait instability. Indeed, this mechanism allows patients to reduce the occurrence of falls. The need for this strategy, which results in excessive muscle co-contraction, increased metabolic costs and cartilage degeneration processes, could conceivably be overcome through the use of supportive braces specially designed to provide greater joint stability.

Keywords Co-activation · Gait · Cerebellar Ataxia · Antagonist Muscles · Stability

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Introduction

Antagonist muscle co-activation is an important component of motor control [1]. Indeed, simultaneous co-contraction of antagonist muscles, by stiffening joints, produces upper limb stability during the execution of tasks requiring positional accuracy [2] and reduces lower limb instability during walking [3–5]. However, excessive and/or prolonged co-contraction, particularly during walking, can impair functional performance and carries an increased metabolic cost [5, 6]. Moreover, since muscle forces are the greatest contributors to joint loads during weight-bearing activities, the presence of abnormal co-activation may also result in increased forces

within joints, which may, in turn, lead to cartilage degeneration [7].

Elderly people, as well as motor-impaired patients have been found to show high levels of muscle co-activation in knee and in ankle joints [5, 8–11]. Increased muscle co-contraction in patients with neurological disorders, such as Parkinson's disease [12], can be interpreted in two ways: as the primary deficit due to impaired reciprocal inhibition [13] and/or as an attempt to reduce instability in the lower limbs during walking (i.e., a compensatory mechanism) [12]. One of the neurological diseases that impact most on locomotion is cerebellar ataxia, which is typically characterised by irregular and unstable gait and a high variability of all time–distance parameters [14–17], alterations known to be closely linked to a high risk of falls [18]. Moreover, patients with cerebellar ataxia typically have reduced muscle tone (hypotonia) and severely impaired inter-joint coordination [17, 19]. All these alterations suggest that ataxic patients, to obtain better control of dynamic stability and multi-joint coordination, may have a real need to reduce their degrees of freedom of movement. Thus, in these patients, increased co-contraction of antagonist muscles could be interpreted as a compensatory strategy rather than a primary deficit of the neuromuscular system. This strategy may be related to variability of the step parameters, as well as to disease severity and rate of falls. In order to test these hypotheses, we analysed antagonist muscle co-activation, variability of step parameters and lower limb joint ranges of motion in patients with cerebellar ataxia during walking. Moreover, to explore the issue of co-contraction in greater depth, we analysed the temporal and amplitude features of the myoelectric activity of the single muscles. Data shedding light on this aspect, currently lacking in the literature, would be interesting and useful, particularly with a view to developing rehabilitation strategies, mechanical devices, or footwear that may provide a greater joint stability, thus reducing the need for excessive antagonist muscle co-activation and preventing joint degradation.

Materials and Methods

Subjects

Seventeen patients (13 males, 4 females; mean age 51.4± 10.9 years) non-consecutively admitted to a outpatient clinic (Centre for Neurogenetic Disorders, University of Rome "Sapienza", Polo Pontino, Latina) were enrolled (see Table 1). Nine had a diagnosis of autosomal dominant ataxia (spinocerebellar ataxia (SCA); five patients with SCA1 and four patients with SCA2), while the other eight had sporadic adult-onset ataxia of unknown aetiology (SAOA). As patients with SCA and SAOA may present impairment of other systems, we included only those who had, clinically, an almost 'pure' form of ataxia. We excluded patients with major involvement of neurological systems other than the cerebellar one and/or orthopaedic disorders also liable to cause gait impairment (two patients with SCA, one had not been enrolled). No patient had visual impairment, while almost all had oculomotor abnormalities such as gaze nystagmus or square-wave jerks during pursuit movements. All the patients showed cerebellar atrophy on MRI. All could walk without assistance or walking aids.

Almost all patients enrolled were undergoing physical therapy during the study, which included upper and lower limb exercises and balance and gait training. Two patients stopped doing physical therapy between 3 and 6 months before the study.

Disease severity was rated using the International Cooperative Ataxia Rating Scale (ICARS) [20]. Since gait and posture are balance-related, ICARS items and limb kinetics is a coordination-related ICARS item [21], we combined the gait and posture scores to obtain an indicator of balance deficit and used the lower limb kinetics score as an indicator of coordination deficit (Table 1).

Seventeen age-matched, healthy adults (14 males and 3 females; mean age 51.6±11.2 years) were recruited as controls. The two groups had similar heights (H) and masses (M) (H: ataxic patients 167.6±8.0 cm, controls 167.8±7.4 cm, $p>0.05$; M: ataxic patients 70.5±13.3 kg, controls 69.3± 7.8 kg, $p>0.05$). All the participants gave their informed consent prior to taking part in the study, which complied with the Helsinki Declaration and had local ethics committee approval.

Gait Analysis

An optoelectronic motion analysis system (SMART-D System, BTS, Italy) consisting of eight infrared cameras (300 Hz) was used to detect the movements of 22 reflective spherical markers placed over anatomical landmarks according to Davis et al. [22]. Anthropometric data were collected for each subject [23].

Surface myoelectric signals were acquired at a sampling rate of 1,000 Hz, using a 16-channel Wi-Fi transmission surface electromyograph (FreeEMG300 System, BTS, Milan, Italy). A pre-processing filtering and denoising procedure was performed. The lower and upper cut-off frequencies of the Hamming filter were 10 and 400 Hz, respectively, and the common mode rejection ratio was 100 dB. After skin preparation, bipolar Ag/AgCl surface electrodes (H124SG, Kendall ARBO, Donau, Germany), prepared with electroconductive gel, were placed over the muscle belly in the direction of the muscle fibres according to the European Recommendations for Surface Electromyography (SENIAM) [24] and the atlas of muscle innervation zones [25].

Table 1 Patients' characteristics

Patient	Age (years)	Gender	Diagnosis	Onset (years)	Duration (years)		ICARS			
						Falls 1 year	Gait	Posture	Lower limb kinetics	Total
1	44	M	SCA2	35	9	8	4	8	5	21
2	54	F	SAOA	40	14	20	5	10	8	30
3	69	F	SAOA	60	9	2	3	3	1	11
4	48	M	SAOA	30	18	1	1	0	1	6
5	65	F	SAOA	62	3	1	3	5	0	12
6	65	M	SAOA	60	5	1	3	6	2	17
7	45	M	SAOA	30	15	15	4	9	7	26
8	32	M	SCA1	30	2	0	2	4	0	7
9	46	M	SAOA	17	29	0	4	5	2	18
10	41	M	SCA1	35	6	2	1	1	1	6
11	57	M	SAOA	47	10	0	3	4	4	12
12	37	M	SCA2	30	7	0	3	6	2	20
13	69	M	SCA2	25	44	0	7	6	1	27
14	49	M	SCA1	41	8	0	4	5	2	18
15	56	F	SCA2	43	13	1	9	15	4	39
16	53	M	SCA1	40	13	5	5	6	3	26
17	46	M	SCA1	27	19	0	3	3	3	21

The bipolar electrodes were placed bilaterally on the vastus lateralis (VL), biceps femoris (BF), tibialis anterior (TA) and gastrocnemius medialis (GM) muscles.

Patients and controls were required to walk barefoot at self-selected speed along a walkway approximately 10 m in length. Assuming that this speed would be slower in the patients, we instructed the controls to walk barefoot at low speeds, too. In this way the parameters could be compared between the groups without the potential velocity bias (see 'Speed Matching Procedure' for details). Before formal measurements were started, practice sessions were performed to familiarise the participants with the procedure. Then, six walking trials were acquired per subject. To ensure that gait parameters were collected during steady-state walking, the first and last two steps of each trial, corresponding to the acceleration and deceleration phase, were excluded from the analysis. To avoid fatigue, groups of three trials were separated by a 1-min rest.

Speed Matching Procedure

The speed was matched between groups as follows: for each control group subject, we considered only those trials in which their gait velocity fell within the range identified by the ataxic patients' mean gait speed ± SD. On this basis, we selected two controls walking at self-selected speed and 15 controls walking at low speed. In this way, the mean speed values were not statistically different between groups (ataxic patients 0.93 ±0.28 m/s; controls 0.94±0.21 m/s; $p>0.05$ on Mann–Whitney test).

Data Analysis

Three-dimensional marker trajectories were acquired using a frame-by-frame tracking system (SMART Tracker - BTS, Milan, Italy). Data were processed using 3-D reconstruction software (SMART Analyzer, BTS, Milan, Italy) and MATLAB software (MATLAB 7.4.0, MathWorks, Natick, MA, USA). Kinematic and electromyographic (EMG) data were normalised between two consecutive heel strikes and were analysed considering the right and left limb together.

Variability of Time–Distance Parameters

To obtain an index of intra-subject variability, we calculated the coefficient of variation (CV) as the ratio between the standard deviation and the mean values of the following parameters: step length, step width, cycle duration, and stance, swing and double-support phase percentage durations.

Joint Kinematic Parameters

To assess the lower limb joint kinematics, we ascertained the hip, knee, and ankle joint centres of rotation and calculated the following angular ranges of motion (ROMs) in the sagittal plane during the gait cycle: (1) hip flexion/extension (H_fe);

(2) knee flexion/extension (K_fe); (3) ankle flexion/extension (A_fe).

EMG Processing

The raw EMG signals were processed by subtracting their average value; they were then full-wave rectified and filtered with a 10-Hz LP Butterworth filter. Signals were time normalised to 100 samples with respect to the gait cycle duration.

Antagonist Co-Activity Index EMG amplitude was expressed as a percentage of the peak EMG of the intra-individual ensemble average (peak dynamic method) [26]. We refer to the EMG processed in this way as "muscle activity" (a). Thereafter, VL-BF and TA-GM co-activation (CA) values were then determined sample by sample using the following formula modified from the one proposed by Rudolph et al. [27, 28]:

$$\mathrm{CA} = \left[(a_\mathrm{H} + a_\mathrm{L})/2\right] * \left(a_\mathrm{L}/a_\mathrm{H}\right)$$

where a_H and a_L represent the highest and the lowest activity between the two antagonist muscles.

A co-activity index (CAI) for the following four sub-phases of the gait cycle was then obtained by calculating the mean values of the co-activity level in the corresponding cycle windows: first double-support (DS1), single-support (SS), second double-support (DS2) and swing (SW). In order to have a global measure of the co-activity level, the CAI was also evaluated during the entire gait cycle.

Data from the six trials considered were averaged to obtain each subject's mean value.

EMG Amplitude and Timing Parameters In order to investigate the temporal and amplitude features of the myoelectric activity of the single muscles, we measured the following EMG parameters within the gait cycle: (1) peak value, calculated as the maximum value of the filtered EMG signal; (2) full-width at half-maximum (FWHM), i.e. the measure of the width of the peak at the half height position; (3) peak event, detected as the occurrence of the maximum within the gait cycle; and (4) centre of activity [29], i.e. the weighted average of the gait samples, where the 'weights' are the filtered EMG values. Since the peak event and the centre of activity can cross the boundary of a gait cycle, the 0–100 % scale of the gait cycle was transformed into a 0–360 ° angular scale, with 0, 180 and 360 ° corresponding to 0, 50 and 100 % of the gait cycle respectively [30]. Thus, the centre of activity was calculated by integrating the formula adopted by Labini et al. [29] with the circular transformation, obtaining the following expression:

$$coa = \tan^{-1}\left(\frac{\Sigma_{i=0}^{100} EMG_i \cdot \sin\theta_i}{\Sigma_{i=0}^{100} EMG_i \cdot \cos\theta_i}\right)$$

where θ_i is the i-th sample of the 0–100 % gait cycle scale transformed into a 0–360 ° angular scale. This allowed us to use circular statistics tools [31] to describe the timing characteristics of EMG signals (see below).

Statistical Analysis

All the analyses were performed using SPSS 17.0 software (SPSS Inc. Chicago, IL, USA). The Kolmogorov–Smirnov test was used to analyse the normal distribution of the data. The parametric Student t test was adopted to investigate between-groups differences in the variability of time–distance parameters, joint ROMs, EMG amplitude and co-contraction parameters. The Watson–Williams test [32] for circular data was used to investigate between-groups differences in EMG timing parameters.

The Spearman test was used to investigate correlations between the CAIs and the ICARS scores (i.e. gait and posture scores combined and lower limb kinetics score). Partial correlations were used to analyse correlations between the CAIs and disease onset and duration, self-reported number of falls per year, CV, and ROMs controlling for the influence of disease severity (balance- and coordination-related ICARS scores).

A p value of less than 0.05 was considered statistically significant. When multiple comparisons were made, the Bonferroni correction was used.

Results

Variability of Time–Distance Parameters

The patients showed significantly higher CV of step length ($p=0.001$), step width ($p=0.002$), and stance ($p=0.015$) and swing ($p=0.016$) percentage durations compared with the controls (Fig. 1).

Joint Kinematic Parameters

A significantly reduced ankle flexion/extension ROM was observed in the patients compared with the controls (ataxic patients 19.0±5.8 °, controls 26.4±4.7 °, $p<0.001$). Hip and knee flexion/extension ROMs were not significantly different between the groups (H_fe: ataxic patients 40.9±6.5 °, controls 40.3±4.7 °, $p=0.761$; K_fe: ataxic patients 55.3±7.9 °, controls 55.4±5.3 °, $p=0.956$). Figure 2 shows the mean joint

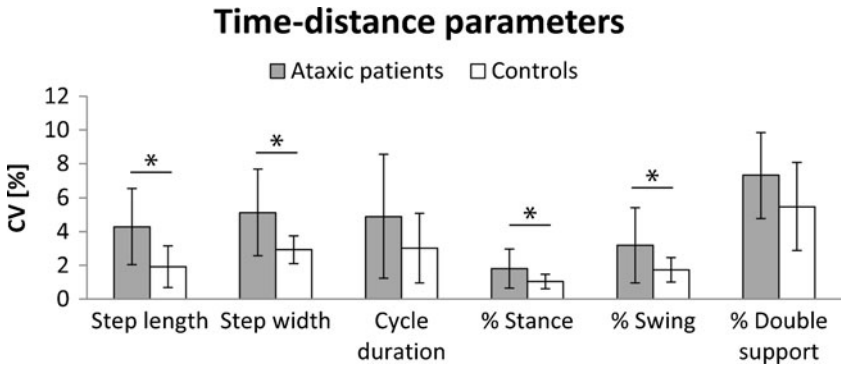

Fig. 1 Coefficient of variation of the time–distance parameters in patients and controls. *Significant differences

kinematics traces of a representative ataxic patient and a control subject.

Antagonist Muscle Co-Activation

Figure 3 shows the mean muscle activity traces of a representative ataxic patient and a control subject. The ataxic patients showed significantly higher co-activation index values throughout the gait cycle both for the TA-GM (ataxic patients 15.78±4.87, controls 10.23±3.06; $p<0.001$) and the VL-BF (ataxic patients 18.20±6.20, controls 13.83±4.15; $p=0.02$) pairs of antagonist muscles.

Analysis of the co-activation indexes in the different gait sub-phases revealed significantly higher values in the ataxic patients, compared with the controls, in the first double-support ($p<0.001$), single-support ($p=0.002$), second double-support ($p=0.002$) and swing ($p=0.004$) phases for the TA-GM muscles and in the first double-support ($p<0.001$), single-support ($p<0.001$) and swing ($p=0.004$) phases for the VL-BF muscles (Fig. 4). In Fig. 5, the co-activation of ankle and knee joint antagonist muscles is plotted during the gait cycle.

EMG Amplitude and Timing Parameters

As illustrated in Fig. 6, the ataxic patients, compared with the controls, showed significantly higher peak values for the VL ($p<0.001$), BF ($p<0.001$) and TA ($p=0.001$) muscles. Moreover, the FWHM was significantly greater in the patients than in the controls for all the investigated muscles (all, $p<0.001$). As regards to the timing parameters, the BF peak event ($p=0.001$, see Fig. 7) and the VL and BF centres of activity (VL $p=0.021$; BF $p=0.003$, see Fig. 8) were significantly delayed in the patients compared with the controls.

Correlations

With regard to the clinical variables, significant positive correlations were found between the onset of disease and $VL\text{-}BF_{DS2}$, between the balance-related ICARS items

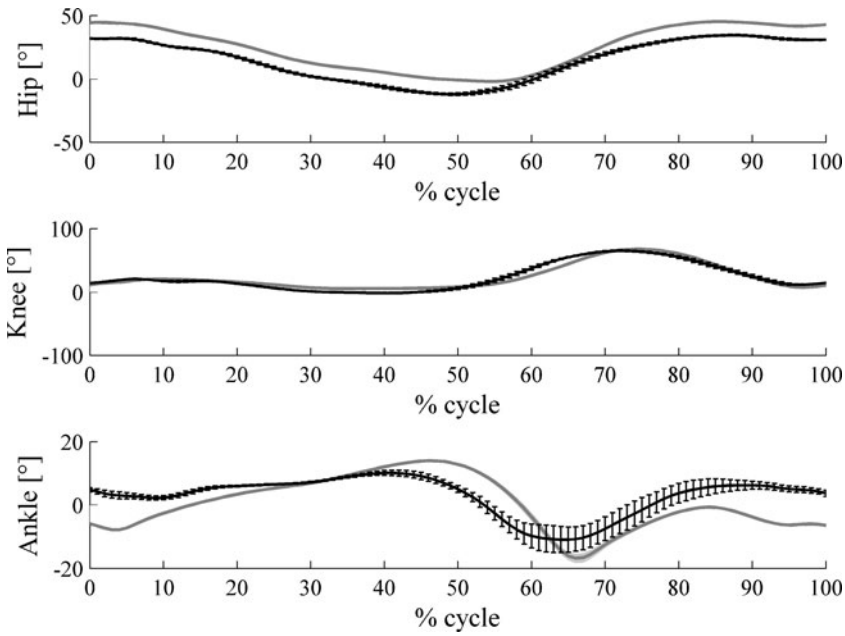

Fig. 2 Mean kinematic plot of lower limb joint angular displacement in the sagittal plane during the gait cycle of a representative ataxic patient and a control subject. The *grey curve* refers to the control subject and the *black curve* to the ataxic patient

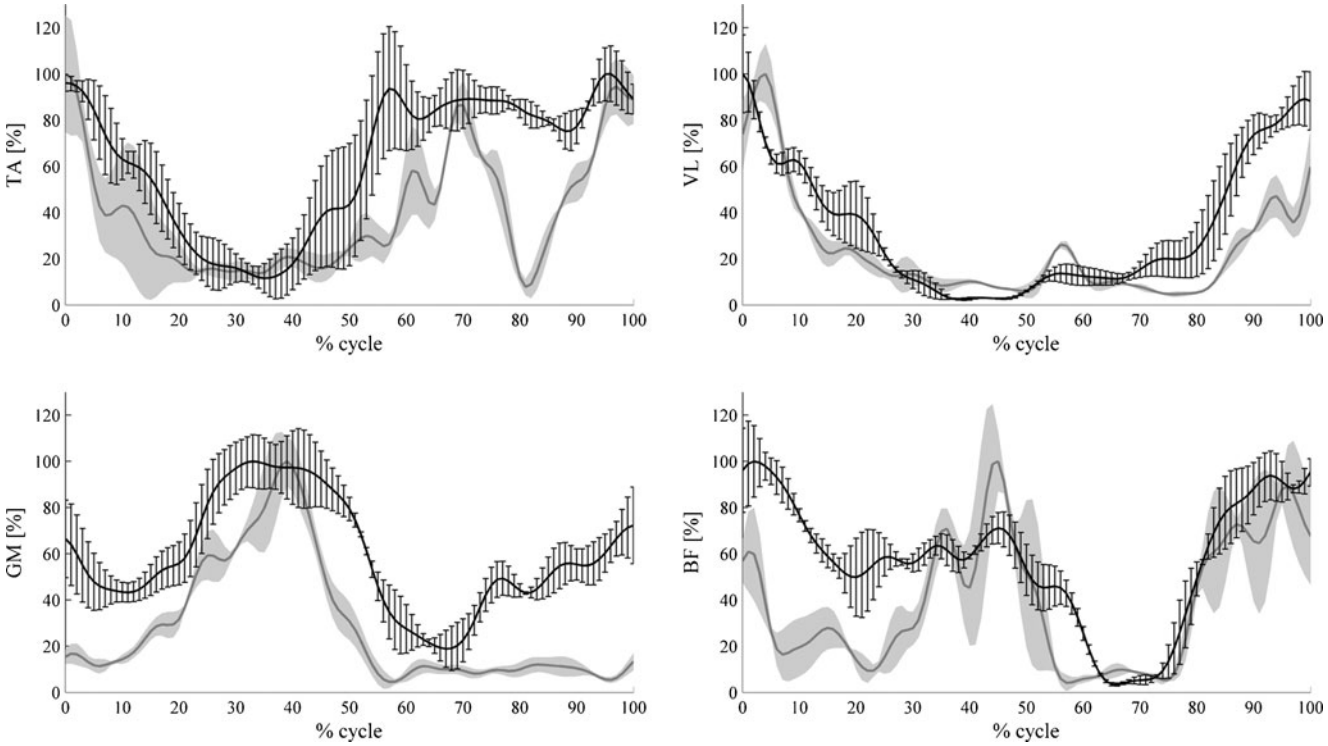

Fig. 3 Mean muscle activity traces of a representative ataxic patient and a control subject. The *grey curve* refers to the control subject and the *black one* to the ataxic patient

(combined score) and TA-GM_{SS}, TA-GM_{DS2}, VL-BF_{SS}, between the lower limb coordination-related ICARS score and TA-GM_{SS} and VL-BF_{SS}, and between the ICARS total score and TA-GM_{SS}, TA-GM_{DS2} and VL-BF_{SS}. Significant negative correlations were observed between the number of self-reported falls per year and VL-BF_{DS1} and VL-BF_{SS} (Table 2).

With regard to the CV values, significant positive partial correlations were found between the CV of stance percentage and TA-GM_{DS1} ($R=0.533$, $p=0.033$), TA-GM_{SS} ($R=0.691$, $p=0.004$), TA-GM_{SW} ($R=0.533$, $p=0.032$), and between the CV of % swing and TA-GM_{SS} ($R=0.636$, $p=0.011$) and TA-GM_{SW} ($R=0.550$, $p=0.033$). Moreover, significant negative partial correlations were found between TA-GM_{DS1} and both knee and ankle ROM (knee: $R=-0.522$, $p=0.046$; ankle: $R=-0.521$, $p=0.047$). Similarly, significant negative partial correlations were found between TA-GM_{SS} and both knee and ankle ROM (knee: $R=-0.577$, $p=0.024$; ankle: $R=-0.698$, $p=0.004$). No other significant correlations were found.

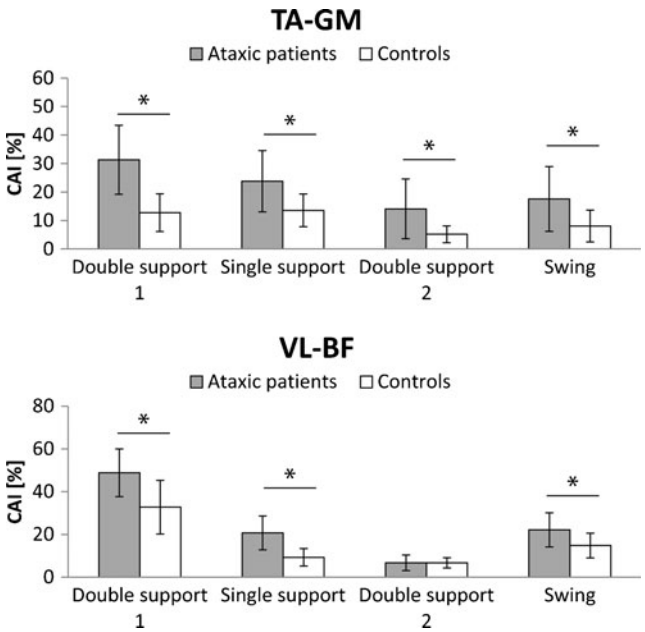

Fig. 4 CAI of the ankle and knee antagonist muscles calculated in the four sub-phases of the gait cycle in patients and controls. *Significant differences

Discussion

To our knowledge, this is the first study that investigates the co-contraction of lower limb joint antagonist muscles during gait in patients affected by cerebellar ataxia.

Our main findings can be summarised as follows: (1) knee and ankle joint muscle co-activation is increased in ataxic patients compared with controls; (2) increased antagonist muscle co-activation is associated with a significantly wider

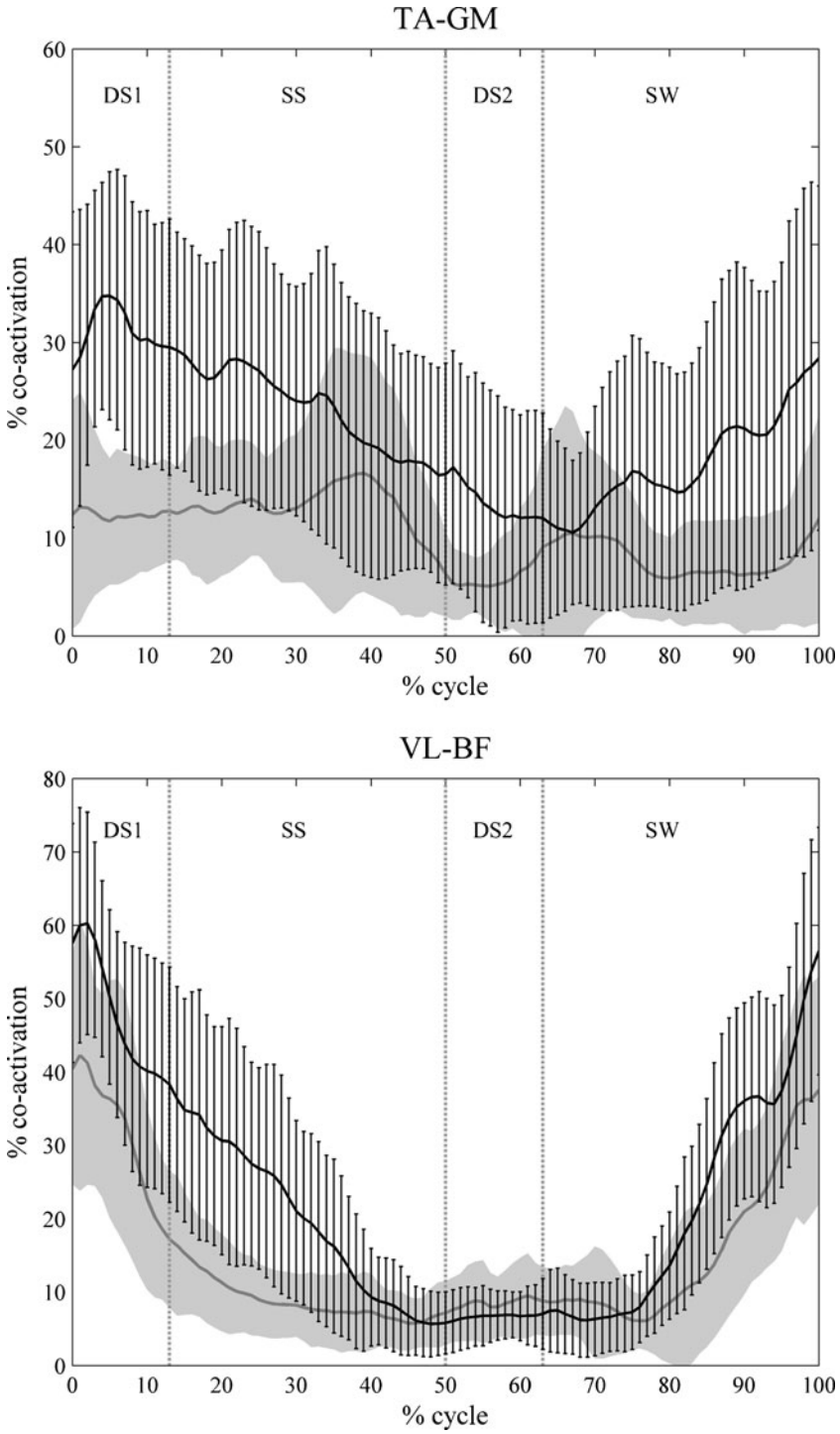

Fig. 5 Co-activation of ankle and knee joint antagonist muscles during the gait cycle. The *grey curve* refers to the controls and the *black curve* to the ataxic patients

and higher EMG peak for all the muscles and with a delay of BF muscle activity; (3) the knee and ankle co-contraction pattern is correlated with disease severity (ICARS scores); (4) the higher the co-contraction index of knee joint muscles during the first double-support and single-support sub-phases, the lower the number of falls per year in ataxic patients.

In general terms, antagonist muscle co-activation during walking constitutes an attempt by the neuromuscular system to provide mechanical stability by stiffening joints [6]. An abnormal co-contraction pattern has been demonstrated in categories of people who have a great need for active muscular stabilisation, such as the elderly [5], individuals who have undergone knee arthroplasty [11], patients with stroke or traumatic brain injury [10], and patients with Parkinson's disease [13]. Increased antagonist co-activity during the gait cycle, observed in our patients for both the knee and ankle

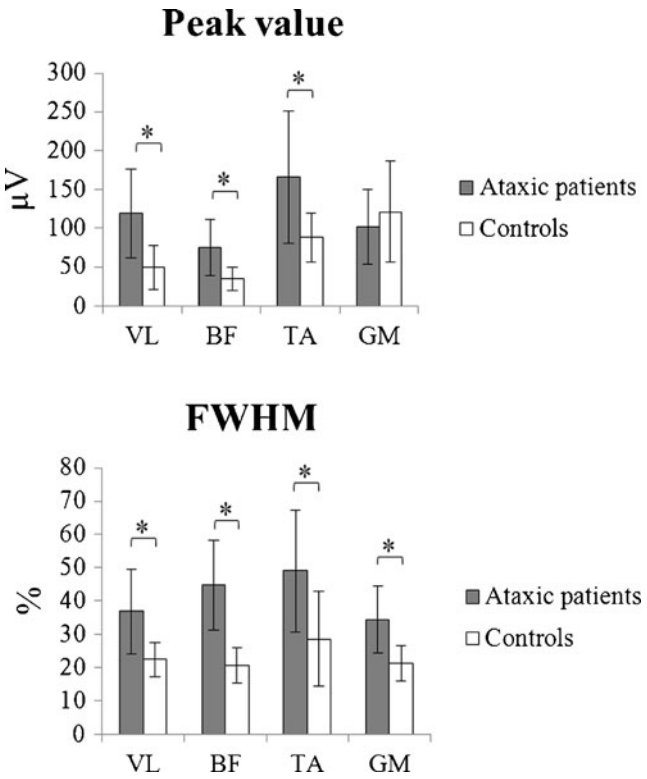

Fig. 6 Peak values and FWHM of the four considered muscles in patients and controls. *Significant differences

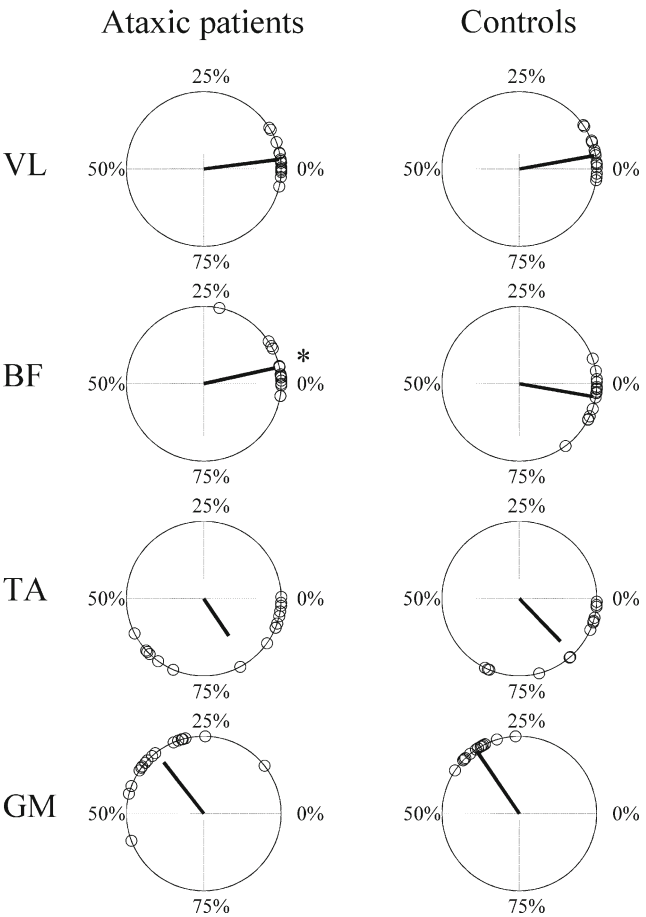

Fig. 7 EMG peak event in ataxic patients and controls. Each *dot* on the circle represents the subject's peak event expressed as a percentage of the normalised gait cycle. The *vector* is the mean resultant vector; it indicates the mean direction of the dots and its *length* is an indicator of the spread of the dots. *Between-groups significant differences

joint muscles, may be a consequence of a lack of balance and/or of impaired joint coordination due to cerebellar degeneration. Indeed, we observed that ataxic patients, compared with healthy subjects, needed to activate antagonist muscles more and for longer, possibly in an attempt to compensate for the instability due to the lack of muscle coordination. On analysing the CAI in the sub-phases of the gait cycle, we found that the ankle and knee joint muscles were more co-contracted in patients than in controls during all four gait sub-phases. On analysing the individual muscles of the knee joint, we observed that the peak event and the centre of activity of the BF muscle moved from 90–100 % (in controls) to 0–10 % of the gait cycle in the patients, i.e., towards the loading response. This indicates that the BF muscle was most active in the early phase of the gait cycle in the patients, and that its activity thus overlapped the abnormally prolonged VL muscle activity (Figs. 7 and 8). This increased co-activation during the first double-support phase suggests that ataxic patients stiffen the knee joint in order to compensate for the instability created by the load transfer from one limb to the other.

As regards to the single-support phase, which is a particularly unstable postural configuration due to the fact that the bodyweight is supported by one limb, when plotting the co-contraction function as continuous variable (Fig. 5) it emerged that, in this phase, the ataxic patients' co-activation values were highest during mid-stance (10–30 %). This co-contraction pattern is the result of the abnormally increased activity observed in the TA muscle (Fig. 6), which overlaps the activity of the GM muscle in this phase. This finding strongly indicates that ataxic patients need to increase their stability during the forward progression that accompanies the loading of their bodyweight onto a single leg. For this reason, they may stiffen their lower limbs in order to limit and control the forward acceleration of the body, acting mainly on the ankle and knee joints in this phase.

We also observed an increase in ankle and knee joint antagonist muscle co-activation in patients, compared with controls, during the swing phase. The plot of the co-contraction functions during the gait cycle (Fig. 5) shows that this increase is particularly pronounced in the late swing phase, suggesting that the joints may possibly be stiffened in advance to prevent the instability induced by the mechanical perturbation in the stance phase. As regards to the knee joint, this interpretation fits well with the observed abnormal enlargement of the FWHM in the VL muscle, whose activity

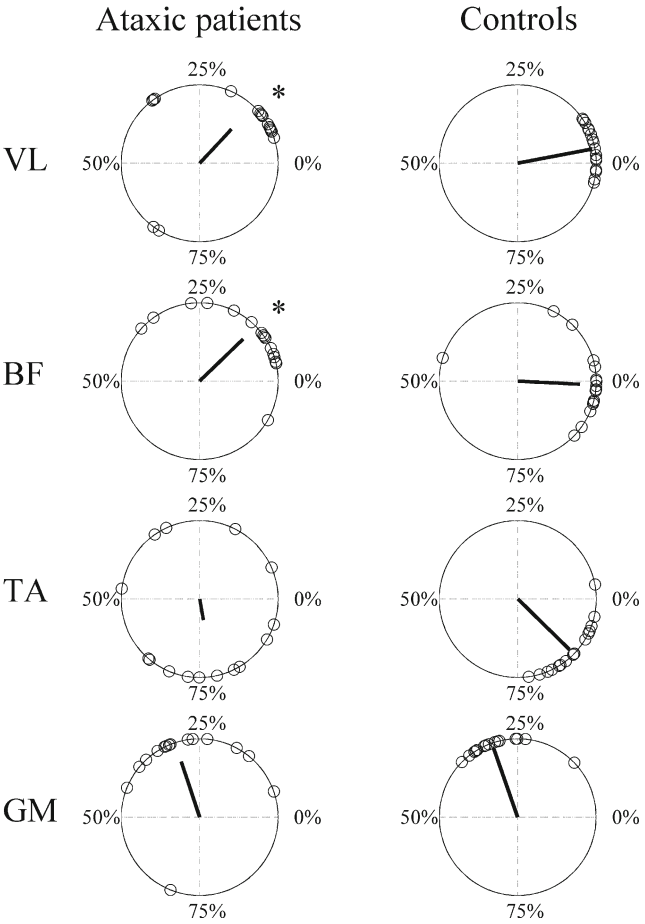

Fig. 8 EMG centre of activity in ataxic patients and controls. Each *dot* on the circle represents the subject's centre of activity expressed as a percentage of the normalised gait cycle. The *vector* is the mean resultant vector; it indicates the mean direction of the dots and its *length* is an indicator of the spread of the dots. *Between-groups significant differences

Table 2 Significant correlations (with p values) between the clinical variables and the co-activity indexes

Clinical variable	CAI
Onset*	VL-BF_{DS2} 0.690 (0.004)
Duration*	NS
ICARS gait + posture (balance)	TA-GM_{SS} 0.694 (0.002)
	TA-GM_{DS2} 0.491 (0.045)
	VL-BF_{SS} 0.649 (0.005)
ICARS lower limb (coordination)	TA-GM_{SS} 0.599 (0.011)
	VL-BF_{SS} 0.706 (0.002)
ICARS total	TA-GM_{SS} 0.771 (<0.001)
	TA-GM_{DS2} 0.654 (0.004)
	VL-BF_{SS} 0.638 (0.006)
Falls in 1 year*	VL-BF_{DS1} −0.552 (0.033)
	VL-BF_{SS} −0.590 (0.021)

*Partial correlation

NS no significant correlation

starts in the late swing phase and lasts beyond its expected termination (i.e., until mid-stance); as a result of this, its centre of activity is delayed (Figs. 6, 7, and 8).

Given the lack of muscle coordination in ataxic patients, it could be claimed that the co-activation pattern observed in our study is merely the result of random superimposition of highly variable individual patterns of muscle activation. However, analysis of the activity of the individual muscles showed a marked enlargement of the EMG peaks in all the muscles. This behaviour may be interpreted, at least in part, as an effort to stiffen the whole limb as compensatory mechanism due to the lack of inter-joint coordination. However, further studies should aim to discriminate, within this abnormally enlarged EMG activity, the component related to the primary coordination deficit from the compensatory mechanism.

Interestingly, in our study, of all the muscles considered, the TA was the one that showed the highest and widest peak values across the whole gait cycle. We speculated that this may be interpreted in terms of an attempt to maintain balance in the sagittal plane by forward displacing the centre of mass and thus avoiding backward instability and the risk of falling backwards. However, this interpretation, which needs further investigation, goes beyond the scope of the present study.

In this study, we found that the abovementioned increased co-activation pattern observed in ataxic patients was related to disease severity (Table 2), i.e., the more severe the disease, the more co-contracted the knee and ankle antagonist muscles.

As previously documented [14, 17], the ataxic patients we studied showed a significantly increased variability of the time–distance parameters compared to the controls. This high variability is an indication of these patients' inability to maintain dynamic balance through a regular walking pattern and has been proved to be a risk factor for falls [18]. We found a significant positive partial correlation between stance and swing duration variability and TA-GM co-activation during the DS1, SS, and SW sub-phases. This result suggests that gait variability, per se, induces increased co-contraction of the ankle muscles, which is possibly a compensatory attempt to reduce the irregularity of gait. This result, together with the negative correlation found between ankle joint muscle co-activation and both ankle and knee joint ROMs, as well as the reduced ankle joint ROM (Fig. 2), clearly suggests that ankle joint stiffening plays a key role in the ataxic patient's attempt to increase gait stability, even at the expense of an efficient ankle movement [33].

In our study, we found a negative correlation between the number of falls per year and the CAI of the knee joint muscles during the single support sub-phase. This result, showing that higher levels of co-activation are linked to a lower occurrence of falls, indicate that knee muscle co-contraction may be an effective compensatory mechanism to reduce the risk of falls caused by a lack of balance and limb coordination. An alternative interpretation of the results could be that the

observed increased antagonist co-activation is a primary deficit, rather than compensation, of cerebellar ataxia disease; and the positive correlation between co-activity indexes and disease severity could be considered as supporting this hypothesis. However, the negative correlation between CAI and falls incidence seems not to support this hypothesis, suggesting instead a compensatory role for co-activation. Moreover, it should be considered that an increased co-activation is not a distinctive feature of ataxic gait, but it has been observed also in patients with orthopaedic disorders [11], in which co-activation appeared to be a mechanism aimed to reduce variability and increase stability. In addition, co-activation is used also by healthy subjects to regulate limb stiffness in tasks requiring arm positional accuracy [2]. In the light of the above mentioned considerations, we believe that the most plausible view is that the ataxic patients increase their muscles co-activation to stiffen the limbs in the attempt of reducing instability.

In our study, the EMG timing parameters were reported as circular data to handle bursts of muscle activity that cross the end of a gait cycle. In literature, a reliability analysis has been performed only on the peak and events amplitudes [34], whereas no data are available regarding the centre of activity and co-activation indexes. This would be an area of recommendation for future study.

Conclusions

Taken together, our data suggest that ataxic patients adopt a co-contraction-based strategy in order to reduce the dynamic instability due to the loss of cerebellar functions. This strategy is increasingly more pronounced as the disease progresses and results in a substantial and beneficial decrease in the occurrence of falls. However, abnormal co-activation of antagonist muscles carries some negative effects, such as the increased metabolic cost [5, 6] and the risk of cartilage degeneration [7]. Moreover, increased antagonist muscle co-contraction may affect motor performances. In the light of the above considerations, it would be useful to design and develop orthoses or braces that may provide external joint stability and thus reduce the need for excessive muscle co-contraction. Such aids should be made using special flexible materials that provide joint stability without affecting motor performance. This may reduce the abovementioned energy cost, risk of joint degeneration and occurrence of falls. Further studies are warranted to investigate these hypotheses.

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