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1 **Agonist and antagonist activation at the ankle monitored along the swing phase in**
2 **hemiparetic gait**

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1 **Abstract**

2 *Background:* Descending command in hemiparesis is reduced to agonists and misdirected to
3 antagonists. We monitored agonist and antagonist activation along the swing phase of gait,
4 comparing paretic and non-paretic legs.

5 *Methods:* Forty-two adults with chronic hemiparesis underwent gait analysis with bilateral
6 EMG from tibialis anterior, soleus and gastrocnemius medialis. We monitored ankle and knee
7 positions, and coefficients of agonist activation in tibialis anterior and of antagonist activation
8 in soleus and gastrocnemius medialis over the three thirds of swing phase. These coefficients
9 were defined as the ratio of the root-mean-square EMG from one muscle over any period to
10 the root-mean-square EMG from the same muscle over 100 ms of its maximal voluntary
11 isometric contraction.

12 *Findings:* As against the non-paretic side, the paretic side showed lesser ankle dorsiflexion
13 and knee flexion ($P < 1.E^{-5}$), with higher coefficients of agonist activation in tibialis anterior
14 ($+100 \pm 28\%$, $P < 0.05$), and of antagonist activation in soleus ($+224 \pm 41\%$, $P < 0.05$) and
15 gastrocnemius medialis ($+276 \pm 49\%$, $P < 0.05$). On the paretic side, coefficient of agonist
16 activation in tibialis anterior decreased from mid-swing on; coefficients of antagonist
17 activation in soleus and gastrocnemius medialis increased and ankle dorsiflexion decreased in
18 late swing ($P < 0.05$).

19 *Interpretation:* During the swing phase in hemiparesis, normalized tibialis anterior
20 recruitment is higher on the paretic than on the non-paretic leg, failing to compensate for a
21 marked increase in plantar flexor activation (cocontraction). The situation deteriorates along
22 swing with a decrease in tibialis anterior recruitment in parallel with an increase in plantar
23 flexor activation, both likely related to gastrocnemius stretch during knee re-extension.

1 **Clinical Trial Registration-URL:** <http://www.clinicaltrials.gov>. Unique identifier:

2 [NCT03119948](#)

3

4 **Key-words:** Electromyography, muscle spasticity, paresis, dorsiflexion, stretching, antagonist

5 activation.

1 **1. Introduction**

2 In chronic hemiparesis, movement impairment results mainly from disturbance of the
3 descending command and from soft tissue hypoextensibility, in particular from structural
4 changes in muscle (Bourbonnais and Vanden Noven 1989; Fitts et al., 1989; Gracies 2005a).
5 The muscle disorder causes visco-hyperelasticity that reduces passive muscle extensibility
6 and has long been demonstrated to play a highly important role in limiting movements during
7 gait, particularly the progression of passive dorsiflexion during stance (Berger et al., 1984).
8 The neurological disorder involves a quantitative reduction of the supraspinal descending
9 motor command to the target agonist motor neuron, which defines *paresis* (Bourbonnais and
10 Vanden Noven 1989; Fitts et al., 1989; Gracies 2005a). In parallel, this command spreads
11 excessively to other motor neurons, in particular those activating antagonist muscles, causing
12 *cocontraction* (Knutsson and Mårtensson 1980; Hammond et al., 1988; Gracies 2005b). From
13 a body of evidence from isometric studies, it is known that both paresis of the agonist and
14 cocontraction of the antagonist are worsened by the stretched position of the antagonist,
15 phenomena that have been termed *stretch-sensitive paresis* and *spastic cocontraction*,
16 respectively (Gracies 2005b; Vinti et al., 2013, 2015). However, during the progression of
17 gait cycle in hemiparesis, actual quantifications of the normalized recruitment of agonists and
18 antagonists have been relatively scarce in the literature, whether in hemiparetic adults or
19 children (Knutsson and Richards 1979; Berger et al., 1984; Shiavi et al., 1987; Unnithan et al.,
20 1996a, b; Damiano et al., 2000; Lamontagne et al., 2000; Chow et al., 2012; Ma et al., 2017;
21 Souissi et al., 2018; Vinti et al., 2018). A number of these previous accounts were based on
22 stance only (Chow et al., 2012), or both stance and swing phase analyses and ended up
23 interpreting cocontraction as an adaptive strategy, a *compensation* for the paresis disorder or
24 the lack of stability (Lamontagne et al., 2000; Chow et al., 2012; Ma et al., 2017; Souissi et
25 al., 2018). In contrast, we believe that cocontraction is a *part* of the neurological disorder and

1 adds up to paresis to worsen weakness around joint, which might be particularly clear during
2 swing phase. The swing phase of the paretic limb is a critical phase in which the risk of
3 tripping is increased and in which plantar flexors are supposed to be at rest (Shiavi et al.,
4 1987; Vinti et al., 2013, 2015). The paucity of specific swing phase muscle activation
5 measurements in hemiparesis is unfortunate as these measurements could lead to specific
6 therapeutic approaches, such as agonist stimulation if the agonist was found to be under-
7 recruited, or antagonist weakening or lengthening if the antagonist was found to be
8 excessively activated and/or if this excessive recruitment appeared to be tension-related.

9 We use normalized coefficients of muscle activations, designed to measure *how much of a*
10 *given motoneuron pool* is involved in agonist or antagonistic activation at a given time, with
11 respect to its maximal possible isometric recruitment as an agonist (Vinti et al., 2013, 2015,
12 2018). The present study focuses on the kinematics and electromyography of the swing phase
13 of gait and investigates these coefficients of muscle activation for the agonist tibialis anterior,
14 and for the antagonists soleus and gastrocnemius medialis bilaterally, throughout the
15 progression of swing phase during gait at comfortable speed in chronic hemiparesis.

16 The first hypothesis was about kinematics as precise knowledge about the changes in knee
17 kinematics along the three thirds of the swing phase has been scarce from previous literature:
18 we expected to find lesser knee flexion and ankle dorsiflexion on the paretic side, with earlier
19 onset of knee re-extension and a specific worsening of the dorsiflexion deficit in the last third
20 of swing compared with the non-paretic side. The second hypothesis was that recruitment of
21 tibialis anterior in swing would be abnormally high in the paretic compared with the non-
22 paretic limb, in order to oppose passive antagonist resistances from stiffer and less extensible
23 antagonists. The third hypothesis was that, as a consequence of the previous behaviour,
24 cocontraction of the soleus and gastrocnemius medialis would be higher than in the non-
25 paretic limb, based on the effort-dependance of antagonist cocontraction in spastic paresis

1 (Vinti et al., 2013, 2015). Two phenomena recently individualized in spastic paresis led us to
2 a final hypothesis: these features are *spastic cocontraction*, i.e. a level of antagonist
3 cocontraction that increases with antagonist stretch in isometric paradigms (Gracies 2005b;
4 Vinti et al., 2013) and *stretch-sensitive paresis* i.e. a level of agonist recruitment that
5 decreases with increasing stretch imposed on the antagonist (Vinti et al., 2015). We thus
6 finally hypothesized that the normalized recruitment of tibialis anterior would decrease along
7 the swing phase, as previously suggested when considering Berger et al. findings (Berger et
8 al., 1984), while the cocontraction of soleus and gastrocnemius medialis would increase in
9 parallel, all due to gradually increasing tension within the gastrocnemius muscle, in parallel
10 with the knee re-extension of the swing phase.

11 **2. Methods**

12 *2.1. Participants*

13 This prospective descriptive cohort study was a physiological sub-study of a randomized,
14 controlled, parallel-group, double-blind trial aiming to evaluate the effects of rectus femoris
15 and soleus injections with botulinum toxin vs placebo on gait in hemiparesis (GENUFLEX
16 protocol, ClinicalTrials.gov: NCT03119948). This sub-study used the baseline evaluations of
17 the participants of GENUFLEX study conducted from 2015 to 2019 in the *Analyse et*
18 *Restauration du Mouvement* Laboratories of the Neurorehabilitation Department at Henri
19 Mondor Hospital, Créteil, France.

20 Inclusion criteria were: age ≥ 18 years, hemiparesis due to a non-evolutive central nervous
21 system lesion, time since lesion ≥ 6 months, ability to walk 10 meters barefoot without any
22 assistance, cognitive abilities to understand the verbal instructions for a walking test
23 according to the investigator's judgment, and absence of botulinum toxin injections within the
24 last 3 months prior to enrolment.

1 The study was conducted in compliance with the Declaration of Helsinki (2008), Good
2 Clinical Practice guidelines, the approval of the local ethical committee and regulatory
3 requirements (registration number, ID-RCB-P101107, *Comité de Protection des Personnes*
4 *Ile-de-France V*). All patients gave informed consent before study entry. The present
5 manuscript conforms to the *Strengthening the Reporting of Observational Studies in*
6 *Epidemiology* guidelines.

7 2.2. Evaluation

8 All subjects underwent a clinical assessment of the paretic ankle and a kinematic analysis
9 with surface EMG of both lower limbs during barefoot walking at comfortable speed without
10 any assistance, all during the same visit.

11 2.2.1. Clinical assessment

12 Study subjects were assessed at the paretic ankle using the *Five-Step Assessment* (FSA)
13 (Gracies et al., 2010), an expansion of the Tardieu scale, in supine position with the knee
14 flexed to measure resistances from soleus and with the knee extended to measure resistances
15 from pooled gastrocnemius and soleus. The investigator measured the first three technical
16 parameters of the FSA in the following order: first, the passive range of dorsiflexion (slow
17 stretch, X_{V1}), then the angle of catch at fast stretch (X_{V3}), and finally the range of active
18 dorsiflexion (X_A).

19 2.2.2. Kinematic gait assessment

20 The procedure of gait analysis was based on the recommendations of the International
21 Society of Biomechanics, having shown good repeatability and reliability in post-stroke
22 subjects (Wu et al., 2002). Gait was analyzed by the same investigator using a tridimensional
23 motion capture system (10 cameras 4MPx, Cortex software package, Motion Analysis
24 Corporation, Santa Rosa, CA, USA). The trajectories of 26 markers placed on anatomical
25 landmarks using the Helen Hayes marker set were collected (sampling frequency, 100 Hz)

1 and filtered using a 4th order zero-lag Butterworth low pass filter, with a 6 Hz cut-off
2 frequency (Wu et al., 2002). At least eight gait cycles from each lower limb were used for the
3 kinematic and EMG analysis. Five key parameters were computed: walking speed, cadence,
4 and, for each lower limb, step length and the mean angle position in ankle dorsi-plantar
5 flexion and in knee flexion-extension, calculated over the three thirds of the swing phase (T1,
6 [0-33%]; T2, [34-66%]; T3, [67-100%]).

7 2.2.3. Electromyography assessments

8 Bilateral muscle activity was recorded using wireless surface electromyography (EMG,
9 sampling frequency, 1000 Hz) from pairs of bipolar electrodes coated with electro-conductive
10 gel (WBA, Mega Electronics Ltd, Kuopio, Finland) placed 2 cm apart over each of tibialis
11 anterior (TA), soleus (SO) and gastrocnemius medialis (GM) at specific sites, according to the
12 European recommendations for surface electromyography (Hermens et al., 2000). Skin was
13 cleansed and abraded before electrode placement. EMG signals were first recorded during the
14 maximal voluntary isometric efforts in standing position with hip, knee and ankle fixed at 0°
15 (anatomic angles). Patients were first asked to perform two 4-second maximal isometric
16 efforts against resistance, one in dorsiflexion and one in plantar flexion. Then, EMG was
17 monitored throughout the kinematic gait assessments. EMG signals were centred, rectified
18 and filtered using a 2nd-order zero-lag Butterworth low pass filter, with a 30 Hz cut-off
19 frequency.

20 The *Coefficient of AGonist activation* in TA (CAG_{TA}) and the *Coefficient of ANtagonist*
21 *activation* in SO and GM (CAN_{SO} , CAN_{GM}) were calculated from the Root Mean Square
22 EMG (RMS-EMG) signals over each of the three thirds of the swing phase of gait (T1, T2,
23 T3). CAG_{TA} , CAN_{SO} and CAN_{GM} over any period were defined as the ratio of the RMS-EMG
24 from the muscle of interest over that period to the RMS-EMG from the same muscle over the
25 100 ms around the peak of its maximal voluntary isometric contraction (Figure 1).

1 2.3. Statistics

2 For this descriptive cohort study no formal sample size calculations were performed. After
3 verification of normality using Kolmogorov-Smirnov tests, kinematic and EMG parameters
4 were compared between paretic and non-paretic sides and between the three thirds of swing
5 phase using repeated measures ANOVAs (side*period). Post-hoc comparisons were
6 performed using Bonferroni corrections to evaluate the effects of side and period, and their
7 interaction. For each muscle, EMGs were analyzed only in subjects for whom data were
8 complete on both sides for each of the three periods. Statistical significance was set at $P<0.05$.
9 Statistical analysis was conducted using Statistica software package (version 7.0, StatSoft,
10 Inc., Tulsa, OK, USA).

11

12 3. Results

13 Forty-two individuals with chronic hemiparesis participated in the study and only their data
14 from the baseline evaluations were analyzed (Table 1).

15 3.1. Clinical assessment

16 Against soleus resistances, X_{V1} was $104\pm6^\circ$ (mean \pm SD, $n=40$); X_{V3} , $90\pm6^\circ$ and X_A , $91\pm10^\circ$.
17 Against gastrocnemius resistances, X_{V1} was $94\pm5^\circ$; X_{V3} , $82\pm6^\circ$ and X_A , $77\pm12^\circ$.

18 3.2. Kinematics

19 All subjects performed the kinematic gait analysis. Mean walking speed was 0.66 ± 0.26 m/s,
20 cadence was 1.47 ± 0.27 step/s and step length was 0.47 ± 0.12 m on the paretic side and
21 0.41 ± 0.16 m on the non-paretic side. Compared with the non-paretic side, dorsiflexion was
22 reduced on the paretic side in T2 ($P<0.001$; Table 2A, Figure 2A) and T3 ($P<0.001$), and knee
23 flexion was reduced throughout all periods (T1, $P<0.001$; T2, $P<0.001$; T3, $P=5.E^{-14}$, Figure
24 2B). The increase in dorsiflexion between T1 and T2 (non-paretic, $+10\pm5^\circ$, $P<0.001$; paretic,
25 $+2\pm5^\circ$, $P=0.009$) and the knee re-extension between T2 and T3 (non-paretic, $-28\pm7^\circ$,

1 $P<0.001$; paretic, $-12\pm 10^\circ$, $P<0.001$) were lower on the paretic side (side*period, $P=1.E^{-15}$).
2 Dorsiflexion decreased between T2 and T3 on the paretic side only ($-2\pm 3^\circ$, $P=0.04$).

3 *3.3. Electromyography*

4 Due to occasional loss of EMG data through electrode movements during gait, EMGs could
5 be fully analyzed bilaterally for TA in 81% of subjects ($n=34$), SO in 90% of subjects ($n=38$)
6 and GM in 98% of subjects ($n=41$).

7 *3.3.1. Agonist activation*

8 Compared with the non-paretic side, CAG_{TA} was higher on the paretic side throughout the
9 three periods of swing (T1, $P=2.E^{-11}$; T2, $P=2.E^{-6}$; T3, $P=9.E^{-4}$, Table 2B1, Figure 3A).
10 Throughout swing CAG_{TA} decreased on the paretic side only (from T1 to T2, non-paretic
11 side, -0.10 ± 0.17 , ns; paretic, -0.18 ± 0.21 , $P=7.E^{-6}$; from T1 to T3, non-paretic side, -
12 0.07 ± 0.21 , ns; paretic, -0.21 ± 0.31 , $P=2.E^{-7}$; side*period, $P=0.009$).

13 *3.3.2. Antagonist activation*

14 Compared with the non-paretic side, CAN_{SO} was higher on the paretic side in T2 ($P=0.023$;
15 Table 2B1, Figure 3B) and T3 ($P=2.E^{-12}$) and CAN_{GM} was higher on the paretic side in each
16 of the three periods (T1, $P=4.E^{-4}$; T2, $P=1.E^{-8}$; T3, $P=1.E^{-13}$, Figure 3C). From T2 to T3, both
17 $CANs$ increased on the paretic side only (CAN_{SO} , $+0.36\pm 0.42$, $P=2.E^{-10}$; side*period, $P=1.E^{-4}$;
18 CAN_{GM} , $+0.16\pm 0.28$, $P=3.E^{-5}$; side*period, $P=0.003$; Figures 3B, 3C, 4).

19 The data from a typical participant expressed as mean \pm standard deviation of 12 gait cycles,
20 for the coefficient of agonist activation in tibialis anterior (CAG_{TA}) and the coefficients of
21 antagonist activation in soleus (CAN_{SO}) and in gastrocnemius medialis (CAN_{GM}) on the
22 paretic and non-paretic sides, show the reliability of the coefficients (Table 2B2).

23

24 **4. Discussion**

1 The present study of gait in both paretic and non-paretic limbs reports rare data monitoring
2 normalized agonist and antagonist activations and their changes along the three thirds of the
3 swing phase, together with their kinematic correlates, in a cohort of 42 patients with chronic
4 hemiparesis. Reduced amplitude of active dorsiflexion is concomitant with high levels of
5 normalized agonist TA activation (Table 2, Figures 2A, 3A), in the context of markedly
6 increased antagonist SO and GM activation (Table 2, Figures 3B, 3C), compared to the non-
7 paretic limb. The descending command would initially enhance agonist recruitment in the
8 paretic limb in an attempt to overcome pathologically increased passive resistance from stiffer
9 antagonists, i.e. from antagonists with excessive levels of viscoelasticity at rest. As the swing
10 phase progresses, plantar flexor cocontraction worsens while TA activation diminishes.

11 *4.1. Methodology: how to measure cocontraction - coefficient of antagonist activation*

12 A number of approaches to quantify agonist-antagonist coactivation have been used
13 (Knutsson et al., 1979; Berger et al., 1984; Falconer and Winter 1985; Shiavi et al., 1987;
14 Hammond et al., 1988; Levin and Hui-Chan 1994; Unnithan et al., 1996a, b; Damiano et al.,
15 2000; Lamontagne et al., 2000; Chow et al., 2012; Vinti et al., 2013, 2015; Kitatani et al.,
16 2016; Ma et al., 2017; Souissi et al., 2018). A recent method based on an EMG-driven
17 modelling approach estimating muscle forces generated around ankle joint during gait did not
18 report increased cocontraction in triceps surae during swing phase (Souissi et al., 2018).
19 However, this method omits the distorted relationship between EMG and force in visco-
20 hyperelastic paretic muscles (Berger et al., 1984) and the musculoskeletal model used has not
21 been validated, especially in the context of a central nervous system lesion with known
22 neuromuscular disorders (Souissi et al., 2018). Another interesting approach has been to
23 estimate coherence in the beta band between agonist and antagonist ankle muscles,
24 demonstrating increased coherence in paretics as compared with normal (Kitatani et al.,

1 2016). However, this method, while suggesting muscle coactivation by a single command
2 center, is not exactly a *quantification* of cocontraction.

3 Early attempts consisted of measuring the *time* of muscle coactivation during specific periods
4 (Lamontagne et al., 2000). Others developed *cocontraction indices* involving adding and/or
5 dividing EMG from one muscle and that from another muscle (Berger et al., 1984; Falconer
6 and Winter 1985; Shiavi et al., 1987; Unnithan et al., 1996a, b; Damiano et al., 2000). Such
7 mathematical constructions ignored the lack of comparability between two EMG signals
8 coming from different muscles in different stretching positions, i.e. the different biological
9 meanings of myoelectric signals detected on one side of the joint and those detected on the
10 other (Merletti et al., 1993; Merletti 2004). Such type of error is minimized when both signals
11 undergo some form of normalization (Levin and Hui-Chan 1994; Chow et al., 2012; Vinti et
12 al., 2013, 2015; Gross et al., 2015; Chalard et al., 2020).

13 To circumvent this issue, one may restrict this investigation to the question of how much of a
14 motor neuron pool is involved in antagonist cocontraction, during an opposite effort (Vinti et
15 al., 2013, 2015, 2018; Chalard et al., 2020). This approach involves the same muscle of
16 interest in the numerator and the denominator of the coefficient. This denominator is the
17 maximal voluntary contraction of the muscle as an agonist, tested for example in an isometric
18 position similar to the position in which the muscle may act as an antagonist during function.

19 One may object that this method of EMG normalization could overestimate cocontraction,
20 regardless of the presence of paresis (Knutson et al., 1994; De Luca 1997). Indeed, the
21 reference denominator remains a *maximal voluntary* effort of questionable reliability, rather
22 than an *M-max* that is the most appropriate method to estimate the maximal synchronized
23 recruitment of the whole motor unit pool (Pierrot-Deseilligny et al., 1976). The *M-max* was
24 not used in the present study because of practical and technical difficulties: discomfort and
25 difficulty to obtain *M-max* responses from plantar flexor knee extended.

1 *4.2. Excessive tibialis anterior activation in the paretic leg*

2 To interpret the excessive coefficient of TA activation during swing phase, the first
3 explanation could be an underestimation of the denominator while calculating the coefficient
4 of agonist activation. Indeed, because of the stretch-sensitive paresis (Table 2B1, Figure 3A,
5 Gracies 2005b; Vinti et al., 2015), the “maximal” TA recruitment obtained at 90° dorsiflexion
6 (standing) might in fact have been sub-maximal depending on the level of tension of the
7 plantar flexor at that angle in spastic subjects. Thus, it might have been preferable to test
8 maximal TA recruitment at around -8° (plantar flexion), which is the position at which swing
9 began in our subjects (Table 2, Figure 2A). Still, if we accept that there is excessive activation
10 of TA, this could be explained at least in part by reflex phenomena: the plantar flexion that
11 occurs in the very late stance phase (paretic limb, -8°; non-paretic limb, -7°) could come into
12 a context of stretch reflex hyper-excitability in TA and produce an excessive stretch reflex in
13 that muscle. Such reflex could be contributing to an over-recruitment of TA in the first third
14 of the swing phase. Another likely component is an adapted behaviour trying to overcome
15 excessive passive plantar flexor resistances to dorsiflexion. The present study does not bring
16 data allowing to favour any of these hypotheses.

17 *4.3. Changes in antagonist and agonist activation along the swing phase*

18 Cocontraction in SO and GM was markedly higher on the paretic side from the start of the
19 swing phase, with further, abnormal increase throughout swing (Figures 3B, 3C, 4). In
20 parallel, normalized TA recruitment proved also higher but gradually decreased along swing
21 on the paretic side (Figure 3A, 4). The neurological disorder of cocontraction in antagonist
22 plantar flexors is likely potentiated by their histological disorder, which modifies their elastic
23 properties (increased visco-hyperelasticity) early after stroke (Malouin et al., 1997).
24 Abnormally high tension upon stretch then increases low and high threshold afferent
25 discharge from these muscles (Gioux and Petit 1993).

1 On the paretic side, increased gastrocnemius tension along swing, might relate to the knee re-
2 extension movements along with swing (Table 2A, Figures 2B, 3C, Ateş et al., 2018). The
3 gastrocnemius tension increase-related afferent activity is likely inhibitory on dorsiflexor
4 motor neurons (Delwaide and Oliver 1988; Gracies 2005b; Morita et al., 2006) raising their
5 firing threshold (i.e. *stretch-sensitive paresis*) (Vinti et al., 2015) and excitatory on plantar
6 flexors, lowering their firing threshold in late swing (i.e. *spastic cocontraction*) (Vinti et al.,
7 2013, 2015). In the specific case of soleus, increased afferent gastrocnemius activity during
8 knee re-extension may fail to inhibit - and in fact trigger heteronymous facilitation of - soleus
9 motor neurons (Meunier et al., 1993; Pierrot-Deseilligny and Burke 2005), contributing to
10 their increased coactivation.

11 *4.4. Descending origin of the antagonist overactivity in swing phase*

12 The presence of antagonist EMG does not discriminate between reflex (spasticity) and
13 descending (cocontraction) origins, particularly at a time when the antagonist is stretched.
14 However, a number of arguments favour the descending origin. The hypothesis of antagonist
15 cocontraction as a descending phenomenon is consistent with the well-known lack of
16 correlation between stretch hyperreflexia and active movement impairment in hemiparesis
17 (Broberg and Grimby 1983). In addition, the plantar flexor stretch reflex is still repressed
18 during swing in spastic paresis (Sinkjær et al., 1995; Dietz 2002). Further, the present results
19 show that soleus activation doubles in late swing (Table 2B1, Figure 3B), even though this
20 muscle is actually *shortened* in that phase through a *decrease* in dorsiflexion on the paretic
21 side (Figure 2A). One might then wonder whether soleus overactivity might be partly
22 prompted by heteronymous facilitation from gastrocnemius stretched through the knee re-
23 extension (Meunier et al., 1993; Pierrot-Deseilligny and Burke 2005). However, the gain of
24 this phenomenon has been questioned (Pierrot-Deseilligny and Burke 2005). More likely,

1 antagonist plantar flexor activity from a non-dorsiflexing ankle must have a primarily
2 descending origin, which represents the spastic cocontraction form (Vinti et al., 2015).

3 *4.5. Significance of antagonist cocontraction during the swing phase of gait in hemiparesis*

4 In healthy subjects, involuntary physiologic antagonist cocontraction is observed as a postural
5 anticipatory mechanism or in skill learning (Dietz 2002; Pierrot-Deseilligny and Burke 2005).

6 The role of cocontraction is then supposed to stabilize unstable dynamics using an energy-
7 efficient strategy of selective impedance control (Burdet et al., 2001). However, a number of
8 quantitative and qualitative features seem to distinguish the antagonist cocontraction
9 described during swing phase in paretic patients in the present study from the joint-stabilizing,
10 physiologic cocontraction seen in healthy subjects. One feature is the magnitude potentially
11 reached by antagonist cocontraction in paresis, which might go well beyond *stabilization* to
12 overwhelm agonist contraction and *reverse* the intended movement (Nothnagel 1872; Vinti et
13 al., 2013, 2015).

14 These *quantitative* differences between the “normal” cocontraction and the cocontraction
15 observed in hemiparesis have been measured. In studies of isometric efforts in healthy
16 subjects, the level of antagonist activation reached 20%, increasing with effort and remaining
17 grossly unchanged with antagonist muscle position (Vinti et al., 2013, 2018). As could be
18 expected, the present results in the non-paretic limb during swing phase show slightly higher
19 cocontraction in plantar flexors during swing phase than in isometric measurements in healthy
20 subjects (Vinti et al., 2013, 2018), reaching 27%, and remaining unchanged over that phase
21 (Table 2B1, Figures 3B, 3C). These levels are half of those in the paretic limb. Another
22 important difference between the non-paretic and paretic patterns of cocontraction is the
23 *absence of stretch-sensitivity* of the cocontraction in the non-paretic limb during gait
24 demonstrated here, as well as in healthy subjects during isometric tasks (Vinti et al., 2015,
25 2018).

1 Finally, the pathophysiology of abnormal cocontraction in hemiparesis may involve lesion-
2 induced branching of descending fibers onto afferent antagonist motor neurons and increased
3 recurrent (Renshaw) inhibition (Aoki et al., 1986; Katz and Pierrot-Deseilligny 1999). As
4 patients no longer modulate recurrent inhibition during effort (Katz and Pierrot-Deseilligny
5 1999), a greater background Renshaw level might contribute to spastic cocontraction, because
6 recurrent Renshaw cell collaterals suppress Ia inhibitory interneurons directed to the
7 antagonist (Hultborn et al., 1971). In that context, studies have demonstrated the *lack* of
8 normal increases in reciprocal Ia inhibition and presynaptic inhibition on Ia afferents directed
9 to the antagonist during voluntary contractions in spastic paresis, sometimes converted into
10 reciprocal facilitation (Crone et al., 2003; Gracies 2005b). The physiology of cocontraction in
11 healthy subjects trying to adapt to an unsteady environment seems opposite, as involuntary
12 cocontractions are associated with *increased* presynaptic inhibition of Ia afferents and
13 *increased* fusimotor drive (Llewellyn et al., 1990).

14 4.6. Study limitations

15 The present study did not use a group of healthy subjects as controls. It must be however be
16 understood that the primary objective here was to compare the paretic leg with the non-paretic
17 leg. In addition, this group of authors has previously accumulated extensive experience in
18 healthy subjects using this very method of cocontraction index measurement (coefficient of
19 antagonist activation) (Vinti et al., 2013, 2015, 2018). Even though the validity of the
20 measures of coefficients of agonist and antagonist activation obtained from bipolar electrodes
21 as against values obtained using high density EMG has been shown (Vinti et al., 2018), there
22 are no formal reliability studies for these coefficients as yet. In addition, this study focused on
23 ankle and knee movements in the sagittal plane only. Hip movement analysis in the frontal
24 plane in particular might have added more information regarding the mechanisms of toe
25 clearance in hemiparesis.

1

2 **5. Conclusions**

3 The present analysis of normalized agonist and antagonist activations during hemiparetic gait,
4 circumventing the lack of comparability between raw EMG traces obtained from two sides of
5 the leg, first demonstrates that the stretch-sensitive paresis of TA and the spastic
6 cocontraction of triceps surae increasingly impede paretic foot clearance across swing.
7 Practically, these findings coming from the selective quantification of these two phenomena
8 might help therapists to focus on the adequate interventions to improve gait. Indeed, the
9 increasing disorder of the central command transmission with increasing gastrocnemius
10 tension as the knee re-extends during swing phase progression may suggest to use treatments
11 aiming to decrease plantar flexor tension upon stretch, which might be adequate to reduce
12 passive and active resistances to dorsiflexion *and* to enhance agonist dorsiflexor recruitment
13 during the swing phase of hemiparetic gait. In addition, clinicians might use any strategy to
14 specifically diminish plantar flexor cocontraction in hemiparetic gait (e.g. motor training or
15 injection of blocking agents).

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4

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1 **Figure legends**

2 **Fig. 1 Processing of the EMG - Individual data from the soleus**

3 (A) EMG of the soleus from one paretic gait cycle

4 (A1) Raw EMG signals centered, (A2) EMG rectified, (A3) EMG filtered using a 2nd-order
5 zero-lag Butterworth low pass filter, with a 30 Hz cut-off frequency. (A4) The Root Mean
6 Square (RMS) of the rectified and filtered EMG over each of the three-thirds of the swing
7 phase (T1, T2, T3) is highlighted in grey. The numerator of the Coefficient of ANtagonist
8 activation of soleus is the RMS_{swing} of the selected swing phase period, T1, T2 or T3.

9 (B) EMG of the soleus from maximal voluntary isometric contraction

10 From a recording during maximal voluntary isometric contraction (MVC) of the soleus, The
11 Root Mean Square (RMS_{MVC}) of the rectified and filtered EMG over the 100 ms around the
12 peak is the denominator of the Coefficient of ANtagonist activation of soleus.

13

14 **Fig. 2 Kinematics in the sagittal plane**

15 Mean joint position calculated over the three thirds of swing phase (T1-3) in ankle dorsi-
16 plantar flexion (αA) and in knee flexion-extension (βB) during gait barefoot at comfortable
17 speed (n=42). Results expressed as mean \pm standard error of the mean. DF, dorsiflexion; PF,
18 plantar flexion. Repeated measures ANOVAs: change since previous period, *** $p < 0.001$, **
19 $p < 0.01$, * $p < 0.05$; inter-limb comparison at each period: ‡, $p < 1.E^{-5}$.

20

21 **Fig. 3 Coefficients of agonist and antagonist activation**

22 Coefficients of agonist activation in tibialis anterior (αA , CAG_{TA} , n=34) and of antagonist
23 activation in soleus (βB , CAN_{SO} , n=38) and in gastrocnemius medialis (ϵC , CAN_{GM} , n=41)
24 over the three thirds of swing phase (T1-3) during barefoot gait at comfortable speed. Results

1 expressed as mean \pm standard error of the mean. Repeated measures ANOVAs: change since
2 previous period, *** $p < 0.001$; inter-limb comparison at each period: †, $p < 0.05$, ‡, $p < 1.E^{-5}$.

3

4 **Fig. 4 EMG measurements - Individual data**

5 Samples of rectified EMG from tibialis anterior, soleus and gastrocnemius medialis during
6 one paretic gait cycle at comfortable speed. Grey areas: surface under rectified EMG over
7 swing phase used for the calculations of the Coefficient of AGonist activation in tibialis
8 anterior and the Coefficients of ANtagonist activation in soleus and in gastrocnemius
9 medialis. Hatched areas: expected periods of muscle activity during one cycle in healthy
10 subjects (Sutherland 2001). T1, T2, T3: three thirds of swing phase.

1 **Table 1 Subject characteristics and gait measurements**

2

Subjects (n)	42
Age (y)	50 ± 15
Time since paresis onset (y)	7 ± 7
<i>Gender</i>	
Female (n)	14
Male (n)	28
<i>Paretic side</i>	
Left (n)	28
Right (n)	14
<i>Cause</i>	
Ischemic stroke (n)	21
Hemorrhagic stroke (n)	10
Non-evolutive tumor (n)	6
Traumatic brain injury (n)	5

3

4 Values expressed as mean ± standard deviation.

1 **Table 2 Kinematics and electromyography**

Side: Period:	Non paretic			Paretic			Side*Period <i>P</i>
	T1	T2	T3	T1	T2	T3	
A. Mean position in sagittal plane							
Ankle (deg)	-7±7	2±4	4±5	-8±10	-5±9	-8±8	1.E ⁻¹⁵
Knee (deg)	51±6	53±6	25±9	28±11	27±14	15±8	1.E ⁻¹⁵
B. Coefficients of muscle recruitment							
B1. Cohort data							
CAG _{TA}	0.32±0.18	0.23±0.13	0.26±0.09	0.60±0.41	0.42±0.34	0.39±0.30	0.009
CAN _{SO}	0.14±0.13	0.15±0.13	0.25±0.18	0.24±0.19	0.29±0.25	0.65±0.48	1.E ⁻⁴
CAN _{GM}	0.18±0.32	0.19±0.23	0.27±0.28	0.32±0.20	0.41±0.38	0.57±0.35	0.003
B2. Individual data							
CAG _{TA}	0.44±0.09	0.14±0.04	0.22±0.06	0.88±0.16	0.36±0.08	0.14±0.03	-
CAN _{SO}	0.26±0.10	0.09±0.02	0.17±0.04	0.15±0.05	0.24±0.05	0.45±0.14	-
CAN _{GM}	0.07±0.02	0.05±0.01	0.25±0.05	0.34±0.05	0.45±0.09	0.77±0.09	-

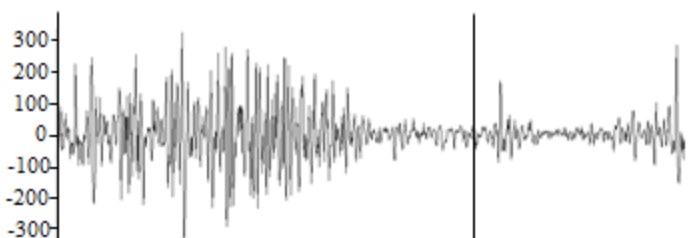
2

3 Results expressed as mean±standard deviation for ankle mean position (n=42), knee mean
4 position (n=42), coefficient of agonist activation in tibialis anterior (CAG_{TA}) and coefficients
5 of antagonist activation in soleus (CAN_{SO}) and in gastrocnemius medialis (CAN_{GM}) from the
6 cohort data and from individual data, calculated over the 3 thirds of swing phase (T1-3)
7 during barefoot gait at comfortable speed at the non-paretic and paretic sides.

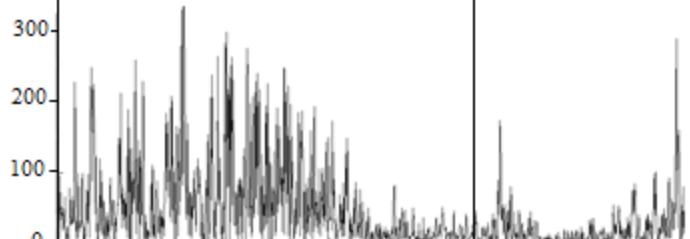
8 Cohort data: CAG_{TA}, n=34, CAN_{SO}, n=38, CAN_{GM}, n=41. Repeated measures ANOVAs are
9 used for inter-limb and inter-period comparisons.

A. EMG from soleus over one paretic gait cycle

A1. Raw EMG centered



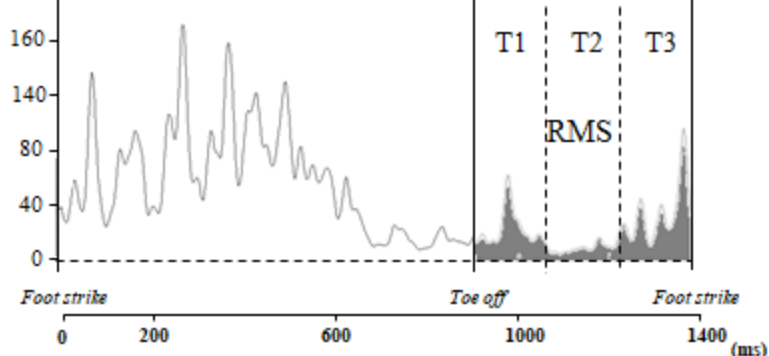
A2. EMG rectified



A3. EMG filtered
(low pass 30 Hz)



A4. RMS_{swing} over swing
(grey area)



B. EMG from soleus during maximal voluntary isometric contraction

RMS_{MVC} over 100ms
around the peak
(grey area)

