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DIFFERENCES IN HUMAN ANTAGONISTIC ANKLE DORSIFLEXOR COACTIVATION BETWEEN LEGS; CAN THEY EXPLAIN THE MOMENT DEFICIT IN THE WEAKER PLANTARFLEXOR LEG?

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SUMMARY

The present study examined the hypothesis that the antagonistic ankle dorsiflexor coactivation level during maximum isometric voluntary plantarflexion (MVC) is a function of ankle angle. Six male subjects generated plantarflexion and dorsiflexion MVC trials at ankle angles of −15 deg (dorsiflexed direction), 0 deg (neutral position), +15 deg (plantarflexed direction) and +30 deg having the knee flexed at an angle of 90 deg. In all contractions surface EMG measurements were taken from tibialis anterior and soleus which were considered representative muscles of all dorsiflexors and plantarflexors, respectively. Antagonistic dorsiflexor coactivation was expressed as normalized EMG and moment. Calculations of the antagonistic dorsiflexor moment were based on the tibialis anterior EMG–dorsiflexor moment relationship from contractions at 50, 40, 30, 20 and 10 % of the dorsiflexion MVC moment. In both legs dorsiflexor coactivation level followed an open U-shaped pattern as a function of ankle angle. Differences of 9 and 14 % ($P < 0{\cdot}05$) were found in the measured net plantarflexion MVC moment between legs at ankle angles of −15 and +30 deg, respectively. No difference ($P > 0{\cdot}05$) was found in the calf circumference between legs. Differences were found in the antagonistic dorsiflexor coactivation between legs at ankle angles of −15 and +30 deg. In the weaker leg the antagonistic EMG measurements were higher by 100 and 45 % ($P < 0{\cdot}01$) and the estimated antagonistic moments were higher by 70 and 43 % ($P < 0{\cdot}01$) compared with the weaker leg at −15 and +30 deg, respectively. This finding was associated with a decreased range of motion (ROM) in the weaker leg (14 %, $P < 0{\cdot}01$), such that no difference ($P > 0{\cdot}05$) was found in dorsiflexor antagonistic coactivation between legs at end-range ankle angles. The findings of the study (i) have to be taken into consideration when estimating musculoskeletal loads in the lower extremity, (ii) imply that stretching training can result in a stronger plantarflexion at end-range ankle angles through inhibition of the dorsiflexors, and (iii) imply a neural drive inadequacy during a plantarflexion MVC at end-range angles.

INTRODUCTION

Musculotendon units control movement by exerting forces resulting in joint moments. The moment generating capacity around a joint is experimentally quantified using dynamometers on which the agonist muscle group can be tested under dynamic or static conditions. Although the dynamometer moment reading is traditionally used for quantifying the agonists' moment generating capacity, coactivation of antagonists can complicate interpretation of results essential for evaluating the effectiveness of an exercise stimulus in terms of athletic training or

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rehabilitation programmes. Coactivation of the antagonist during a contraction of the agonists results in a negative moment in relation to the moment developed by agonists, reducing the net resultant moment output. Antagonistic coactivation during an exercise task depends (i) on the training background of the subject (Baratta, Solomonow, Zhou, Letson, Chuinard & D'Ambrosia, 1988; Ostering, Caster & James, 1995); (ii) the type of contraction involved in the task (Snow, Cooper, Quanbury & Anderson, 1993, 1995; Kellis & Baltzopoulos, 1996, 1997); (iii) the contraction intensity (Solomonow, Baratta, Zhou & D'Ambrosia, 1988; Grabiner & Weiker, 1993) and velocity (Snow *et al*. 1993; Kellis & Baltzopoulos, 1996); (iv) the joint tested (Solomonow *et al*. 1988; Eloranta, 1989); (v) the muscles involved (Snow *et al*. 1993, 1995); and (vi) the joint angle at which testing is done (Solomonow *et al*. 1988; Eloranta, 1989). Coactivation can decrease as a function of skill, practice and co-ordination (Person, 1958; Patton & Motensen, 1971; Solomonow *et al*. 1988; Carolan & Cafarelli, 1992; Amiridis *et al*. 1996). A consequence of the latter could be that on the dominant limb less antagonistic activation might occur than on the non-dominant limb during contraction of the agonist muscles. Clearly, differences in coactivation may contribute to the observed differences in the net measured moment generating capacity around a joint between limbs (Fugl-Meyer, Gustafsson & Burstedt, 1980).

Traditionally, quantification of the antagonists' negative contribution to the net moment generated around a joint has been done while the agonists are concentrically or eccentrically contracted (Ostering, Hamill, Lander & Robertson, 1986; Baratta *et al*. 1988; Solomonow *et al*. 1988; Snow *et al*. 1993, 1995; Kellis & Baltzopoulos, 1996, 1997). In contrast, fewer studies have reported antagonistic coactivation during agonistic maximal isometric contraction (Solomonow, Guzzi, Baratta, Shoji & D'Ambrosia, 1986; Eloranta, 1989; Grabiner, Campbell, Hawthorne & Hawkins, 1989; Carolan & Cafarelli, 1992; Grabiner, Koh & Miller, 1992).

Although the effect of joint angle on antagonistic coactivation during maximum isometric contraction of agonists has been examined in the knee (Eloranta, 1989; Grabiner *et al*. 1992) and the elbow (Solomonow *et al*. 1986), there are no such reports on the ankle joint. The hypothesis in the present study was that the antagonistic ankle dorsiflexor coactivation level during a maximum isometric plantarflexion is a function of ankle angle. A verification of our hypothesis would be important in deriving the true force generating capacity of the ankle plantarflexors and estimating realistically musculoskeletal loads in the lower extremity as a function of ankle angle.

METHODS

Subjects

Six healthy males, from whom informed consent had previously been obtained, volunteered to participate in this study. All subjects were sedentary and none had any clinical history of musculoskeletal injury or any orthopaedic abnormality in the lower extremities. Their average (mean $\pm$ S.D.) age, height and body mass were 19 ± 2 years, 174 ± 5 cm and 71 ± 4 kg, respectively. The study was approved by the local ethics committee.

Moment measurements

Subjects were positioned and secured on an isokinetic dynamometer (Lido Active, Loredan Biomedical, Davis, CA, USA) for isometric ankle plantarflexion and dorsiflexion measurement purposes. The body was placed in the prone position and the knee joint of the tested leg was flexed at an angle of 90 deg. Isometric plantarflexion and dorsiflexion moments were taken at ankle angles of -15 deg (dorsiflexed direction), 0 deg (neutral position, the footplate of the dynamometer perpendicular to the tibia), +15 deg (plantarflexed direction) and +30 deg. Measurements were taken in both legs. Plantarflexion

and dorsiflexion MVC moments (maximum isometric voluntary plantarflexion and dorsiflexion contractions, respectively; the best out of three maximal efforts) were measured at all the above ankle angles. All MVC trials were performed in a randomized order. The stronger leg was identified according to the criterion of the highest observed plantarflexion MVC moment. The subjects were then asked to maintain for about 2–3 s with either leg contractions at 80, 60, 50, 40, 30, 20 and 10 % of the dorsiflexion MVC moment at ankle positions of −15, 0, +15 and +30 deg. Subjects were given visual feedback of target and elicited force on a computer screen. All maximal and submaximal contractions were separated by a 1 min period. A familiarization study was performed 7 days before the data collection day to acquaint the subjects with the measurements involved in the study.

EMG measurements

Bipolar Ag–AgCl surface electrodes with a centre-to-centre distance of 2 cm were used for EMG recording. EMG signals were obtained from two muscles; the tibialis anterior muscle (TA) and the soleus muscle (SOL). TA EMG signals were obtained having positioned the recording electrodes parallel to the tibia over the TA muscle belly, 10 cm below the distal edge of the patella. For SOL the electrodes were placed over the midline of the calf, 4 cm distal to the insertion point of the two heads of gastrocnemius into the Achilles' tendon. A ground electrode was attached above the knee. Before electrode placement, the skin was shaved and cleaned with alcohol to reduce impedance. Agonistic and antagonistic TA and SOL EMG signals were recorded during plantarflexor and dorsiflexor MVC trials at ankle angles of −15, 0, +15 and +30 deg. EMG signals were additionally obtained from TA and SOL at all four ankle angles during isometric dorsiflexions at contracting intensities corresponding to 80, 60, 50, 40, 30, 20 and 10 % of the dorsiflexion MVC moment. The EMG signals were recorded by amplifiers with a gain of 1000 and a frequency band of 20–500 Hz. The analog EMG signal was collected at a sampling frequency of 1000 Hz and converted to digital form using a twelve-bit analog-to-digital converter. The digital EMG signal was then full-wave rectified and integrated over 10 ms intervals. Integrated EMG signals at a given contraction intensity were averaged over a 1 s period during which EMG recording remained approximately constant.

Antagonistic coactivation expressed as normalized EMG

Antagonistic coactivation in each muscle at a given ankle angle was expressed as a percentage of the EMG activity when the respective muscle was acting as agonist at a maximal intensity (MVC) at that ankle angle.

Antagonistic coactivation expressed as moment

The TA EMG activity at a given ankle angle was reduced to the respective moment using the TA EMG–dorsiflexion moment relationship when TA was acting as agonist at that ankle angle. Since the predicted antagonistic moment of a muscle is expected to correspond to a moment within the range 0–50 % of MVC when the muscle acts as an agonist, EMG data below a moment corresponding to 50 % of dorsiflexor MVC were considered to determine the TA EMG–dorsiflexion moment relationship (Kellis & Baltzopoulos, 1997). Data were fitted with a second-degree polynomial (Fig. 1).

Statistical treatment

Values are reported as means ± S.D. Differences in the plantarflexion MVC moment between legs (two levels) and between different ankle angles (four levels) were tested using two-way analysis of variance. Two-way analysis of variance was also used to test for each coactivation level expression method, differences in antagonistic coactivation between legs (two levels) and between different ankle angles (four levels). Simple effects tests were used to identify where interaction effects occurred. Tukey's *post hoc* tests were used to determine significant differences between mean values. The statistically significant difference level was set at $P < 0{\cdot}05$.

RESULTS

Plantarflexion MVC moment (*Fig. 2*)

In either leg the plantarflexion MVC moment was dependent on ankle angle. In the stronger leg, as ankle angle increased from −15 to +30 deg, plantarflexion MVC decreased from 160 ± 9 to 32 ± 3 N m ($P < 0{\cdot}01$). In the weaker leg, as ankle angle increased from −15 to

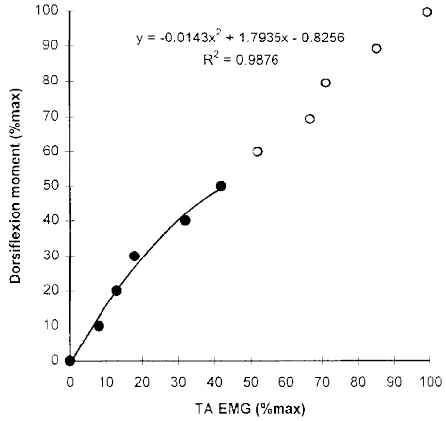

Fig. 1. Example of the TA EMG–dorsiflexion moment relationship used to estimate the antagonistic dorsiflexor moment. Data from one subject during static dorsiflexions with the stronger plantarflexor leg at the neutral ankle position (0 deg) are presented. Data up to 50 % of dorsiflexion MVC (●) were fitted using a second-degree polynomial (see Discussion and Fig. 6).

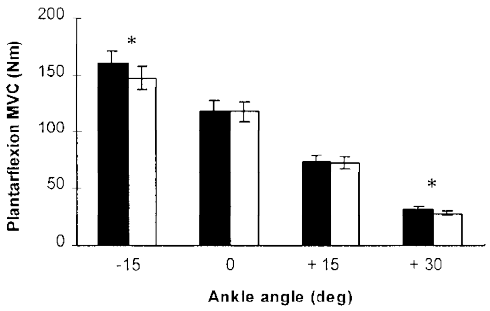

Fig. 2. Plantarflexion MVC moment of both stronger (■) and weaker (□) plantarflexor legs as a function of ankle angle. Values are means ± S.D. ($n = 6$). * Significant difference ($P < 0{\cdot}01$) between legs at a given ankle angle.

+30 deg, plantarflexion MVC decreased from 147 ± 8 to 28 ± 3 N m ($P < 0{\cdot}01$). Plantarflexion MVC at ankle angles of 0 and +15 deg did not differ ($P > 0{\cdot}05$) between legs. In contrast, at angle ankles of −15 and +30 deg, plantarflexion MVCs in the stronger leg were higher by 9 ± 4 % ($P < 0{\cdot}05$) and 14 ± 3 % ($P < 0{\cdot}05$), respectively, than in the weaker leg.

Antagonistic coactivation normalized EMG (Fig. 3)

In the stronger leg, the antagonistic dorsiflexor normalized EMG signal showed no difference ($P > 0{\cdot}05$) over the range from −15 deg of dorsiflexion to +30 deg of plantarflexion and varied between 18 ± 4 and 20 ± 5 % of the respective maximum agonistic EMG signal. However, this was not the case for the weaker leg where dorsiflexor coactivation was higher ($P < 0{\cdot}01$) at the extreme angles studied (−15 and +30 deg) than at the mid-range angles (0 and +15 deg). In this leg, coactivation ranged between 18 ± 5 and 40 ± 4 %

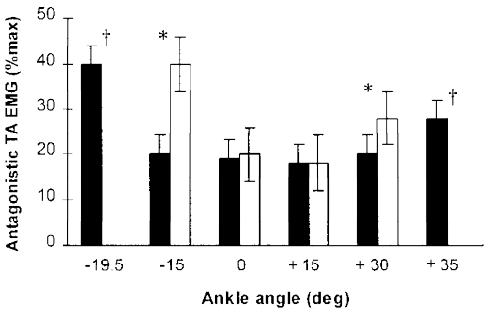

Fig. 3. Antagonistic normalized TA EMG signal (expressed as a percentage of that muscle's maximum agonistic EMG) in both stronger (■) and weaker (□) plantarflexor legs during plantarflexion MVC trials at ankle angles in the range from −15 to +30 deg. In a subsequent experiment the ROM of the ankle of the stronger leg was measured. The end-range dorsiflexed and plantarflexed angles were −19·5 and +35 deg, respectively (see Discussion). The single filled bars at ankle angles of −19·5 and +35 deg represent antagonistic normalized TA EMG signals during plantarflexion MVC trials with the stronger leg at these angles. Notice that the antagonistic EMG signal follows approximately an open U-shaped pattern in the range from −15 to +30 deg in the weaker leg and from −19·5 to +35 deg in the stronger leg. Notice also that coactivation levels at ankle angles of −15 and +30 deg in the weaker leg are almost identical with coactivation levels at ankle angles of −19·5 and +35 deg, respectively, in the stronger leg. Values are means ± S.D. ($n = 6$). *Significant difference ($P < 0·01$) between legs at a given ankle angle. †Significant difference ($P < 0·01$, one-way ANOVA followed by Tukey's *post hoc* analysis) at a given ankle angle compared with the mid-range ankle angles in the stronger leg.

($P < 0·01$) of the respective maximum agonistic EMG signal. Antagonistic EMG did not differ ($P > 0·05$) between legs at ankle angles of 0 and +15 deg. In marked contrast, at angle ankles of −15 and +30 deg, antagonistic EMG signals in the weaker leg were higher by 100 ± 28 % ($P < 0·01$) and 45 ± 18 % ($P < 0·01$), respectively, than in the stronger leg.

Antagonistic coactivation moment (*Fig. 4*)

In the stronger leg, the calculated antagonistic dorsiflexor normalized moment showed no difference ($P > 0·05$) over the range from −15 deg of dorsiflexion to +30 deg of plantarflexion and varied between 24 ± 6 and 27 ± 5 % of the respective measured dorsiflexor MVC. However, this was not the case for the weaker leg where the calculated antagonistic dorsiflexor moment was higher ($P < 0·01$) at the extreme angles studied (−15 and +30 deg) than at the mid-range angles (0 and +15 deg). In this leg the antagonistic dorsiflexor moment ranged between 23 ± 5 and 45 ± 3 % ($P < 0·01$) of the respective measured dorsiflexor MVC. Antagonistic moment did not differ ($P > 0·05$) between legs at ankle angles of 0 and +15 deg. In marked contrast, at angle ankles of −15 and +30 deg, the antagonistic moments in the weaker leg were higher by 70 ± 27 % ($P < 0·01$) and 43 ± 14 % ($P < 0·01$), respectively, than in the stronger leg.

DISCUSSION

In the present study a marked difference was found in the antagonistic dorsiflexor coactivation level at the extreme ankle angles studied between the stronger and the weaker plantarflexor leg. The validity of these results depends on the amount of cross-talk between the muscles studied during plantarflexion and dorsiflexion contractions with each leg. We believe that no cross-talk

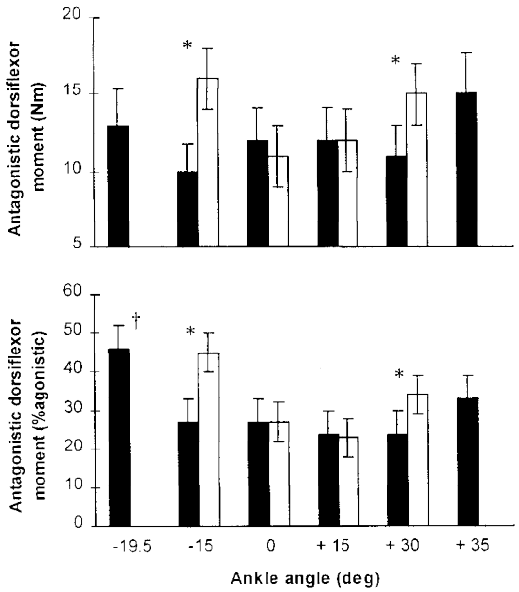

Fig. 4. Estimated antagonistic dorsiflexor moment (upper panel) and normalized moment (expressed as a percentage of dorsiflexion MVC moment) (lower panel) of both stronger (■) and weaker (□) plantarflexor legs during plantarflexion MVC trials at ankle angles in the range from −15 to +30 deg. In a subsequent experiment the ROM of the ankle of the stronger leg was measured. The end-range dorsiflexed and plantarflexed angles were −19·5 and +35 deg, respectively (see Discussion). The single filled bars at ankle angles of −19·5 and +35 deg represent antagonistic normalized dorsiflexor moments during plantarflexion MVC trials with the stronger leg at these angles. Notice that the estimated antagonistic moment follows approximately an open U-shaped pattern in the range from −15 to +30 deg in the weaker leg and from −19·5 to +35 deg in the stronger leg. Notice also that coactivation levels at ankle angles of −15 and +30 deg in the weaker leg are almost identical with coactivation levels at ankle angles of −19·5 and +35 deg, respectively, in the stronger leg. Values are means ± S.D. ($n = 6$). *Significant difference ($P < 0{\cdot}01$) between legs at a given ankle angle. † Significant difference ($P < 0{\cdot}01$, one-way ANOVA followed by Tukey's *post hoc* analysis) at a given ankle angle compared with the mid-range ankle angles in the stronger leg.

could have occurred between electrodes over TA and SOL because of the spatial anatomical position of these muscles. TA is electrically isolated from SOL by other muscles that lie in-between and by the tibia (Salmons, 1995). Moreover, Moritani, Oddson & Thorstensson (1990) showed that a near-maximal electrical stimulus applied over gastrocnemius resulted in an M-wave, even in the much closer and less isolated SOL, with an amplitude of only 6 % of the M-wave amplitude in gastrocnemius.

Dorsiflexion results from the combined action of four muscles (tibialis anterior, extensor digitorum longus, extensor hallucis longus and peroneus tertius; Salmons, 1995). The force exerted by each of these muscles and the generated moment around the ankle joint is not directly measurable in humans. Bouisset (1973) introduced the concept of muscle equivalency for studying synergists acting across a joint. According to this simplification, the activity of a group of synergists can be reduced to the function of the predominant or representative muscle for this particular function. Adopting such a simplification, antagonistic TA EMG can be considered as a representative measure of the mechanical activity of the whole coactivated dorsiflexor muscle group during plantarflexion. Antagonistic TA EMG activity at a given

Table 1. *Antagonistic normalized dorsiflexor EMG during a plantarflexion MVC at different ankle angles in two subjects*

		Antagonistic EMG* (% max)					
Subject	Electrode placement	ERD	−15 deg	0 deg	+15 deg	+30 deg	ERP
A	TA EMG	32	15	16	17	16	22
	DORS EMG	29	14	19	16	16	25
B	TA EMG	—	40	23	17	30	—
	DORS EMG	—	36	20	19	32	—

* Expressed as a percentage of the respective maximum agonistic EMG signal; see Methods. ERD and ERP denote end-range dorsiflexed and end-range plantarflexed angles, respectively. Measurements were taken from the stronger plantarflexor leg in Subject A, and from the weaker plantarflexor leg in Subject B. EMG data were collected using two different electrode placements. In the first configuration, the recording electrodes were placed over the TA belly (TA EMG). In the second configuration the recording electrodes were placed over all four dorsiflexor muscles (DORS EMG). Notice that there is neither a substantial nor a systematic difference in EMG coactivation levels between different electrode configurations for either subject at any given ankle angle.

ankle angle can then be reduced to the respective moment using the TA EMG–dorsiflexion moment relationship when TA acts as agonist at that ankle angle.

The acceptance of antagonistic TA EMG signal as a measure of the mechanical activity of all four dorsiflexor muscles is an important, yet speculative assumption. An ideal way to relate dorsiflexor EMG recordings with the whole dorsiflexor mechanical activity would be to collect with a single electrode configuration signals from all four individual dorsiflexors. Such an EMG data collection approach can be followed for synergists adjoining each other (e.g. triceps surae complex) but for the ankle dorsiflexors this is not the case; the muscle bellies of tibialis anterior and extensor digitorum longus lie midway between knee and ankle but extensor hallucis longus and peroneus tertius arise from the middle half and the distal third of the fibula, respectively (Salmons, 1995). Placement of the TA electrode 2 cm more laterally and 10 cm below its original position in the present study, an increase in the inter-electrode distance to 5 cm and placement of the second electrode over peroneus tertius as reported by Jungers, Meldrum & Stern (1993) could give a more appropriate electrode configuration for obtaining simultaneously EMG signals from all four dorsiflexors. Using this electrode placement over the dorsiflexors, we collected additional EMG data during all contractions with the stronger leg in one subject and during all contractions with the weaker leg in a different subject. In neither subject was any substantial or systematic difference found in antagonistic normalized EMG activity between TA and the whole dorsiflexor group during a plantarflexion MVC at any given ankle angle (Table 1). Thus, in the present study TA was taken as representative of the whole dorsiflexor group.

In the present study the selection of SOL as representative of the antagonistic activity of the whole ankle plantarflexor group was based on the mono-articular function of SOL around the ankle joint. At the adopted knee position of 90 deg, the bi-articular gastrocnemius, although electrically active, has negligible contribution to the generated plantarflexion moment (Hof & Van der Berg, 1977; Gravel, Arsenault & Lambert, 1987). A reduction of the antagonistic TA EMG signal to the respective moment value would only result in realistic estimations if plantarflexors were electrically silent during dorsiflexion. In the present study SOL was active during graded isometric dorsiflexion. However, the normalized SOL EMG signal during a

Table 2. *Antagonistic normalized SOL EMG in both stronger and weaker plantarflexor legs during isometric dorsiflexion at the moment level produced by dorsiflexors during a plantarflexion MVC at different ankle angles*

	Antagonistic SOL EMG* (% max)					
	−19·5 deg	−15 deg	0 deg	+15 deg	+30 deg	+35 deg
Stronger leg	2·6 ± 0·3	2·2 ± 0·6	2·6 ± 0·3	2·7 ± 0·2	2·5 ± 0·4	2·6 ± 0·2
Weaker leg	—	2·3 ± 0·4	2·5 ± 0·3	2·2 ± 0·2	2·4 ± 0·4	—

*Expressed as a percentage of the respective maximum agonistic EMG signal; see Methods section. Values are means ± S.D. ($n = 6$).

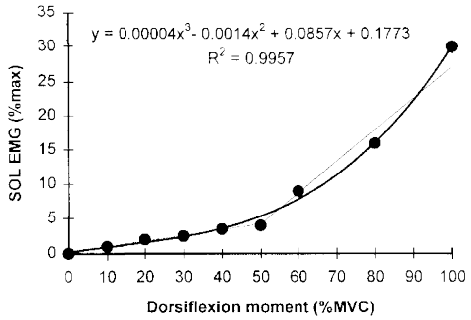

Fig. 5. Antagonistic normalized SOL EMG signal (expressed as a percentage of that muscle's maximum agonistic EMG) as a function of dorsiflexion moment. Values represent measurements taken in one subject during static dorsiflexions with the stronger plantarflexor leg at the neutral ankle position (0 deg). Third-degree polynomial fit to the data shows that the coactivation moment level of dorsiflexors in that specific subject (33 %) corresponds to a normalized SOL EMG signal of 2·9 %. Notice the change in the slope of the two consecutive linear components of the graph. Based on such a slope change, a fit up to 50 % of dorsiflexion MVC was performed to the data to construct the normalized TA EMG–dorsiflexion moment curve (see Discussion and Fig. 1).

dorsiflexor contraction at the moment level of coactivated dorsiflexors during a plantarflexion MVC was less than 3 % in either leg at any given ankle angle (Table 2 and Fig. 5). The experimental design of the study does not allow quantification of the plantarflexion moment that would result from this SOL activation. Assuming, however, a similar agonistic EMG–MVC moment relationship between TA and SOL, it can be estimated that the recorded antagonistic SOL EMG signal would result in a plantarflexion moment of 1–6 N m. It should be recognized that there will always be such a systematic effect on calculated moments as a consequence of the prevailing antagonistic coactivation.

Antagonistic coactivation can be mediated by (i) excitation of Ib afferents from Golgi tendon organs; (ii) spinal or supraspinal driven firing of Renshaw cells that inhibits reciprocal inhibition of antagonists (Henatsch & Langer, 1985; Rothwell, 1987); and (iii) a direct central motor drive, common for both agonists and antagonists (the common drive hypothesis: Basmajian & DeLuca, 1981). It should be noted, however, that in the present experiment the highest antagonistic dorsiflexor normalized EMG signal in the weaker leg was recorded at the maximum angles of −15 deg of dorsiflexion and +30 deg of plantarflexion (Fig. 2). Increased coactivation at maximum joint angles could imply the involvement of a neural drive for

Table 3. *Range of motion* (*ROM*), *range of dorsiflexion* (*ROD*) *and range of plantarflexion* (*ROP*) *in the ankle of the stronger and weaker plantarflexor legs of the subjects*

	Stronger leg			Weaker leg		
Subject	ROM (deg)	ROD (deg)	ROP (deg)	ROM (deg)	ROD (deg)	ROP (deg)
1	55	19	36	47	15	32
2	50	18	32	46	16	30
3	59	20	39	51	16	35
4	54	21	33	47	17	30
5	56	20	36	50	17	33
6	53	19	34	47	15	32
Mean	54·5*	19·5*	35*	48	16	32
S.D.	3	1	3	2	1	2

Presented data are mean values across three measurements. * Significantly larger values ($P < 0{\cdot}01$) in the stronger leg than in the weaker leg.

antagonist muscles mediated via joint mechanoreceptors. Experimental results have been equivocal, either providing support to a neuromotor link between joint mechanoreceptors and antagonists (Rothwell, 1987; Solomonow *et al*. 1987, 1988; Tyler & Hutton, 1989; Ostering *et al*. 1995) or not supporting such a mechanism (Grabiner *et al*. 1989, 1992; Grabiner & Weiker, 1993). If such a neural mechanism does exist, it might be expected to operate to a similar extent at a given ankle angle in both legs and this was clearly not the case in the present experiment. An explanation for a lower coactivation in the stronger leg at ankle angles of −15 and +30 deg than in the weaker leg could be that there is a smaller range of motion (ROM) in the ankle of the weaker leg than in that of the stronger leg; joint proprioreceptors acting as pressure sensors and joint position detectors would be excited at a given plantarflexed or dorsiflexed position to a higher extent in the ankle with the smaller ROM than in the ankle with the larger ROM (Rothwell, 1987). This in turn would result in higher antagonist EMG activity in the ankle with the smaller ROM than in the ankle with the larger ROM. To test this hypothesis we measured in either leg of the subjects of the study the ROM of the ankle joint using the Lido footplate system, having placed the body in the adopted body position during EMG data acquisition. ROM measurements were taken 7 days after the day on which MVC and EMG data were collected. The average ROM in the ankle of the stronger leg was 54·5 deg (from −19·5 deg of dorsiflexion to +35 deg of plantarflexion) whereas the average ROM in the ankle of the weaker leg was 48 deg (from −16 deg of dorsiflexion to +32 deg of plantarflexion). ROM, range of dorsiflexion and range of plantarflexion in the ankle of the stronger leg were respectively 14, 22 and 9 % larger ($P < 0{\cdot}01$, Student's t test) than in the weaker leg (Table 3). The finding of a larger range of dorsiflexed and plantarflexed angles in the stronger leg than in the weaker leg, and the consistency between the end-range positions in the ankle of the weaker leg (−16 and +32 deg) and the maximum angles examined in the present study (−15 and +30 deg) provide support for our hypothesis of a negative relationship between ROM and antagonistic co-contraction.

The finding of an increased ROM in the ankle of the stronger leg is in line with observations by Fugl-Meyer *et al*. (1980). Differences in the plantarflexion MVC moment between legs in the present study were localized at ankle angles of −15 and +30 deg. The magnitude of differences in the plantarflexion MVC moment between legs (9 and 14 % at ankle angles of

−15 and +30 deg, respectively) is consistent with observations of Fugl-Meyer *et al.* (1980) on physically inactive subjects. These authors reported that at the maximum dorsiflexed position tested, the difference in the plantarflexion MVC moment between legs was about 8 %. The authors speculated that such a difference could be attributed to a larger muscle excursion (greater working range due to a wider length–tension relationship) in the stronger plantarflexor leg than in the weaker leg. This could be the result of a unilateral and systematic operation of the ankle of the stronger/dominant leg at end-range positions as happens, for example, when kicking and dribbling in soccer. In fact, the subjects in the present study did report that the stronger plantarflexor leg was also dominant with respect to kicking a ball. However, the subjects had no athletic background or any sport habit and their participation in recreational physical activities at the time of the study was not regular. Moreover, a comparison of the calf circumference between legs in our subjects showed no difference measured to the nearest millimetre (see also Kitai & Sale, 1989). The present findings suggest that a decreased excursion in the plantarflexors of the weaker leg compared with the stronger leg may not account *per se* for the difference in the net measured moment between legs at end-range angles, as speculated by Fugl-Meyer *et al.* (1980). The operation, however, of a neural mechanism with an excitatory effect upon the opposing acting antagonist muscles, is directly dependent on muscle excursion and can explain the present findings and those reported by Fugl-Meyer *et al.* (1980). A cause-and-effect relationship between a smaller ankle ROM and an increased antagonistic EMG activity at ankle angles of −15 and +30 deg in the weaker leg compared with the stronger leg was further supported by comparing EMG measurements at the actual end-range dorsiflexed and plantarflexed positions between legs. Additional EMG measurements taken in the stronger leg at the measured end-range positions (−19·5 and +35 deg) were compared with EMG measurements at −15 and +30 deg in the weaker leg. Since the difference between the maximum angles studied and the end-range ankle angles measured in the weaker leg was very small (1 deg for the dorsiflexed direction and 2 deg for the plantarflexed direction), EMG measurements at ankle angles of −15 and +30 deg were considered representative of the electrical activity of the muscle at the actual end-range ankle angles of −16 and +32 deg. Statistical analysis (Student's t test) revealed no difference ($P > 0{\cdot}05$) in antagonistic coactivation between legs, expressed either as normalized EMG or normalized moment at: (i) the end-range dorsiflexed positions and (ii) end-range plantarflexed positions (Figs 3 and 4).

If an increased antagonistic coactivation in the weaker leg did account for the difference in the plantarflexion MVC moment between legs at the ankle angles of −15 and +30 deg, then in a situation where the plantarflexors in each leg were the sole contributors to the resultant moment output, a comparison in plantarflexion MVC moments at the above angles between legs should reveal no difference. Indeed, the difference in the measured net plantarflexion MVC output between legs at the ankle angles of −15 and +30 deg disappeared ($P > 0{\cdot}05$) when comparing corrected moments generated by selectively activated plantarflexors (calculated as measured net plantarflexion MVC moment minus estimated antagonistic dorsiflexor moment; Table 4). The results of this comparison provide additional support for the hypothesis that differences in the measured net plantarflexion moment output were attributed to differences in the level of dorsiflexor coactivation between legs. An alternative way to test this hypothesis would be to compare between legs maximum plantarflexion moments generated at the ankle angles of −15 and +30 deg by the agonists only; in the absence of dorsiflexor coactivation, through, for example, nerve blocking, the recorded

Table 4. *Measured net plantarflexion MVC moment and the respective corrected agonistic moment* (*net plantarflexion MVC moment minus estimated antagonistic moment*) *of both stronger and weaker plantarflexor legs at different ankle angles*

	Maximum isometric moments (N m)					
	−19·5 deg	−150 deg	0 deg	+15 deg	+30 deg	+35 deg
Measured in the stronger	150 ± 9 *	160 ± 9 *†	119 ± 6 *	74 ± 4 *	32 ± 3 *†	30 ± 2 *
Corrected in the stronger	160 ± 8	169 ± 8	131 ± 6	86 ± 5	43 ± 3	40 ± 3
Measured in the weaker	—	147 ± 8 *	118 ± 8 *	72 ± 3 *	28 ± 3 *	—
Corrected in the weaker	—	164 ± 7	128 ± 7	83 ± 4	43 ± 3	—

Values are means ± S.D. ($n = 6$). * Significant difference ($P < 0{\cdot}01$) between measured and corrected moments for a given leg at a given ankle angle. † Significant difference ($P < 0{\cdot}01$) in the measured plantarflexion MVC moment between legs at a given ankle angle. Notice that the difference in the net measured plantarflexion MVC moment between legs at a given ankle angle disappears ($P > 0{\cdot}05$) when comparing the respective corrected moments.

plantarflexion moments at a given ankle angle should be similar between the stronger and the weaker plantarflexor legs. This hypothesis needs further investigation.

The results of the present study have important implications for the analysis of loads in the musculoskeletal system and the maximum voluntary contraction efficiency at end-range positions over a joint's ROM.

Inaccurate information with respect to the true moment generating capacity of a muscle would inevitably result in erroneous estimations of the loads imposed in the musculoskeletal system during maximal efforts. The impact of the antagonists' negative contribution in calculating musculoskeletal loads can be realized using data reported by Fukunaga, Roy, Shellock, Hodson & Edgerton (1996) on Achilles' tendon forces in man during maximum voluntary isometric plantarflexion. By subtracting the corresponding normalized antagonistic dorsiflexor moment at an ankle angle of +30 deg as calculated in the present study from the reported net plantarflexion MVC moment, it was calculated that both the Achilles' tendon force and plantarflexor specific tension would be higher by 10 % in the stronger leg and by 14 % in the weaker leg. These differences indicate that estimation of forces generated by muscles acting around the ankle joint can be unrealistic when assuming negligible antagonistic coactivation.

Traditionally, strengthening of an agonist muscle involves resistance exercise training of the target muscle. However, an increase in the maximum moment generated around a joint could be achieved without strengthening the agonist itself. A reduction in the moment generated by the co-contracted antagonist would result in an increased net moment generated around a joint. A stretching exercise programme designed to increase the ROM in a joint, could decrease the neural drive for antagonistic co-contraction and might thereby enhance the net moment output at the end-range angles over the joint's ROM.

It should also be realized that antagonistic coactivation may inhibit maximum activation of the agonists (Tyler & Hutton, 1989). Clearly, the marked increase in antagonistic coactivation at the end-range ankle positions as observed in the present study, could result in increased inhibition of agonist activation, further reducing the net moment due to 'neural insufficiency' (see Enoka & Fuglevand, 1993).

In conclusion, in the present study antagonistic dorsiflexor coactivation during a plantarflexion MVC (i) was a function of ankle angle and followed an open U-shaped pattern

and (ii) was higher in the weaker plantarflexor leg at ankle positions of −15 deg of dorsiflexion and +30 deg of plantarflexion than in the stronger leg. The difference in antagonistic coactivation level between legs disappeared when coactivation was expressed as a function of the end-range dorsiflexed and plantarflexed angle in each individual leg. Similarly, the difference in the net plantarflexion MVC moment between legs disappeared when the respective antagonistic dorsiflexor moment was taken into account. The findings of the present study can account for the difference in plantarflexion MVC between legs and have important consequences for musculoskeletal modelling of the lower extremity and the efficiency of maximal effort plantarflexions.

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